AN INVESTIGATION INTO THE PSYCHOBIOLOGICAL MECHANISMS UNDERPINNING THE RELATIONSHIP BETWEEN TYPE D PERSONALITY AND PHYSICAL HEALTH COMPLAINTS IN THE GENERAL POPULATION

S. F. ALLEN

PhD

2017

AN INVESTIGATION INTO THE PSYCHOBIOLOGICAL MECHANISMS UNDERPINNING THE RELATIONSHIP BETWEEN TYPE D PERSONALITY AND PHYSICAL HEALTH COMPLAINTS IN THE GENERAL POPULATION

SARAH FRANCES ALLEN

A thesis submitted in partial fulfilment of the requirements of the University of Northumbria at Newcastle for the degree of Doctor of Philosophy

Research undertaken in the Faculty of Health and Life Sciences

September 2017

Abstract

Type D personality is characterised by high levels of social inhibition and negative affectivity, and is traditionally associated with poor prognosis and negative outcomes in cardiac patients. Research has also demonstrated links between Type D personality and adverse health outcomes among other clinical populations, and in apparently healthy individuals. A number of psychophysiological mechanisms have been suggested to underpin the relationship, including sympathetic dysregulation and maladaptive immune activation. However, previous findings are relatively inconclusive, and further exploration of potential psychobiological mechanisms is warranted. This project therefore aims to elucidate the potential mechanisms underpinning the relationship between Type D personality and poor physical health in the general population.

In the current project, physical symptom clusters were derived from an existing tool designed to assess everyday health complaints, in order to ascertain the relationship between specific physical symptom clusters and Type D. Subsequently, a cross-sectional online questionnaire-based study was conducted to assess the associations between Type D personality and physical symptoms, in addition to a number of psychological and behavioural outcomes identified in the literature. Relationships between Type D and specific stress-related symptom clusters were observed. Subsequently, a one-year follow up was conducted to determine the dynamic nature of the Type D-health relationship and the potential mediating factors involved. Type D was related to metabolic, gastrointestinal and cold/flu symptom clusters, and anxiety and stressful life events were found to play mediating roles.

Given the findings regarding Type D and stress-related symptoms, the subsequent phase of the project comprised an experimental study, which objectively examined sympathetic arousal in response to an acute stress task, in addition to inflammatory activation. Findings indicated that sympathetic dysregulation may be involved in the Type D-health relationship.

Finally, a positive writing intervention was implemented to assess whether the influence of Type D on physical symptoms may be attenuated by means of increasing positive emotions. The utility of the intervention was demonstrated for reducing cold symptoms in Type D individuals over one month.

Overall, this project provides novel contributions to the literature on Type D and adverse health, demonstrating links with specific symptom clusters, further evidence of a potential mechanism of sympathetic dysregulation and the value of a positive psychology invention in Type D individuals.

Table of Contents

List of Tables

List of Figures

Published Work

Chapter 3 is published as:

Allen, S. F., Wetherell, M. A. & Smith, M. A. (2017). The Cohen–Hoberman inventory of physical symptoms: Factor structure, and preliminary tests of reliability and validity in the general population. *Psychology & Health*, *32*(5), 567-587.

The following scientific communications have also arisen from this programme of work:

Allen, S. F., Wetherell, M. A., Lovell, B. & Smith, M. A. (2015) "The relationship between Type D personality and subjective health complaints in the general population"- Poster presented at BSPID conference 2015; and PsyPAG conference 2015.

Allen, S. F., Wetherell, M. A., Lovell, B. & Smith, M. A. (2016) "The psychobiological mechanisms underpinning the link between Type D personality and increased physical symptoms" presented at BPS Psychobiology Annual Scientific meeting 2016.

Allen, S. F., Wetherell, M. A., Lovell, B. & Smith, M. A. (2017) "Type D personality is associated with a maladaptive haemodynamic profile in response to acute stress" presented at HLS Faculty conference, Northumbria University.

Allen, S. F., Wetherell, M. A., Lovell, B. & Smith, M. A. (2017) "Examination of salivary alpha amylase as a biomarker of sympathetic activation to acute stress in Type D individuals" presented at PsyPAG conference 2017, and BPS Psychobiology Annual Scientific meeting 2017.

Acknowledgements

Firstly, I would like to express my sincere gratitude to my supervisors; Dr. Michael Smith and Dr. Mark Wetherell for the continued support and help they have provided, and for their patience, humour, motivation, and expertise. Their guidance has helped me immeasurably throughout the time spent conducting research and writing this thesis. I could not have imagined having a better supervisory team for my PhD study.

I would also like to thank Dr Brian Lovell, my third supervisor, for his insights, Dr Kris McCarty for his technical expertise, and the other staff at Northumbria who helped out with my study protocols.

I would also like to thank all the wonderful people of CoCo research centre for their continued friendship and support, for the weird and interesting discussions, and all the fun we have had in the last three years.

I am also greatly appreciative for the support received from my lovely friends, in particular my flatmate Heather Lynn, who has kept me sane, and provided many a bottle of wine and sympathetic ear to guide me through the most stressful times.

Finally, I would like to thank my parents for their love, support, and encouragement throughout my PhD experience and my life in general.

Declaration

I declare that the work contained in this thesis has not been submitted for any other award and that it is all my own work. I also confirm that this work fully acknowledges opinions, ideas and contributions from the work of other.

Any ethical clearance for the research presented in this thesis has been approved. Approval has been sought and granted by the Faculty Ethics Committee on 11 November 2014.

I declare that the Word Count of this Thesis is 64,489 words

Name: Sarah Frances Allen

Signature:

Date:

Chapter 1: A review of the relationship between Type D personality and physical health; background, aims and objectives.

This first chapter will introduce the concept of Type D personality and provide a comprehensive review of the literature pertaining to the influence of Type D personality on physical health in cardiac patients and other clinical populations, in addition to within the general population. A number of behavioural and psychobiological mechanisms have been proposed as underpinning the relationship between Type D and poor health, and the research exploring these pathways will also be discussed. The aims and objectives of this thesis project as a whole are proposed, and summaries of each of the studies are provided.

1.1 Introduction to Type D personality

In 1995, a new 'type' of personality construct named Type D, 'distressed' personality was introduced (Denollet, Sys & Brutsaert, 1995). The construct was derived from previous theoretical and empirical studies which investigated coping style in coronary patients (Denollet et al., 1995; Denollet, 1993). Type D personality is characterised by the interaction of the two stable, global personality traits; negative affectivity (NA) and social inhibition (SI) (Denollet et al., 1996). Type D individuals have the tendency to experience negative emotions characterised by dysphoria, anger, anxiety, hostility, and general distress across situations and time (Denollet, 1998a); whilst also inhibiting the expression of these emotions in social situations due to fear of rejection or disapproval (Mols & Denollet, 2010a). The Type D personality construct was initially proposed to emphasise the role of normal traits rather than psychopathology in poor clinical prognosis of cardiac patients (Denollet, 2000; Denollet et al., 1996) and it has been asserted that the construct does not represent a psychiatric disorder. Since its initial proposal a great body of research has demonstrated the prognostic validity of the construct in cardiac patient populations. However, there is accumulating evidence that Type D may be an important risk factor for poor health in other illness groups, as well as in apparently 'healthy' individuals, that is, individuals who are free from any chronic conditions.

1.2 Development of Type D, and research in cardiac populations

The Type D construct was developed through psychological examination and follow up of outcomes in patients with ischemic heart disease, and was validated in cardiovascular disease patients (Pedersen & Denollet, 2003). Social inhibition is thought to moderate the influence of negative affectivity on the relationship between Type D and negative health outcomes and it is suggested that individual differences in the way that negative emotions are expressed and dealt with can be just as important as the negative emotions themselves (Sher, 2005).The personality construct was initially linked to increased risk of poor outcomes in cardiac patients but also increased risk factors (e.g. lifestyle-related) for developing cardiovascular disease in otherwise healthy individuals (Mols, Thong, van de Poll-Franse, Roukema & Denollet, 2012).

It was initially proposed that the specific synergistic effect of NA and SI is pertinent to predicting negative cardiac outcomes and an abundance of early studies demonstrated the prognostic value of the construct in cardiac patient populations (Denollet, 2005). It is not surprising that this combination of traits is particularly detrimental to cardiac health, given that an abundance of research has demonstrated that aspects of psychological distress (e.g. depression, anxiety, negative affect) are established predictors of cardiac events (Strik, Denollet, Lousberg & Honig, 2003). Further, lower social support has been associated with increased morbidity and mortality (Berkman, Leo-Summers & Horwitz, 1992) and worse cardiac symptoms (Lindsay, Smith, Hanlon & Wheatley, 2001).

Since it was initially proposed, the Type D personality concept has been increasingly investigated and discussed as an independent risk factor for poor prognosis in cardiac patients, including increased morbidity and mortality (Denollet, 1994; Kupper & Denollet, 2007; Pedersen & Denollet, 2004). It is proposed that Type D personality is associated with a three-fold increase in mortality risk (Pedersen & Denollet, 2006). Previous research has also indicated links between the personality type and a range of specific health outcomes including; increased risk of cancer in men with coronary heart disease (Denollet, 1998); impaired health

status in chronic heart failure patients (Schiffer et al., 2005); increased vital exhaustion in patients with ischemic heart disease (Pedersen & Middel, 2001); and heightened risk of nonfatal myocardial infarction (Grande, Romppel & Barth, 2012).

Further, Type D personality has also been related to increases in negative psychological outcomes in cardiac patients, for example as a predictor of increased distress in ICD (Implantable Cardioverter Defibrillator) patients, (Pedersen, Theuns, Erdman & Jordaens, 2008; Pedersen, van Domburg, Theuns, Jordaens & Erdman, 2004) and chronic anxiety following coronary intervention (Spindler, Pedersen, Serruys, Erdman & van Domburg, 2007). Type D personality has also been linked to increased depressive symptoms in chronic heart failure (Schiffer et al., 2005), impaired psychological status in a large sample of Icelandic cardiac patients (Svansdottir, van den Broek, Karlsson, Gudnason & Denollet, 2012) and levels of anxiety 12 months post-coronary intervention (van Gestel et al., 2007).

The links between Type D personality and negative cardiac outcomes are thought to be independent of disease severity; however, the underlying mechanisms linking Type D to health are not entirely clear. A number of behavioural pathways have been proposed, including poor health behaviours, such as smoking, reduced physical activity and poor treatment adherence (e.g. Gilmour & Williams, 2012; Williams, O'Connor, Grubb & O'Carroll, 2011). For example, Type D personality has been associated with an unhealthy lifestyle in a sample of cardiac patients (Svansdottir et al., 2012). Svansdottir et al., (2012) assessed health-related behaviours four months following angiography and found that Type D patients were more likely to smoke, use antidepressants and sleep medication at follow up as compared to non-Type Ds. Research into whether these behaviours may be a precipitating factor present in apparently healthy Type D individuals may aid in understanding the link between the personality disposition and poor health.

Psychobiological pathways including elevated cortisol levels (Denollet & Kupper, 2007; Molloy, Perkins-Porras, Strike & Steptoe, 2008; Whitehead, Perkins-Porras, Strike, Magid & Steptoe, 2007), cardiovascular stress reactivity (Habra, Linden, Anderson &

Weinberg, 2003) and sympathetic dysregulation (Kupper, Pelle & Denollet, 2013) have also been proposed as potential contributing factors to the relationship between Type D and poor cardiac outcomes. The biological systems underpinning these potential mechanisms are described in more detail in chapter 6. A number of studies have also implicated inflammatory processes in the link between Type D and poor cardiac health (Denollet et al., 2003). For example; Denollet, Vrints, and Conraads, (2008) found that Type D was independently associated with substantially increased TNF-a (tumour necrosis factor-alpha) activity, which has been implicated in the pathogenesis of chronic heart failure and concluded that the disease-promoting influence of Type D personality mirrored the pro-inflammatory trajectory seen in ageing.

Given the abundance of literature proposing the link between Type D and poor health in cardiac populations, research investigating the influence of Type D on health prior to disease onset (i.e. in apparently healthy individuals) is warranted to further understand how the construct may manifest as a risk factor. Furthermore, it is also clear that the potential neuroendocrine, cardiovascular and inflammatory mechanisms warrant further consideration within individuals from the general population, to examine whether dysregulation of these systems could be considered as early warning signs of Type D related health impairments.

1.3 Measurement of Type D and an overview of criticisms

Type D personality has been measured using a number of different instruments across studies. Some studies have used independent questionnaires to measure the components of the construct; negative affect and social inhibition, including measures of neuroticism and introversion respectively. Other questionnaires implemented across a number of earlier studies have included the State Trait Anxiety Inventory (Spielberger, 1970), the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1976), Anxiety Sensitivity Index (Peterson & Heilbronner, 1987), Penn State Worry Questionnaire (Meyer, Miller, Metzger & Borkovec, 1990), and Beck Depression Inventory. However, as using a single validated measure eliminates inconsistent results associated with the use of different tools (Smith & MacKenzie,

2006), Denollet (1998a) realised the necessity to develop a combined instrument for Type D assessment, comprising two separate NA and SI scales. The Type D scale 16 (DS16) was initially developed with the aim of characterising individual risk when examining psychosocial influences in cardiac disease (Denollet, 1998a). Denollet (2000) also went on to develop the DS24; which included additional items relating to the lower-order traits of dysthymia and tension (within the NA scale) and withdrawal (within the SI scale), and the DS14, which will be used throughout this project.

The Type D scale-14 (DS14; Denollet, 2005), comprises 7 items measuring NA and 7 measuring SI and was specifically developed to assess Type D in a reliable and standardized way that poses little burden to respondents (this will described in more detail in Chapter 2). Type D was traditionally assessed as a categorical variable across all Type D scales, with scores on both scales above a particular threshold being classified as Type D, and all others classified as non-Type D. Whereas, it is recommended that individuals scoring above the median on the SI and NA scales of the DS16 are classified as Type D (Denollet, 1998a), the later developed DS14 classification has been standardised, and those scoring above 10 on both scales are labelled Type D (Denollet, 2005).

Type D personality has been found to be a stable taxonomy over an 18-month period in cardiac patients, and category classification is not influenced by mood changes (Martens & Kupper, 2007). Furthermore, a twin study (Kupper, Denollet, de Geus, Boomsma & Willemsen, 2007) demonstrated that Type D personality is considerably heritable, with equally heritable subcomponents (NA and SI). NA levels appeared to be determined by additive genetic effects and non-shared environment, whereas social inhibition differences were shown to be predominantly determined by non-additive genetic effects and non-shared environment. This suggests that Type D is to a large degree a stable and genetically predisposed combination of traits (Kupper et al., 2007).

A number of conceptual, methodological, and statistical issues have however been raised concerning the traditional categorical approach to Type D personality. The categorical

approach has been criticised for using arbitrary cut-offs and for not accurately representing how the combination of SI and NA synergistically interact, leading to worse outcomes than each of the components in isolation. Furthermore, there is a consensus in the psychometric and personality theory literature that typologies generated from high-low, 2 × 2 crossings of continuous variables are not appropriate and are prone to spurious findings (Coyne et al., 2011). There is also no direct evidence at present to support the theory that Type D is categorical, particularly when it comes to differentiating between true cases and non-cases (Ferguson et al., 2009).

In response to criticisms of the categorical approach to Type D personality, Ferguson et al., (2009) tested the dimensionality of Type-D personality, using taxometric procedures, to assess whether Type D personality is taxonic or dimensional. It was recommended that Type D should be conceptualised as the interaction of continuous negative affectivity and social inhibition, rather than as a categorical variable. This is supported by Coyne et al., (2011) who also propose that Type D is best understood in continuous, dimensional terms. This culminates in the validation of the dimensional approach in which NA scores are multiplied by SI scores to produce a Type D interaction score enabling 'levels' of Type D personality to be compared with continuous outcome measures. Multiple regression analyses can also be conducted to assess the specific contribution of the Type D interaction while controlling for the effects of NA and SI in isolation (how this was implemented in analyses is described in more detail in Chapter 2). However, recent research utilising this method of analysis has so far produced an array of null findings in (albeit small) samples of the general population (e.g. Stevenson & Williams, 2014). In line with this recommendation analyses in the current project will be undertaken using both approaches where possible (with the exception of the intervention study discussed in Chapter 8 as this requires a 2 by 2 groups design in order to assess the efficacy of an intervention as opposed to a control task between Type D groups).

A number of early reviews aiming to assess the relationship between Type D personality and hard endpoints (i.e. all-cause mortality, cardiac death and non-fatal myocardial infarction)

consistently concluded that Type D personality was associated with an increased risk of adverse cardiac events (Denollet, 2000; Grande et al., 2012). However, these reviews have received criticism due to the majority of work coming from a single research group using only patients from the Netherlands and Belgium (Grande et al., 2012) which was suggested as indicative of publication bias. Although Type D is still viewed as a significant predictor of adverse physical and psychological outcomes in cardiac patients (Pedersen & Denollet, 2006), there have been a few recent contradictory findings. The evidence is now a little uncertain with regards to the importance and implications of the personality concept as a whole (Grande et al., 2012; Smith, 2011). Whereas early findings indicate strong associations between Type D and cardiac health related outcomes, it has been claimed that these studies may have overestimated the prognostic significance of the construct (Coyne & de Voogd, 2012; Coyne et al., 2011; Grande et al., 2012), and more recent studies controlling for the first order separate effects of SI and NA have shown that the synergistic effect of the two is not significantly related to indices of ill health (e.g. Grande & Romppel, 2011; Stevenson & Williams, 2014). On the other hand, other studies have found that Type D can significantly predict aspects of health (e.g. medication adherence), after controlling for the individual effects of SI and NA (Williams et al., 2011). In light of these inconsistencies in the literature, the clinical utility of the Type D personality construct in cardiac patients is still relatively ambiguous.

Further criticisms of the Type D construct as a whole include whether Type D may plausibly predict negative health outcomes independent of similarly known biomedical risk factors, including severity of disease (Coyne et al., 2011). It is argued that the strength of existing literature on the relationship between negative affect and poor health (e.g. Farmer & Ferraro, 1997; Miller et al., 2009) may lead to a dubious view of a new concept such as Type D claiming to be better predictor of poor health outcomes (Coyne et al., 2011). Coyne et al., (2011) therefore recommends caution when considering some of the first small-scale studies by Denollet's group which have not yet been replicated independently (Coyne et al., 2011). Furthermore, additional analyses incorporating depression measures instead of negative

affectivity should be considered (Coyne et al., 2011). It has also recently been proposed that identification of combined personality profiles; specifically the combination of Type A and Type D, may be useful in clinical research and practice, particularly in cardiac patients (Steca et al., 2016). Although this concept is not within the scope of current project, it is interesting to consider moving forward, particularly if implementation of personality assessments in clinical practice is to be considered.

Further, whether the concept is sufficiently distinct from other measures of negative affect, including neuroticism, anxiety and depressive symptoms is also disputed. Notably, it is suggested that Type D may be better measured by the well-established five factor model of personality (FFM; McCrae & Costa, 1987) specifically; introversion (or inverse extroversion) and neuroticism (roughly equivalent to SI and NA, respectively). In light of these criticisms research has been conducted to examine how Type D personality may be incorporated within the context of such existing personality theories. De Fruyt and Denollet (2002) found that as expected; the NA scale of the DS16 (Denollet, 1998) correlated most highly with neuroticism, and social inhibition correlated most highly with both extraversion and neuroticism. Furthermore, Chapman et al., (2007) found that in addition to high levels of neuroticism and low extraversion, Type D individuals also appeared to exhibit lower levels of agreeableness and conscientiousness as well. Overlaps between personality traits are not unexpected, however as other personality traits are thought to be implicated in health outcomes (e.g. Korotkov, 2008) the influence of these traits must be considered in Type D research.

Furthermore, Howard and Hughes, (2012) found that Type D personality could be represented using introversion and neuroticism. Associations between Type D and anxiety, depression and perceived stress were mainly accounted for by neuroticism (Howard & Hughes, 2012). However, the relationship between Type D and cardiac output were attributable to the synergistic effect of NA and SI. This may suggest that the observed effects of Type D personality on some outcomes may be solely influenced by NA (or neuroticism) whereas the synergistic effect of both traits can have distinct effects on others (i.e.

cardiovascular function). This therefore advocates that the Type D construct may still deserve a place in the health literature after all.

Ultimately, despite the criticisms of the construct, there is a large body of research indicating associations between Type D personality and poor cardiovascular outcomes (e.g. Mols & Denollet, 2010a, Grande, Romppel & Barth, 2012). Furthermore, Type D individuals have been identified as having a higher propensity for distress, and poorer stress coping strategies (Polman, Borkoles & Nicholls, 2009). Given that heightened distress is a risk factor for poor health outcomes, it makes conceptual sense to investigate why some individuals are at such higher risk. Even though some Type D research yields null findings, it is suggested that abandoning Type D research is premature (Borkoles, Polman, Ski & Thompson, 2012). It is therefore important to conduct further research to identify whether, and to what degree, there is an association between Type D and poor cardiovascular outcomes, and attenuate some of the risks.

1.4 Type D personality in non-cardiovascular clinical populations

Although the majority of earlier research focused the influence of Type D personality on cardiac health, it has also become clear that due to their vulnerability to chronic distress, Type D individuals have an increased risk of a wider range of negative health outcomes (Denollet, 2005). Research into the direct links between Type D personality and negative clinical outcomes via biological (e.g. immune activation, Denollet & Kupper, 2007) and behavioural (e.g. unhealthy lifestyle, Williams et al., 2008) mechanisms of disease led to the assumption that Type D personality could also be associated with negative outcomes among non-cardiovascular patient populations (Mols & Denollet, 2010a). A number of Type D studies have consequently been conducted within other clinical populations.

One particularly noteworthy meta-analytic review (Mols & Denollet, 2010a) found that correlations were present between Type D personality and the reporting of either a negative or more severe outcome within a variety of patient groups, in the majority of the high-quality studies reviewed. For example; in three separate studies; chronic pain patients, tinnitus

patients and sufferers of diabetic foot syndrome classified as Type D scored higher on measures of depression and anxiety (Barnett, Ledoux, Garcini & Baker, 2009; Bartels, Pedersen, et al., 2010; Simson et al., 2008). This may be unsurprising given the overlap between Type D personality and negative mood states including dysphoria, anxious apprehension, irritability and dysthymia (Denollet, 2000), an issue that, as previously mentioned, the construct is often criticised for. Type D personality was also found to be more prevalent in patients in comparison to controls in studies examining tinnitus (Bartels, Middel, Pedersen, Staal & Albers, 2010), and recurrent vulvovaginal candidiasis (Ehrström, Kornfeld & Rylander, 2007).

Type D has been associated with maladaptive health behaviours and emotional distress in adults with diabetes (Nefs et al., 2015). Increased perceived frequency and severity of side effects of CPAP (continuous positive airway pressure treatment) in obstructive sleep apnoea patients with Type D personality have also been observed. Further, in a sample of older primary care patients, Chapman, Duberstein, and Lyness, (2007) found that Type D personality was related to higher number of reported co-morbid conditions, in addition to poorer subjective health ratings and psychological functioning. Another study found that Type D cancer survivors reported more comorbid conditions and distress due to health complaints, in addition to increased visits to healthcare professionals, in comparison to their non- Type D counterparts (Mols, Thong, et al., 2012). However, the direction of causality within these identified relationships is unclear due to the cross-sectional and self-report nature of many studies. Specifically, it could be that individuals with high levels of negative affect, due to their illness experience, may be biased towards the reporting of more negative health outcomes. This, therefore, warrants prospective research to be undertaken to understand how Type D status may lead to adverse health and vice versa.

Alternatively, other studies observed no differences in outcomes between Type D and non-Type D individuals. For example in a study of asthma sufferers no differences in Type D scores were observed between non-atopic and atopic asthmatics (Barone et al., 2008).

However, within this study atopic asthmatics also scored significantly higher on the anxiety sensitivity index, providing evidence of a distinction between Type D and anxiety in clinical groups. This may also suggest that the effect of Type D personality may differentiate between illnesses and symptoms, highlighting the requirement to assess distinct aspects of health, particularly in non-diseased populations.

1.5 Associations between Type D and health in the general population

In addition to those studies which have suggested that Type D personality is predictive of health outcomes in clinical populations, more recently, studies have begun to examine the effects of Type D in the general population of apparently 'healthy individuals'. Denollet (2005) originally claimed that the prevalence of Type D personality in the general population was around 20%, which was considerably lower than that found in CHD patients (28%) and Hypertensives (54%). However, in more recent studies have estimated the prevalence to be up to 42.8% in the general population (Booth & Williams, 2015). This somewhat high prevalence indicates the importance of researching the health effects of Type D in healthy populations in addition to those with documented cardiac illnesses.

Type D personality has been associated with increased anxiety, depression and somatisation (Michal, Wiltink, Grande, Beutel & Brähler, 2011), maladaptive stress reactivity (Habra et al., 2003; Howard & Hughes, 2013; Kelly-Hughes, Wetherell & Smith, 2014; O'Leary, Howard, Hughes & James, 2013), poor sleep (Condén, Ekselius & Aslund, 2013), dysfunctional coping strategies and lower social support (Williams & Wingate, 2012) and poor health behaviours (Booth & Williams, 2015) in the general population. All of which have been postulated to potentially mediate the relationship between Type D and physical health. However, despite these findings, the relationship between Type D personality and indices of health in the general population has received little attention. In particular, it appears necessary to investigate the interaction of behavioural and psychobiological mechanisms by which Type D personality may manifest in physical symptoms and poor health.

1.5.1 Physical health in the general population

It is claimed that Type D personality is a vulnerability factor for poor physical health and may be related to disease-promoting mechanisms in apparently healthy individuals (Mols & Denollet, 2010b). In a meta-analytic review, it was demonstrated that in the nineteen selected studies with adequate to high methodological quality, the presence of Type D characteristics had a negative influence on both mental and physical health. Outcomes included poorer health status, increased reporting of somatic complaints and more flu-like illness (Mols & Denollet, 2010b).

Existing research has also demonstrated that Type D personality is related to an increase in self-reported physical symptoms and poorer perceived health in both adults and children (Stevenson & Williams, 2014; van den Broek, Smolderen, Pedersen & Denollet, 2010; Williams & Wingate, 2012). In a recent study, Williams, Abbott, and Kerr, (2015) found that Type D individuals reported significantly poorer subjective health than non-Type Ds (i.e. using the traditional categorical approach) and when analysed as a dimensional construct, Type D was positively correlated with physical symptoms and negatively correlated with quality of life. A subsequent study by Stevenson and Williams (2014) also found that individuals classified as Type D reported increased physical symptoms and poorer quality of life, however, levels of Type D were not significantly related to the outcome variables when the separate effects of SI and NA were controlled. (Stevenson & Williams, 2014).

Previous studies have also established associations between Type D personality and increased severity of psychosomatic and musculoskeletal pain symptoms in a large Swedish community sample (Condén, Leppert, Ekselius & Åslund, 2013). Increased distress due to climacteric symptoms and decreased sexual functioning in peri- and post- menopausal women (Borkoles et al., 2015) who were classified as Type D has also been demonstrated. Furthermore, Type D personality has been associated with individuals with voice complaints being less likely to receive appropriate medical treatment (Thomas, de Jong, Kooijman & Cremers, 2006); and a decreased likelihood of students seeking regular medical check-ups

(Williams et al., 2008).

There are also documented links between the personality disposition and increased risk of somatisation, alcohol abuse, and panic disorder (Michal et al., 2011), all of which have substantial negative consequences for health. Furthermore, Michal et al., (2011) demonstrated that Type D personality was associated with increased help-seeking behaviour and use of health care services, suggesting that Type Ds suffer from more health problems. Conversely, in line with the symptom perception hypothesis (Gijsbers van Wijk & Kolk, 1997) it could also be considered that 'distressed' individuals may be more sensitive to particular bodily sensations and may over report or be more anxious (hence increased healthcare utilisation) regarding the physical symptoms they experience.

In terms of cardiac related health in the general population, Hausteiner, Klupsch, Emeny, Baumert & Ladwig, (2010) found Type D to be associated with a number of cardiovascular risk factors and psychopathological symptoms in a community sample, including depression, anxiety, exhaustion and poor subjective health. It was concluded that the personality disposition has established itself as a relevant and independent risk marker for poor cardiac health. Furthermore, Einvik and Dammen, (2014) found that in a non-clinical population Type D personality was independently associated with a higher likelihood of ventricular arrhythmias (abnormal heart rhythms), which may be implicated in the increased cardiovascular disease risk observed in persons with Type D personality. In light of these findings, despite the inconsistencies, it is clear that the potential risk that Type D personality poses for health in the general population warrants further attention.

1.5.2 Stress-related mechanisms

As Type D individuals have an increased propensity to distress it seems likely they may react differently to stressful events. An abundance of research has found that Type Ds are more likely to report higher levels of perceived stress and anxiety (Mols & Denollet, 2010b). Links have also been found between Type D and atypical self-reported stress levels, and stress related illnesses (Armon, 2014; Polman, Borkoles & Nicholls, 2010). Psychobiological

evidence has also indicated associations between Type D personality and dysregulation of psychobiological stress pathways in the general population, including hyper-reactivity of the hypothalamic-pituitary-adrenal (HPA) axis (Bibbey, Carroll, Ginty & Phillips, 2015; Habra et al., 2003), and maladaptive sympathetic nervous system (SNS) activity (Bibbey et al., 2015; Howard & Hughes, 2013; Kelly-Hughes et al., 2014).

1.5.2.1 Subjective stress and stress-related illness

Previous work has observed a positive correlation between perceived stress and Type D personality scores (Williams & Wingate, 2012), and Type D individuals reported higher feelings of subjective stress in an experimental study as compared to non-Type D individuals (Habra et al., 2003). Jellesma (2008) examined the reporting of somatic complaints in a sample of pre- and early adolescents and found that those with a Type D personality reported increased negative mood states and non-productive thoughts, relative to their non-Type D counterparts. Further, Kelly-Hughes et al., (2014) also found that lower mood and amplified background stress were associated with Type D personality; however this relationship was not significant when the independent effects of NA and SI were controlled. Nevertheless, it seems feasible that heightened levels of stress may act as a mediating mechanism by which Type D personality may influence health.

Type D has also been linked to an increase risk for the development of post-traumatic stress disorder in a Dutch sample of prison workers (Kunst, Bogaerts & Winkel, 2009) . The study does however, indicate a stress related mechanism and highlights the contribution of other perpetuating environmental factors, as the risk was found to increase when individuals were exposed to inmate aggression; a particularly stress-inducing aspect of the profession (Kunst et al., 2009). These findings relate to the evidence that Type D personality can also influence aspects of psychological functioning in an occupational context. For example; Type D personality was associated with increased work absences in a sample of industrial workers from Germany (Hanebuth, Meinel & Fischer, 2006), and Type D has also been linked to job burnout (Polman, Borkoles & Nicholls, 2010).

Burnout is a distinct affective response to chronic stress. It is a complex construct comprising symptoms of physical, cognitive and emotional exhaustion and fatigue (Maslach, Schaufeli & Leiter, 2001). It is regarded as a direct consequence of prolonged, perpetual exposure to stress, particularly experienced within a work context. Burnout is implicated in physical health impairment, including increased risk of cardiovascular disease (Melamed, Shirom, Toker, Berliner & Shapira, 2006). Therefore, it seems logical that development of burnout could be an important factor in explaining the relationship between Type D, stress and poor health, particularly in otherwise healthy populations.

A number of studies have demonstrated associations between Type D personality, increased occupational stress and symptoms of burnout. For example Ogińska-Bulik, (2006) found that Type D was associated with higher levels of burnout in healthcare professionals and a study by Polman, Borkoles and Nicholls, (2010) demonstrated that Type D moderated the relationship between perceived stress and burnout levels in students, with Type D individuals experiencing higher levels of burnout at lower stress levels (Polman et al., 2010). However, both of these studies suffered generalisability issues as they used small, non-representative samples. However, another study did find that Type D was positively associated with higher levels of job burnout (Armon, 2012), but it was also found that physical activity may attenuate the effects. Additionally, Van den Tooren and Rutte (2015) found in a large sample of the Dutch working population that even when effect of job demands and resources were controlled, Type D individuals in comparison to non- Type Ds experienced higher levels of emotional exhaustion and lower levels of work engagement.

1.5.2.2 Psychobiological stress reactivity

One psychobiological mechanism which has gained much support, proposes that Type D personality influences stress-appraisal in ways that moderate cardiovascular and neuroendocrine responses to stress (Howard & Hughes, 2013). The stress response is governed by two key mechanisms: the sympathetic-adrenal-medullary (SAM) axis and the hypothalamic-pituitary-adrenal (HPA axis). The SAM axis initiates the immediate stress

response which constitutes changes in sympathetic activity including increases in heart rate and decreases in peripheral functions to prepare the body for fight or flight. The HPA axis regulates the longer-acting response to stress which releases steroid hormones including cortisol into the blood stream and can influence process such as inflammation (Compas, 2006). Dysregulation of these biological systems are implicated in a multitude of illnesses and inflammatory disorders, and it is likely they may represent pathways by which emotional distress may lead to adverse health outcomes (Bibbey et al., 2015; Habra et al., 2003).

In an abundance of clinical research, exaggerated cardiovascular reactivity to stress has been linked to an increased risk of developing cardiovascular disease via a number of various manifestations such as hypertension, systemic atherosclerosis, left ventricular hypertrophy, and coronary artery calcification (e.g Carroll et al., 2012; Treiber & Kamarck, 2003). Increased cortisol reactivity to stress has also been linked to hypertension, coronary artery calcification and increased risk of cardiovascular disease (Hamer, Endrighi, Venuraju, Lahiri & Steptoe, 2012; Hamer, O'Donnell, Lahiri & Steptoe, 2010). Therefore, given the links between Type D personality and poor health outcomes in cardiac patients, it is not surprising that maladaptive cardiovascular and cortisol responses to acute stress have been found in Type D individuals.

With respect to the effect of Type D on cortisol indices, one study found that SI and NA were found to be independently related to greater cortisol reactivity in men; however the stressor task implemented did not evoke a significant cortisol response over time, and therefore this relationship should be interpreted with caution (Habra et al., 2003). Nevertheless, a more recent study found that Type D individuals mounted a positive cortisol response to a socially evaluative stressor, whereas, non-Type D individuals and those in a low social condition did not (Bibbey et al., 2015), indicating that Type D individuals could experience heightened HPA axis activity in response to social stress.

In terms of the SAM axis response to stress, Habra et al., (2003), found that men high in NA demonstrated heightened blood pressure reactivity, whereas those high in NA exhibited

reduced HR change to a mental arithmetic challenge. Although the study did not identify the effects of Type D per se on the cardiovascular reactivity indices (i.e. the synergistic effect of SI and NA did not significantly predict the outcome measures), and the effects were restricted to males, it does still appear feasible that Type D may play some role in cardiovascular stress reactivity (Habra et al., 2003). This is further evidenced by Williams, O'Carroll, and O'Connor (2009) who also found that Type D individuals exhibited an exaggerated cardiac output, in addition to higher levels of subjective stress during a cognitive stress task. Bibbey et al., (2015) also found that Type D individuals exhibited greater cardiovascular stress in a high social evaluation condition in comparison to non-Type Ds in a condition with low social evaluative threat, which may suggest that the nature of the task is important. Furthermore, it has also been demonstrated that Type D individuals showed cardiovascular sensitisation to a recurring stressor (Howard & Hughes, 2013); however, again this relationship was only observed in males. Despite potential shortfalls in these studies, they do nevertheless implicate a mechanism of psychosomatic cardiovascular pathogenesis (Howard & Hughes, 2013).

In addition to exaggerated stress responses, Type D has also been related to lower ('blunted') responses. Blunted cardiovascular reactivity to acute stress could be evidence of sympathetic dysregulation and a growing body of evidence has identified associations between blunted reactivity and indices of ill health; including poorer self-reported health; obesity; depression and eating disorders (e.g. Ginty, Phillips, Higgs, Heaney & Carroll, 2012; Phillips, 2011). Furthermore, in a study by Kupper, Denollet, Widdershoven and Kop (2015), low diastolic blood pressure reactivity to acute stress was associated with poor outcomes in heart failure patients. Blunted cardiovascular reactivity is attributed to an adaptation to recurrent or chronic stress which given the propensity of Type D individual to perpetual distress, may be a plausible cause of such an effect (Lovallo, Farag, Sorocco, Cohoon & Vincent, 2013). This may therefore imply that Type D-cardiovascular health associations could also be underpinned by a blunted cardiovascular stress response.

For example; one study found that Type D women exhibited lower systolic blood

pressure responses to stress, in comparison to non-Type D women (O'Leary et al., 2013), and a study by Kelly-Hughes et al., (2014) found an inverse relationship between levels of Type D and blood pressure in response to an acute stressor in both men and women. Furthermore, a study by Howard, Hughes and James (2011) found that Type D personality was related to decreased heart rate reactivity to stress, again in women. It could be theorised that the converse findings regarding whether Type D is associated with an exaggerated or a blunted cardiovascular response could possibly be due to different tasks. For example, the tasks used in Williams et al., (2009) and Bibbey et al. (2015) contained socially salient elements, whereas the task in the study by Howard et al., (2011) did not. On the other hand, as the effects were found in either females or males, this could also be evidence of gender moderating the nature of sympathetic arousal in Type D individuals. A premise which may be further supported by Kupper, Pelle and Denollet's (2013) who found that Type D males demonstrated higher parasympathetic activity, in addition to larger sympathetic activity in response to a cold pressor task in comparison to female participants and non-Type D males. The few studies mentioned here indicate the possibility of a maladaptive stress response in Type D. However, a more detailed overview of the studies examining Type D and psychobiological stress reactivity can be found in Chapter 6, in addition to a potential explanation of the of the stress response systems.

In summary, it appears that there is an association between Type D personality and maladaptive responses to stress, however, the existing findings are mixed with regards to the nature of the effect. Further examination of the stress systems and the psychobiological mechanisms involved are required in Type D individuals. It is likely than any findings may provide further knowledge in regards to elucidating the pathways by which Type D can lead to ill health.

1.5.3 Psychological and behavioural mechanisms

1.5.3.1 Coping strategies and Social support

Stress-coping strategies and levels of social support are known to influence the relationship between distress and ill health (Miller et al., 2009). For example, Berkman and Syme, (1979) found that less socially integrated individuals had higher mortality rates, and Cornwell and Waite (2009) demonstrated links between social disconnectedness and perceived isolation with lower levels of subjective physical health. Increases in social support are found to attenuate negative health effects most successfully when perceived availability of interpersonal resources required in the face of stressful events is high (Cohen & Wills, 1985). Therefore, as Type D individuals are socially inhibited, they may experience a lack of support when dealing with stressful events, which may therefore contribute to deterioration of their health.

Coping strategies fundamentally comprise of avoiding, reacting to or simply adjusting to the source of stress and thereby attempting to reduce its effects (Compas, 2006). However, some strategies can be maladaptive (e.g. self-blame, substance use and denial) (Carver, 1997), and are inadequate in reducing distress and can be consequentially detrimental to health. Adaptive coping strategies can, however, buffer the effects of stress and negative emotions on poorer health. For example, admitting to experiencing stress has been found to alleviate some of the negative health consequences that stress appears to induce (McGonigal, 2013). Therefore, it would appear that if adaptive coping strategies and support are available to deal appropriately with stress, reductions in stress-related illness may be observed. Furthermore the stress hormone oxytocin (which is enhanced by social contact and social support) may also play a role in stress resilience and reducing the adverse health consequences of the stress response, and cardiac health in particular (McGonigal, 2013). It is therefore feasible that the links between Type D personality and ill health may be mediated by maladaptive coping strategies which could also be exacerbated by lower levels of social support characteristically suffered by socially inhibited individuals (Denollet, de Jonge, et al., 2009).

Previous research has found links between Type D personality, maladaptive coping strategies and lower levels of social support. For example; Polman et al., (2010) found that Type D individuals were more likely to use more passive and maladaptive avoidance coping strategies such as resignation and withdrawal, and these partially mediated the relationship between Type D and increases in perceived stress. However, in this particular study levels of social support were not implicated in the relationship. Although, a study by Williams and Wingate (2012) found that levels of Type D personality were inversely correlated with social support, in addition to problem-focused and emotion-focused coping, increased self-reported perceived stress and avoidant coping. Social support and avoidant coping fully mediated the relationship between Type D and physical symptoms, and social support and emotion-focused coping partially mediated the relationship between Type D and perceived stress (Williams & Wingate, 2012).

In summary, it appears justified to examine how coping strategies and levels of social support may mediate the relationship between Type D personality and the reporting of physical symptoms. If links are demonstrated, these factors could play an important part in attempts to intervene and attenuate the negative effects of Type D on ill-health in the future.

1.5.3.2 Health behaviours

In addition to the direct influences of psychosocial factors on health and biological systems, evidence suggests that psychological distress is likely to increase an individual's likelihood of engaging in unhealthy behaviours. Behaviours associated with Type D include increased propensity for alcohol and drug use, poorer diet, and lower physical activity. All of which could lead to cardiovascular, immunological, and endocrinological consequences and ultimately ill health (Kiecolt-Glaser & Glaser, 2002). Previous research has demonstrated a tendency for Type D individuals to engage in more unhealthy behaviours such as smoking, poor eating habits and participating in less physical activity in comparison to non-Type D individuals (Gilmour & Williams, 2012). For example, one study demonstrated a link between Type D and reduced exercise status and sedentary behaviour in males (Borkoles, Polman &

Levy, 2010) and the researchers suggested that an exercise intervention may be beneficial to reduce negative health effects associated with Type D. However, another study found no differences in activity or exercise participation between Type D or non-Type D females (Borkoles et al., 2015).

Type D personality may also represent a risk factor for unhealthy eating (Booth & Williams, 2015). Results from the study by Booth and Williams (2015) showed an association between Type D personality and lower healthy food intake, including higher consumption of fat and sugar, and lower consumption of fruit and vegetables. The relationship was found to be mediated by coping, suggesting that Type D individuals may use unhealthy eating as a coping mechanism in the face of adverse events. Further, a large scale cross-sectional study also demonstrated a higher prevalence of metabolic syndrome in Type D individuals, which was attributed to poorer lifestyle choices such as reduced physical activity, consuming a less varied diet, and a higher fat intake (Mommersteeg, Kupper & Denollet, 2010). This suggests that Type D individuals may exhibit a behavioural as well as a physiological vulnerability to negative health outcomes.

It appears feasible that Type D individuals may engage in fewer health promoting and more adverse health behaviours due to high levels of negative affect and social inhibition. Previous research has indicated links between depressive symptoms and low social support with poor health behaviours such as partaking in less exercise and irregular sleeping patterns (Allgower, Wardle & Steptoe, 2001). The propensity of Type D individuals to inhibit emotions may accentuate this tendency further by reducing the likelihood that the individuals will engage with activities designed to lessen performance of these behaviours (e.g. attending support groups or the gym) due to their preference for avoiding social contact. For example, levels of social anxiety have been found to correlate with avoidance of sporting activities (Norton, Burns, Hope & Bauer, 2000). It is therefore plausible that participation in adverse health behaviours may be involved in the link between Type D and physical health, and may represent an additional behavioural mechanism which may exacerbate psychobiological

influences. Consequentially, consideration of health behaviours in the general population is warranted, to further understand the influence of this factor on the relationship between Type D and physical health.

1.5.3.3 Sleep problems

Reduced sleep quality has been associated with social and emotional difficulties and higher levels of stress and distress (Gregory & O'Connor, 2002; Lund, Reider, Whiting & Prichard, 2010; Vgontzas et al., 2008). The HPA axis plays an important role in maintaining alertness and modulating sleep (Buckley & Schatzberg, 2005). As Type D may be associated with dysregulated HPA activity, it seems plausible that sleep may also be involved in the link between the personality disposition and ill health within the general population. Existing research has demonstrated that Type D is associated with increased sleeping problems (Fruyt et al., 2002) and shorter sleep duration in adolescents (Condén, Ekselius, et al., 2013). A potential explanation for this is that Type D personality may adversely affect sleep due to insufficient stress-coping strategies (Condén, Ekselius, et al., 2013) leading to abnormalities with the HPA axis. Dysregulation of the HPA axis could lead to disruptions to circadian rhythms, resulting in increased periods of wakefulness and reduced sleep quality (Buckley & Schatzberg, 2005). Poor sleep is also often associated with short-term increases in the activity of the major stress systems, including the SAM axis and the HPA axis (Meerlo, Sgoifo & Suchecki, 2008) (See chapter 6 for a review of these systems). Sleep abnormalities can directly activate these biological systems, but can also affect the reactivity of these systems to other stressors over time (Meerlo et al., 2008). Therefore, as it is postulated that these stress systems may be dysregulated in Type D individuals, potentially leading to the observed ill-health effects, it is possible that poor sleep may contribute to this mechanism.

However, it is also plausible that the reverse causality may be true, in that sleep problems may lower mood (e.g. Bonnet, 1985) and conversely evoke an increase in Type D attributes in individuals. For example, sleep problems have been associated lower perceived quality of life as well as poorer physical and mental health (Paiva, Gaspar & Matos, 2015),

and disturbed sleep may lower positive affect and psychological well-being (Buysse, Reynolds, Monk, Berman & Kupfer, 1989, O'Leary et al., 2013). Furthermore, it is feasible that fatigued or sleep deprived individuals may be less inclined to attend social events and interact with others, therefore leading to higher scores on measures of social inhibition. In summary, it is appears justified to examine whether there may be an association between self-reported sleep and Type D personality, and if a link is found, further longitudinal studies may be warranted to elucidate the causal direction of this relationship.

1.5.3.4 Anxiety, depression and other psychological distress

Not surprisingly, research has demonstrated that increased indices of anxiety and depression tend to accompany Type D personality, particularly in clinical populations, as previously discussed above (e.g. Howard & Hughes, 2012; Pedersen et al., 2004; Spindler et al., 2007). Type D individuals experience higher levels of anxiety and depressive symptoms in addition to emotional and social difficulties (Denollet, 1998, 2005). Type D personality has been linked to increased symptoms of anxiety and depression but also, social alienation and vital exhaustion (Pedersen, van Domburg, Theuns, Jordaens & Erdman, 2004) in addition to chronic stress, hostility and pessimism (Fruyt et al., 2002).

Studies have shown that poorer subjective well-being, reduced self-esteem, life dissatisfaction (Pedersen & Denollet, 2003), and poorer body image perceptions (Borkoles et al., 2010) are also more prevalent within Type D populations. Type D personality has also been associated with more retrospectively reported alienation from parents in childhood (van den Broek et al., 2010) Furthermore, in a sample of Icelandic coronary patients, Type D personality was independently associated with increased self-reported risk of anxiety, depression and stress (Svansdottir et al., 2012).

Earlier studies postulated that depression may underpin the link between Type D and mortality (e.g. Jonge, Denollet & Melle, 2007), however, in conjunction with the recent criticisms surrounding Type D research, this premise has been disputed (Coyne et al., 2011). For instance, a study of COPD patients demonstrated that although Type D personality was

associated with depressive symptoms, it did not mediate the relationship between depressive symptoms and mortality (De Voogd et al., 2009), thus bringing into question the real-world implications of the construct in terms of hard endpoints (e.g. morbidity and mortality). While it appears that Type D may lack predictive value in terms of explaining mortality via depressive symptoms as initially expected, important associations may still exist between Type D and illness progression, links which may be mediated by other aspects of psychological distress and/or similar factors. For example, higher levels of anxiety and depression have also been linked to increased pain sensitivity (e.g. Lee et al., 2009), sleep problems (e.g. insomnia; Taylor, Lichstein, Durrence, Reidel & Bush, 2005) and maladaptive coping strategies (e.g. self-blame, rumination, catastrophising; Garnefski, Legerstee, Kraaij, Van Den Kommer & Teerds, 2002). It is therefore likely that anxiety and depression may also be involved in the complex pathways underpinning the link between Type D personality and poorer health outcomes, and consideration of these factors in research examining these pathways is required.

1.6 Key points and conclusions

In light of the findings regarding the link between Type D and poor health in cardiac and clinical populations it appears important to examine the influence of Type D in apparently healthy individuals.

Despite the criticisms of the Type D construct, and the scepticism regarding its predictive ability, it is clear that further examination of the relationship between Type D personality and physical health is required. The literature pertaining to Type D in the general population suggests a number of psychobiological and behavioural mediating factors may be involved (see figure 1), but clarification is needed. It therefore appears necessary to investigate the interaction of behavioural and psychobiological mechanisms by which Type D personality may manifest in physical symptoms and potentially lead to negative health outcomes in later life. Further, research examining this relationship could have important implications for clinical practice.

It appears that the personality disposition may have particularly noteworthy effects on stress-related outcomes, and therefore the Type D-stress relationship will be a particular focus within this project. Nonetheless, it does appear likely that a number of other mechanisms may also be involved. Therefore, the mechanisms and potential mediating factors presented thus far are summarised in the model in figure 1.1 below.

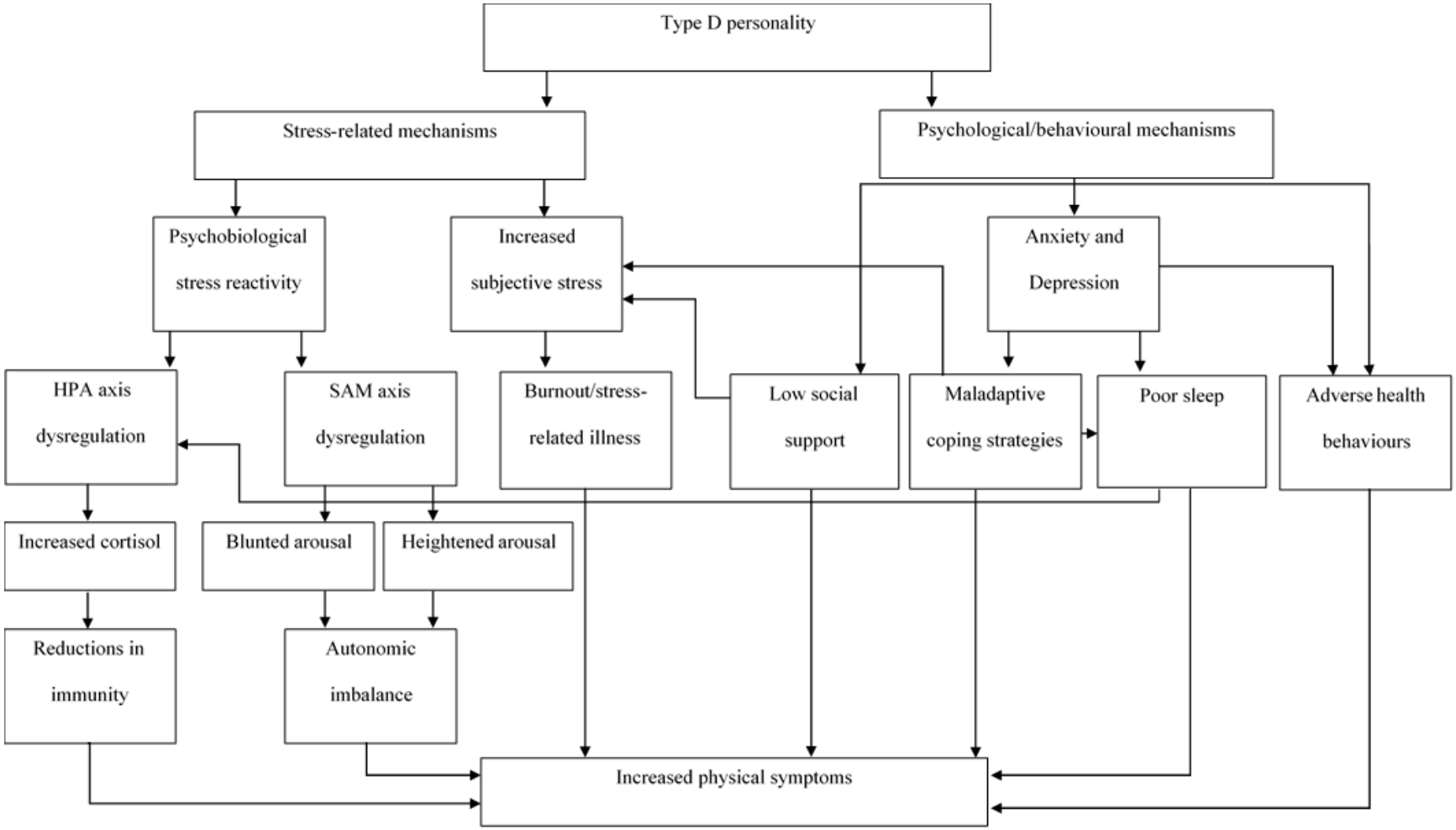

Figure 1.1 The potential mechanisms and mediating factors involved in the relationship between Type D personality and increased physical symptoms, as derived from the literature.

1.7 Project overview and rationale

This thesis aims to investigate the prevalence and effects of Type D personality on physical health in the general population. The associations between Type D and physical health problems, and the underlying mechanisms have not yet been adequately investigated in otherwise healthy individuals. This project will provide an original contribution to this area and address this gap in the literature by providing insight into the associations between Type D personality and physical health, as well as the underlying psychobiological pathways. Conceptualisation of the Type D construct will also be considered, and analyses will be conducted with a particular focus on the dimensional approach to the Type D construct.

This programme will commence with a large scale assessment of the relationships between Type D and a number of the outcomes discussed above. The project will then attempt to bridge a number of gaps in the literature in order to elucidate potential mediating relationships and investigate a number of important potential psychobiological mechanisms. In addition, once these pathways have been established, an intervention designed to attenuate the negative effects associated with Type D will be tested.

1.7.1 Aims and objectives

This programme of research aims to investigate the mechanisms underpinning the link between Type D personality and physical health within the general population. More specifically the programme aims to address the following research objectives:

i) Establish the presence of relationships between Type D personality and aspects of subjective physical health in the general population as well as potential psychophysiological and behavioural mediators determined from the above review of the literature.

ii) Prospectively investigate the relationships between Type D personality and physical health to determine the development and possible dynamic nature of the relationships and the potential mediating factors involved.

iii) Experimentally explore the potential psychobiological mechanisms which may underpin the association between Type D personality and the aspects of physical health identified in earlier studies, using objective measures of sympathetic arousal, cardiovascular stress reactivity and immune function within a group of healthy individuals.

iv) Identify and assess the efficacy of a positive psychological intervention which may aid in attenuating some of the negative influences that Type D personality may exert on the physical and psychological health outcomes demonstrated within the project.

1.7.2 Chapter Summaries

The individual chapters within this project attempt to collectively address the aims of the project described above. An overview of the aims of each of the chapters is outlined below.

1.7.2.1 Chapter 2 - An overview of the general methods implemented in the investigation of the relationship between Type D personality and physical symptoms.

As a standard collection of self-report instruments were used across all studies to assess the outcomes investigated within this project, the second chapter in this thesis will describe each of these in detail. This will include how Type D personality was assessed, conceptualised and analysed in each of the four studies. The ethical considerations applied to this project and how data was treated (including management of outliers) will also be explained.

1.7.2.2 Chapter 3 - The assessment of physical symptoms in the general population; exploration of the factor structure and psychometric properties of the Cohen-Hoberman inventory of physical symptoms.

The main outcome measure of this project comprises self-reported physical symptoms.

The majority of established tools assessing physical symptoms tend to consider all symptoms collectively; however, given that different symptoms manifest via different psychobiological pathways, consideration of different symptom clusters is perceived as necessary. This third chapter will therefore describe the suitability of the Cohen-Hoberman inventory of physical symptoms (CHIPS; Cohen & Hoberman, 1983) as the main self-report health assessment tool within this project; and describe the findings of a psychometric evaluation study which establishes the potential underlying factor structure and psychometric properties of the instrument.

1.7.2.3 Chapter 4 - Initial cross-sectional examination of the relationships between Type D personality and physical health symptoms within the general population.

Chapter 4 attempts to address the first aim outlined above. As the existing literature is missing an all-encompassing large scale assessment of Type D personality within the general population, this Chapter describes a cross-sectional, online-questionnaire based study which was conducted in order to establish the presence of any relationships between Type D personality and physical symptoms. In addition, links between Type D personality and a number of stress-related (subjective stress, perceived stress reactivity), psychological (anxiety, depression, sleep quality) and behavioral variables (social support, coping strategies, health behaviours) are also explored.

1.7.2.4 Chapter 5 - A longitudinal investigation into the relationship between Type D personality and 'stress-related' physical symptom clusters: A one-year follow up study.

A necessity for prospective research into the relationship between Type D personality and physical health has been highlighted. This is particularly important in order to determine how Type D may influence deterioration in subjective health over time. Consequently, Chapter 5 aims to address the second aim of thesis project and discusses the findings of a follow up phase of the initial, cross-sectional, online-questionnaire based study outlined in the previous chapter. This longitudinal study further explores the relationships between Type D personality and physical symptoms. An additional aspect comprises the consideration of

potential mediating influences of a selection of the stress-related and psychological variables assessed within the first study.

1.7.2.5 Chapter 6 - A review of the mechanisms of the stress response, including the techniques for inducing acute stress and measurement of stress biomarkers.

It is suggested extensively in the existing Type D literature that stress reactivity is likely to play a role in the relationship between Type D personality and poorer health outcomes, within cardiac, other-clinical, and general populations. Chapter 6 provides an overview of the literature describing the systems involved in the psychobiological stress response. In order to provide a thorough background for the subsequent chapter, methods for measuring stress reactivity and inducing stress in the laboratory are also reviewed.

1.7.2.6 Chapter 7 - An experimental investigation of sympathetic arousal, cardiovascular stress reactivity and inflammatory activation in Type D individuals.

Chapter 7 addresses the third aim of this project by detailing the investigation of potential psychobiological mechanisms that may underpin the links between Type D personality and physical symptoms in light of the findings of Chapters 4 and 5. The primary aim of the chapter is to examine sympathetic activation in response to acute stress in relation to Type D personality. This is accomplished via the measurement of a range of beat-to-beat cardiovascular parameters (including blood pressure, heart rate and the underlying determinants of haemodynamic profile), in addition to salivary alpha amylase levels (a reliable biomarker of sympathetic activity). Levels of C-Reactive protein (an inflammatory biomarker indicative of poor cardiac health) in relation to Type D personality is also investigated in light of the pro-inflammatory mechanism hypothesis discussed earlier. This chapter aims to provide novel findings regarding sympathetic dysregulation as a potential contributing factor to the influence Type D personality exerts on health.

1.7.2.7 Chapter 8 – Examination of the efficacy of an online positive writing intervention for enhancing well-being in Type D individuals.

Chapter 8 addresses the final aim of the project as outlined above. This chapter concerns the final empirical study of this project which was conducted to ascertain whether the influence of Type D personality on the physical symptoms identified within the project and aspects of psychological distress could be alleviated by a positive writing intervention. Language analysis was also conducted as a secondary aim to ascertain whether the language use of Type D individuals may differ from non-Type Ds. Overall, this chapter aims to determine the efficacy of a positive writing intervention in Type D individuals from the general population.

1.7.2.8 Chapter 9 – General discussion of findings

This Chapter summarises the findings of each individual study, and explains how this project has made a novel contribution to knowledge. An in depth consideration of the conceptualisation of the Type D construct is outlined and the implications of this research as whole are discussed. Furthermore, strengths and limitations of the study designs, methodologies implemented and analytic strategies are also highlighted.

Chapter 2: An overview of the general methods implemented in the investigation of the relationship between Type D personality and physical symptoms

This chapter will describe the ethical considerations applied to each study within this project and the self- report measures that were utilised across the four studies. Fundamental aspects of data treatment across all studies and the ways in which Type D personality was defined and analysed will also be explained.

2.1 Ethical considerations

All studies were approved prior to commencement by the Northumbria University Faculty of Health and Life Sciences Ethics Committee. Participants were provided with full information before commencing each study, required to provide informed consent and advised of their rights to withdraw. All participants were fully debriefed once their participation in the study was complete. All participants were allocated a participant number to maintain anonymity, and in the online studies were required to provide a unique code word in order to identify their data. All electronic data was stored on a password protected computer and paper-based data in a locked filing cabinet in accordance with the Data Protection Act 1998.

2.2 Self-report measures.

The following self-report measures were used throughout the studies undertaken as part of this project. These measures were either completed online on Qualtrics.com or were paper-and-pencil based.

2.2.1 Type D personality

Type D personality was assessed using the Type D Scale-14 (DS14; Denollet, 2005). The DS14 is a brief psychometrically sound measure of negative affectivity (NA) and social inhibition (SI) devised specifically for the purpose of defining Type D personality. The instrument comprises a 7-item subscale measuring negative affectivity covering aspects of

dysphoria, worry and irritability (e.g. 'I often feel unhappy') and a 7-item subscale measuring social inhibition comprising discomfort in social situations, reticence and lack of social poise (e.g. 'I often feel inhibited in social situations'). In initial validation studies (Denollet, 2005) both the NA and SI scales were found to be internally consistent with Cronbachs α= 0.88 and 0.86, and stable over a 3-month period (test–retest reliability coefficients of 0.72 and 0.82, respectively).

2.2.2 Physical symptoms

The Cohen-Hoberman Inventory of Physical Symptoms (CHIPS; Cohen & Hoberman, 1983) was used as the main self-reported health outcome measures in this project. The CHIPS is a list of 33 common physical symptoms and asks respondents to rate 'how much that problem has bothered or distressed you during the last two weeks including today?'. The symptoms include items such as 'back pain' and 'diarrhoea' (see table 2 in Chapter 3 for full item list) and are rated on a 5-point Likert scale ranging from (0) 'not been bothered by the problem' to (4) 'extremely bothered by the problem' for how much that item bothered the individual during the past two weeks. The total score is the sum of the responses on the 33 items (possible score range 0-132). Cronbach's alpha for the overall CHIPS scale was .92 indicating good internal consistency.

In order to distinguish between different 'clusters' of symptoms, factor analysis was performed on data obtained in the first online questionnaire-based study of this project in order to identify which physical symptom groups may be related to Type D personality. The factor structure and psychometric properties of the CHIPS are reported and discussed in chapter 3.

2.2.3 Subjective health complaints

The Subjective Health Complaints inventory (SHC; Eriksen, Ihlebæk & Ursin, 1999) was used to assess the content validity of the CHIPS factor structure. The inventory comprises 29 items concerning subjective somatic and psychological complaints experienced during the last 30 days. The inventory provides five individual scores indicating severity and frequency of five subcategories of symptoms. Severity of each complaint is rated on a 4-point scale (0=

'not at all' to 3 = 'serious') and is also scored for duration (number of days on which the symptom was experienced) during the last 30 days. The subscales were scored by simple summation of the raw scores for severity for each of the items. Higher scores indicate increased subjective severity of these health complaints with maximum scores of 24 for musculoskeletal pain, 21 for pseudoneurology, 21 for gastrointestinal problems, 15 for allergy and 6 for flu-like symptoms. A duration score could also be calculated from the scale by multiplying severity (0 - 3 on single items) by duration (number of days/10). Eriksen et al., (1999) indicated each of the subscales exhibited adequate internal consistency as follows: flu-like symptoms (2 items; α= 0.67) musculoskeletal pain (8 items; α= 0.74), pseudoneurology (7 items; α=0.73), gastrointestinal problems (7 items; α= 0.62) and allergies (5 items; α= 0.58).

2.2.4 Anxiety and depression

Levels of psychological distress were gauged across studies using the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1994). The HADS comprises 14 items each with 4 answers coded between 0 and 3 (positively worded items are reversed scored). Seven items measure anxiety (α= 0.83) and 7 items measure depression (α=0.82), a separate score is derived for each of the scales with higher scores indicating higher levels of anxiety and depression, respectively.

2.2.5 Perceived stress

The Perceived Stress Scale (PSS; Cohen, Kamarck & Mermelstein, 1983) was used to assess levels of perceived background stress in all studies. The PSS is a 10-item scale that assesses how respondents have experienced and dealt with stressful situations in the past month and includes items such as "felt nervous and stressed?" and "felt that you were unable to control the important things in your life?". Response choices are on a 5-point Likert scale and range from (0) "never" to (4) "very often" and a number of items are reverse scored. Scores are calculated by adding up the 10 item ratings, and range from 0 to 40, with higher scores indicating higher levels of perceived stress. Cronbach's alpha for the PSS was reported as 0.85 indicating high internal consistency.

2.2.6 Perceived stress reactivity

Subjective stress reactivity was measured by the Perceived Stress Reactivity Scale (PSRS; Schlotz, Yim, Zoccola, Jansen & Schulz, 2011). The PSRS comprises 23 items with five subscales and one overall total score. Each item provides four possible answers coded 0 to 3 (12 items are reversed scored). The five separately scored subscales of reactivity include 4 items of Prolonged Reactivity (PrR, α=.69), 5 items of Reactivity to Work Overload (RWO, α=.82), 5 items of Reactivity to Social Conflict (RSC- α=.77), 4 items of Reactivity to Failure (RFa, α=.73) and 5 items of Reactivity to Social Evaluation (RSE α=.72). The sum of all subscales provides a total perceived stress reactivity score (PSRS-Tot α=.91) with higher scores indicating increased levels of reactivity to stress.

2.2.7 Maladaptive coping strategies

The Brief COPE (Carver, 1997) is a shortened 28-item scale of the full 60-item COPE scale (Carver & Scheier, 1989). The Brief COPE comprises 14 coping types including self-distraction, active coping (e.g. 'I've been taking action to try to make the situation better'), denial (e.g. 'I refuse to believe that it has happened'), substance use (e.g. 'I use alcohol or other drugs to make myself feel better'), use of emotional support (e.g. 'I get emotional support from others'), use of instrumental support (e.g. 'I get help and advice from other people'), behavioural disengagement (e.g. 'I give up trying to deal with it'), venting (e.g. 'I express my negative feelings'), positive reframing ('I've been looking for something good in what is happening') planning (e.g. 'I've been thinking hard about what steps to take'), humour (e.g. 'I've been making jokes about it'), acceptance (e.g. 'I've been learning to live with it'), religion ('I've been praying or meditating') and self-blame (e.g. 'I've been criticising myself). Participants self-report their coping strategies on a 4-point Likert-type scale which ranges from 1 (I don't do this at all) to 4 (I do this a lot). Each of the items are summed together to derive an overall score, with 8 items reverse scored. In the present project the items of the following 'maladaptive' coping strategies were summed; self-distraction, denial, substance use, use of emotional support, behavioural disengagement, venting and self-blame. Higher

scores indicate increased use of maladaptive coping strategies (range: 14–56). Cronbach's alpha for the scale was reported as .76 indicating good internal consistency.

2.2.8 Quality of social support

Questions about quality of social network and social support (SNSS) taken from Dalgard, Björk and Tambs (1995) were used to measure quality of social support. The questions included 12 items regarding participants' perceptions of their relationship to friends, family and neighbours. Questions were answered on either a 'yes' or 'no' basis, or on a 3 or 4 point Likert scale. Eight out of the 12 items were reversed scored and higher scores indicated greater quality of social support. Four questions asked about family, five about friends and three related to neighbours. Item responses were summed to produce a total score relating to quality of social support. Adequate internal consistency was reported as 0.67 (Dalgard, Björk & Tambs, 1995).

2.2.9 Health behaviours

Health behaviours were measured by a set of 10 questions utilising a similar scale to the Diabetes Self-Care and Activities Scale (Toobert et al., 2000) adapted to only incorporate behaviours applicable to the general population. The questions assessed alcohol consumption, smoking, physical activity and good dietary habits. Three questions were related to alcohol consumption, three related to smoking, two were in regards to physical activity and two more ask about diet. Each question required a multiple choice response from each respondent; 'Yes' or 'No' on whether this behaviour was performed (alcohol and tobacco use only) or number days this behaviour occurred in an appropriate given time scale (week/ fortnight/ month). The scores were summed to produce a total score for each health behaviour. Higher scores on the alcohol and tobacco subscales indicated increased participation in poorer health behaviours, and higher scores on the physical activity and good dietary habits subscales indicated increased participation in healthy behaviours. See Appendix B for Health Behaviour questions.

2.2.10 Sleep quality

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989)is a self-rated questionnaire which was used to assess sleep quality in participants over a one-month period. It contains nineteen items which generate seven component scores to distinguish 'good' from 'poor' sleep as follows: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The answers are scored on a 0 (positive extreme) to 3 (negative extreme) Likert scale and the sum of the seven component scores yields one total score. Higher scores indicate higher incidence of sleep problems (i.e. poorer sleep quality). The seven component scores had an overall Cronbach's alpha of .83 (between .35 and .76 for each component) and test-retest reliability coefficient of .85 (between .65 and.84 for each component) (Buysse et al., 1989). The total score was used in this project to ascertain the influence of Type D personality on general sleep related problems.

2.2.11 Pain sensitivity

The Pain Sensitivity Questionnaire (PSQ; Ruscheweyh et al., 2009) is a self-rating instrument for the assessment of pain sensitivity. The PSQ comprises 17 items describing a situation in which an individual may feel pain. Participants are required to indicate on a ten-point scale how painful they would imagine this situation to be (0=not at all painful at all, 10 = most severe pain imaginable). Three items are included as filler questions and are not scored. Higher scores indicate higher levels of self-reported pain sensitivity. The PSQ covers 2 components; a minor pain (7 items) and moderate pain (7 items) score to provide a total pain sensitivity score (14 items). Cronbach's alpha was reported as 0.92 for total pain, 0.81 for minor pain and 0.91 for moderate pain.

2.2.12 Big 5 personality traits

To assess the Big 5 personality traits, the Big-five Mini-markers (Saucier, 1997) was utilised. This scale is a brief version of Goldberg's (1992) 100 unipolar adjective markers developed to measure the big-five factors of personality. The scale comprises a list of 40

adjective markers, of which eight correspond to each of the five factors; Extraversion, Agreeableness, Conscientiousness, Emotional stability, and Openness. Responses are measured on a 9 point Likert scale (1 = 'Extremely inaccurate' to 9 = 'Extremely accurate') and negatively worded items are reverse scored. Scores are summed and larger scores indicate higher levels of that particular trait. The five subscales are assessed separately and all show good internal consistency, with Cronbach's alpha levels reported between .76 and .86 (Saucier, 1997).

2.2.13 Trait anxiety

The State-Trait Anxiety Inventory Short Form (STAI-SF; Marteau & Bekker, 1992) comprises two 3-item subscales, measuring state anxiety. The STAI-SF requires participants to rate how they 'feel right now' in regards to 6 statements on a four-point scale ranging from 'not at all' to 'very much so'. Reverse scoring was used for positively worded items (e.g. 'I feel content'), so that the highest level of anxiety for an individual item was represented by a score of 4. Total scores were calculated by summing together the scores for all 6 items. Total scores ranged from 6 to 24.

2.2.14 Retrospective health

A number of retrospective health questions were used in the online follow-up study as a supplementary assessment of physical health and health care utilisation in participants over a one-year period. These comprised a set of 11 questions, each of which implemented a 5-point multiple choice response. Questions included those associated with perceptions of general health (e.g. how do you rate your current general health), frequencies of illness (how often have you suffered a non-serious illness?), use of healthcare services (how often have you visited your GP or other healthcare professional?) and seeking of medical advice (how often have you sought advice about health problem?). Example responses included scales of frequency (e.g. 'never' to 'often') and perceptions of quality (e.g. 'very good' to 'very poor'). Each question provided data comprising categorical responses and were analysed separately using Chi squared tests. See Appendix C for Retrospective Health Questions.

2.3 Treatment of data

2.3.1 Missing data

For individual missing values, a mean substitution method was implemented when single answers were omitted from each measure. Missing values were substituted by the within subjects mean score of each subscale for all questionnaires with the exception of the physical symptoms for which missing values were replaced with 0. It has been previously stated that up to 50% of items on each subscale on the SHC (Eriksen et al., 1999) and 20% on the PSRS scales (Schlotz et al., 2011) can be substituted. For the remaining measures no particular cut-off was stipulated in the published article, therefore the missing responses for up to 2 items per questionnaire were inputted; otherwise the individual's data for that measure were excluded. This technique is in line with similar missing data methods used in a variety of questionnaire based research studies (Roth, 1994).

2.3.2 Treatment of outliers

There has been much debate in the literature regarding the treatment of outliers, however, it is argued that they must be considered in large data sets such as those obtained within the present project. Outliers can cause problems for statistical analyses by potentially increasing error variance, reducing power, decreasing normality, and are likely to alter the likelihood of both type I and type II errors (Osborne & Overbay, 2004). Self-report data; which constitutes the majority of data collected in the present project, can be subject to self-report bias and social desirability, as well as human scoring errors, therefore extreme values observed in this data may require exclusion. Similarly, unusual values observed in biological data (such as the salivary alpha amylase data collected in the experimental study) may be also constitute a special case or a mistake in the data collection or assaying procedure and therefore removal of these data points must also be considered. Consequentially, before statistical analyses were conducted on the data collected within this project, scatterplots and histograms were firstly examined to determine the distribution of data, and to identify extreme data points. Where obvious outliers were present, a common, simple and relatively effective rule (Osborne &

Overbay, 2004) was followed in which data points that fell 3 standard deviations above or below the mean were removed. To ensure consistency across studies, this technique was applied to all psychological factors and biological data in each study within the present project, before statistical analyses were performed.

This outlier elimination procedure has however been known to produce problems in highly skewed distributions (Osborne & Overbay, 2004). Physical symptom data is generally subject to positively skewed distributions, as many participants, (particularly those who are considered healthy), will report zero, or very few, health complaints (Eriksen et al., 1999). It was therefore decided that extreme values may represent an aspect of the intrinsic variability of the physical symptoms data, and removal of outliers using this technique would risk the loss of real data points. Outliers were therefore not removed from the physical symptoms data, a decision which is in line with other research examining physical symptoms and health complaints within the general population (e.g. Ihlebaek, Eriksen & Ursin, 2002).

2.3.3 Analysis of Type D personality

Although initially developed as a taxonomy, the Type D construct has been suggested to be better represented as a dimensional rather than a categorical construct (Ferguson et al., 2009). Each approach is explained below and both were used in analyses in the present project.

2.3.3.1 Categorical construct

To distinguish Type D individuals from non-Type D individuals, the traditional dichotomous approach was used. This traditional categorical assessment of Type D classifies individuals scoring 10 or above on both SI and NA scales of the DS14 as Type D, and those who score below 10 on both, or either one of the scales as non- Type D. The categorical representation of Type D was utilised in statistical analyses (independent samples t-tests, factorial ANOVAs and Chi-squared analyses) in this project to ascertain whether there were significant differences between the two groups on the outcome measures.

2.3.3.2 Dimensional construct

In order to analyse Type D as a dimensional construct a continuous measure of Type D was computed using the arithmetic product of SI and NA scores. Correlational analyses which were conducted across the studies in this project were undertaken with this variable. In order to ascertain the influence of the dimensional Type D interaction (i.e. synergistic effect of NA and SI) on the outcome variables explored in this project, hierarchical regression analyses (using the enter method) were performed, controlling for the separate effects of NA and SI. In these analyses NA and SI were entered as predictors in step 1, and the Type D (NA × SI) interaction score was then added at step 2, to determine whether the synergistic effect had a significantly greater predictive value than that of NA and SI considered separately on each outcome variable.

Chapter 3: The assessment of physical symptoms in the general population; exploration of the factor structure and psychometric properties of the Cohen-Hoberman inventory of physical symptoms

---

This chapter will discuss how physical symptoms were assessed within the current project. Firstly, the importance of exploring clusters of particular symptoms will be addressed, and a number of measures considered. Secondly, the potential underlying factor structure and psychometric properties of the CHIPS will be described and its suitability for use as the main self-report health assessment tool within this project.

This chapter is based on the following published paper:

Allen, S. F., Wetherell, M. A. & Smith, M. A. (2017). The Cohen–Hoberman inventory of physical symptoms: Factor structure, and preliminary tests of reliability and validity in the general population. *Psychology & Health*, *32*(5), 567-587.

3.1 Introduction

Subjective health complaints and physical symptoms are very common; can often be acknowledged as 'normal', and are not necessarily associated with any specific diagnosis of illness. However, the experience of physical symptoms that do not meet the diagnostic criteria for particular illnesses or disorders can have noticeable negative effects on physical and psychological well-being, hence the importance of this project. In the general population as a whole, everyday physical symptoms are highly prevalent, can be bothersome and can even cause substantial distress. The most common symptoms measured in a community sample of over 13,000 individuals were found to be joint pains (36.7%), back pain (31.5%), headaches (24.9%), chest pain (24.6%), pain in the limbs (24.3%), stomach pain (23.6%), fatigue (23.6%), and dizziness (23.2%), with almost one third of symptoms being attributed to a psychosomatic or unexplained cause (Kroenke & Price, 1993). As the aim of this project is to explore the influence of aspects of personality, namely Type D characteristics, there is a

requirement for reliable assessment tools to do so. The experience of physical symptoms has been linked to indices of psychological well-being including fluctuations in both negative (Leventhal, Hansell, Diefenbach, Leventhal & Glass, 1996; Watson & Pennebaker, 1989); Watson & Pennebaker, 1989) and positive (Watson, 1988) affect, other personality factors including neuroticism (Brown & Moskowitz, 1997) and optimism (Rasmussen, Scheier & Greenhouse, 2009). An abundance of literature has also linked typically psychological concepts such as chronic stress to increased incidences of a variety of psychological and physical health outcomes (Juster, McEwen & Lupien, 2010) including depression and anxiety; increased susceptibility to viruses such as the common cold (Kiecolt-Glaser et al., 1987) and cardiovascular disease (Steptoe & Kivimäki, 2012). This further exemplifies the necessity for easy to use self-reported health assessment tools in research such as this.

However, there is no general consensus on how to measure physical symptoms. Some large surveys simply rely on standalone questions, and other measures tap into other aspects of ill-health, such as quality of life or levels of disability (e.g. The Health Assessment Questionnaire; Fries, Spitz, Kraines & Holman, 1980; The Health status index; Kaplan, Bush & Berry, 1979). A number of health related assessment tools are currently available including the Nottingham Health Profile (Hunt, Mcewen & Mckenna, 1985), the Short Form Health Survey (Ware & Sherbourne 1992) and the Child Health Questionnaire (Landgraf, Abetz & Ware, 1996). However, these tools are typically implemented in clinical samples and are less appropriate for use in the general population. A number of brief tools have also been developed to specifically and simply measure the experience of symptoms (e.g. SHC; Eriksen et al., 1999) which are particularly useful for research in healthy populations, as they fundamentally gauge the event to which general everyday symptoms can be bothersome for individuals on a straightforward and easily-scored rating scale.

A number of complications can arise when attempting to adequately measure physical symptoms as a construct in the general population. The frequency and severity of physical symptoms tend not to follow a normal distribution (i.e. data is often skewed), as many people do not suffer from the more frequent symptoms at any one time (Eriksen et al., 1999). This

poses particular problems for statistical analyses of symptom data in a research context. Furthermore, all symptoms are not necessarily demonstrable of general ill health and can be interpreted differently. If they are all similar, a high overall score could represent an isolated illness with similar symptoms. On the other hand, if an individual experiences many different symptoms it could be representative of more general poor health status. It could be argued that the only reliable way to measure health is to consider the experience of symptoms on a case by case basis, although this is time consuming and complicated, particularly in large data sets. It is therefore beneficial to consider physical symptoms as groups or clusters of similar ailments, which may logically be related to a common underlying cause and/or occur simultaneously.

It is particularly important to understand more specifically the types of health complaints that Type D individuals might experience. For example, given that Type D is associated with cardiac problems (e.g. Borkoles, Polman, Ski & Thompson, 2012), it may be expected that Type D individuals from the general population may report more cardiac related symptoms.

3.1.1 Symptom clusters

General everyday symptoms, although collectively representative of poor health, are neither entirely interrelated nor completely separate. It can be argued that some physical symptoms may be distinct, but some may also cluster together into similar symptom groups. This is particularly useful for the diagnosis of illnesses, as attributing ill-health to single, or even a number of independent symptoms, can be seen as inadequate. Furthermore, different symptoms can be differently associated with not only distinct illnesses but also other extrinsic factors. For example; musculoskeletal complaints can often be associated with job type or working conditions (Johansson & Rubenowitz, 1994), and there is evidence of links between anxiety symptoms and low levels of social support (Zimet, Dahlem, Zimet & Farley, 1988)

Ursin et al., (1988) were the first to conduct a factor analysis of subjective health complaints in otherwise healthy individuals and found four independent factors: ‘muscle

pain'; gastrointestinal problems; allergies/colds and pseudoneurological complaints. Similarly, Eriksen et al., (1999) found that subjective health complaints comprising the subjective health complaints (SHC) inventory could be split into five separate factors: musculoskeletal pain; allergies; gastrointestinal problems; pseudoneurology and flu; (Eriksen et al., 1999). Whilst the SHC is a useful tool for assessing general health complaints it does include some diagnoses-dependent items which may only be experienced in certain clinical populations (e.g. eczema, asthma, dyspepsia, obstipation). Furthermore, the 'allergies' subscale contains theoretically dissimilar items which seem unlikely to co-occur (e.g. chest pain and eczema) and as such, this cluster label is somewhat ambiguous.

A range of intrinsic and extrinsic factors can also contribute to the variations of the self-report of physical symptoms (Pennebaker, 1982). It could be argued that tools which assess subjective experience (e.g. severity, frequency, inconvenience etc.) of physical symptoms may be correlated with or demonstrable of psychologically based concepts, and in fact measure levels of intolerance to discomfort or distress, therefore only representing an individual's perception of their symptoms. It could therefore be suggested the scores from a self-report scale of physical symptoms could also be related to levels of psychological morbidity (i.e. anxiety and depression), perceived stress and pain sensitivity.

3.1.2 The Cohen-Hoberman Inventory of Physical Symptoms

The Cohen & Hoberman Inventory of Physical Symptoms (CHIPS; Cohen & Hoberman, 1983) was designed as a measure of perceived burden due to the experience of a range of physical symptoms. The measure has been used to assess experience of physical symptoms in different populations and has been utilised in research into individual differences and health (e.g. Smolderen, Vingerhoets, Croon & Denollet, 2007; Stevenson & Williams, 2014; Williams, Abbott & Kerr, 2015).

The CHIPS is an easily administered tool for quickly determining participant's experiences of everyday physical symptoms. However, it is somewhat limited in its original form, as it provides only a global score based on all 33 symptoms, whereas it seems logical

that certain symptoms will likely co-occur given that they may have a common underlying cause.

For research into the links between Type D personality and health it can be proposed that it would be useful to consider whether reliable and valid symptom clusters emerge on this instrument in order to provide a symptom sensitive measure of physical health complaints that can ameliorate the shortcomings of the of the SHC's ambiguous 5-factor subscale solution. Therefore, the factor structure of the CHIPS will be compared to the subscales of the SHC to indicate the benefits of utilising the CHIPS within this project.

3.1.3 Aims and objectives

The primary aim of the current chapter was to define the potential underlying factor structure and psychometric properties of the CHIPS; for use as a self-report assessment tool of general physical symptoms within this project.

3.2 Method

Participants were 535 healthy adults aged between 18 and 65 years (80.6% female, mean age = 29.80 [±12.90]) recruited using a variety of recommended online platforms (Branley, Covey & Hardey, 2014), which included dedicated participation sites (e.g. callforparticipants.com), social media (e.g. Facebook, Twitter, Reddit, and LinkedIn), university and research group mailing lists, student participation pools as well as various websites and online forums (e.g. Mums.net). Snowball sampling was also used to maximise recruitment by encouraging participants to refer the survey to friends and family friends, and/or share the study on social media. The study was also advertised via the distribution of posters and leaflets within Northumbria University. Participation was entirely voluntary and participants who wished to take part accessed the survey via an anonymous link.

A brief in-house questionnaire was used to gather demographic information including age, gender, BMI, residency, household income and employment status (see Appendix A). Participant demographic information can be observed in table 3.1.

Table 3.1. Participant demographics

		n
	Age (M, SD)	29.80 (±12.90)
Gender	Males	104
	Females	431
Employment status	Students	283
	Employed	223
	Retired/unemployed	29
Residency	UK	482
	Europe (non-UK)	13
	North America	18
	Australia	8
	Asia	9
	South America	3
	Africa	1
BMI	18- 25	345
	26-30	111
	31 +	68
Household income	£0-20k per year	213
	£20k+ per year	253
Total	N =	535

First and second year undergraduate psychology students received course credit for their participation in the study; otherwise all participants were unpaid volunteers. As the study aimed to gauge levels of common physical symptoms in the general population of healthy individuals, exclusion criteria included; those with diagnosed (albeit self-reported) mental health issues, physical conditions or sleep disorders, as these factors could influence the levels

of symptoms reported. Ineligibility was defined as receiving a formal medical diagnosis. These exclusion criteria were presented in all recruitment adverts, emails and study instructions.

3.2.1 Measures

Participants completed all 33 items of the CHIPS (Cohen & Hoberman, 1983). The SHC (Eriksen et al., 1999) was implemented to assess the concurrent validity of the CHIPS factor structure. The perceived stress scale, pain sensitivity questionnaire (Ruscheweyh, Marziniak, Stumpenhorst, Reinholz & Knecht, 2009), and the hospital anxiety and depression scale (Zigmond & Snaith, 1983) were used to determine to discriminant validity of the CHIPS instrument as a measure of physical symptoms. These measures are fully described within Chapter 2.

3.2.2 Treatment of data

Missing values for CHIPS was minimal (.102%) and ranged from .0 to .6% for each item. Missing values were managed in the PCA using pairwise deletion, as there were so few missing values to retain as much information as possible. Data for the other questionnaires were included on the basis that there were no more than two missing values for each measure per participant. Missing values for the SHC were replaced with 0, otherwise mean substitution was implemented. As discussed in Chapter 2 this is in line with similar missing data methods used in a variety of questionnaire-based studies (Roth, 1994). Therefore, 535 cases were entered into the analyses for the CHIPS and HADS data, 534 for the SHC, 524 for the PSS, and 521 for the PSQ.

Partially following recommendations by Hinkin (1995, 1998) exploratory factor analysis (Principal Components Analysis with Varimax rotation and Kaiser Normalization) was conducted on the CHIPS data in the first instance using IBM SPSS 22. The internal consistency, construct validity with the subscales of the SHC inventory, and discriminant validity with measures of pain sensitivity, psychological distress and perceived stress, were also explored.

The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis (KMO = .897 (meritorious according to Hutcheson and Sofroniou, 1999). Bartlett's test of Sphericity also showed a significant result (BS (528) = 6479.893, p<.001). Therefore, both tests suggested the suitability of the data for PCA. The 33 items of the scale were entered into the factor analysis and factor loadings greater than .35 were considered significant, as this level of significance has previously been claimed as appropriate for sample sizes greater than 250 (Hair, Anderson, Tatham & Black, 1998). Factors with eigenvalues above 1.00 were extracted in line with Kaiser's (1958) criterion resulting in an 8 factor solution.

The internal consistency of the factors was examined using Cronbach's alpha (Cronbach, 1951). To examine construct validity, Pearson's correlations were conducted between the factors and subscales of the SHC, and discriminant validity was assessed by conducting correlations between the factors and the PSS, HADs and PSQ.

3.3 Results

3.3.1 Factor structure

The factor loadings are presented in table 2. Eight factors were extracted and accounted for 58.16% of the overall variance.

Due to the inconsistent nature of acute physical symptoms in the general population, there was evidence of overlap between the factors. The items of 'faintness' and 'feeling weak' demonstrated secondary 'cross loadings' (above .40) on 'sympathetic/cardiac symptoms' and 3 other items ('acne', 'poor appetite' and 'pulled ligaments') had secondary 'cross loadings' that exceeded .35. These items were included on the factor for which they demonstrated the highest loading. Inclusion of these items onto the factor for which they demonstrated the highest loading made conceptual sense, in relation to the other items in their cluster, and this approach improved Cronbach's alpha for the factors. The final 8 factors were identified and labelled as shown in table 3.2.

Table 3.2. Factor loadings for each of the eight factors main items (factor loadings lower than .35 were suppressed).

	Rotated factor loading							
	Factor 1: Sympathetic/cardiac symptoms	Factor 2: Muscular pain	Factor 3: Metabolic symptoms	Factor 4: Gastro-intestinal symptoms	Factor 5: Vasovagal symptoms	Factor 6: Cold/flu	Factor 7: Headache	Factor 8: Minor haemorrhagic symptoms
Pains in heart or chest	.768							
Heart pounding or racing	.707							
Shortness of breath when not exercising or working hard	.648							
Hands trembling	.601							
Numbness or tingling in parts of your body	.573							
Blurred vision	.536							
Hot or cold spells	.431							
Muscle tension or soreness		.718						
Pulled (strained) muscles		.675						
Severe aches and pains		.642						
Muscle cramps		.620						
Back pain		.557						
Pulled(strained) ligaments		.532						.374

Sleep problems (can't fall asleep, wake up in middle of night or early in morning)			.692					
Weight change (gain or loss of 5 lbs+)			.604					
Feeling low in energy			.582					
Constant fatigue			.572					
Poor appetite	.368		.390					
Diarrhoea				.733				
Stomach pain				.624				
Acid stomach or indigestion				.596				
Constipation				.516				
Nausea				.498				
Dizziness	.397				.662			
Faintness	.425				.661			
Acne					.484		.354	
Felt weak all over	.451				.473			
Stuffy head or nose						.867		
Cold and/or cough						.846		
Migraine headache							.770	
Headache							.658	
Nosebleed								.657
Bruises								.495
Eigen values	9.246	1.985	1.705	1.552	1.313	1.228	1.159	1.005
Cronbach's α	.827	.752	.736	.714	.743	.837	.690	.309

3.3.2 Reliability

Cronbach's alpha for the 8 factors were; 'sympathetic/cardiac' symptoms (7 items) α= .827, muscular pain (6 items), α= .752, 'metabolic symptoms' (5 items) α= .736, 'gastrointestinal symptoms' (5 items), α=.714, 'vasovagal symptoms' (4 items), α=.743, 'cold/flu' (2 items) α=.837, 'headache' (2 items) α= .690, 'minor haemorrhagic symptoms' (2 items) α= .309. These values can be considered to represent acceptable or good internal consistency for all factors other than 'minor haemorrhagic symptoms'. There were mainly small but significant correlations evident between the symptom factors, as can be seen in table 3.3. Moderate positive correlations (between .50 and .69) were demonstrated between 'sympathetic/cardiac symptoms', 'muscular pain', 'metabolic symptoms', 'vasovagal symptoms' and 'gastrointestinal symptoms'. Negligible and low positive correlations (.00-.29) and .30 - .49 respectively) were evident between the remainder of the factors.

Table 3.3 Pearson's correlations between the sum scores (n=535) of the individual factors

	1.Sympathetic /cardiac	2.Muscular pain	3. Metabolic	4.Gastro-intestinal	5. Vasovagal	6. Cold/flu	7. Headache	8.Minor haemorrhagic
1. Sympathetic/cardiac	-	.523**	.561**	.552**	.631**	.223**	.390**	.319**
2. Muscular pain		-	.474**	.413**	.464**	.122**	.363**	.240**
3. Metabolic			-	.486**	.562**	.271**	.418**	.260**
4. Gastrointestinal				-	.601**	.248**	.372**	.253**
5. Vasovagal					-	.285**	.448**	.335**
6. Cold/flu						-	.262**	.157**
7. Headache							-	.131**
8. Minor haemorrhagic								-

*p<0.05; **p<0.01

3.3.3 Construct validity

To further establish construct validity, the factors identified were correlated with the 5 subscales of the SHC. As evident in table 3.4, high (>.70) significant positive correlations were demonstrated between the total scores on both scales (*r*= .722) and the 'cold/flu' factor of the CHIPS and the flu-like subscale of the SHC (*r*= .702) Moderate (.50-.69) significant positive correlations were demonstrated between the 'sympathetic/cardiac symptoms' and the pseudoneurology subscale of the SHC (*r*= .511), the 'muscular pain' factor and the musculoskeletal SHC subscale (*r*=.625), 'metabolic symptoms' and the pseudoneurology subscale (*r*=.660). Low significant positive correlations were evident between the pseudoneurology subscale and the 'vasovagal symptoms', and between the musculoskeletal subscale and both the 'headache' factor (*r*=.445) and 'minor haemorrhagic symptoms' (*r* =.375) factors.

3.3.4 Discriminant validity

Correlations were conducted between the derived factors and anxiety, depression, perceived stress, and pain sensitivity to assess the discriminant validity of the physical symptoms structure, see table 3.5.

The majority of the factors showed negligible correlations (.00 - .29) with perceived stress, anxiety, depression and pain sensitivity. However, perceived stress demonstrated low (.30 - .49) positive correlations with 'sympathetic/cardiac symptoms', 'vasovagal symptoms' and 'headaches' and a moderate correlation with 'metabolic symptoms'. Low positive correlations were also observed between anxiety levels and all factors apart from 'cold/flu' and 'minor haemorrhagic symptoms', and between depression levels and 'metabolic symptoms'.

Table 3.4. Pearson's correlations between the sum scores of the 5 SHC subscales and the 8 CHIPS factors

	1. Sympathetic /cardiac	2. Muscular	3 Metabolic	4. Gastro-intestinal	5. Vasovagal	6. Cold /flu	7. Headache	8. Minor haemorrhagic	Total
Cold/flu	.156**	.077	.154*	.161**	.189**	.702**	.150**	.109*	.285**
Musculoskeletal	.321**	.625**	.365**	.267**	.301**	.148**	.445**	.375**	.512**
Pseudoneurology	.511**	.401**	.660**	.361**	.463**	.179**	.334**	.149**	.624**
Gastrointestinal	.323**	.321**	.342**	.654**	.286**	.164**	.213**	.150**	.476**
Allergies	.447**	.318**	.301**	.293**	.322**	.162**	.204**	.272**	.439**
Total	.535**	.578**	.592**	.520**	.481**	.318**	.439**	.212**	.722**

*p<0.05; **p<0.01

Table 3.5. Pearson's correlations between the sum scores of the CHIPS symptom factors with scores on measures of perceived stress (PSS), anxiety, depression (HADs) and pain sensitivity (PSQ).

	1. Sympathetic/ cardiac	2. Muscular	3. Metabolic	4. Gastrointestinal	5. Vasovagal	6. Cold /flu	7. Headache	8. Minor haemorrhagic
Perceived stress	.319**	.276**	.518**	.277**	.354**	.218**	.306**	.133**
Anxiety	.434**	.355**	.566**	.327**	.421**	.167**	.328**	.192**
Depression	.269**	.179**	.489**	.196**	.283**	.119**	.156**	.053
Pain sensitivity	.121**	.110*	.110*	.069	.094*	.103*	.126**	.035

*p<0.05; **p<0.01

3.5 Discussion

The aim of the current study was to assess the internal factor structure of the Cohen-Hoberman Inventory of Physical Symptoms (CHIPS). This was proposed as necessary due to the frequency of subjective physical symptoms in the general population, and the need for the assessment and differentiation of symptom clusters for this programme of research.

The eight factor solution that emerged showed generally good internal consistency (with the exception of the 'minor haemorrhagic symptoms' scale) and demonstrated similarities wth the five factor solution of the SHC. The CHIPS does not include the additional scoring aspect of symptom duration that the SHC measures, however, its absence is not acknowledged as a limitation to the CHIPS, particularly as this aspect of the SHC scoring appears to accentuate the skewedness of the data (Eriksen et al., 1999).

Similarities were demonstrated between the SHC subscales and CHIPS factor solution, for example both scales contain factors pertaining to specifically gastrointestinal symptoms, cold and flu symptoms, and musculoskeletal complaints. However, this is not surprising given these are very common physical symptoms (Ursin, 1997). Conversely, the CHIPS solution formed a separate coherent 'headache' factor whereas the migraines item was included in the musculoskeletal SHC subscale. Symptoms that were included in the pseudoneurological SHC subscale and other logically similar items separated out onto 'sympathetic/cardiac' (e.g. heart pounding/extra heartbeats), 'vasovagal symptoms' (e.g. dizziness) and 'metabolic symptoms' (e.g. sleep problems) factors of the CHIPS. Chest pain, which was included in the allergies subscale of the SHC was also more logically included in the 'sympathetic/cardiac' factor. Furthermore, the allergies subscale includes specific conditions such as eczema and asthma which, although may develop over the lifetime, tend to be specific to sufferers of the conditions. The CHIPS however, measures only general symptoms, all of which could potentially be suffered by any given individual within a healthy population.

In terms of construct validity, from a theoretical perspective, due to the nature of subjective symptoms of physical health, it is not surprising that significant correlations were observed between the individual factors that were identified. The largest associations are

particularly noteworthy: between ‘vasovagal symptoms’ and both sympathetic/cardiac’ and ‘gastrointestinal symptoms’. Even though the latter two are logically and statistically distinct, they both overlap with some aspects of the ‘vasovagal symptoms’ factor. A few of the factors were also found to be weakly, but significantly, associated with measures of perceived stress and anxiety, adding further support to the burgeoning literature linking both of these elements of psychological well-being to physical health (e.g. Segerstrom & O’Connor, 2012). Furthermore, ‘metabolic symptoms’ were associated with levels of depression. It is well known that negative emotional states can lead to an increase in self-reported ill health. A plethora of research has linked poor health outcomes to higher indices of negative affect, and demonstrates that a physical decline in health can be accompanied by major depression (Penninx et al., 1999).

This study has also shown that the subjective distress from physical symptoms is only weakly associated with levels of pain sensitivity, suggesting that the number of health complaints people will report is not simply due to individual biological factors involved in tolerance to discomfort (Ruscheweyh et al., 2009). As levels of perceived stress and anxiety were found to be associated with some, but not all of the factors, it is evident that individual psychological and environmental concepts can play a role in the perception of one’s physical symptoms. However, only some of the physical symptom factors were associated with these variables, which support the suggestion that examination of distinct symptom clusters may be particularly beneficial for research investigating links between psychological factors and ill health. This extends the rationale for examining symptom clusters across the present research project, as Type D personality may affect different symptoms via distinct psychophysiological pathways.

Furthermore, it can be argued that the proposed factor structure of the CHIPS is also particularly useful when investigating the links between stress and health outcomes. For example, research has found stress to be related to higher indices of gastrointestinal complaints (Whitehead, 1994) and flu symptomology (Smolderen et al., 2007). Further, as psychological morbidity (i.e. negative affectivity and depression) has been linked to physical health

problems (e.g. Dua, 1994; Sullivan, LaCroix, Russo & Walker, 2001), the use of this approach to self-reported symptoms in the present project may aid in elucidating potential psychobiological mechanisms which may underpin the Type D-health pathways.

Statistical analyses may be conducted on the resulting CHIPS scores for each symptom group but must be treated cautiously, particularly in healthy populations. Analyses using the 'minor haemorrhagic' factor scores are not recommended when utilising the proposed factor structure to define symptom clusters, due to the poor internal consistency demonstrated in the current study. However, the definition of the factor and the items 'bruises 'and nosebleeds' have not been excluded from the measure, as they can provide valuable data regarding individual's experiences of these symptoms, particularly as both are related to risks associated with the use of commonly used medicines, such as Aspirin (Meade, Roderick, Brennan, Wilkes & Kelleher, 1992)

For many of the single items on the CHIPS, many people will not suffer or report any symptoms, and high numbers of zero scores may cause the resulting data to be highly positively skewed. However, in some research studies it could be useful to split groups based on total scores or individual symptom cluster scores (e.g. low, medium, and high levels of symptoms) and analyse the data categorically, or combine the scores with other measures of subjective physical symptoms.

It must be acknowledged that the sample of the general population utilised in this study comprised mainly females residing in the UK, and therefore cannot be entirely generalisable. The number of females outweighed males approximately four-fold, which although is not unusual in online studies (Smith, 2008), and was a similar ratio to that of the paper detailing the SHC subscales (Eriksen et al., 1999), may present a source of potential bias. However, Eriksen et al., (1999) chose a combined gender analysis to represent their final factor structure, as it most closely matched that of their female sample. Therefore, although females tend to report more symptoms than males in general (Kroenke & Spitzer, 1998) it is suggested that there is little reason to believe clustering of symptoms may necessarily differentiate between genders.

The self-administered nature of the CHIPS fundamentally brings with it the usual issues surrounding the use of self-report measures; however, although this may limit its use as a diagnostic tool, it may prove to be particularly valuable in research into individual differences in subjective health. The current study only postulates a potential factor structure, and it may therefore, be beneficial for future studies to undertake confirmatory factor analysis to confirm the factor structure hereby presented. It may also be beneficial to seek further evidence for construct validity in different populations such as cross cultural and clinical samples, and in larger samples. With a validated factor structure, researchers can consistently differentiate between symptom clusters, and provide insights into specific aspects of self-reported ill health, in the general population. Therefore, the clusters derived here will be utilised within the present project to investigate the influence of Type D personality on different symptom clusters.

3.5.1 Conclusions

The CHIPS is a simple, inexpensive and practical tool for the quick assessment of the experience of physical symptoms, with a particular focus on the amount of subjective distress associated with each symptom, in the general population. The measure gauges the most frequently reported physical symptoms (Nixon, Mazzola, Bauer, Krueger & Spector, 2011) all of which have the propensity to be experienced throughout the population of otherwise healthy individuals, which is a particular advantage over the similar SHC inventory. The 33 items can be categorised into eight relatively distinct symptom-types as follows; ‘sympathetic/cardiac’, ‘muscular pain’, ‘metabolic’, ‘gastrointestinal’, ‘vasovagal’, ‘cold/flu’ ‘headache’ and ‘minor haemorrhagic symptoms’. All factors include conceptually similar items, and with the exception of minor haemorrhagic symptoms, demonstrate adequate internal consistency, and the construct validity of each of the derived subscales has been ascertained. This instrument, and the proposed factor structure, will therefore be utilised as a valid assessment of physical symptoms throughout the present project.

Chapter 4: Initial cross-sectional examination of the relationships between Type D personality and physical symptoms within the general population

This Chapter discusses the findings of a large scale, cross-sectional, online-questionnaire based study which aimed elucidate the relationships between Type D personality and physical symptoms. In addition, a number of stress related, psychological and behavioural variables identified in the Type D literature were examined with respect to their relationship with Type D personality. Analyses were conducted utilising both the categorical and dimensional approaches. Categorical analyses indicated Type D individuals reported more physical symptoms than non-Type D individuals, and differences on the other psychological and behavioural measures were as expected. These were mainly supported by correlational analyses, however utilising the more stringent regression analyses, Type D was only significantly related to certain symptom clusters. Findings indicate Type D significantly predicted higher levels of cardiac/sympathetic, metabolic, muscular and headache symptoms. These symptoms can be attributed to stress-related causes and consequences, and the implications of these findings are discussed accordingly.

4.1 Background

An abundance of research has observed links between Type D personality and various aspects of health. Type D is a well-established risk factor in cardiac health (Denollet, 1998a; Kupper & Denollet, 2007; Pedersen & Denollet, 2003), and is also associated with poorer outcomes in other clinical (e.g. Bartels et al., 2010; Denollet et al., 2009; Mols & Denollet, 2010a) and non-clinical (Stevenson & Williams, 2014; Williams & Wingate, 2012) populations. Type D has also been associated with increases in anxiety and depression (Howard & Hughes, 2012; Michal et al., 2011; Polman et al., 2010), perceived stress and stress reactivity (Howard & Hughes, 2013; Kelly-Hughes et al., 2014), and decreases in social support (Mommersteeg, Herr, Bosch, Fischer & Loerbroks, 2011), use of maladaptive coping

strategies (Polman et al., 2010; Williams & Wingate, 2012), poor health behaviours (Gilmour & Williams, 2012; Horwood, Anglim & Tooley, 2016; Williams et al., 2008) and poorer sleep quality (Condén, Ekselius & Aslund, 2013).

Given the relevance of previous findings regarding links between Type D and negative health outcomes, it appears necessary to further investigate the significance of the Type D construct among otherwise healthy individuals, with a particular focus on self-reported symptoms, to clarify the extent to which possible declines in physical and psychological health may transpire in these individuals. In light of the accumulating evidence that the synergistic effect of negative affect and social inhibition can lead to an increased risk of adverse health outcomes, it also appears pertinent to further expand our knowledge of the psychological and behavioural factors involved. This is particularly important, in attempt to understand the development of the pathways underpinning the relationship between Type D, and the manifestation of physical symptoms in the general population.

However, previous studies are subject to different methods, samples, and analyses and do not cumulatively provide a clear picture of the factors related to Type D personality. Therefore, further investigation and clarification of nature of relationships between all of these factors and Type D personality in the general population is warranted.

4.1.1 Aims and objectives

The aim of the present study is to establish the presence of relationships between Type D personality and physical symptoms, as well as to identify potential psychological and behavioural factors which may also be related to Type D personality. These additional psychological and behavioural factors are being investigated as potential mechanisms underpinning the link between Type D personality and ill-health. The findings of the present study, will therefore inform the development of mediation models, which will be tested longitudinally in the following chapter. It is hypothesised that Type Ds will report increased physical symptoms and perceived stress, in addition to lower social support and pain sensitivity, poorer sleep and health behaviours, and higher use of coping strategies.

4.2 Method

4.2.1 Design

A cross-sectional online questionnaire-based design was employed, in which Type D personality was the independent predictor variable. The outcome variables were physical symptom scores, levels of perceived stress reactivity, perceived stress, use of coping strategies, social support, sleep problems, pain sensitivity, health behaviours, anxiety, and depression. Both categorical classification of Type D status and continuous measurement of the Type D construct were used in analyses (See chapter 2).

4.2.2 Participants

A total of 535 healthy participants took part in this study. The participant sample and demographic characteristics are fully described in Chapter 3. Using the traditional dichotomous approach to Type D classification, 244 (190 females) individuals were classified as Type D which equated to 45.6% of participants within the current study.

4.2.3 Materials and procedure

Participants were able to follow a link to the online survey on Qualtrics.com, where full study information was provided. Informed consent was required via a multiple choice selection, and an option to provide an email address to allow participation in a subsequent one year follow up study (see Chapter 5) was provided.

Type D personality was assessed using the Type D Scale-14 (DS-14; Denollet, 2005) and the Big-five Mini-markers (Saucier, 1994) was used to assess other aspects of personality. The Cohen & Hoberman Inventory of Physical Symptoms (CHIPS; Cohen & Hoberman, 1983) was used as the main outcome measure of self-reported health. Health behaviours were measured by a set of 10 questions adapted from the Diabetes Self-Care and Activities Scale (Toobert, Hampson & Glasgow, 2000). Subjective stress and stress reactivity were measured by the Perceived Stress Scale (PSS; (Cohen, Kamarck & Mermelstein, 1983) and Perceived Stress Reactivity Scale (PSRS; Schlotz, Yim, Zoccola, Jansen & Schulz, 2011) respectively.

Levels of psychological distress were examined using the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). The Brief COPE inventory (Carver, 1997) was used to gauge participants' use of functional coping strategies, and questions about quality of social network and social support (SNSS) taken from Dalgard, Bjork & Tambs, (1995) measured quality of social support.

The Pain sensitivity Questionnaire (PSQ; Ruscheweyh et al., 2009) and Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman & Kupfer, 1989) were also included as measures of pain sensitivity, and subjective sleep quality respectively (see Chapter 2 for a detailed description of each measure). Once all questionnaires were completed participants viewed an online debrief.

4.2.4 Treatment of data

Data was downloaded from Qualtrics.com, assessed for missing data, and scored accordingly (see Chapter 2). Participants were encouraged to answer all questions but could skip individual questions if required. From the total 535 participants, 508 participants responded to all questionnaires within the survey, and 27 participants completed between 80% and 100%. Partial data sets in which less than 80% of questionnaires were either viewed or attempted were removed and excluded from analyses, as the respondents were considered to have not appropriately engaged with the study. For the remaining participants, missing data was imputed according to the procedure outlined in Chapter 2.

Data from the DS14 (Denollet, 2005), Big 5 mini markers (Saucier, 1994), CHIPS (Cohen & Hoberman, 1983), Brief-COPE (Carver, 1997), HADS (Zigmond & Snaith, 1983) and PSRS (Schlotz et al., 2011) were included for all 535 participants. 534 participants completed the QSNSS (Dalgard et al., 1995), 524 completed the PSS (Cohen et al., 1983), and 521 fully answered the PSQ (Ruscheweyh et al., 2009). Full completion of the PSQI (Buysse et al., 1989) was achieved for 524 participants. Data were also checked for likely duplicate responses, and were removed accordingly.

4.2.5 Statistical analyses

Based on a number of categorical demographic characteristics (i.e. gender, employment status, household income, BMI), independent samples t-tests and one-way ANOVAs were conducted to assess differences in Type D scores between the groups. Pearson's correlations were also conducted to investigate the relationships between SI, NA and Type D scores, in addition to each of the big five personality factors. Primary analyses then comprised independent samples t-tests between Type D and non-Type D individuals on all health, psychological and behavioural continuous outcome variables. Pearson correlational analyses were also conducted to investigate the relationships between the continuous Type D personality (NA × SI) scores and the outcome variables. Finally, hierarchical regression analyses were performed, to ascertain the additional variance in the physical symptoms that Type D could predict over above the individual contributions of NA and SI (see chapter 2).

4.3 Results

4.3.1 Demographics

An independent samples t-test revealed that in the current study, the non-Type D participants (*M*= 31.57, *SD*= ±13.59) were significantly older than the Type D participants (*M* = 27.71, *SD* = ±11.75) (*t* (533) =3.524, *p*<.001, *d*=.030). Pearson's correlational analyses indicated that Type D score was negatively correlated with age (*r*=-.185, *p*<.001).

There was an unequal gender split in the current study, with females accounting for 80.56% of total participants. An independent samples t-test indicated that the mean Type D scores for male participants (*M*= 164.82, *SD*= ±142.67) and female participants (*M*= 158.08, *SD*= 144.27) were not significantly different (*t* (533) =0.428, *p*=.667, *d*=.004). Similarly, a one-way ANOVA also showed that Type D scores did not significantly differ between participants with a normal (BMI 18-25), overweight (BMI 26-30) and obese (BMI 31<) BMI (*F*, 2,521) = 2.771, *p* = .064, ηp^2=.011).

Retired/unemployed participants (*M*= 213.69, *SD*= ±187.52) and students (*M*= 175.13,

SD= ±144.32) reported higher average Type D scores than those participants 'in active paid work' (M =132.35, SD = 132.10). A one-way ANOVA revealed a significant difference between the Type D scores across the three groups (F (2,532) = 7.898, p<.001, ηp^2=.029). Tukey post hoc analyses indicated that the significant differences lay between the 'in work' participants and the students (p=.002), and the 'in work' participants and retired /unemployed participants (p=.011). Furthermore, an independent samples t-test revealed that participants with a lower household income had higher average Type D scores (M=166.39, SD= ±139.10) than those who reported a higher household income (M= 133.93, SD= ±131.82), and this difference was significant (t (464) = 2.581, p=.010, d=0.240).

4.3.2 Personality factors

Correlational analyses presented in table 4.1 indicate negative correlations between SI and Extraversion (-.789) and between NA and Emotional Stability (-.734). The Type D interaction was most highly negatively correlated with extraversion (-.642) followed by emotional stability (-.538).

Table 4.1. Correlation matrix showing relationships between Type D, SI, NA and big 5 factors.

Measures	SI	NA	Type D	*M*	*SD*
Extraversion	-.789**	-.347**	-.642**	44.56	11.94
Agreeableness	-.389**	-.316**	-.380**	55.80	9.93
Conscientiousness	-.146**	-.170**	-.187**	52.30	9.64
Emotional stability	-.339**	-.734**	-.538**	44.45	12.09
Openness	-.098*	.006	-.095*	50.24	9.34

** p>.01 * p>.05

4.3.3 Physical symptoms

Type D participants reported significantly more physical symptoms as measured by the CHIPS than non-Type D participants. This was demonstrated for all eight symptom factors with the exception of haemorrhagic symptoms. The results of the independent samples t-tests

are reported in table 4.2.

Table 4.2. Independent samples t-tests showing differences in physical symptoms between Type D groups (Mean [±SD]).

Measures	Non-Type D	Type D	*t*	*df*	*p*	*d*
Physical symptoms	14.64[14.54]	21.84 [16.27]	-5.407	533	<.001	.468
Cardiac/sympathetic	1.69 [3.83]	2.75 [4.02]	-3.126	533	.002	.271
Muscular	2.34 [3.25]	3.71 [4.27]	-4.217	533	<.001	.365
Metabolic	4.48 [4.09]	6.63 [4.51]	-5.763	533	<.001	.500
Gastrointestinal	1.64 [2.99]	2.36 [2.92]	-2.782	533	.006	.241
Vasovagal	1.22 [2.42]	1.98 [2.82]	- 3.349	533	.001	.290
Cold	1.78 [2.12]	2.16 [2.29]	-1.976	533	.049	.171
Headache	1.18 [1.55]	1.94 [2.14]	-4.774	533	<.001	.414
Haemorrhagic	0.35 [0.90]	0.39 [0.87]	-.0507	533	.624	.004

As observed in table 4.3 Pearson's correlational analyses indicated significant positive correlations between SI scores and total physical symptoms score, and each symptom cluster with the exception of gastrointestinal and haemorrhagic symptoms. Furthermore, NA scores and total Type D score (NA × SI) demonstrated significant positive correlations with all symptom factors, with the exception of haemorrhagic symptoms.

Table 4.3. Correlations between SI, NA, total Type D scores and physical symptoms.

Symptoms	SI	NA	Type D	*M*	*SD*
Physical symptoms	.186**	.448**	.369**	17.93	15.75
Cardiac/sympathetic	.117**	.290**	.235**	2.18	3.95
Muscular	.156**	.292**	.273**	2.96	3.81
Metabolic	.184**	.481**	.361**	5.46	4.41
Gastrointestinal	.065	.247**	.193**	1.97	2.98
Vasovagal	.150**	.325**	.294**	1.57	2.63
Cold	.095*	.220**	.147**	1.96	2.20
Headache	.165**	.318**	.311**	1.52	1.88
Haemorrhagic	.000	.082	.052	0.37	0.88
Mean	11.38	12.33	149.77	-	-
SD	6.33	6.327	126.72	-	-

*p<0.05; **p<0.01

Hierarchical regression analyses were performed on each physical symptom factor, with NA and SI entered in the first step, and the Type D (NA × SI) interaction score entered in the second step. Regression coefficients, R square and R Square Change for each model are shown in table 4.4.

Table 4.4. Hierarchical regression analyses predicting self-reported physical symptoms

Symptom factor		*β*	*SE b*	*β*	*t*	*p*	R^2 (ΔR^2)
Physical symptoms							
Step 1	SI	-.066	.111	-.026	-.597	.551	
	NA	1.154	.109	.458	10.623	<.001	.200**
Step 2	SI	-.631	.223	-.244	-.2832	.005	
	NA	.679	.195	.269	3.475	.001	
	Type D	.045	.015	.361	2.917	.004	.213 (.013**)
Sympathetic/Cardiac							
Step 1	SI	-.016	.030	-.025	-.542	.588	
	NA	.181	.029	.289	6.248	<.001	.078**
Step 2	SI	-.141	.059	-.220	-2.373	.018	
	NA	.075	.052	.121	1.447	.149	
	Type D	.010	.004	.323	2.422	.016	.088(.010*)
Muscular							
Step 1	SI	.016	.028	.025	.554	.580	
	NA	.172	.028	.288	6.255	<.001	.089**
Step 2	SI	-.094	.057	-.153	-1.661	.097	
	NA	.080	.050	.133	1.608	.109	
	Type D	.009	.004	.296	2.230	.026	.098 (.009*)
Metabolic							
Step 1	SI	-.038	.031	-.053	-1.243	.215	
	NA	.346	.030	.491	11.543	<.001	.222**
Step 2	SI	-.148	.062	-.205	-2.401	.017	
	NA	.254	.054	.359	4.681	<.001	
	Type D	.009	.004	.252	2.054	.041	.228 (.006*)
Gastrointestinal							
Step 1	SI	-.028	.023	-.057	-1.217	.224	
	NA	.133	.022	.277	5.959	<.001	.067**
Step 2	SI	-.104	.046	-.212	-.2.262	.024	
	NA	.069	.040	.144	1.708	.088	
	Type D	.006	.003	.256	1.908	.057	.073 (.006)
Vasovagal							

Step 1	SI	.002	.020	.004	.086	.932	
	NA	.139	.019	.328	7.206	<.001	.109
Step 2	SI	-.097	.040	-.223	-2.446	.015	
	NA	.056	.035	.132	1.616	.107	.122(.014**)
	Type D	.008	.003	.375	2.866	.004	
Cold/Flu							
Step 1	SI	-.006	.017	-.015	-.329	.742	
	NA	.078	.017	.221	4.696	<.001	.046**
Step 2	SI	.019	.034	.053	.561	.575	
	NA	.099	.030	.280	3.287	.001	
	SI × NA	-.002	.002	-.114	-.834	.405	.047(.001)
Headache							
Step 1	SI	.011	.014	.036	.797	.426	
	NA	.096	.014	.316	6.953	<.001	.111**
Step 2	SI	-.059	.028	-.192	-2.105	.036	
	NA	.036	.025	.120	1.463	.144	
	Type D	.006	.002	.377	2.882	.004	.125(.014**)
Haemorrhagic							
Step 1	SI	-.009	.007	-.060	-.1.249	.212	
	NA	.015	.007	.105	2.184	.029	.009
Step 2	SI	-.027	.014	-.186	-1.922	.055	
	NA	-.001	.012	-.004	-.043	.965	
	Type D	.001	.001	.208	1.500	.134	.014(.004)

*p<0.05; **p<0.01

NA was a significant predictor of total physical symptoms at Step 1, with the overall regression model predicting a significant 20% of the variance in physical symptoms; $F(2, 522) = 65.35$, $p < .001$, $\eta p^2 = .200$. The overall regression model was also significant at Step 2; $F(3, 522) = 47.03$, $p < .001$, $\eta p^2 = .212$ and the Type D interaction term significantly predicted an additional 1.3% variance in physical symptoms. A similar finding was observed for sympathetic/cardiac symptoms, with NA being the only significant predictor at Step 1, accounting for 7.8% of the variance, within a significant regression model; $F(2, 522) = 22.112$, $p < .001$, $\eta p^2 = .078$. The regression model was also significant at Step 2; $F(3, 521) = 16.834$, $p < .001$, $\eta p^2 = .082$, and the addition of Type D significantly predicted an additional 1.0%

variance in sympathetic/cardiac symptoms (p=.016).

NA was also a significant predictor of muscular symptoms at Step 1 within a significant regression model explaining 8.9% of the variance; $F(2, 522) = 25.63$, $p<.001$, $\eta p^2=.089$. The regression model was also significant at Step 2; $F(3, 521) = 18.88$, $p<.001$, $\eta p^2=.098$, and Type D significantly predicted an additional 0.9% of variance in muscular symptoms. Similarly, for metabolic symptoms, 22.2% of the variance was accounted for in step 1, with NA the only significant predictor in a significant regression model; $F(2, 522) = 74.38$, $p<.001$, $\eta p^2=.221$. At step 2 the regression model remained significant; $F(3, 521) = 51.30$, $p<.001$, $\eta p^2=.228$, and Type D significantly predicted metabolic symptoms, explaining an additional 0.6% of the variance.

Table 4 also shows that 6.7% of the variance of gastrointestinal symptoms was accounted for in step 1, with NA as the only significant predictor in the significant regression model; $F(2, 522) = 18.74$, $p<.001$, $\eta p^2=.067$. The model remained significant at Step 2; $F(3, 521) = 13.76$, $p< .001$ $\eta p^2=.073$, and Type D accounted for an additional 0.6% of the variance, however this only approached significance. It is also evident that 10.9% of the variance of vasovagal symptoms was explained in step 1, with NA as again the only significant predictor in the model; $F(2, 522) = 31.67$, $p< .001$, $\eta p^2=.108$. At step 2, the model remained significant; $F(3, 521) = 24.21$, $p< .001$, $\eta p^2=.122$, and Type D significantly predicted an additional 1.4% of the variance. Similarly, for headaches, 11.1% of the variance was accounted for in step 1, with NA the only significant predictor in a significant regression model; $F(2, 522) = 32.47$, $p< .001$, $\eta p^2=.111$. At step 2 the regression model remained significant; $F(3, 521) = 25.72$, $p< .001$, $\eta p^2=.123$, and Type D again significantly predicted headache symptoms, explaining an additional 1.4% of the variance.

NA was also a significant predictor of cold/flu symptoms at Step 1 within a significant regression model, $F(2,522) = 12.64$, $p< .001$, $\eta p^2=.046$, and explained 4.6% of the variance. The regression model was also significant at Step 2; $F(3, 521) = 8.65$, $p< .001$, $\eta p^2=.047$, however, Type D did not significantly predict any additional variance in cold/flu symptoms. Finally, for haemorrhagic symptoms, the regression model at step 1 was non-significant; $F(2,$

522) = 2.45, p=.087, ηp^2=.009, and did not reach significance with the addition of Type D at Step 2; $F(3, 521) = 2.39$, p=.068, ηp^2=.014.

4.3.4 Subjective stress-related outcomes

As observed in table 4.5., Type D participants scored significantly higher on measures of perceived stress reactivity (across all subscales of the PSRS), and levels of background stress, in comparison to non-Type D participants.

Table 4.5. Independent samples t-tests indicating significant differences between Type D and non-Type D individuals on perceived stress reactivity and background stress (Mean [±SD]).

Measures	Non Type D	Type D	*t*	*df*	*p*	*d*
Prolonged (PRr)	2.78 [1.29]	3.32 [1.33]	-4.784	533	<.001	.414
Work overload (RWO)	3.74 [2.22]	5.56 [2.09]	-9.706	533	<.001	.841
Social conflict (RSC)	5.33 [2.07]	6.64 [2.08]	-7.261	533	<.001	.629
Failure (RFa)	4.04 [1.32]	5.03 [1.67]	-7.638	533	<.001	.662
Social evaluation (RSE)	4.03 [2.30]	5.91 [2.31]	-9.373	533	<.001	.812
Total reactivity (PSRS)	19.93 [6.69]	26.45 [6.75]	-11.193	533	<.001	.970
Perceived Stress (PSS)	15.72 [6.41]	22.07 [6.36]	-11.331	522	<.001	.981

Table 4.6, shows that significant positive correlations were observed between all self-reported stress-related outcomes and Type D, SI and NA scores.

Table 4.6. Correlations between SI, NA, Type D and subjective stress-related outcomes.

Symptoms	SI	NA	Type D	*M*	*SD*
Prolonged (PRr)	.183**	.333**	.257**	3.02	1.33
Work overload (RWO)	.330**	.591**	.477**	4.57	2.34
Social conflict (RSC)	.248**	.508**	.388**	5.93	2.18
Failure (RFa)	.300**	.475**	.403**	4.49	1.57
Social evaluation (RSE)	.437**	.485**	.516**	4.88	2.49
Total reactivity (PSRS)	.417**	.654**	.571**	22.90	7.46
Perceived Stress (PSS)	.383**	.672**	.556**	18.55	7.06

*$p<0.05$; **$p<0.01$

Hierarchical multiple regression analyses were also completed for each of the stress-related outcomes. However, in all of the models the Type D interaction score did not significantly predict any additional variance in the outcomes over and above that of NA (the only significant predictor at step 1, when entered into the model at step 2.

4.3.5 Psychological and behavioural outcomes

Table 4.7 shows that Type D individuals scored significantly higher on measures of coping strategy use, sleep problems, anxiety, depression, optimism and pain sensitivity (both moderate and minor), in comparison to non-Type Ds. Type Ds also scored significantly lower than non-Type Ds on measures of social support, alcohol consumption and good dietary behaviours. No significant differences were observed between the groups on measures of smoking or physical activity.

Table 4.7. Independent samples t-tests showing differences between Type D and non-Type D individuals on the psychological and behavioural outcome measures (Mean [±SD]).

Measures	Non-Type D	Type D	*t*	*df*	*p*	*d*
Social Support	28.19 [3.27]	24.77 [3.79]	11.195	533	<.001	.970
Maladaptive coping	24.33 [6.53]	28.03 [6.77]	-6.412	533	<.001	.555
Alcohol consumption	6.57 [5.15]	5.60 [4.72]	2.241	533	.025	.194
Smoking	2.79 [5.76]	3.01 [5.77]	-0.438	533	.662	.037
Good diet behaviours	6.09 [3.81]	5.43 [3.56]	2.028	533	.043	.176
Physical activity	5.87 [3.51]	5.53 [3.56]	1.148	533	.251	.099
Sleep problems	5.13 [3.05]	6.39 [3.39]	-4.507	533	<.001	.390
Anxiety	7.16 [3.54]	10.30 [3.74]	-9.930	532	<.001	.861
Depression	2.92 [2.71]	5.57 [3.38]	-10.050	530	<.001	.873
Pain sensitivity	44.82 [19.10]	48.81 [20.89]	-2.280	522	.023	.199

As observed in table 4.8, Pearson's correlational analyses indicated significant negative correlations between quality of social support and SI, NA and Type D scores. Use of maladaptive coping strategies was not significantly associated with SI, but demonstrated significant positive correlations with NA and total Type D scores. With respect to health behaviours, significant negative correlations were observed between SI, and alcohol, smoking and diet; between NA and diet; and between Type D, and alcohol and diet. Sleep problems and pain sensitivity both demonstrated significant positive correlations with SI, NA and Type D, and anxiety and depression scores were all positively correlated with NA, SI and Type D.

Table 4.8. Correlations between SI, NA, Type D and psychological and behavioural factors

Measures	SI	NA	Type D	*M*	*SD*
Social Support	-520**	-458**	-.545**	26.63	3.91
Maladaptive coping	.026	.244**	.119**	57.06	13.23
Alcohol consumption	-.147**	.003	-.100*	5.73	4.16
Smoking	-.096*	.054	-.018	2.62	5.34
Good diet behaviours	-.094*	-.094*	-.093*	5.79	3.71
Physical activity	-.003	-.035	-.033	5.71	3.42
Sleep problems	.139**	.360**	.246**	5.70	3.27
Anxiety	.305**	.683**	.492**	8.59	3.95
Depression	.385**	.547**	.502**	4.13	3.31
Total pain	.104*	.144**	.118**	46.61	20.00

*p<0.05; **p<0.01

Hierarchical multiple regression analyses were also completed for each of the psychological outcome variables. However, in all of the models the Type D interaction score did not significantly predict any of the variables when entered into the model at step 2.

4.4 Discussion

4.4.1 Summary

The primary aim of the current chapter was to investigate the relationship between Type D personality and self-reported physical symptoms, in addition to a number of psychological and behavioural factors, in a large sample of healthy adults. Analyses were conducted utilising both the categorical and dimensional approach to the construct, as recommended by Ferguson et al. (2009) with the aim of elucidating the outcomes which may be a consequence of the synergistic combination of negative affect and social inhibition, and therefore play a role in the pathways by which Type D can lead to poor health outcomes.

4.4.2 Type D prevalence

The prevalence of Type D personality in the current study was 45.6%, meaning that almost half of the participants exhibited the combination of high negative affect and high social inhibition. This percentage is higher than those generally found in other studies undertaken within the general population. For example, a study of Dutch adults reported a prevalence as low as 18.1%, (van den Broek et al., 2010), although the majority of studies report around 25% (Mols & Denollet, 2010b). However, the percentage in this study is comparable to a number of recent studies undertaken in the UK/Ireland and in which 38.5% (Williams et al., 2008) and 42.8% (Booth & Williams, 2015), and in Australia where 39.7% (Horwood, Chamravi & Tooley, 2015) of participants were characterised as Type D.

4.4.3 Demographics

There appeared to be a weak negative relationship between Type D score and age, and individuals classified as Type D were found to be significantly younger within the current study. Research is relatively mixed with respect to the relationship between personality and age, with some suggesting that personality is relatively stable over the lifespan (Harris, Brett, Johnson & Deary, 2016), whereas others state that some traits differ between younger and older individuals. It has however, been demonstrated that emotional stability may increase with age (Carstensen et al., 2011), and although sociability appears to conversely decrease (Warr, Miles & Platts, 2001), this could partially account for the age differences observed between the Type D groups within this study. Although Type D personality has been found to be stable over both an 18-month (Martens & Kupper, 2007), and a nine-year period (Kupper & Boomsma, 2011), this may suggest that in future smaller studies, an age-related criterion may need to be implemented. However, it could be implied that further work should examine the temporal stability of the construct over the lifespan.

Type D scores did not appear to differ on the basis of participants' BMI or gender. However, participants with higher household income and those in active paid work were found to have lower Type D scores than those with a lower household income; and students,

unemployed and retired individuals, respectively. This is also not unsurprising given higher levels of negative affect, stress and psychological distress have been demonstrated in both lower income (Orpana, Lemyre & Gravel, 2009) and student (Andrews & Wilding, 2004) populations. However, this brings into question the representativeness of the current study's sample. Sample representativeness is a common issue in Type D studies (Hausteiner et al., 2010) and will be addressed further in Chapter 9.

4.4.4 Big five personality factors

Although negative affectivity and social inhibition are described as global personality traits (Denollet, 1998a), they are not entirely distinct. The two facets exhibit a degree of overlap with each other as well as the 'big five'; conscientiousness, openness, agreeableness, neuroticism (emotional stability) and extraversion (Svansdottir et al., 2013). As one would expect, correlational analyses demonstrated the presence of strong relationships between SI and extraversion, and NA with emotional stability, supporting the assumption that Type D individuals exhibit a relatively stable, trait-like level of distress (Denollet, 2005). This may suggest that the domains of negative affectivity and social inhibition could be assessed by measures of emotional stability and extraversion to a certain degree. However, although the correlations were strong (above .70), there was not perfect concordance between these variables, suggesting that the DS14 traits are not entirely synonymous with their respective big five counterparts. Furthermore, both SI and NA showed moderate relationships with the other traits, with the exception of openness to experience, exemplifying the large degree of overlap between personality traits.

Other personality factors have also been associated with poorer health outcomes, for example lower levels of conscientiousness (Bogg & Roberts, 2013; Korotkov & Hannah, 2004). Additionally, as neuroticism refers generally to a lack of positive psychological adjustment, individuals high in neuroticism have also been found to score higher on measures of psychological distress and have a propensity for rating subjective health as poor (Costa & McCrae, 1980). It must therefore be acknowledged that the influence of other traits may also

play a role in the links between personality and health hereby presented.

4.4.5 Self-reported physical symptoms

Categorical analyses indicated that participants characterised as Type D reported more physical symptoms across all symptom clusters with the exception of haemorrhagic symptoms, than non-Type Ds. Correlational analyses utilising the dimensional Type D construct reflected these findings by demonstrating positive (albeit weak to moderate) relationships between the Type D scores and level of symptoms reported.

These findings support previous research demonstrating associations between Type D personality and increases in ill health and physical symptoms within the general population (e.g. Stevenson & Williams, 2014; Williams & Wingate, 2012), as well as poorer subjective health in patient populations (e.g. Chapman, Duberstein & Lyness, 2007). This also indicates that the conceptualisation of Type D as a dimensional construct demonstrates a similar predictive value to the dichotomous Type D typology. However, in the regression models, the interactive Type D construct accounted for additional variance, above that of NA and SI, for some but not all self-reported symptom clusters.

The predictive value of Type D was found to significantly contribute to the total physical symptom score in this study over and above that of its constituent components. Conversely, Stevenson and Williams, (2014) recently found the synergistic effect of Type D personality did not significantly predict physical symptoms when analysed in this way, although with a somewhat smaller sample. In the current study the interactive Type D construct also predicted additional variance for the sympathetic/cardiac, muscular, metabolic, vasovagal and headache symptoms, but not for the gastrointestinal, cold/flu and haemorrhagic symptoms.

The significant influence of Type D on the sympathetic/cardiac symptoms, corresponds with the abundance of literature linking Type D personality to poor cardiac health and cardiovascular outcomes (Borkoles et al., 2012; Denollet, 1998a; Howard & Hughes, 2013; Kelly-Hughes et al., 2014; Kupper & Denollet, 2007; Nyklíček, Vorselaars & Denollet, 2011;

Pedersen & Denollet, 2003; Sher, 2005). As this finding is apparent here within a sample of seemingly healthy individuals, it may indicate that the influence of Type D in cardiac-related health begins to manifest substantially prior to disease onset, also supporting evidence of Type D personality as an independent cardiovascular risk marker (Hausteiner et al., 2010). Recent research has also indicated cardiac related impairments in Type D individuals in non-clinical populations including increased prevalence of ventricular arrhythmias (Einvik & Dammen, 2014).

The vasovagal symptoms ('dizziness', 'faintness', 'feeling weak all over' and 'acne') are seemingly demonstrable of issues with vitality and exhaustion, which have been associated with both acute (Kudielka et al., 2006) and chronic (Bellingrath, Weigl & Kudielka, 2009) stress. Similarly, increased vital exhaustion (a psychological state characterised by low energy and fatigue) has been demonstrated in Type D patients with ischemic heart disease (Pedersen & Middel, 2001). Symptoms included on both the sympathetic/cardiac (e.g. 'heart pounding' and 'blurred vision') and vasovagal (e.g. 'dizziness' and 'faintness') symptom clusters are theorised to be underpinned by autonomic dysfunction and governed by the sympathetic nervous system (SNS) (Shinoura & Yamada, 2005) which is heavily implicated in the stress response (Hall, Cruser, Podawiltz, Mummert & Jones 2012). Headaches were also found to be predicted by the Type D interaction in the current study, symptoms of are also often attributed to heightened stress levels (Björling, 2009).

Further adding to this theory; the items on the metabolic symptom factor, may also be similar to the negative health effects associated with chronic stress. For example; weight change, poor appetite and sleep problems are often associated with prolonged experiences of stress (Kim & Dimsdale, 2007; Serlachius, Hamer & Wardle, 2007) and, along with feeling low in energy and constant fatigue, are common symptoms of burnout (Lundgren-Nilsson, Jonsdottir, Pallant & Ahlborg, 2012). The finding that Type D is associated with these symptoms therefore corresponds to the findings of a number of studies that have demonstrated links between Type D personality and symptoms of burnout and related mental health issues (Geuens, Braspenning, Van Bogaert & Franck, 2015; Ogińska-Bulik, 2006; Polman et al.,

2010). Similarly, the significant influence of the Type D interaction term on the muscular pain symptoms, supports the findings of Condén, Leppert, et al., (2013) who found that in a large community sample of Swedish adolescents, Type D individuals suffered increased musculoskeletal symptoms. However, as these studies employed the categorical approach to Type D analyses, the current study provides novel evidence in this area using the dimensional approach.

Despite gastrointestinal problems such as IBS being prevalent in Type D populations (e.g. Sararoudi et al., 2011), this study found that the Type D interaction did not significantly influence the reporting of gastrointestinal symptoms. However, as its contribution was approaching significance, further research may be warranted. Type D was also not significantly predictive of cold and flu symptoms over and above the contribution of NA, which may suggest that NA in isolation may be responsible for the differences between the Type D groups observed. NA has previously demonstrated predictive value in the development of cold and flu like illnesses (Cohen, Tyrrell & Smith, 1993), however it is uncertain whether this may be due to NA-induced cognitive biases involving over sensitivity to symptoms or misinterpretations of benign sensations (Cohen, Doyle & Skoner, 1995). Nevertheless, it appears that SI does not account for any additional variance in the increased reporting of these symptoms, reinforcing findings of Smolderen, Vingerhoets, Croon, and Denollet (2007). Their study found that NA and perceived stress were associated with increased self-report of flu-like illnesses, whereas socially inhibited individuals tended to report less flu-like illnesses than less socially inhibited individuals.

As previously stated, it may be suggested that the influence of Type D personality on physical health complaints in the general population may be primarily due to NA. It has also been proposed that sensitive individuals high on traits such as neuroticism may be biased towards increased reporting of symptoms (Brown & Moskowitz, 1997). However, as Type D personality was not related to haemorrhagic symptom reporting, this may not be the case. It could be suggested that the symptoms; nosebleeds and bruises, are more likely to be a consequence of trauma, rather than an underlying biological cause representing poor health.

However, if the Type D-health relationship was mainly due to individuals over reporting or exhibiting exaggerated sensitivity, it may be expected that this would be reflected in the levels of haemorrhagic symptoms reported.

This study provides cross-sectional evidence of relationships between Type D and physical symptoms, however there is a need for longitudinal research in order to provide a clearer understanding of causality. This will therefore be explored in Chapter 5. If the relationships between Type D and physical symptoms are confirmed by a longitudinal study, it could be suggested that screening for Type D personality in a medical diagnostic context could be beneficial in the prediction and treatment of illnesses, particularly cardiac and somatic symptoms, in later life.

4.4.6 Stress-related outcomes

Type D individuals reported significantly higher levels of background stress, as well as perceived stress reactivity, relative to non-Type D participants. This indicates that Type D individuals experience more stress, and perceive themselves to be more reactive in various situations. These findings were also confirmed by the correlational analyses, which demonstrated strong positive relationships between Type D personality and both background stress (PSS scores) and perceived stress reactivity (PSRS scores). An abundance of previous categorical Type D studies on perceived stress have demonstrated similar findings (Habra et al., 2003; Kelly-Hughes et al., 2014; Ogińska-Bulik, 2006; Polman et al., 2010). However, the regression analyses indicated the NA× SI interaction term did not significantly increase the variance explained by NA and SI independently. This corresponds to the findings of Kelly-Hughes et al., (2014) regarding levels of background stress, and also extends our knowledge by indicating that Type D is similarly associated with perceptions of stress reactivity.

Despite the Type D interactive term not adding any significant predictive value to measures of background stress or perceived stress reactivity, as Type D was related to the symptom clusters identified earlier, it seems plausible that these factors could be implicated in the Type D-health relationship. As discussed, stress appears to be a common underlying

determinant of the symptoms that appear to be associated specifically with the Type D construct when analysed utilising the robust dimensional approach.

Biological research has supported the notion that Type D is associated with maladaptive stress responses, for example exaggerated cardiovascular responses to acute stress (Habra et al., 2003; Williams et al., 2009), which have in turn been implicated in a plethora of negative health outcomes (Carroll et al., 2012; Lepore, Miles & Levy, Jodi, 1997; Treiber et al., 2003). As the cardiovascular stress response is governed by the sympathetic-adrenal-medullary system (Buske-Kirschbaum, Geiben, Höllig, Morschhäuser & Hellhammer, 2002), it may be suggested that this system and indices of sympathetic arousal may be implicated as mechanisms underpinning the link between Type D and cardiac/sympathetic, metabolic and vasovagal symptoms identified.

In the literature, the relationship between Type D and the acute stress response is not entirely clear. Whereas some previous studies have found Type D to be related to exaggerated cardiovascular and neuroendocrine stress reactivity (Habra et al., 2003; Williams et al., 2009), Type D has also been linked to blunted reactivity (Kelly-Hughes et al., 2014; O'Leary et al., 2013). Blunted cardiovascular and neuroendocrine stress reactivity is a well-documented consequence of chronic stress, often exemplified in caregivers (Ruiz-Robledillo, Bellosta-Batalla & Moya-Albiol, 2015), and has been found to lead to reduced immunity (Kiecolt-Glaser et al., 1987) and other negative health consequences (Phillips, Ginty & Hughes, 2013). Consequentially, further research is required to elucidate the psychophysiological pathways by which Type D influences cardiovascular stress reactivity, and will be investigated, in conjunction with other objective measures of sympathetic activation in Chapter 6.

4.4.7 Psychological and behavioural outcomes

Type D individuals reported lower levels of social support, higher use of maladaptive coping strategies and reduced sleep quality relative to non-Type D participants, which supports previous findings (Condén, Ekselius, et al., 2013; Mols & Denollet, 2010b, Nefs, Pouwer, Pop & Denollet, 2012; Williams et al., 2008). This was confirmed by the correlation

analyses indicating significant relationships between Type D and these outcomes.

On the health behaviour measures Type D and non-Type D participants did not significantly differ on levels of physical activity or smoking frequency, contrary to the findings of a number of previous studies (e.g. Borkoles et al., 2010; Svansdottir et al., 2012). Surprisingly, Type D individuals demonstrated lower alcohol consumption than non-Type D individuals, contradicting studies by Bruce, Curren & Williams, (2013) and Michal et al., (2011), who found Type D personality to be associated with increased alcohol use. However, as this sample mainly comprised younger students, and excluded those with any significant health problems, including alcohol dependence, this finding maybe a consequence of social inhibition, as alcohol is regarded as a social drug.

However, in accordance with other findings regarding health behaviour (e.g. Mommersteeg et al., 2010), Type D individuals also demonstrated lower scores on a measure of 'good' dietary habits (i.e. avoidance of fatty foods and higher fruit and vegetable consumption) in the current study. These initial categorical findings therefore provide only partial support for the suggestion that performance of poor health behaviours may be involved in the relationship between Type D personality and ill health (Gilmour & Williams, 2012; Williams et al., 2008). However, a non-standardised instrument was implemented to measure health behaviours. The questions were developed using the scale from an established diabetes questionnaire (Toobert et al., 2000), as it was simple and easily scored. Government recommended guidelines on dietary habits, exercise and alcohol intake informed the content of the questions, and they were straightforward to understand and answer in a similar way to the original diabetes management related questions. However, it is important to note that these questions were developed in house and have not been validated, so the conclusions drawn from these analyses should be considered with this in mind.

Given the nature of the Type D construct it was not surprising that Type D participants exhibited higher levels of anxiety, depression and pain sensitivity than non-Type D individuals. These findings support an abundance of previous research which has demonstrated increased physiological distress in Type D cardiac patients (e.g. Denollet &

Jonge, 2009; Mommersteeg et al., 2012; Pedersen & Denollet, 2003), other clinical populations (Bartels et al., 2009; Mols & Denollet, 2010a; Mols et al., 2012; Nefs et al., 2012; Pedersen & Ong, 2006) and healthy individuals (Michal et al., 2010, 2011; Mols & Denollet, 2010b). Increased levels of psychological distress, particularly in socially inhibited individuals, may underpin the early findings which suggested poor prognosis in Type D cardiac patients (Grande et al., 2012).

Regression analyses revealed that the Type D interaction did not significantly predict any of the psychological and behavioural factors over and above NA and SI. This may support the burgeoning theory that the Type D construct may simply represent another measure of psychological distress (Ferguson et al., 2009) and highlights the importance of controlling for the unique variance explained by NA and SI (Dulfer et al., 2015).

4.4.8 Strengths and limitations

A major strength of the current study was the reasonably large sample of participants from the general population. These participants were not limited to undergraduate students or the UK/Ireland population so the results may be regarded as more generalisable in comparison to similar recent studies (e.g. Polman et al., 2010; Stevenson & Williams, 2014). Secondly, and most importantly, the use of a dimensional conceptualisation of Type D in addition to the traditional taxonomic approach has made the findings more statistically sound. The traditional approach, in which individuals were essentially grouped into different categories based on arbitrary cut-off scores, is not recommended in personality research (Widiger & Trull, 2007) and is a specific criticism of the Type D taxonomy (Coyne & de Voogd, 2012; Coyne et al., 2011). The use of multiple regression analyses assessing Type D as a continuous measure allows for the statistical control of the independent effects of NA and SI, which is particularly important given the well documented links between negative emotions and physical health.

Certain limitations must also be acknowledged, including the usual issues that arise with the reliance on subjective measures, such as the risk of self-report biases and social desirability. On a related note, as this study is essentially comparing individuals differing in

levels of distress it must also be considered that distress can influence perceived health status (Farmer & Ferraro, 1997; Tessler & Mechanic, 1978), and therefore any self-reported health impairments may be exaggerated in this population. However, self-report methodology maintains substantial merit and is regarded a highly reliable technique for assessing perceptions of general health in population studies (Miilunpalo, Vuori, Oja, Pasanen & Urponen, 1997). Nevertheless, to overcome some of these issues and strengthen the robustness of the findings, the use of objective measures, particularly health and stress related measures, should be explored. For example; recording of cardiovascular function, physical activity monitoring and assessment of biomarkers (e.g. of inflammation and sympathetic activation) may be implemented. A number of these measures have been employed in a subsequent study within the present project (see Chapter 6).

Due to the cross-sectional design of the present study, like many other studies investigating Type D and subjective health, a cause and effect relationship cannot be reliably inferred. The Type D personality taxonomic construct has been found to be relatively stable over time (Kupper & Boomsma, 2011; Martens & Kupper, 2007); however, its stability in patients before and after cardiac surgery has been found to be changeable (Dannemann et al., 2010). Therefore, it may be possible that the characteristics exhibited by Type D individuals may actually develop from illness or symptom experience. A prospective study of the links between Type D and the symptoms hereby examined, is discussed in Chapter 5 in attempt to clarify causality.

The current study utilised an online questionnaire based approach which enables access to a large number of potential participants who may otherwise be difficult to recruit. Online questionnaire research is relatively easy to administer, reduces burden on participants, and increases respondents' perceptions of anonymity (Branley et al., 2014). Nevertheless, it must be considered that use of online research limits the sample to only an internet based population, and therefore excludes those who do not regularly use the internet, reducing the generalisability. The ability to complete the study remotely (i.e. not under the supervision of the researcher) also opens up the possibility of technical issues interfering with data collection,

uncertainty regarding participants' intentions and easier study withdrawal (i.e. not completing the full survey). In the current study some partial data sets were lost due to respondents discontinuing part way through, occurrence of which may have been reduced if the researcher had been present. Further, there is also the potential risk of 'ballot stuffing' (i.e. taking part multiple times to deliberately manipulate overall results) in remote online research, however in the present study demographic data were checked and any likely duplicate responses were removed.

Additionally, there was a much larger proportion of females than males in the current sample, which is common in online survey studies (Smith, 2008). However, there is no strong evidence to suggest gender differences in the prevalence of Type D personality (Hausteiner et al., 2010); therefore, the effects of this disparity may be reasonably limited. Further, despite the survey being open to participants worldwide, it is not entirely representative of the worldwide population as the majority of respondents were UK residents. The implications of these issues with generalisability are further discussed in Chapter 9.

4.4.9 Conclusions

In summary, Type D individuals report significantly higher severity of physical symptoms than non-Type D individuals. Specifically, when analysed using the dimensional approach, there is evidence of associations between Type D personality and particular symptom clusters, independent of the separate effects of negative affectivity and social inhibition, within a large sample of otherwise healthy adults. Although categorical Type D personality was also found to relate to increased background stress and perceived stress reactivity, poorer coping strategies, sleep problems, lower social support and increased psychological distress, dimensional analyses suggest that these outcomes were not predicted by the interaction of NA and SI when the contribution of both components was controlled.

This study extends our knowledge of the relationship between Type D personality and physical health, and is the first to investigate particular clusters of ill-health symptoms within a Type D context. The findings demonstrate that Type D personality is associated with some,

but not all symptom clusters. In terms of future directions, there is a clear requirement for longitudinal research to confirm the relationships identified in the present study, which will be the objective of the study reported in Chapter 5. Upon clarification of these relationships, the psychophysiological mechanisms purported to underpin the link between Type D personality and physical health, particularly cardiovascular reactivity and sympathetic arousal, investigated and the findings discussed in Chapter 6.

Chapter 5: A longitudinal investigation into the relationship between Type D personality and 'stress-related' physical symptom clusters: A one-year follow up study

---

This Chapter discusses the findings of a follow up to the initial, cross-sectional, online-questionnaire based study documented in Chapter 4. This longitudinal component aims to further elucidate the relationships between Type D personality and physical symptoms. The potential mediating influences of stressful life events, depression, anxiety and perceived stress were also investigated. Analyses were again conducted utlising both the categorical and dimensional conceptualisations of Type D personality and bootstrapped mediation analyses were undertaken. Findings indicated longitudinal relationships between Type D and metabolic, gastrointestinal and cold/flu symptoms, and stressful life events and anxiety were identified as potential mediating factors. Furthermore, Type D individuals were more likely to report worse health status, higher frequency of illnesses and work absences, and seek medical information.

5.1 Background

Previous research has established that Type D personality is a predictor of physical health status, including the reporting of more somatic symptoms, general health complaints and immune related illnesses (Condén, Leppert, et al., 2013; Stevenson & Williams, 2014; Williams & Wingate, 2012). Type D personality has also been associated with increased psychological distress and aberrant physiological responses to stress (e.g. Habra, Linden, Anderson & Weinberg, 2003; Howard, Hughes & James, 2011; Howard & Hughes, 2013; Kelly-Hughes, Wetherell & Smith, 2014; Schiffer, Pedersen, Broers, Widdershoven & Denollet, 2008). As discussed in Chapter 4, findings from a cross-sectional online survey of healthy adults in the general population demonstrated significant relationships between Type D personality and increased reports of physical symptoms, as well as heightened levels of perceived stress and psychological distress. Most notably the dimensional Type D construct

remained a significant predictor of specific symptom clusters (cardiac/sympathetic, metabolic, vasovagal, headaches) which are considered stress-related, when controlling for the separate effects of NA and SI.

In the Type D literature, there is a notable lack of longitudinal research into the health effects of Type D personality in the general population. The cross-sectional nature of the majority of studies in this area makes it difficult to reliably infer cause and effect. Cross-sectional designs also limit the capacity to investigate potential 'mediating' mechanisms which may underpin the observed relationships (Maxwell & Cole, 2007). Therefore, a longitudinal assessment of the associations between Type D personality and both health and stress related outcomes is warranted, in order to ascertain the mechanisms via which Type D may lead to deleterious health consequences.

Therefore, in light of the findings that Type D is significantly related to some specific physical health outcomes it is timely to investigate whether Type D personality can predict changes in physical symptoms and perceptions of health one year later. It was envisaged that this study would overcome previous limitations in cross sectional studies, by enabling longitudinal mediation analyses to allow a better understanding of the mechanisms underpinning the relationships between Type D personality and physical health.

5.1.1 Aims and objectives

The primary aim of this chapter is to establish whether Type D personality is a predictor of physical symptoms and negative health outcomes in the general population. More specifically, a longitudinal design was employed to enable an investigation of whether Type D personality leads to a deterioration of subjective psychological and physical health over a one-year period. A further aim of this study was to examine the contribution of perceived stress, psychological distress and experience of stressful life events as mediating factors of the relationship between Type D personality and physical symptoms. Finally, the longitudinal nature of the study enabled an investigation of the temporal stability of the Type D personality construct.

5.2 Method

5.2.1 Design

A longitudinal online questionnaire-based design was employed with Type D personality at time 1 as the main predictor variable, and physical symptoms, perceived stress and psychological distress at time 2 as outcome variables. A number of retrospective health questions were also measured at time 2 as additional outcome variables. Type D personality was again assessed as both a categorical and a dimensional construct depending on the research question being addressed.

5.2.2 Participants

All participants who participated in Study 1 (healthy adults aged 18-65years from the general population) and provided contact details indicating that they were willing to take part in a follow-up, were contacted. The date at which the participants completed the original study was recorded and they were contacted via email one year after this date (+/- 7 days). The email included the participant number and the link to the survey itself. From the initial pool of 535 participants, 293 provided contact details indicating a willingness to take part in this follow-up study and 160 responded to the invitation to take part in the follow-up. Participants were allowed up to 35 days to complete the follow-up. Mean response time following the email invitation to take part in the follow-up was 5.45 days.

No course credit or remuneration was offered to participants for taking part in the follow up. Exclusion criteria stipulated that individuals with a history of mental health issues, known chronic or immune related illnesses and diagnosed sleep disorders should refrain from taking part (including those diagnosed since the completion of the first survey). The sample was again predominantly female (127 females, 33 males). Using the traditional dichotomous approach for classifying Type D personality using the cut off scores of >10 on both scales of the DS14. 72 participants (51 females) were classified as Type D and 88 (76 females) were classified as non-Type D.

5.2.3 Materials and procedure

Participants who chose to follow the link in the invitation email were directed to the online survey on Qualtrics and were required to input the participant number they had been allocated. Study information and a multiple choice consent option were embedded in the online survey for the participants to read and complete. Following this, participants completed a number of questions to ascertain their date of birth, height, weight, occupation and gender. Type D personality was then assessed using the Type D Scale-14 (DS-14 Denollet, 2005) and The Cohen Hoberman Inventory of Physical Symptoms (CHIPS; Cohen & Hoberman, 1983) measured total health complaints and clusters of physical symptoms. The Perceived Stress Scale (PSS; Cohen, Kamarck & Mermelstein, 1983), the Hospital Anxiety and Depression Scale (HADS; (Zigmond & Snaith, 1983) and the Stressful life events checklist (Holmes & Rahe, 1967) were also completed. (See Chapter 2 for a detailed description of the measures employed). A set of in-house multiple choice retrospective health questions were also included (see appendix for full list of questions). Once all questionnaires were completed participants viewed an online debrief.

5.2.4 Treatment of data

Data was downloaded from Qualtrics.com and assessed for missing data, and scored accordingly (See Chapter 2). A total of 158 participants completed all questionnaires within the survey. One participant did not complete the PSS and HADS and two participants did not complete the life events checklist and prospective health questions. A further three participants began the survey but did not provide enough data to be included in analyses as they were considered to have not appropriately engaged with the study.

5.2.4.1 Statistical analyses

Differences between respondents and non-respondents from the initial online cross-sectional study (see Chapter 4) were examined utilising a number of paired samples t-tests. This assessed for any differences between baseline and follow-up for age, BMI, Type D, NA, and SI scores, physical symptoms, perceived stress, anxiety, depression and stressful life

events.

Chi squared analyses were used to analyse the association between respondent status (i.e. whether individuals were respondents or non-respondents) and the categorical demographic variables: gender, employment status, household income and country of residence (with adjusted standardized residuals where necessary).

Primary analyses comprised independent samples t-tests to examine differences between Type D and non-Type D individuals on levels of physical symptoms, anxiety, depression and perceived stress at follow up in addition to stressful life events experienced over the past year. A number of 2 × 2 repeated measures ANOVAs were also conducted to assess the differences in these variables from baseline to follow up. Type D category was the between subjects factor, and the within subjects factor was time (baseline or follow up). Physical symptoms, anxiety, depression and perceived stress were again the dependent variables. Pearson's correlations between the continuous Type D construct at baseline and both the physical symptoms and psychological variables at follow up were also undertaken.

For the retrospective health questions Pearson chi-squared analyses were used to assess the association between Type D category and participants' responses to each aspect of retrospective health. Adjusted standardised residuals were conducted to identify which responses exhibited differences. The residual scores indicate significance at $p<.05$* if they lie outside ±1.96, significance at $p<.01$** if they lie outside ±2.58 and significance at $p<.001$*** if they lie outside ±3.29.

Finally, mediation analyses using the PROCESS macro for SPSS (Hayes, 2013) were used to investigate whether the perceived stress, anxiety, depression or stressful life events mediated the relationship between Type D, and the physical symptom clusters. Each mediator and outcome variable was entered into a separate model for analysis, and in line with recommendations for longitudinal research (Fitzmaurice, Laird & Ware, 2012), baseline measures of the appropriate symptom cluster were entered as covariates into each model. This analysis was performed using the recommended 5,000 bootstrap resamples.

In order to conclude whether the potential mediators fully mediated the relationship

between Type D and physical symptoms, the following conditions were required: i) the bootstrapped confidence intervals relating to the indirect effect must not overlap with 0, and ii) the direct effect when the mediator was included (path *c*') needed to become non-significant. In the case that only the first condition was met, then it could be concluded that partial mediation had occurred. The direct effect shows the direct relationship between Type D personality and CHIPS scores via path *c'* when each mediator is included in the model. The indirect effect shows the indirect relationship between Type D personality and CHIPS scores via each mediator (i.e. path *a*b*). The bootstrap mediation analyses performed are represented diagrammatically in Figure 5.1

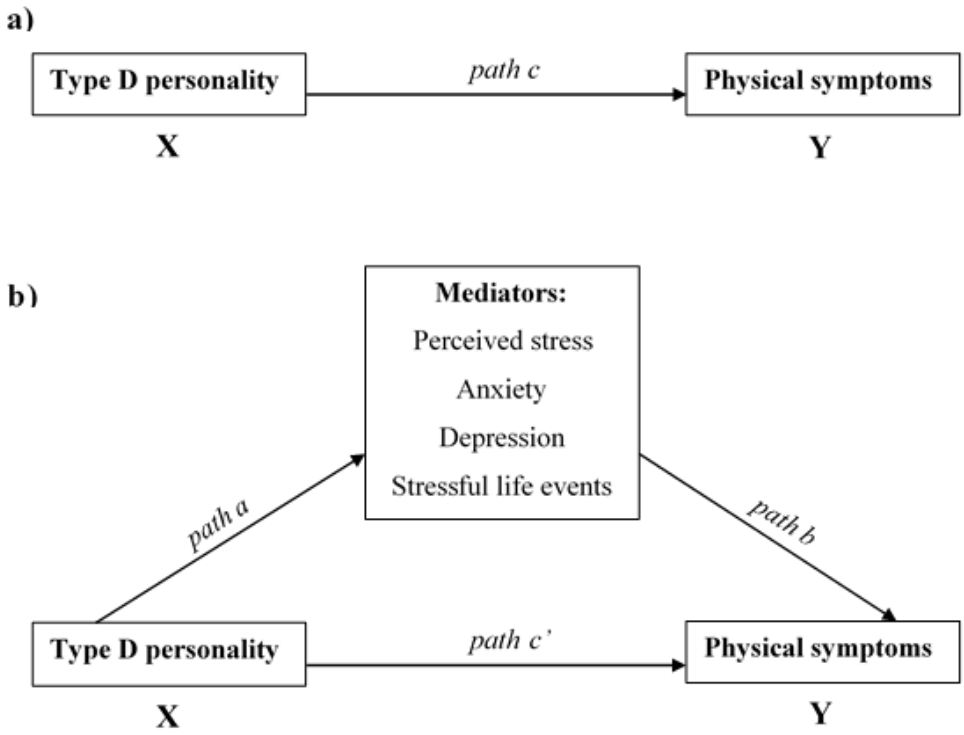

Figure 5.1. Non-mediated (a) and mediated (b) pathways between Type D personality and physical symptoms (each symptom cluster was considered in a separate statistical model). Path c' represents the direct effect of Type D personality on physical symptoms with the mediator included in the model. The indirect effect is the product of path a and path b. Each mediator was considered in a separate statistical model. All models controlled for the baseline symptom score in model of Y only.

5.3 Results

5.3.1 Respondents Versus Non respondents

Independent samples t–tests revealed that respondents (M_{age} = 34.05, *SD*= ±13.71) were significantly older than non -respondents (M_{age}=28.00, *SD*= ±12.13), t (533) = -5.073, p<.001, and respondents (M= 17.61 *SD*= ±7.32) scored significantly lower than non-respondents (*M*=19.01, *SD*= ±6.99) on the Perceived Stress Scale, *t* (522) =2.077, *p*= .038. No significant differences were evident between respondents and non-respondents on any other continuous measures.

Chi squared analyses indicated no significant association i) between gender and respondent status X^2 (1) =.865, *p*= .352, or ii) between Type D and respondent status X^2 (1) =.034, *p*= .854.

A significant relationship was however observed between respondent status and household income; X^2 (2) = 13.580, *p*<.001. Adjusted standardised residuals indicated that non-respondents 'undeclared' their income (z = 3.0**) significantly more than expected and respondents did so less than expected (z = -3.0**). Also, respondents stated their income as 'above £20,000' (z =3.1**) more than expected and non-respondents did so less than expected (z = -3.1**).

A significant association was observed between respondent status and employment status; X^2 (2) = 35.056, p<.001. Adjusted standardised residuals indicated that non-respondents were in 'active paid work' (*z*= 5.8***) significantly more than expected, and respondents were in 'active paid work' less than expected (*z*= -5.8***). Additionally, respondents were 'students' (*z*= 4.7***) more than expected and non-respondents were less than expected (*z*= -4.7**).

A significant association was observed between respondent status and residency; X^2 (2) =15.242, *p*=.002. Adjusted standardised residuals indicated that non-respondents indicated their residency as 'UK' (*z*= 2.5*) significantly more than expected, and respondents were from the 'UK' less than expected (*z*= -2.5*). Conversely, respondents indicated their residency as

'Europe' (z= 3.7***) more than expected and non-respondents did so less than expected (z= -3.7***). The number and relative percentage of respondents in each demographic category are shown in table 5.1.

Table 5.1. The number of respondents and non-respondents to the follow up study (% of N) in each demographic category.

		Respondents	Non- respondents	Total
Gender	Female	125 (23.4%)	306 (57.2%)	431(80.6%)
	Male	35 (6.5%)	69 (12.9%)	104 (19.4%)
	Total	160 (29.9%)	375 (70.1%)	535 (100%)
Income	>£20K	58 (10.8%)	155 (29.0%)	213 (39.8%)
	£20K+	92 (17.2%)	161 (30.1%)	253 (47.3%)
	Undeclared	10 (1.9%)	59(11.%)	69 (12.9%)
	Total	150 (29.9%)	375 (70.1%)	535 (100%)
Employment	Student	91 (17.0%)	132 (24.7%)	223 (41.7%)
	Employed	54 (10.1%)	229 (42.8%)	283 (52.9%)
	Unemployed/retired	15 (2.8%)	14 (2.6%)	29 (5.4%)
	Total	160 (29.9%)	375 (70.1%)	535 (100%)
Residency	UK	136 (25.4%)	345 (64.5%)	481 (89.9%)
	Europe	10 (1.9%)	3 (0.6%)	13 (2.4%)
	Other	14 (2.6%)	26 (4.9%)	40 (7.5%)
	Total	160 (29.9%)	375 (70.1%)	534 (100%)
Type D	Type D	72 (13.5%)	172 (32.1%)	244 (45.6%)
	Non Type D	88 (16.4%)	203 (37.9%)	291 (54.4%)
	Total	160 (29.9%)	375 (70.1%)	535 (100%)

5.3.2 Temporal stability of Type D

Paired samples t-tests indicated no significant differences between scores at baseline with

scores at follow up on the measures of negative affect; *t*=-0.333 (159), *p*=.739, *d*=.053, social inhibition; *t*=1.032 (159), *p*=.304, *d*=.175, or Type D (SI × NA); *t*= 0.916 (159), *p*=.361, *d*=.145. Pearson's correlations analyses indicated strong correlations between baseline and follow-up scores; (SI; *r*= .857**, NA; *r*=.837**, Type D; *r*=.828**) indicating the DS14 exhibited excellent test-retest reliability over the year period.

5.3.3 Physical symptoms

As shown in table 5.2, independent samples t-tests indicated that Type D individuals reported significantly higher levels of physical symptoms (including all symptom clusters) at follow up, in comparison to non-Type D individuals.

Table 5.2. Independent samples t-tests between Type D and non-Type D individuals on physical symptoms at follow up (Mean [±SD])

Measures	Non-Type D	Type D	*t*	*df*	*p*	*d*
Cardiac/Sympathetic	1.52 [2.98]	3.49 [4.83]	-3.153	158	.002	.502
Muscular	2.49 [3.19]	4.08 [4.42]	-2.646	158	.009	.421
Metabolic	4.27 [3.59]	7.13 [4.38]	-4.527	158	<.001	.720
Gastrointestinal	1.24 [2.08]	2.74 [3.29]	-3.505	158	.001	.558
Vasovagal	1.85 [1.99]	2.79 [2.39]	-2.712	158	.007	.432
Cold/flu	1.07 [1.62]	1.86 [2.00]	-2.766	158	.006	.440
Headache	1.03 [1.31]	1.53 [1.49]	-2.229	158	.027	.354
Haemorrhagic	0.22 [0.81]	0.56 [1.06]	-2.298	158	.023	.367
Physical symptoms	13.13 [12.59]	23.74 [18.19]	-4.348	158	<.001	.692

These were supported by correlational analyses which demonstrated significant relationships between all physical symptoms at follow up and the continuous Type D scores and NA scores at baseline. Baseline SI scores were significantly associated with most physical symptoms at follow up, with the exception of muscular and vasovagal symptoms. Correlation coefficients and mean (SD) physical symptoms for all participants are presented in table 5.3.

Table 5.3.Correlations between SI, NA, total Type D scores and physical symptoms (Mean [±SD]).

Symptoms	SI	NA	Type D	*M*	*SD*
Cardiac/sympathetic	.235**	.411**	.403**	2.41	4.03
Muscular	.140	.299**	.292**	3.21	3.86
Metabolic	.253**	.577**	.493**	5.56	4.20
Gastrointestinal	.268**	.408**	.447**	1.91	2.78
Vasovagal	.127	.384**	.279**	2.28	2.22
Cold	.171*	.219**	.260**	1.43	1.84
Headache	.177*	.219**	.260**	1.26	1.41
Haemorrhagic	.170*	.199*	.275**	0.37	0.94
Physical symptoms	.283**	.513**	.502**	17.90	16.20
Mean	11.69	12.38	157.84	-	-
SD	6.91	6.81	149.24	-	-

*p<0.05; **p<0.01

Repeated measures ANOVAs demonstrated that Vasovagal symptoms significantly increased from baseline (M= 1.53) to follow up (M= 2.32); Wilks lambda; F(1,158) =19.739, p<.001 ηp^2=.111. Cold/flu symptoms significantly decreased from baseline (M= 1.93) to follow up (M=1.47); Wilks lambda; F(1,158) = 6.682, p=.011, ηp^2=.040. No significant interaction effects between Type D and time were evident for any of the symptom clusters.

Hierarchical multiple regression analyses were also conducted. In the final model (step 2), NA (at baseline) was the only significant predictor for the CHIPS (β=.411, p =.002), and cardiac (β=.336, p=.020), metabolic (β=.526, p<.001), vasovagal (β=.527, <.0010), and headache (β=.706, p=.039) symptoms at follow up. For the remaining symptoms (muscular, gastro-intestinal, cold/flu and haemorrhagic), SI, NA and Type D did not significantly contribute to the final model.

5.3.4 Psychological distress and stress outcomes.

As observed in table 5.4 independent samples t-tests indicated that Type D individuals reported significantly higher levels of anxiety, depression, and perceived stress at follow up in addition to increased stressful life events experienced in the past year, in comparison to non-Type Ds.

Table 5.4. Independent samples t-tests between Type D and non-Type D individuals on measures of anxiety, depression, perceived stress and stressful life events at follow up (Mean [±SD]).

Measures	Non-Type D	Type D	*t*	*df*	*p*	*d*
Anxiety	6.00[4.27]	9.53[4.76]	-4.922	157	<.001	.786
Depression	2.61[2.83]	5.60[3.84]	-5.637	157	<.001	.899
Perceived stress	13.61[6.93]	20.77[7.00]	-6.467	157	<.001	1.03
Stressful life events	168.79[120.72]	221.99[147.17]	-2.595	155	.010	.414

These findings were supported by correlational analyses which demonstrated significant relationships between anxiety, depression and perceived stress at follow up with the continuous Type D, SI and NA scores at baseline. Stressful life events were significantly correlated with NA and Type D scores but not SI scores. Correlation coefficients and sample means are shown in table 5.5.

Table 5.5. Correlations between SI, NA, total Type D scores and anxiety, depression, perceived stress and stressful life events (Mean [±SD]).

Symptoms	SI	NA	Type D	*M*	*SD*
Anxiety	.306**	.664**	.546**	7.60	4.82
Depression	.405**	.496**	.509**	3.96	3.64
Perceived stress	.400**	.626**	.611**	16.86	7.81
Stressful life events	.021	.271**	.180*	192.85	130.1

*p<0.05; **p<0.01

Hierarchical multiple regression analyses indicated that in the final model (step 2), NA

was the only significant predictor for anxiety (β=.620, <.001), and perceived stress (β=.457, p<.001). However, for depression and stressful life events, SI, NA and Type D did not significantly contribute to the model.

5.3.5 Mediation analyses

Table 5.6 demonstrates that all symptom clusters at baseline significantly predicted the respective symptom clusters at follow up. However, Type D personality was only a significant predictor of metabolic, cold/flu and gastrointestinal symptoms (path c) one year later when baseline scores were controlled. See figure 5.1 for diagrammatical representation of the mediation models.

Table 5.6. The relationship between each symptom cluster at follow up with i) Type D at baseline (path c) and ii) the respective symptom cluster score at baseline (covariate).

	Total effect (path *c*)				Relationship with covariate (baseline)			
Factor	*B*	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*
All Symptoms	.0138	.0079	1.744	.083	.7052	.0896	7.872	<.001
Cardiac	.0047	.0038	1.230	.221	.5699	.1642	3.471	<.001
Metabolic	.0047	.0019	2.430	.016*	.6176	.0684	9.033	<.001
Muscular pain	.0014	.0017	0.810	.419	.5448	.0825	6.600	<.001
Gastro	.0058	.0017	3.465	<.001*	.4783	.1448	3.303	.001
Vasovagal	.0008	.0013	0.596	.552	.4668	.1403	3.327	.001
Cold/Flu	.0025	.0009	2.690	.008*	.2803	.0763	3.673	<.001
Headaches	.0006	.0009	0.704	.483	.4312	.0892	4.834	<.001
Haemorrhagic	.0010	.0007	1.381	.169	.5568	.2375	2.345	.020

As evident in table 5.7 Type D personality at baseline significantly predicted anxiety, depression, perceived stress, and stressful life events one year later (path a).

Table 5.7. The relationship between i) Type D personality and each mediating variable (path *a*).

	Type D personality (path *a)*			
Mediator	*β*	*SE*	*t*	*p*
Anxiety	.0154	.0020	7.6971	<.001
Depression	.0134	.0016	8.446	<.001
Perceived Stress	.0305	.0047	6.440	<.001
Stressful life events	.1568	.0755	2.0756	.040

Anxiety was found to be significantly related to total physical symptoms, cardiac/sympathetic, vasovagal, metabolic, and muscular pain symptoms, whereas depression was only significantly related to cold/flu symptoms. Perceived stress was related to cardiac/sympathetic and vasovagal symptoms, and stressful life events were significantly related to all symptoms with the exception of cold/flu and haemorrhagic symptoms.

Table 5.8 shows mediation analyses for the relationships between baseline Type D personality and i) metabolic, ii) gastrointestinal, and iii) cold/flu symptoms at follow up, via the mediating variables.

There was a significant indirect effect (a*b) of Type D on metabolic symptoms (whereby the bootstrapped confidence interval for the indirect effect did not include 0) via both anxiety (BaC CI [.0017,.0070]) and stressful life events (BaC CI [.0001, .0020]). The direct effect (path *c'*) between Type D and the metabolic symptoms became non-significant when considered through the anxiety pathway (*p*=.130), but remained significant through the stressful life events pathway (*p*=.015). This indicates that anxiety fully mediated; and stressful life events partially mediated, the relationship between baseline Type D and metabolic symptoms at follow up. Depression and perceived stress were not significantly related to metabolic symptoms (path *b*), and the indirect effects (a*b) failed to reach significance, therefore mediation via these pathways was not observed (see table 5.8).

There was a significant indirect effect (a*b) of baseline Type D on gastrointestinal symptoms at follow up, via stressful life events (BaC CI [.0001,.0014]). The direct effect (path

c') between Type D and gastrointestinal symptoms remained significant (p=.006), therefore stressful life events partially mediated the relationship. Depression, anxiety and perceived stress were not significantly related to gastrointestinal symptoms (path *b*), and the indirect effects (a*b) also failed to reach significance.

Depression was significantly (*p*=.043) related to cold and flu symptoms (path *b*), however the indirect effect (a*b) between Type D and cold/flu symptoms one year later, via the depression pathway failed to reach significance and the direct effect (path *c'*) remained significant (*p*=.002) when depression was included in the model. Anxiety, perceived stress and stressful life events were not significantly related to cold/flu symptoms (path *b*), and the indirect effects (a*b) also failed to reach significance. Therefore, mediation of the relationship between baseline Type D and cold/flu symptoms at follow up via these pathways was not observed.

These results can be observed in table 5.8 and table 5.9.

Table 5.8. Mediation between Type D and metabolic, gastrointestinal and cold/flu symptoms by PSS, anxiety, depression and stressful life events.

		Relationship between mediator and symptom (path *b*)			Direct effect (*c'*path)					Indirect effect (a*b) [1]			
Symptoms	Mediator	*β*	*SE*	*p*	*β*	*SE*	*p*	*LLCI*	*ULCI*	*β*	*SE*	*LLCI*	*ULCI*
Metabolic	Anxiety	.2662	.0837	.002	.0028	.0018	.130[a]	-.0008	.0064	.0041	.0013	.0017	.0070
	Depression	-.0503	.1120	.654	.0051	.0023	.027	.0006	.0097	-.0007	.0015	-.0037	.0023
	PSS	.0428	.0466	.361	.0037	.0022	.091	-.0006	.0080	.0013	.0015	-.0011	.0048
	SLE	.0041	.0017	.017	.0046	.0019	.015[b]	.0009	.0082	.0006	.0004	.0001	.0020
Gastrointestinal	Anxiety	.0604	.0720	.403	.0050	.0020	.013	.0011	.0090	.0009	.0010	-.0012	.0028
	Depression	.0666	.0796	.404	.0049	.0021	.019	.0008	.0090	.0009	.0011	-.0012	.0031
	PSS	-.0170	.0437	.698	.0063	.0025	.012	.0014	.0112	-.0005	.0011	-.0025	.0015
	SLE	.0029	.0013	.034	.4068	.1454	.006[b]	.0021	.0087	.0004	.0003	.0000	.0014
Cold/flu	Anxiety	.0455	.0436	.299	.0018	.0012	.128	-.0005	.0042	.0007	.0006	-.0004	.0021
	Depression	-.0903	.0442	.043	.0037	.0011	.002	.0014	.0059	-.0012	.0006	-.0025	-.0001
	PSS	.0454	.0267	.090	.0011	.0013	.396	-.0014	.0036	.0014	.0008	.0000	.0032
	SLE	.0016	.0011	.155	.0021	.0009	.0195	.0003	.0039	.0003	.0002	.0000	.0010

[1]The indirect (mediation) effect is significant if the bootstrapped confidence intervals do not include 0. [a]Full mediation. [b]Partial mediation.

Table 5.9. Relationships between other symptom clusters and the mediating variables (path *b*)

Symptoms	Mediator	Relationship between mediator and symptom (path *b*)		
		β	*SE*	*p*
Physical symptoms	Anxiety	.7451	.3109	.018
	Depression	-.0328	.4020	.935
	PSS	.1917	.1378	.166
	SLE	.0198	.0074	.008
Cardiac/Sympathetic	Anxiety	.2605	.0866	.003
	Depression	.0590	.1217	.628
	PSS	.0908	.0431	.037
	SLE	.0051	.0022	.024
Vasovagal	Anxiety	.1558	.0544	.005
	Depression	.0919	.0600	.128
	PSS	.0722	.0325	.028
	SLE	.0047	.0011	<.001
Muscular	Anxiety	.1589	.0797	.048
	Depression	.1369	.0898	.130
	PSS	.0348	.0425	.415
	SLE	.0047	.0018	.011
Headache	Anxiety	.0599	.0362	.101
	Depression	.0373	.0440	.398
	PSS	.0275	.0167	.102
	SLE	.0020	.0009	.029
Haemorrhagic	Anxiety	.0160	.0149	.238
	Depression	-.0160	.0172	.356
	PSS	-.0127	.0147	.386
	SLE	.0004	.0005	.452

5.3.6 Retrospective Health Questions

5.3.6.1 Perceived general health

Type D category was significantly related to i) perceptions of current general health (X^2 (4) =13.765, p=.004.), ii) perceptions of health over the past 12 months (X^2 (4) =11.637, p=.012) and iii) satisfaction with current health status (X^2 (4) =9.321, p=.046). Type Ds were more likely to rate their current general health as 'fair', their health over the past year as 'not very good', and be dissatisfied' with their current health status. Conversely, Type Ds were also less likely to provide ratings of 'very good' for these questions. However, there were no differences in how participants compared their health status to one year earlier (X^2 (4) =7.299, p=.099).

5.3.6.2 Frequency of illnesses

Type D category also showed significant interactions with the frequency of i) feeling unwell or run-down (X^2 (4) =9.716, p=.035), ii) suffering a non-serious illness (not requiring a prescription) (X^2 (4) =12.015, p=.013) and iii) illness-related work absences (X^2 (4) =17.838, p<.001). Type Ds were more likely to have felt unwell or run-down 'frequently' and were less likely to have 'never' suffered a non-serious illness or taken time off work. However, Type D category did not relate to the frequency of participants suffering an illness requiring prescription medication; X^2 (4) =2.749, p=.654.

5.3.6.3 Healthcare utilisation

Type D category was significantly associated with the seeking of medical information (without visiting a medical professional) (X^2 (4) =15.444, p=.003). Type Ds were more likely to have 'never', or 'once or twice' sought medical information. Type D was however related to neither frequency of GP (X^2 (4) =2.781, p=.643) or hospital (X^2 (3) =3.213, p=.377) visits over the past year.

The number (and percentage of total) of responses to each retrospective health question for Type D and non-Type Ds can be observed in table 5.10.

Table 5.10. The number (and percentage) of responses to each retrospective health question

		Non-Type D	Type D	Total
Perception	Very good	28 (17.7%)	9 (5.7%)	37 (23.4%)
of current	Good	42 (26.6%)	34(21.5%)	76 (48.1%)
health	Fair	14 (8.89%)	24 (15.2%)	38 (24.1%)
	Bad	2 (1.3%)	4(2.5%)	6 (3.8%)
	Very Bad	0 (0.0%)	1 (0.6%)	1 (0.6%)
Perception	Very good	32 (20.3%)	13 (8.2%)	45 (28.5%)
of health	Good	37 (23.4%)	33 (20.9%)	70 (44.3%)
over past	Fair	14 (8.9%)	19 (12.0%)	33 (20.9%)
year	Not very good	2 (1.3%)	7 (4.4%)	9 (5.7%)
	Not good at all	1 (0.6%)	0 (0.0%)	1 (0.6%)
Change in	Much better	10 (6.4%)	2 (1.3%)	12 (7.6%)
health over	Somewhat better	14 (8.9%)	20 (12.7%)	34 (21.7%)
past year	About the same	44 (28.0%)	36 (22.9%)	80 (51.0%)
	Somewhat worse	16 (10.2%)	14 (8.9%)	30 (19.1%)
	Much worse	1 (0.6%)	0 (0.0%)	1.0 (0.6%)
Satisfaction	Very Satisfied	20 (12.7%)	13 (8.2%)	33 (20.9%)
with their	Satisfied	44 (27.8%)	24 (15.2%)	68 (43.0%)
current	Neither	8 (5.1%)	11 (7.0%)	19 (12.0%)
health status	Dissatisfied	13 (8.2%)	22 (13.9%)	35 (22.2%)
	Very Dissatisfied	1 (0.6%)	2 (1.3%)	3 (1.9%)
Frequency	All the time	3 (1.9%)	3 (1.9%)	6 (3.8%)
of feeling	Frequently	14 (8.9%)	22 (13.9%)	36 (22.8%)
unwell or	Sometimes	35 (22.2%)	33 (20.9%)	68 (43.0%)
run-down	Rarely	32 (20.3%)	14 (8.9%)	46 (29.1%)
	Never	2 (1.3%)	0 (0.0%)	2 (1.3%)

Frequency of suffering a non-serious illness	Seven or more times	1 (0.6%)	3 (1.9%)	4 (2.5%)
	Five or six times	8 (5.1%)	12 (7.6%)	20 (12.7%)
	Three or four times	21 (5.1%)	22 (13.9%)	43 (27.2%)
	Once or twice	43 (27.2%)	34 (21.5%)	77 (48.7%)
	Never	13 (8.2%)	1 (0.6%)	14 (8.9%)
Frequency of illnesses requiring prescription medication	Seven or more times	1 (0.6%)	0 (0.0%)	1 (0.6%)
	Five or six times	1 (0.6%)	3 (1.9%)	4 (2.5%)
	Three or four times	3 (1.9%)	3 (1.9%)	6 (3.8%)
	Once or twice	30 (19%)	28 (17.7%)	58 (36.7%)
	Never	51 (32.3%)	38 (24.1%)	89 (56.3%)
Frequency of work absences	Very frequently	0 (0.0%)	3 (1.9%)	3 (1.9%)
	Quite regularly	0 (0.0%)	1 (0.6%)	1 (0.6%)
	A few times	7 (4.5%)	12 (7.6%)	19 (12.1%)
	Once or twice	35 (22.3%)	27 (17.2%)	62 (39.5%)
	Never	42 (26.8%)	21 (13.4%)	63 (40.1%)
Frequency of GP visits	Very frequently	1 (0.6%)	1 (0.6%)	2 (1.3%)
	Quite regularly	2 (1.3%)	5 (3.2%)	7 (4.4%)
	A few times	20 (12.7%)	15 (9.5%)	35 (22.2%)
	Once or twice	42 (26.6%)	30 (19.0%)	72 (22.2%)
	Never	21 (13.3%)	21 (13.3%)	42 (26.6%)
Seeking medical information	Very frequently	2 (1.3%)	4 (2.5%)	6 (3.8%)
	Quite regularly	8 (5.1%)	9 (5.7%)	17 (10.8%)
	A few times	17 (10.8%)	17 (10.8%)	34 (21.5%)
	Once or twice	19 (12.0%)	29 (18.4%)	48 (30.4%)
	Never	40 (25.3%)	13 (8.2%)	53 (33.5%)

Frequency of hospital visits	Multiple, as inpatient	0 (0.0%)	0 (0.0%)	0 (0.0%)
	Multiple, day-patient	1 (0.6%)	4 (2.5%)	5 (3.2%)
	Once, as inpatient	5 (3.2%)	2 (1.3%)	7 (4.4%)
	Once, as day-patient	11 (7.0%)	9 (5.7%)	20 (12.7%)
	Never	69 (43.7%)	57 (36.1%)	126 (79.7%)

5.4 Discussion

5.4.1 Summary

The primary aim of the current chapter was to explore the longitudinal relationship between Type D personality and physical symptoms over a one-year period. The temporal stability of the Type D scale was also assessed, as well as the potential mediating influence of perceived stress, psychological distress and stressful life events in the Type D-health relationship. Retrospective health status and health care utilisation in relation to Type D was also examined. This study intended to extend the findings of Chapter 4 in attempt to further elucidate the potential mechanisms underpinning the relationship between Type D personality and physical symptoms. Key findings were that Type D personality was longitudinally related to metabolic, gastrointestinal and cold/flu symptoms. Stressful life events and anxiety were found to play mediating roles in some but not all of these pathways. Furthermore, Type D individuals reported generally worse health status, higher frequency of illnesses, and work absences and were less likely to never seek medical information, however this was not reflected in the likelihood of GP or hospital visits.

5.4.2 Type D assessment

Firstly, it could be concluded that the DS-14 has good test-retest reliability over the course of a year, supporting Denollet (2005)'s development of the scale for the purpose of defining Type D personality, and Martens and Kupper (2007) who stated Type D was a stable taxonomy over an 18 month period. However, further research is warranted into the potentially dynamic nature of Type D personality in the longer term, particularly as some studies indicate

that there is little stability of personality from teenage years to older adulthood (e.g. Harris et al., 2016; Mischel, 1969; Putnam, 2011). Nevertheless, it can be concluded that i) the DS-14 is reliable to use for the assessment of Type D in short term longitudinal studies such as this, and ii) Type D personality exhibited adequate stability within the current study.

The analyses conducted to assess any differences between respondents and non-respondents from the original cross-sectional study indicated a number of demographic differences between the two groups. Respondents were older than non-respondents, which has also previously been found in studies examining factors underpinning non-response bias (Jacobsen & Thelle, 1988; Sheikh & Mattingly, 1981), potentially suggesting an age-related difference in motivation.

Respondents also reported lower levels of perceived stress which may suggest that willingness to participate may be influenced by stress levels. A number of differences were also observed on the categorical analyses, suggesting that individuals were more likely to respond than not respond to follow-up if they: i) had higher household income, ii) resided in Europe, and iii) were students. These findings are envisaged to have little impact on the results of the study, however must be considered in the interpretation of the findings.

5.4.3 Self-reported physical symptoms

Type D individuals reported significantly greater severity of physical symptoms on all symptoms clusters in comparison to the non-Type D participants. This was supported by the dimensional analyses which demonstrated significant relationships between Type D scores and the symptom clusters at follow up. These findings also support those demonstrated in Chapter 4. However, it can also be observed that the correlation coefficients indicating the strength of the relationships between Type D and the symptom clusters, with the exception of vasovagal and headache symptoms were larger in the current study.

In terms of the Type D components, NA scores independently correlated with all symptoms, as did SI scores with the exception of muscular and vasovagal symptoms. These findings support previous research that demonstrate NA has continuously been linked to

poorer health in addition to increases in stress and psychological distress (Cohen et al., 1995, 1993; Watson & Pennebaker, 1989). Although it is argued that NA plays the more prominent role in the Type-D health relationship (e.g. Grande, Glaesmer & Roth, 2010), there is sufficient evidence that SI also independently contributes to poor health. For example, there is strong evidence linking increased social support to improved health outcomes (Cohen & Hoberman, 1983; Dalgard et al., 1995; Uchino, 2006), and epidemiological findings suggest poor social support may be a predictor of cardiovascular disease, infectious diseases and inflammatory related illnesses (e.g. Kaplan et al., 1994; Uchino, 2006). Furthermore, other aspects of social inhibition such as social phobia are also associated with negative health outcomes (Weiller, Bisserbe, Boyer, Lepine & Lecrubier, 1996).

This may support the premise that it is specifically the combination of SI and NA that is key rather than the influence of the traits in isolation. However, contrary to the cross-sectional findings in Chapter 4, when the individual components of the Type D construct were controlled for in regression analyses, the Type D interaction score did not significantly predict any of the symptom measures at follow-up. Nevertheless, this could be a consequence of the smaller sample size (Tabachnick & Fidell, 2007).

Inexplicably, in all participants', vasovagal symptoms were found to significantly increase, and cold/flu symptoms decrease from baseline to follow up. However, it appeared that Type D category did not have an influence on differences in physical symptoms reported between baseline and follow up. However, this may not be surprising given that this study was conducted within an apparently healthy population over a relatively short period of time. A longer term follow up may provide a clearer picture of the effect of Type D personality on the development of physical symptoms in the general population. Further, as a large proportion of the original research conducted in Type D concerns clinical outcomes in older patients (Nefs et al., 2015) it is suggested that Type D is largely predictive of later life health, illness experiences and clinical outcomes (e.g. Aquarius, Denollet, Hamming & De Vries, 2005).

The mediation analyses revealed that Type D scores at baseline significantly predicted metabolic, gastrointestinal and cold/flu symptoms at follow up, when baseline measures were

controlled for. This suggests that the extent to which an individual exhibits Type D traits could contribute to the development of these types of symptoms over time. However, in analyses conducted on the data set from the original cross-sectional study (See Chapter 4), of these symptom clusters, only metabolic symptoms were found to be significantly predicted by the Type D interaction when the separate influences of NA and SI were controlled. As discussed in Chapter 4, symptoms included in the metabolic symptom cluster including weight change, poor appetite, sleep problems, and constant fatigue are often associated with increases in stress, and burnout (Kim & Dimsdale, 2007; Lundgren-Nilsson et al., 2012; Serlachius et al., 2007). This finding therefore further supports the suggestion that maladaptive psychobiological stress reactivity may be a mechanism by which Type D personality can influence health, and will therefore be further explored in Chapter 7.

A longitudinal relationship was also observed between Type D and gastrointestinal symptoms, which partially supports previous research. For example, Type D personality has been related to the increased severity of symptoms and impaired quality of life in patients with functional gastrointestinal disorders (Hansel et al., 2010), and irritable bowel syndrome; a common chronic functional gastrointestinal disorder (Sararoudi et al., 2011). Further, it is theorised that psychosocial factors such as emotional distress and stress reactivity play a key role in modulating illness experience and clinical outcomes in irritable bowel syndrome patients (Drossman, Camilleri, Mayer & Whitehead, 2002). Therefore, given Type D individuals' propensity to distress, it could be implied that symptoms associated with the illness may be common and develop rapidly within this group. Furthermore, it has been suggested that symptoms of irritable bowel syndrome may be underpinned by a dysfunction of the autonomic nervous system (ANS) (Mazur et al., 2007), which is also implicated in anxiety related conditions, depressive disorders (e.g. Gorman & Sloan, 2000; Laederach-Hofmann, Mussgay, Büchel, Widler & Rüddel, 2002) and abnormalities of the stress response (see Chapter 6). This further strengthens the proposition that investigation of ANS function in Type D individuals may be warranted. Therefore, this will be an aim of the study reported in Chapter 7. As autonomic arousal and activation of the SNS may be implicated in IBS

(Shufflebotham et al., 2009), potential findings could also be particularly useful in the design of additional resources and treatment plans for IBS sufferers with Type D personality (Sararoudi et al., 2011).

Baseline Type D scores also significantly predicted cold and flu symptoms at follow up, when bassline measures were controlled. Cold and flu like symptoms are often exemplary of viral respiratory infections and are related to a dampened immune system (Kelly & Busse, 2008). Both reduced immunity and cold and flu symptoms can be exacerbated by increases in stress (Kiecolt-Glaser et al., 1987; Smolderen, Vingerhoets, Croon & Denollet, 2007) and abnormalities in the stress response system, mainly underpinned by maladaptation of the HPA axis and increased cortisol levels (See Chapter 6 for further explanation). Additionally, the cold/flu symptom factor includes the item 'stuffy head or nose', a symptom often associated with allergies such as allergic rhinitis (hay fever) and asthma, which share some pathophysiology with chronic systemic inflammation (Ciprandi et al., 2005). Therefore, this may also suggest that the effects of Type D personality may extend to abnormalities with inflammatory processes. It has been previously reported that Type D is associated with both pro-inflammatory cytokines such as IL-6 (Denollet, Schiffer, et al., 2009) and high sensitivity C-Reactive protein (Einvik et al., 2011) in cardiac patients. Furthermore, inflammatory illnesses such as these have shown associations with cardiac outcomes such as increased risk of stroke and elevated blood pressure (Matheson, Player, Mainous, King & Everett, 2008). Therefore, given the research indicating that Type D is a predictor of poor prognosis and depression in cardiac patients (Denollet, Pedersen, Vrints & Conraads, 2006; Dulfer et al., 2015; Kupper & Denollet, 2007), and inflammation (e.g. Dooren et al., 2015), these findings suggest a relationship between Type D and increased low level inflammation in the general population (i.e. prior to disease onset). This suggestion requires further consideration, and will be explored using C-Reactive protein levels in the experimental study described in Chapter 7.

5.4.4 Stress and psychological distress outcomes

Type D individuals reported significantly higher levels of perceived stress, anxiety and

depression in addition to increased stressful life events experienced within the past year, as compared to non-Type D individuals. These findings were also supported by the correlational analyses which indicated significant relationships between NA, SI and Type D scores with the four variables. Substantial cross sectional evidence supports the relationship between Type D, and psychological distress and perceived stress (e.g. Pedersen, van Domburg, Theuns, Jordaens & Erdman, 2004; Polman, Borkoles & Nicholls, 2010; Williams & Wingate, 2012). The present study has extended previous Type D research conducted within the general population by considering the longitudinal relationship between Type D and these outcomes. It is suggested from the present study findings that the continuous Type D construct significantly predicted perceived stress and aspects of psychological distress over a one-year period.

5.4.5 Mediating relationships

A number of potential mediating relationships were identified between Type D and the symptom clusters identified (metabolic, gastrointestinal and cold/flu symptoms) via the psychological variables. Firstly, it was found that anxiety fully mediated the relationship between Type D personality and metabolic symptoms. Anxiety symptoms have been related to both increased somatic symptoms (Haug et al., 2004) and Type D personality (e.g. Pedersen, 2004), although it has been claimed that the latter association may be mainly attributable to the high levels of negative affectivity which are characteristic of Type D (Howard & Hughes, 2012) due to the similarities of the constructs. Further, as items on the metabolic symptoms factor (e.g. feeling low in energy, poor appetite) may be related to stress and burnout, it is not surprising that levels of anxiety may play a mediating role in the relationship. This is supported by research conducted in a large sample of healthy individuals that found anxiety was related to symptoms of burnout (Toker, Shirom, Shapira, Berliner & Melamed, 2005). The study, also demonstrated associations between anxiety, depression and burnout with biomarkers of inflammation (Toker et al., 2005), which further highlights the importance of assessing levels of inflammation in Type D individuals from the general

population (see chapter 7).

Contrary to both expectation and previous research (e.g. Watson, 1988), baseline measures of perceived stress did not significantly predict any of the three symptom clusters. Therefore, perceived stress was not found to mediate the relationships between baseline Type D and either metabolic, gastrointestinal or cold/flu symptoms at follow up. However, stressful life events were found to partially mediate the relationships between Type D and both metabolic and gastrointestinal symptoms. Therefore, these findings partially support research demonstrating associations between increased stress and symptoms such as those included on the metabolic factor (e.g. poor appetite, sleep problems and fatigue; Kim & Dimsdale, 2007; Serlachius et al., 2007), and the influence of stress on gastrointestinal problems such as IBS (Pract, 2016). Further, both gastrointestinal problems (e.g.IBS; Mazur et al., 2007) and some psychosomatic symptoms (e.g. Matsumoto, Ushiroyama, Kimura, Hayashi & Moritani, 2007) have been associated with autonomic imbalance, which as previously mentioned can be a consequence of increased stress (Piazza, Almeida, Dmitrieva & Klein, 2010; Thayer, Yamamoto & Brosschot, 2010). As previously stated this will be further explored as a potential mechanism in Chapter 7.

Stressful life events were found to partially mediate the relationships between Type D and metabolic and gastrointestinal symptoms, whereas perceived stress was not related to either cluster. This suggests that it is the experience of chronic stress over a long period (i.e. at least year) in relation to Type D that is important, given that the PSS assesses only recent perceptions of stress in the previous month. On the other hand, the observation that respondents to the follow up reported lower levels of perceived stress than non-respondents may have influenced these results. However, both stressful-life events and perceived stress were related to both cardiac/sympathetic and vasovagal symptoms, although these two clusters were not significantly predicted by baseline Type D scores when baseline symptoms were controlled. Nevertheless, given the cross-sectional findings documented in Chapter 4 (that Type D interaction scores were related to these clusters over and above NA and SI in isolation) and previous research demonstrating links between Type D and cardiac health (Emons, Meijer

& Denollet, 2007; Grande & Romppel, 2011; Sher, 2005), it seems plausible that cardiovascular stress reactivity may play a role in the relationship.

It was unexpected that the stress-related variables significantly predicted cold/flu symptoms, given that reduced immunity associated with contracting the common cold can often result from increases in stress (Cohen et al., 1993). However, Smolderen, Vingerhoets, Croon, and Denollet (2007) found that NA, and perceived stress were associated with increased self-report of flu-like illnesses, whereas SI was related to lower reporting of less flu-like illnesses which may suggest the relationship between Type D personality and flu-related illnesses is not clear cut.

Depression and Type D personality have both been postulated as independent psychosocial risk factors for cardiovascular disease (Barth, Schumacher & Herrmann-Lingen, 2004; Gesine Grande et al., 2012). Depression has also been implicated in the relationship between Type D personality and poor health outcomes in cardiac patients (De Voogd et al., 2009). However, depression did not mediate the relationship between Type D and any of the symptoms in the present study, which focussed on individuals drawn from the general population. Speculatively, it is possible that depression only emerges as a risk factor for the development of physical symptoms in Type D individuals from clinical populations at a later stage of illness progression.

5.4.6 Retrospective health and treatment seeking

A further aim of the present study was to investigate differences in indicators of ill health, physical health status and health care utilisation over the past year between Type D and non- Type D individuals, as determined by retrospective health questions. It was observed that Type D participants were more likely than the non-Type Ds to rate their current general health and health over the past 12 months as poorer, and were more likely to be dissatisfied with their current health status. It also emerged that Type Ds were more likely than non-Type Ds to report higher frequencies of feeling unwell or run down, experience non-serious illnesses and to be absent from work due to sickness. This corresponds with the findings of a meta-

analysis (Mols & Denollet, 2010b) which demonstrated links between Type D and poorer health status and increased work related problems including absenteeism.

Additionally, Type D individuals were less likely to have never sought medical advice during the past year; however, no relationship was observed between Type D and frequency of GP or hospital visits. This partially opposes a previous study that found Type D individuals were less likely to get a regular medical check-up (Williams et al., 2008), and may suggest that they prefer to seek out medical information from other sources instead. If Type D individuals suffer more illnesses as these findings suggest, but do not seek treatment, it could be proposed that this is likely to be attributed to the social inhibition aspect of the Type D construct. For example; social isolation has been related to reduced health care utilisation and treatment seeking (Cacioppo & Hawkley, 2003) and social support is thought to increase healthcare utilisation (Wallston, Alagna, DeVellis & DeVellis, 1983). It is assumed that individuals will communicate with, and are influenced by members of their social network in their decisions to seek care (Wallston et al., 1983), therefore Type Ds may seek information elsewhere (e.g. online) due to their reduced inclination to interact socially.

No apparent differences between the Type D and non-Type D groups were observed on medication use, visits to the GP, or hospital attendance; however, as Type Ds reported more illnesses, this would again support the suggestion that Type D individuals are more likely to avoid seeking treatment. On the other hand, the lack of differences observed may also be due to the 'healthy' population targeted. The present study findings contradict those of a similar retrospective study (Michal, Wiltink, Grande, Beutel & Brähler, 2011) which found a relationship between Type D personality and both increased health care utilisation and treatment seeking behaviour. However, this previous finding was particularly focussed upon the utilisation of mental health services, and the sample included individuals with diagnosed mental health issues (e.g. clinical depression, panic disorder and alcohol abuse). This could partially explain the disparities between both the current study and the study by Williams and colleagues, (2008) with the findings of Michal et al. (2011).

The finding that Type D individuals were more likely to experience a minor, but not

more serious illness than non-Type Ds, leads to the inference that Type D individuals in the general population may report more minor 'everyday' health complaints and general feelings of being unwell. As discussed in terms of the self-reported symptoms in Chapter 4, this is potentially due to their increased levels of negative affect (Watson & Pennebaker, 1989) and increased sensitivity to sensations attributed to certain symptoms (Cohen, Doyle & Skoner, 1995). Additionally, given that Type D individuals appear to avoid seeking medical advice or treatment, most likely due to their levels of social inhibition (Williams et al., 2008), they may neglect to report more serious, diagnosable illnesses that they fear may warrant medical treatment or visits to healthcare professionals.

5.4.7 Strengths and limitations

Firstly, the longitudinal design adopted in the study is advantageous, particularly in comparison with cross-sectional studies (Farrington, 1991). It has enabled the advancement of our knowledge regarding the links between Type D personality and health including a clearer picture of the directionality of the relationships identified in Chapter 4. The longitudinal design has also enabled the investigation of changes in perceived health status over a year in Type D individuals, which extends previous cross-sectional work (e.g. Chapman, Duberstein & Lyness, 2007; Jellesma, 2008; Michal et al., 2011; Stevenson & Williams, 2014).

The use of the online survey platform Qualtrics was also particularly beneficial for this study as participants were easily invited to participate remotely via the customised link. The use of Qualtrics also eased the process of monitoring the timeframe in which participants completed the follow-up to ensure relative consistency in response times. Nevertheless, the follow up did suffer a substantial rate of attrition. The first cross sectional study boasted a large sample size of 535. However, only 160 of these individuals completed the one year follow up. Additionally, similar to the first phase of the study documented in Chapter 4, the sample comprised a large proportion of females. Research has suggested that females are more likely than males to report somatic symptoms (Gijsbers van Wijk & Kolk, 1997). However,

there is little difference in the prevalence of Type D personality across genders, which limits the potential implications of the gender imbalance of the present study (Mols, Denollet, Kaptein, Reemst & Thong, 2012).

Physical symptoms were measured by the CHIPS which is a simple, practical tool for the assessment of symptom clusters (Allen, Wetherell & Smith, 2017); however the scope of the tool is limited as it only includes 33 symptoms, and as it is not recommended for diagnostic purposes, it is not a reliable measure of any specific illnesses. Further, as it is subjectively scored it can also be affected by individual differences in sensory-processing sensitivity and distress (Benham, 2006). The inclusion of retrospective health questions within this study was beneficial in complementing the assessment of physical symptoms as the only health measure. The addition of this aspect of retrospective health extends previous research into the potential everyday influences of Type D personality on various aspects of health and health behaviour, over and above that of symptom/outcome reporting. However, it must be acknowledged that this cross sectional aspect of the study relied solely on retrospective self-report and therefore causality cannot be reliably inferred. Further, retrospective reporting of health related information is considered less reliable, relative to prospective self-report (Taffe & Dennerstein, 2000). This issue could be overcome by complementing self-report measures with objective biomarker measures of ill health (Piazza et al., 2010). Future studies could therefore support the validity of these findings by employing the assessment of objective physiological markers (e.g. levels of cortisol, inflammatory proteins, cardiovascular function) at a number of time points over a one-year period, in addition to the self-report measures.

Finally, the follow up period of one year was a reasonably short timeframe for investigating the development of poor health in the general population. While a longer term follow-up was not possible within the constraints of this PhD programme, it is recommended that future studies employ a substantially longer follow-up period to maximise our understanding of the longitudinal relationship between Type D personality and the development of physical symptoms. Of course, if future studies experienced an attrition rate equivalent to that of the present study, they would need to recruit a substantially larger sample

of participants to account for this level of attrition over a period of several years.

5.4.8 Conclusions

Type D personality was found to be longitudinally related to a number of self-reported health indices, specifically metabolic gastrointestinal and cold/flu symptoms. It appears that the influence of Type D is stronger for those symptom clusters with a potential stress-related cause, and therefore may support the theory that the Type D personality is linked to maladaptive stress reactivity. Anxiety mediated the relationship between Type D and metabolic symptoms at follow up. However, depression did not mediate any Type D-symptom relationships, suggesting that depression may contribute to the Type D-health relationship later on in disease progression. Interestingly, perceived stress did not mediate the relationship between Type D and any physical symptoms. However, stressful life events partially mediated the relationship between Type D and both metabolic and gastrointestinal symptoms. It has therefore been suggested that the link between Type D and physical symptoms may be underpinned by a stress-related pathway, potentially due to a maladaptive sympathetic nervous system response.

In summary, the present study findings provide further support for the proposition that experimental assessment of the SAM axis in response to acute stress is warranted in Type D individuals. Furthermore, due to the association between anxiety, burnout symptoms, and inflammatory biomarkers it is suggested that levels of inflammation should be assessed in Type D individuals. These objective measurement suggestions will form the basis of the experimental study documented in Chapter 7.

Chapter 6: A review of the mechanisms of the stress response, including the techniques for inducing acute stress and measurement of stress biomarkers.

Existing research has clearly established links between Type D personality and poorer physical and psychological health. The main theme within this thesis proposes that stress and stress reactivity are likely to play a role in this relationship. The negative health effects of stress are well documented, and therefore it seems likely that it will play a role in the relationship between Type D personality and the physical symptoms identified thus far. The present chapter will describe and discuss the systems involved in the stress response, and methods for measuring stress reactivity and inducing stress in the laboratory. Literature on Type D personality and the stress response will also be outlined where appropriate.

6.1 Stress response and health

It is well established that exposure to stress can increase an individual's vulnerability to poor psychological and physical health outcomes (Chrousos, 2009). For example, research has demonstrated that chronic stress is related to increased cold and flu symptomology (Cohen, Tyrrell & Smith, 1993), episodes of clinical depression (Andrews & Wilding, 2004), allergy flare ups and exacerbation of existing autoimmune conditions (Buske-Kirschbaum et al., 2002), in addition to the accelerated progression of aging and deterioration of health. These consequences fundamentally stem from activation of the psychobiological stress system. In response to an actual or perceived stressor, activation of the stress system leads to various changes that enables the body to prepare for survival (McEwen, 2000). This is then followed by self-regulation via negative feedback in order to restore homeostasis.

There are two principal psychobiological pathways of the stress system which originate in the hypothalamus. The first involves the activation of the autonomic nervous system via the sympathetic-adrenal-medullary (SAM) axis and the release of catecholamines, into the blood stream. This system responds quickly to acute stress and prepares the body for what is

described as the ‘fight or flight response’ by increasing sympathetic activity, central nervous system arousal, and skeletal-muscle activity (Cannon, 1932; Taylor et al., 2000). The secretion of catecholamines from the adrenal medulla evokes a number of immediate physiological changes, including reduced blood flow to the gut and extremities; increased blood flow to the muscles, heart, and lungs (increasing blood pressure and heart rate); and changes in blood sugar levels (Cannon, 1932). Repeated activation of this system, however, can lead to poor physical health outcomes including gastrointestinal problems, headaches, and musculoskeletal pain (e.g. Whitehead, 1994). The SAM axis is shown diagrammatically in figure 6.1.

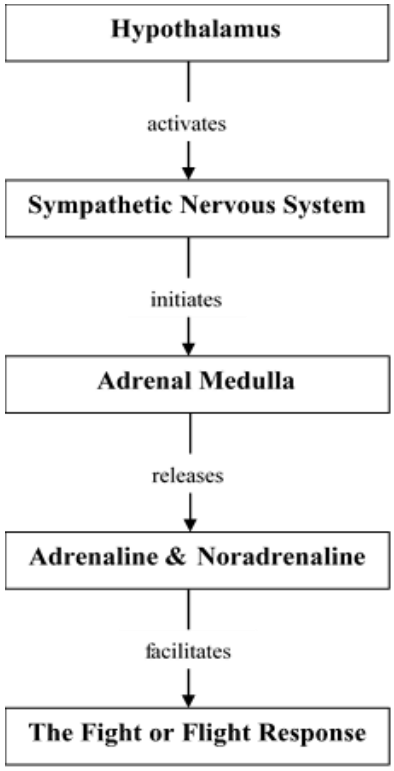

Figure 6.1. The SAM axis response to stress.

The second component involves activation of the hypothalamic-pituitary-adrenal (HPA) axis, which stimulates the secretion of glucocorticoids such as cortisol into circulation (Juruena, 2014). This is a longer acting pathway, which is often continually activated in response to chronic periods of stress, although has a higher threshold of activation in comparison to the SAM axis. (The HPA axis is represented diagrammatically in figure 6.2.) Consequentially, over-activation of this system in particular can lead to negative health

consequences, often associated with abnormalities with the immunological response (Hall et al., 2012). The speed and magnitude of the two pathways can vary depending on the stressor, and are not always activated by every stressor (Sapolsky, 1994).

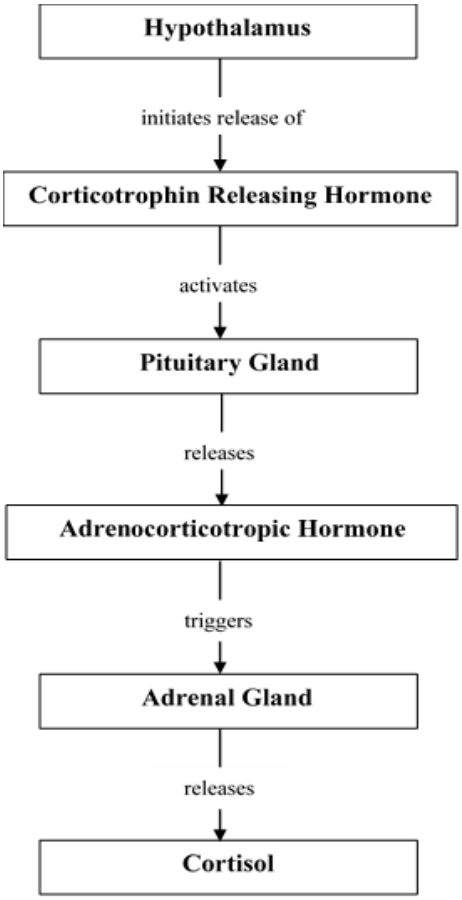

Figure 6.2. The HPA axis response to stress.

The effect of chronic stress on the body can also be explained in terms of allostasis and allostatic load. Allostasis is the process of maintaining physiological stability by changing internal parameters to appropriately fit with environmental demands (McEwen, 2000). In healthy individuals, maintaining allostasis means the body is able to adequately respond and adapt to these demands. However, when repeated allostatic responses are continuously activated in stressful situations the body experiences 'wear and tear' which is termed allostatic load and contributes to ill health (Juster et al., 2010). A normal physiologic (allostatic) response is triggered by a stressor and sustained for an appropriate period and then recovers (top of figure 6.3). However, there are four conditions that can lead to allostatic load; i)

experience of repeated "hits" from numerous stressors (top left); ii) failure to adapt to a stressor (top right); iii) when a response is prolonged due to delayed/no recovery (bottom left) and; iv) when the response to a stressor is inadequate, leading to a compensatory increase in activity of other factors (e.g. heightened levels of cytokines due to inadequate secretion of glucocorticoids).

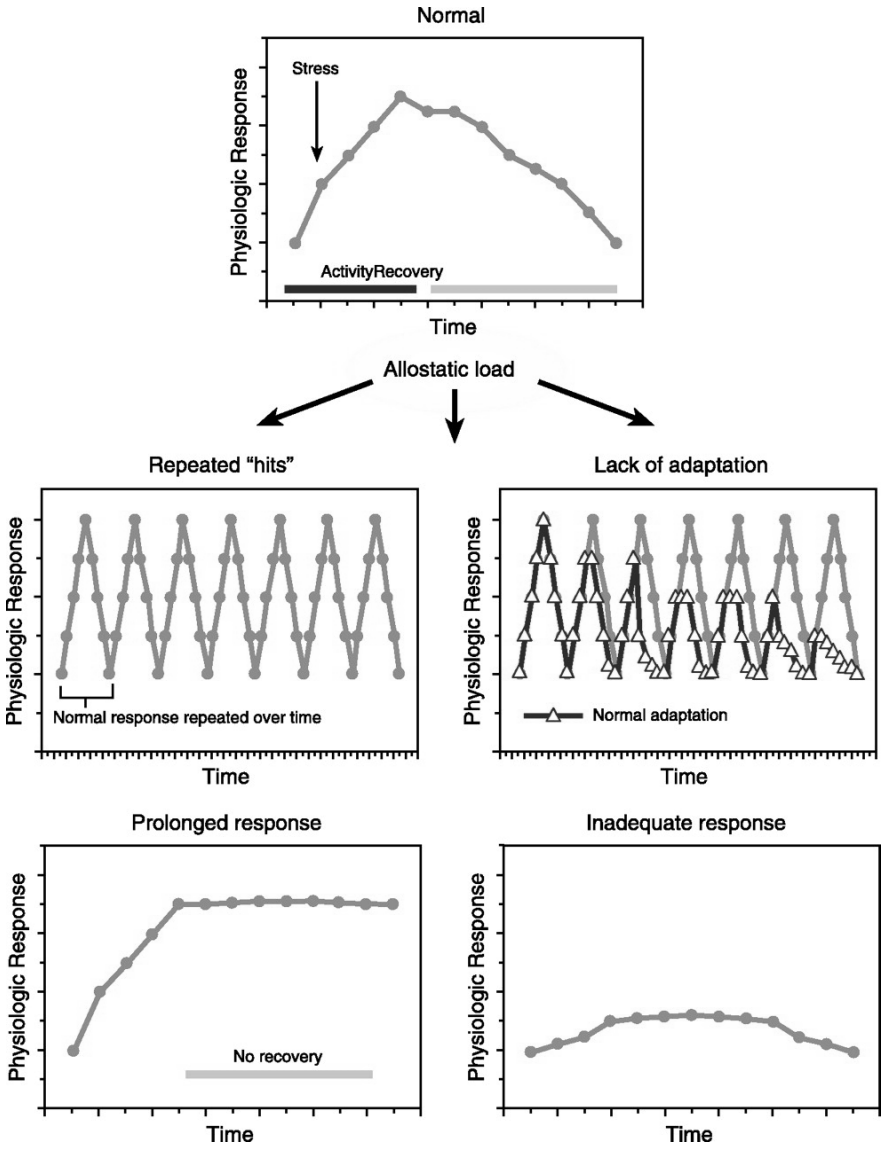

Figure 6.3.5 Illustrations of the four types of allostatic load. *Top middle*; the normal allostatic response. *Top left*; repeated "hits" from multiple stressors. *Top right*; lack of adaptation. *Bottom left;* prolonged response. *Bottom right*; inadequate response. Taken from McEwen (1998).

An abundance of research suggests that both the SAM axis and HPA systems play

substantial roles in the relationship between stress and health (Cohen, Janicki-Deverts & Miller, 2007), and may therefore be mechanisms by which Type D leads to ill health. Furthermore, immediate emotional responses, such as anxiety or frustration, often accompany stress–related physiological changes in the body (Spector, Dwyer & Jex, 1988). This is particularly salient in the context of Type D personality, given that previous research (e.g. Habra et al., 2003; Howard & Hughes, 2012, 2013; Kelly-Hughes et al., 2014) has reported maladaptive stress reactivity in Type D individuals.

6.2 The HPA axis and Cortisol

The HPA axis has received widespread attention in the literature as a mechanism by which stress can affect health. Activation of the system is viewed as the most important endocrine component of the stress response (Juruena, 2014) and can be triggered by numerous physical and psychological stressors. In response to a stressor, corticotropin-releasing hormone (CRH) is released from the hypothalamus, triggering the release of adrenocorticotropin hormone (ACTH) from the anterior pituitary gland, and in turn, cortisol from the adrenal cortex. The secretion of hormones in the HPA axis is subject to a regulatory negative feedback loop, governed by the superchiasmatic nucleus (SCN) and paraventricular nucleus (PVN) (Chrousos & Gold, 1992). Cortisol, the primary effector hormone and the endpoint of this axis, influences multiple systems in the body including the central nervous system, the metabolic system, and the immune system (Sapolsky, Romero & Munck, 2000).

Cortisol exhibits a diurnal profile in healthy individuals which begins with the cortisol-awakening response (CAR); a dramatic increase in cortisol during the first 30–45 minutes after waking. Chronic exposure to stressful situations can result in abnormal (e.g. flattened or exaggerated) CARs as exemplified by Schulz, Kirschbaum, Prüßner & Hellhammer, (1998) who found increased awakening cortisol levels in individuals experiencing chronic work overload. Cortisol levels then decrease throughout the day, which is known as the diurnal cortisol decline (Clow, Thorn, Evans & Hucklebridge, 2004).

Chronic stress can also lead to atypical diurnal cortisol profiles, which have been linked

to numerous health problems, and observed in various clinical populations (e.g. Jarcho, Slavich, Tylova-Stein, Wolkowitz & Burke, 2013; Sephton, 2000; Weissbecker, Floyd, Dedert, Salmon & Sephton, 2006)). Abnormal profiles characterised by heightened levels of cortisol have been observed in relation to reduced sleep quality (Buckley & Schatzberg, 2005), anxiety disorders (Mantella et al., 2008) , and depressive symptoms (Pruessner, Hellhammer, Pruessner & Lupien, 2003). Conversely, blunted profiles have also been linked to poor sleep quality and adverse metabolic health outcomes in middle aged adults (Lasikiewicz, Hendrickx, Talbot & Dye, 2008).

It has been suggested that disruption of the HPA axis could be a pathway underpinning adverse clinical outcomes in Type D individuals. For example, two studies found that type-D personality was positively associated with the CAR magnitude (Whitehead, Perkins-Porras, Strike, Magid & Steptoe, 2007) and diurnal cortisol output (Molloy et al., 2008) in acute coronary syndrome patients.

Cortisol can be measured in saliva to provide a non-invasive index of HPA activity (Kirschbaum & Hellhammer, 1989). Consequently, the majority of research into the HPA axis has utilised this technique as both the diurnal variation and acute reactivity of cortisol can be assessed in this way.

Many studies aimed at examining stress-related responses have investigated individual differences in salivary cortisol responses to an acute stressor in the laboratory (Ginty et al., 2012; Goodin, Smith, Quinn, King & McGuire, 2012; Wetherell, Craw, Smith & Smith, 2017). As such, a couple of studies have investigated acute HPA reactivity in Type D individuals. However, these studies have yielded mixed findings. A study by Habra, Linden, Anderson, and Weinberg, (2003) found that both NA and SI were associated with greater cortisol reactivity to mental arithmetic stress. However, analyses were not sufficiently stringent for reliable conclusions to be drawn as the stressor did not evoke a significant cortisol change and the study did not consider the interactive influence of NA and SI on cortisol reactivity. By contrast, Bibbey, Carroll, Ginty, and Phillips, (2015) found that Type D individuals exhibited a significant change in cortisol levels in response to socially evaluative

stress, in comparison to non-Type Ds and those in a low social evaluative condition. However, the difference in tasks could account for these alternative findings, particularly given that a Type D difference was only observed in a socially evaluative condition in Bibbey's study.

On this note, there is considerable variation in the capacity of laboratory based stress paradigms to reliably evoke salivary cortisol responses (Kudielka et al., 2006). It has been suggested that the inclusion of social-evaluative threat and elements which participants are not able to control are necessary to reliably elicit a cortisol response (Dickerson, Mycek & Zaldivar, 2008). The Trier Social Stress Test (Clemens Kirschbaum, Pirke & Hellhammer, 1993) is a standardised psychosocial stress task which includes both uncontrollable and social-evaluative aspects (public speaking and mental arithmetic), and as such is frequently utilised as a reliable laboratory paradigm to evoke a cortisol response (Kudielka, Hellhammer & Wüst, 2009).

As such, given that the TSST is time consuming and labour intensive it was decided that other stressor paradigms would be explored within the current project, and focus would lie on the SAM axis response to stress rather than cortisol. Furthermore, given that Type D personality has been related to poor cardiac health (e.g. Denollet, Vaes & Brutsaert, 2000), and links with cardiac symptoms have been demonstrated in the earlier studies within this programme, it seems necessary to focus on other measures of sympathetic arousal (including a range of cardiovascular parameters) and inflammatory biomarkers, which have received less attention in the Type D literature.

6.3 The SAM axis

The SAM axis has also been postulated as a mechanism by which stress can effect health (Glaser & Kiecolt-Glaser, 2005). This response is governed by the autonomic nervous system which regulates bodily functions via the modulation of the opposing sympathetic and parasympathetic branches. In response to a stressor, hypothalamic activation of the sympathetic nervous system results in increased secretion of catecholamines from the adrenal medulla including the release of adrenaline and noradrenaline from presynaptic nerve

terminals (Desborough, 2000). Adrenaline and noradrenaline evoke increases in heart rate, respiration, blood flow, and blood pressure, and a simultaneous decrease in digestive activity, in order to mobilise the body to deal with a stressor (Compas, 2006).

If the SAM axis is chronically activated, this can lead to reductions in immune function (Reiche, Morimoto & Nunes, 2005) and dysregulated cytokine production, both of which have serious negative consequences for health (Glaser & Kiecolt-Glaser, 2005). Dysregulation of the sympathetic stress response has also been found to lead to metabolic abnormalities through dysregulation of lipid metabolism and blood pressure over time, which can predispose individuals to cardiovascular disease and diabetes (Licht, De Geus & Penninx, 2013). Research has also found sympathetic dysregulation in disorders such as sleep apnoea (Mansukhani, Kara, Caples & Somers, 2014), panic disorder, and post-traumatic stress disorder (Cohen et al., 2000). These findings highlight that a defective sympathetic stress response can play a role in ill health, and that it is important to consider SAM axis function in relation to the influence of individual differences on stress reactivity.

6.3.1 Measuring SAM axis activity

The most typical method of directly measuring SAM activity is the assessment of cardiovascular function, including blood pressure, heart rate and other haemodynamic parameters. Galvanic skin response, which measures the change in the electrical properties of the skin is another available technique, however this method has been criticised for lack of consistency and practicality (Arunodaya & Taly, 1995).

A further common reliable technique for measuring SAM activity is the measurement of the catecholamines; adrenaline and noradrenaline, in plasma (Mills & Ziegler, 2007). However, this method requires invasive (and potentially stress–inducing) blood collection protocols. Therefore, less invasive techniques for measuring SAM activity have been developed. Saliva sampling is a relatively simple and non-invasive method of measuring biological markers (Beltzer et al., 2010); however direct measurements of adrenaline and noradrenaline in saliva appear not to reflect SAM activity (Schwab, Heubel & Bartels, 1992).

Recently however, there has been a surge in interest in the measurement of alpha amylase in saliva, as a surrogate biomarker of SAM activity (Nater & Rohleder, 2009).

Techniques employed for the measurement of cardiovascular function include portable sphygmomanometers to assess ambulatory blood pressure and heart rate; the Finometer, which can compute a range of haemodynamic parameters via computed aortic-flow waveform from finger arterial pressure measurements (Schutte, Huisman, Van Rooyen, Oosthuizen & Jerling, 2003); and electrocardiography (ECG) which measures electrical activity of the heart (Ewing, Neilson & Travis, 1984). The main cardiovascular biomarkers of the SAM axis are heart rate (HR), diastolic blood pressure (DBP) and systolic blood pressure (SBP), which are also biomarkers of cardiovascular health. HR refers to the number of times the heart beats per minute and is an indicator of blood flow regulation and oxygen delivery to vital organs and peripheral muscle (Piazza et al., 2010). DBP refers to the lowest arterial pressure between heartbeats and exerts the minimum amount of pressure on blood vessel walls during diastole, whereas SBP is the peak arterial pressure during a heartbeat and exerts maximum force on blood vessel walls during systole (Piazza et al., 2010).

6.3.2 Cardiovascular reactivity

In healthy individuals BP and HR have been found to increase in magnitude in response to stressful stimuli. A plethora of research has demonstrated this in response to numerous stressors including public speaking tasks (Al'Absi et al., 1997), cognitively challenging laboratory based tasks (Kamarck & Lovallo, 2003), mental arithmetic, multitasking (Wetherell & Carter, 2014), physical stressors such as the cold pressor (Dembroski, MacDougall, Herd & Shields, 1979) and in anticipation of real-life situations such as exams (Sausen, Lovallo, Pincomb & Wilson, 1992). However, it is well established that individuals can differ with respect to their patterns of acute changes in cardiovascular function, and similarly, it has been demonstrated that different types of tasks can evoke different patterns of cardiovascular reactivity (CVR) (Kamarck & Lovallo, 2003).

Maladaptive cardiovascular reactivity can be indicative of various underlying

psychobiological problems and can have substantial negative implications for health. There is evidence to suggest that exaggerated CVR can predict the development of cardiovascular disease (Blascovich & Katkin, 1993) including preclinical (e.g., increased left ventricular mass and blood pressure) and clinical events in patients with hypertension and coronary heart disease (Treiber et al., 2003). Exaggerated CVR has also been linked to other indices of poor cardiac health including hypertension and atherosclerosis (Kamarck et al., 1997), as well as increased cardiac morbidity (Carney, Freedland & Veith, 2005), and risk of stroke (Everson et al., 2001). Exaggerated responses have also been associated with increased work stress (Vrijkotte, van Doornen & de Geus, 2000) and psychological distress (Lepore et al., 1997). Furthermore, it has been suggested that heightened CVR to acute stress can be attenuated by a number of psychosocial variables including increased social support (Uchino, 1995), perceived control, and levels of physical activity (Rimmele et al., 2009).

Cardiovascular reactivity is assumed to be a mechanism that functions on the basis of adaptive pressures, implying that optimal function occurs at mid-range intensity and function at either extreme is likely to be less adaptive (Phillips et al., 2013). Hence, there is evidence to suggest that blunted reactivity can also have negative health consequences (see Phillips, Ginty & Hughes, 2013 for review). This has been evidenced in a study by Phillips (2011) which demonstrated blunted cardiovascular reactivity in relation to depression, obesity, and poor self-reported health. Furthermore, blunted reactivity has been found to predict smoking relapses (Al'Absi et al., 2005) and has been observed in the children of alcoholics (Sorocco et al., 2006). Blunted reactivity is thought to be particularly problematic for particular health outcomes including obesity. The way in which exaggerated and blunted reactivity can negatively impact health has been conceptualised as an inverted-U model, and is dependent on the health outcome (Douglass Carroll, Lovallo & Phillips, 2009). The model is displayed in Figure 6.4.

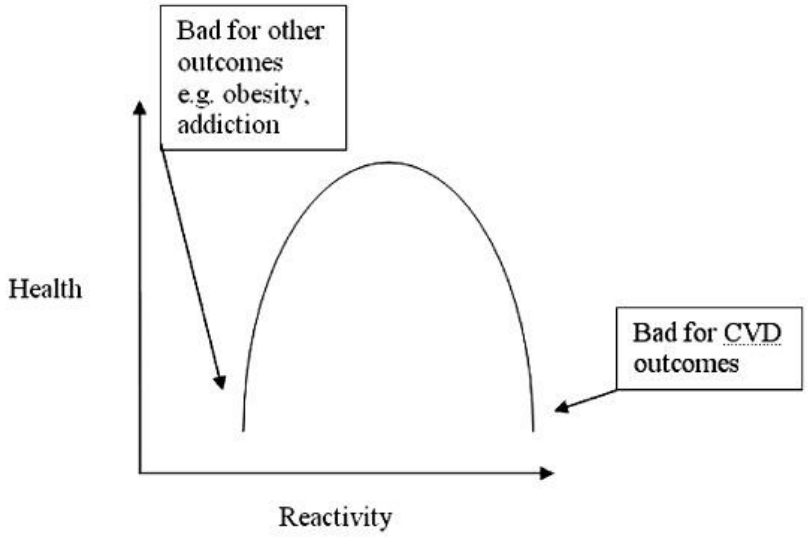

Figure 6.4. Inverted U shape model showing how both exaggerated and blunted reactivity can affect health outcomes (Carroll et al., 2009).

As Type D personality is heavily theorised as a prognostic risk factor for poor cardiac health (Denollet et al., 2006; Denollet, 1998a; Pedersen & Denollet, 2004), and given that distressed individuals are likely to respond atypically to stress, a number of studies have investigated cardiovascular reactivity to stress in relation to Type D.

As previously mentioned in Chapter 1, a relationship between NA and heightened blood pressure reactivity to a mental arithmetic challenge was observed by Habra et al., (2003), and high SI was associated with reduced HR change; however these effects were only observed in men. Heightened cardiovascular reactivity in Type D individuals has also been observed in both men and women in other studies (Bibbey et al., 2015; Williams et al., 2009). However, Bibbey et al., (2015) also examined the contribution of social evaluative threat, finding that Type D individuals only showed greater cardiovascular response to stress in a condition involving high social evaluation threat; suggesting that the type of stress is important. Furthermore, Type D individuals showed a lack of cardiovascular adaptation to a recurring stressor (Howard & Hughes, 2013), further suggesting the complexity of the relationship between Type D and cardiovascular stress reactivity.

Type D has also been related to blunted cardiovascular reactivity. Kelly-Hughes et al.,

(2014) found that Type D was related to lower blood pressure reactivity to an acute multitasking stressor, and O'Leary et al., (2013) found that Type D women exhibited lower systolic blood pressure responses to a stressful task under sleep restriction. Howard, Hughes, and James (2011) also found that Type D personality was related to decreased heart rate reactivity to stress, again in women. It could also be theorised that whether Type D is associated with an exaggerated or blunted cardiovascular response could be due to the presence of social evaluation. Furthermore, the difference may also be attributed to a potential moderating effect of gender, therefore warranting examination of other aspects of sympathetic arousal.

6.3.3 Haemodynamic profile

Although these basic measures of cardiovascular function can be an indication of sympathetic activity, they are underpinned by other more complex haemodynamic parameters, variations in which may be masked by observed changes (or lack of changes) in HR and BP (Gregg, James, Matyas & Thorsteinsson, 1999). Therefore, it is recommended that other haemodynamic parameters are assessed in acute studies to provide a more comprehensive understanding of sympathetic stress reactivity. These parameters include; Stroke volume (SV; the amount of blood, in mL, ejected per heartbeat); cardiac output (CO; the rate, in L/min, of blood flow out of the heart); total peripheral resistance (TPR; the total resistance offered by the systemic circulation); ejection fraction (EJT; the percentage of blood that is ejected per heart contraction); and inter-beat interval (IBI; the time interval, in ms between each heart beat). Furthermore, different stressors (due to differential stimulation of alpha- and beta adrenergic receptors) can produce similar blood pressure responses with differing underlying haemodynamic patterns of TPR and CO (Gregg et al., 1999). It is therefore important to assess haemodynamic parameters in addition to other CVR markers to full understand the effect of acute stress on cardiovascular function (James, Gregg, Matyas, Hughes & Howard, 2012).

The patterning of CO and TPR, is termed Haemodynamic Profile (HP) and underpins the homeostatic regulation of blood pressure, whereby an increase in either parameter should

be accompanied by a decrease in the other (Gregg, Matyas & James, 2002). Abnormalities in the HP can have consequences for health, for example, normal cardiac output in combination with elevated total peripheral resistance is characteristic of hypertension (Julius, 1988). HP essentially measures the nature of the homeostatic compensation underpinning blood pressure change, and can be classified as either 'myocardial' or 'vascular', the latter of which is seen as a typically healthy response (Eliot, Buell & Dembroski, 1982). A myocardial profile (i.e. CO predominates), is demonstrated by an increase in CO together with a decrease in TPR, whereas, a decrease (or no change) in CO accompanied by an increase in TPR is classed as a vascular profile (i.e. TPR predominates). A mixed response is demonstrated by increases in both measures (i.e. a synergistic change in CO and TPR). Responses can vary dependent on the stressor and the individual. For example; responses to 'active' tasks such as mental arithmetic, are believed to be underpinned by beta-adrenergic stimulation, and evoke a marked myocardial response. By contrast, 'passive' stressors including the cold pressor task, typically elicit a more vascular response due to predominantly alpha-adrenergic stimulation (Gregg et al., 2002).

However, more recently, the classification of responses, and in turn, labelling of individuals into responder categories, has been deemed limiting (Gregg et al., 2002) and instead it is thought that responders lie across two continuums. A recent paper has therefore documented a new model of the orthogonal dimensions of HP and compensation deficit (CD) derived from the (multiplicative) interaction of CO and TPR (Gregg et al., 2002). CD represents the extent to which CO and TPR compensate and is essentially a measure of BP change (i.e. whether it increases, decreases, or does not change). This has culminated in the ability to compute a HP score and CD score for individual responders in which positive HP scores indicate a vascular response, and negative HP scores indicate a more myocardial response. Positive CD scores indicate an increase in BP and negative CD scores represent an accompanying BP decrease. The model is illustrated in figure 6.5 and the technique will be used within this programme (Chapter 7).

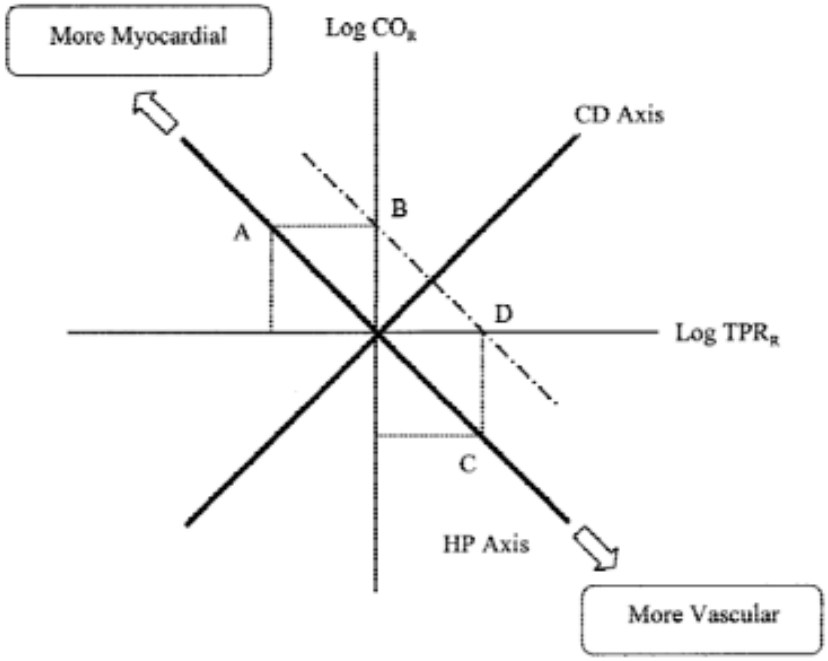

Figure 6.5. An orthogonal model of haemodynamic profile (HP) and compensation deficit (CD) taken from Gregg et al., (2002).

Research regarding the association between Type D personality and haemodynamic parameters is mixed. For example; Williams, O'Carroll, and O'Connor, (2009) found that Type D personality was related to increased cardiac output in response to stress in males, but not females. No differences were observed for TPR, potentially suggesting a CO predominant (myocardial) response, despite a lack of BP reactivity findings. In another study (Howard et al., 2011) Type D females exhibited blood pressure reactivity to a mental arithmetic stressor, as evidenced by positive CD scores, and HP scores were negative, demonstrating a myocardial HP. O 'Leary et al (2013) also found that Type Ds showed an increase in vascular responding in conjunction with lower SBP responses to a stressor. Kupper, Pelle, and Denollet, (2013) found that Type D personality was associated with an exaggerated haemodynamic response to the cold pressor task. However, this may be due the differing task; as previously stated, different tasks can elicit different responses. These interesting but limited findings therefore warrant further investigation into haemodynamic profile in Type D individuals.

6.3.4 Heart rate variability

Heart rate variability (HRV) is an additional measure of cardiac function which may be representative of sympathetic activation as it is governed by the autonomic nervous system (Thayer, Åhs & Fredrikson, 2012). In fact, HRV can be used to assess autonomic imbalances which are associated with poorer health and together with parasympathetic activity can be linked to immune dysfunction and inflammation (Thayer, Yamamoto & Brosschot, 2010). Decreased heart rate variability has been described as an independent risk factor for both physical and psychological morbidity and even mortality in heart disease patients (Grippo & Johnson, 2002; Thayer et al., 2010). Reduced HRV has also been associated with a number of psychological factors including stress, anxiety, (Dishman et al., 2000; Vrijkotte et al., 2000) and depression (Grippo & Johnson, 2002). Further, individual differences in HRV in response to stress have been identified. Martin et al., (2010) examined HRV patterns to stressful imagery experiences in relation to ethnicity and Type D personality. Findings showed that European-American Type D individuals exhibited higher low-frequency (LF) HRV responses (which are suggested to be a measure of sympathetic activity and higher levels of LF HRV are viewed as less healthy) than non-Type D individuals, or African-Americans. This study provides further support for the cardiac health risks associated with Type D personality (e.g. Denollet et al., 2006).

6.3.5 Salivary alpha amylase

As previously mentioned, recent literature has identified Salivary alpha-amylase (sAA) as a reliable surrogate biomarker of SAM axis activity (Nater & Rohleder, 2009). The original hypothesis for this stemmed from the knowledge that the sympathetic and parasympathetic branches of the autonomic nervous system innervate salivary glands. Sympathetic stimulation has shown to increase the secretion of salivary proteins and increases in salivary flow rate are controlled by parasympathetic stimulation (Rohleder, Nater, Wolf, Ehlert & Kirschbaum, 2004). sAA exhibits a stable circadian pattern with moderately low levels upon awakening, a brief drop at 30-min post-awakening, and increases gradually throughout the day (Nater,

Rohleder, Schlotz, Ehlert & Kirschbaum, 2007).

Although the primary function of sAA is enzymatic digestion of carbohydrates (Nater & Rohleder, 2009), concentrations have been found to be predictive of plasma catecholamine levels (Rohleder et al., 2004). For example research has demonstrated that sAA is associated with changes in noradrenaline induced by both physical (exercise) and psychosocial stress (Chatterton, Vogelsong, Lu, Ellman & Hudgens, 1996), suggesting that it is a reliable measure of endogenous adrenergic activity. Results have consistently suggested that sAA increases in response to psychological stress; however, activity appears to be most affected when stressors activate the autonomic nervous system (Nater et al., 2006). A number of studies have used the Trier Social Stress Test (TSST) to assess the usefulness of sAA as a biomarker for psychologically induced stress, with the majority of these studies demonstrating that the paradigm evokes increased levels of sAA(e.g. Rohleder et al., 2004; van Stegeren, Rohleder, Everaerd & Wolf, 2006). A recent study also found noticeable increases in sAA activity in response to a social laboratory based stressor in both sleep restricted and rested participants, and increased baseline sAA levels in the sleep restricted individuals (O'Leary et al., 2013).

Maladaptive sAA reactivity to stress has been observed in populations at risk for negative health consequences. For example, increased sAA responses have been demonstrated in maltreated youths (Gordis, Granger, Susman & Trickett, 2008), whereas, blunted sAA reactivity to the TSST has been observed in individuals at risk for suicide (McGirr et al., 2010). Further, raised awakening and diurnal sAA levels have been observed in women with prenatal depressive symptoms (Braithwaite, Ramchandani, Lane & Murphy, 2015).

sAA levels, as a proxy measure of sympathetic activation in response to acute stress, have yet to be investigated in relation to Type D personality. However, given the findings regarding Type D personality and other indicators of sympathetic stress activation including cardiovascular and haemodynamic reactivity, it seems like a logical next step in the assessment of the potential mechanisms underpinning the link between Type D personality and poor health. This will therefore be explored in Chapter 7 of this thesis.

6.4 Measuring stress in the laboratory

Many cardiovascular reactivity studies have utilised controlled laboratory based stressors. Such tasks are useful owing to the ease of measurement (i.e. via Finometer) experimental design capabilities, and increased control which is difficult to achieve in real-life stressful events. However, laboratory stressors are subject to a number of limitations. Further, there are of course a number of ethical considerations to take into account when inducing stress in participants (Zanstra & Johnston, 2011). The majority of paradigms are therefore cognitively based, including reaction time, mental arithmetic, and vigilance tasks. Another commonly used stressor is the psychologically and physiologically demanding, cold pressor test in which participants are instructed to submerge their hand in vessel of cold water, for as long as they are able. However, these acute stress paradigms tend to lack ecological validity and do not typically evoke the same magnitude of a responses seen in response to stressors in real life (i.e. they do not reliably evoke a cortisol response; Kudielka et al., 2009). Some of these laboratory stressors have a social component (e.g. public speaking) which improves the ecological validity of such tests. However, as participants are aware of the study purpose, stress responses are often still not representative of real world stress. To overcome this, it is further proposed that social evaluation and the receipt of negative feedback can evoke a more realistic response (Dickerson et al., 2008). Therefore, it is recommended that reliable paradigms include a stressful cognitive task, a social evaluative element in addition to some level of uncontrollability (Dickerson & Kemeny, 2004).

The TSST (Kirschbaum et al., 1993), is viewed as the gold standard of laboratory based stressors for eliciting cortisol reactivity. The TSST comprises an interview and arithmetic task in front of a panel of judges, in which participants are also advised their performance is being recorded for later analysis. The uncontrollable nature of the interview in combination with the social-evaluative threat from observation and difficult arithmetic task, evokes a stress response, including activation of the HPA axis (Schubert, Contreras, Franz & Hellhammer, 2011). This paradigm however, is heavily time-consuming and requires a significant amount of preparation and resources, so is therefore difficult to replicate, particularly in smaller

studies.

The Multitasking Framework (MTF; Wetherell & Sidgreaves, 2005) is a cognitively demanding stressor which aims to emulate multitasking stress by requiring participants to attend and respond simultaneously to a number of tasks with varying levels of workload (Wetherell & Carter, 2014; Wetherell & Sidgreaves, 2005). The paradigm is performance-based and cognitively demanding, with the additional advantage of being representative of busy work environments, and is therefore ecologically valid. The task can also be accompanied by negative verbal feedback (social evaluative element), to evoke a reliable psychobiological stress response (Wetherell et al., 2017), and is particularly useful in this project given the findings regarding cardiovascular reactivity to different tasks in Type Ds as previously mentioned. The MTF has been previously used in Type D stress research (Kelly-Hughes et al., 2014), along with numerous studies examining the psychobiological stress response (Wetherell, Atherton, Grainger, Brosnan & Scholey, 2012; Wetherell et al., 2017) in various populations.

6.5 Summary

It has been proposed in previous chapters that the stress response is likely to play a role in the relationship between Type D personality and physical symptoms. This chapter has reviewed the literature to explain a number of mechanisms by which reactivity of two psychobiological stress pathways (the SAM and HPA axes) can affect health. The HPA axis is well established as a mechanism by which atypical stress reactivity and chronic stress can affect health, however this pathway will not be further explored within this project, and rather focus will lie on the SAM axis. Type D personality has been most reliably linked with cardiovascular ill-health, for which dysregulation of sympathetic activity is a candidate mechanism. Further, the findings of previous chapters have indicated a link between Type D and cardiac/sympathetic, metabolic and vasovagal symptoms. The subsequent study will therefore explore measures of sympathetic arousal, given that sympathetic activation is the most likely mechanism by which Type D personality may affect health. A number of different

ways in which to easily and reliably measure SAM activity have been discussed in addition to how to most reliably and realistically induce acute stress in the laboratory. Consequently, SAM axis activity will be measured in relation to Type D personality via cardiovascular reactivity (including haemodynamic profile and a proxy measure of HRV) and levels of sAA in response to a laboratory based stressor with additional critical evaluation.

Chapter 7: An experimental investigation of sympathetic arousal, cardiovascular stress reactivity and immune activation in Type D individuals.

This chapter investigates a number of potential psychobiological mechanisms that may underpin the link between Type D personality and the specific symptom clusters identified in Chapters 3 and 4. To do so, an experimental study was conducted examining sympathetic activation in response to acute stress. A range of beat-to-beat cardiovascular parameters, as well as salivary alpha amylase; a biomarker of sympathetic activation, were assessed. C-Reactive protein (CRP); an inflammatory biomarker indicative of increased cardiac vulnerability, was also assessed. Analyses demonstrated that Type D personality was related to a number of cardiovascular parameters including BP, TPR and SV reactivity, and a potentially maladaptive change in haemodynamic profile over the stress procedure. However, levels of sAA and CRP were not found to be significantly influenced by Type D personality. These findings may implicate some indices of autonomic arousal in the relationship between Type D and poor physical health, and could explain the link with stress-related symptoms, particularly cardiac/sympathetic, vasovagal and metabolic symptom clusters (as identified in Chapter 4 and 5).

7.1 Background

As documented in chapters 3 and 4, it appears that Type D personality is related to specific physical symptom clusters, namely cardiac/sympathetic, metabolic, vasovagal, and headache symptoms. On this basis, it is suggested that Type D is related to some, but not all domains of ill health in the general population. Notably, those symptoms clusters that are associated with Type D have all been related to aspects of stress. On this basis, it could be suggested that psychobiological stress pathways underpin the link between Type D and ill health. However, relatively little is known about how Type D personality influences biological processes, particularly in 'healthy' individuals. It would seem particularly beneficial to

investigate whether, and to what extent, psychobiological stress reactivity may be related to Type D personality. If such effects can be identified early on in the lifespan, prior to chronic illness onset, preventative measures could be introduced to alleviate the adverse effects of dysregulated psychobiological stress reactivity on health outcomes.

Activation of the sympathetic nervous system has been associated with psychological distress and negative health outcomes (Carney, Freedland & Veith, 2005; Mancia, et al., 2007; Remme, 1998), which suggests that this system may also play a role in the Type D-health relationship. As such, numerous studies utilising stress-inducing protocols have demonstrated associations between Type D personality and dysregulated cardiovascular reactivity, with mixed findings (Bibbey et al., 2015; Habra et al., 2003; Howard et al., 2011; Howard & Hughes, 2013; Kelly-Hughes et al., 2014; O'Leary et al., 2013). Furthermore, the majority of studies have often only focused on a small selection of sympathetic measures, mainly cardiovascular parameters (as described in Chapters 1 and 6), and may not, therefore, have been able to provide a complete representation of sympathetic arousal, cardiovascular reactivity and haemodynamic profile in Type D individuals.

As described in chapter 6 Salivary alpha-amylase (sAA) has recently emerged as a reliable biomarker of sympathetic nervous system (SNS) activity (Nater et al., 2007), and increases in sAA concentrations have been observed in response to physiological and psychological stress (van Stegeren et al., 2006). Increased sympathetic arousal is associated with endothelial dysfunction and increased risk for coronary heart disease (e.g. Harris & Matthews, 2004) and has implications for immune related health (e.g. Grebe et al., 2010). This, given the extensive links between Type D and negative cardiac health (as described in Chapter 1) leads to the proposal that stress-induced sAA profiles may differ between Type D and non-Type D individuals. This question will be addressed in the current study.

Type D personality has also been associated with higher levels of pro-inflammatory cytokines (e.g. Conraads et al., 2006; Denollet, Vrints & Conraads, 2008), and it has been theorised that systemic inflammation may be a shared underlying biological pathway in both Type D personality and cardiovascular illnesses. Furthermore, given the combination of cross-

sectional and longitudinal findings indicating associations between Type D and inflammatory/immune related health complaints, assessment of inflammation levels in a group of 'healthy' Type D individuals appears to be warranted. C - reactive protein (CRP) is an inflammatory biomarker that can be relatively easily obtained from blood plasma, and indicates increased risk for cardiovascular disease including atherosclerosis and vascular stroke, and is also related to depression (Kuo et al., 2005). Heightened C- Reactive protein levels have been found to be related to Type D personality in a clinical population of sleep apnoea sufferers; a disorder associated with a high prevalence of cardiovascular risk factors (Einvik et al., 2011). However, to our knowledge CRP has not been examined in relation to Type D personality in the general, young adult population. It is therefore of interest in the present study to investigate this question.

7.1.1 Aims and objectives

The aims of the current study are to examine patterns of cardiovascular reactivity and sympathetic nervous system activity in response to acute stress, as well as levels of immune activation in relation to Type D personality within a population of apparently healthy individuals. It is hypothesised that Type D personality will be associated with an abnormal sAA output in response to stress, representative of a maladaptive sympathetic arousal pathway, and increased low-level inflammation as measured by levels of CRP. Further, heart rate variability; an indicator of autonomic regulation and cardiac/ inflammatory health in response to acute stress, will also be examined in relation to Type D personality. It is also hypothesised that Type D individuals will exhibit a maladaptive cardiovascular response to stress and abnormal haemodynamic profile. The current study attempts to further understand the nature of the relationship between Type D and sympathetic arousal through the simultaneous measurement of the different sympathetic parameters previously detailed in order to achieve a comprehensive view of the mechanisms in play.

7.2 Method

7.2.1 Participants

An opportunity sample of 75 healthy adults from the general population aged between 18-42 years (M_{age}=23.6), 48 females (M_{age}=23.81) and 27 males (M_{age} = 23.19) participated in the study. Of these, 33 were classified as Type D and 42 as non-Type D. All participants were recruited via online and in-house advertising within the Northumbria University campus. Exclusion criteria included history of mental illness, any chronic immune related illnesses, active infections, haemophilia or any blood-related disorders, oral diseases (e.g. gingivitis) or lesions in the oral cavity, resting blood pressure higher than 140/90, current use of steroidal medication or beta-blockers (e.g. propranolol), risk of HIV, hepatitis the Human T - lymphotropic virus or syphilis or had ever had breast cancer and/or a mastectomy.

Participants were asked to refrain from smoking (as smoking can increase blood pressure and heart rate) or consuming any food or drink other than water for 30 minutes prior to attendance at the testing session so as to not influence the sympathetic indices (i.e. alpha amylase and cardiovascular function) and to avoid contamination of saliva samples. It was also required that participants refrained from consuming alcohol for 12 hours, and caffeine or taking part in physical activity for 2 hours prior to attendance at the testing session.

7.2.2 Materials and procedure

7.2.2.1 Standard self-report measures

Participants completed a number of in-house demographic questions to gauge age, gender, occupational status, ethnicity, BMI, menopausal status and smoking status (See Appendix A). The DS14 (Denollet, 2005) was used to assess Type D personality. The CHIPS (Cohen et al., 1993) measure of physical symptoms was also administered, as well as the PSS (Cohen et al., 1983) to measure perceived stress and the HADs (Zigmond & Snaith, 1983) to assess psychological distress as potential outcomes. The STAI-SF (Cohen et al., 1995) was administered to assess state anxiety both pre and post task. Full details of all questionnaires

are described in Chapter 2.

The National Aeronautics and Space Administration Task Load Index (NASA-TLX; Hart & Staveland, 1988) was used as a measure of perceived workload after completion of the Multitasking Framework. The scale includes six items measuring 'mental demand', 'physical demand', 'temporal demand', 'effort', 'performance' and 'frustration'. Participants had to respond using a visual analogue scale (VAS) by placing a mark at the relevant point between 'low' and 'high' on a 100mm line. Each individual item produced a score based on the length (in mm) between the left end of the line and the point at which the participant placed a cross on this line. Higher scores indicate higher levels of perceived workload.

All questionnaires were paper-and-pencil based.

7.2.2.2 Multitasking Framework

The Multitasking Framework (Purple Research Solutions) (see Wetherell & Sidgreaves, 2005) was used as the acute laboratory- based stress paradigm. The Multitasking Framework is a computerised program which induces stress by requiring participants to attend to multiple tasks simultaneously. The framework set-up comprises four modules which can be chosen from a selection of eight, and can be set to varying workload intensities. The tasks are designed to access different cognitive domains including memory, auditory response, visual recognition, mathematical ability and digit vigilance and all have an element of time pressure and / or complexity. Each module is scored for successful completion and negatively scored for incorrect or non-completion. The participant's total score is displayed in the centre of the screen throughout the task. The modules used in this study were mail alert (*top left*), memory search (*top right*), bar tracker (*bottom left*) and telephone number entry (*bottom right*). See Figure 7.1 for screen grab of the four modules used. All modules were set as 'medium' intensity, with the exception of 'Letter Search' which was set as 'high' intensity. This Framework configuration was designed to induce high levels of stress, without being so difficult that participants disengaged. All participants were considered to appropriately engage with all tasks by the researcher.

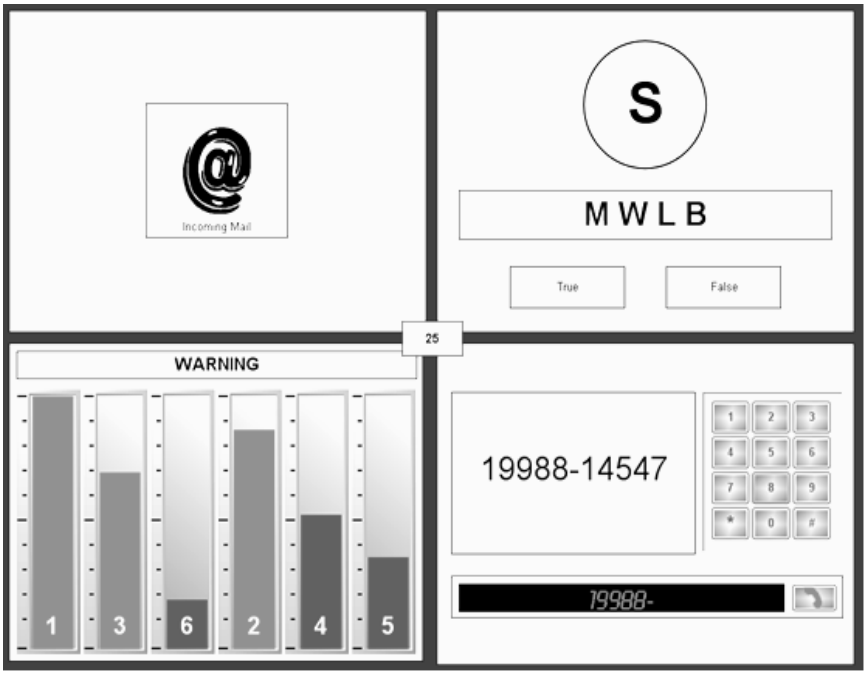

Figure 7.1. Screen grab of the Multitasking Framework showing the four modules that were presented to participants in the current study.

7.2.2.3 Additional social evaluation

Throughout the testing period the participants were exposed to additional social evaluation at regular intervals provided by a researcher seated behind the participant, similarly to previous work (Wetherell et al., 2017). This was in the form of scripted statements providing negative verbal feedback on the participant's performance of the task. The following statements were used:

"Remember you must be as fast & accurate as you can on all of the tasks"

"Your score is a bit on the low side, you need to speed up"

"You should really be working faster"

"Your score is still below the average"

"You only have 2 minutes remaining and you must get as high a score as you can"

7.2.2.4 Saliva Sampling

Saliva sampling was conducted using the passive drool technique. This technique is generally considered the gold standard in saliva sampling for analytes where concentration is

dependent upon saliva flow (Beltzer et al., 2010) as it provides samples of whole unstimulated saliva.

It is currently uncertain whether sAA levels may be affected by saliva flow rate, given that the parasympathetic system increases saliva flow rate in response to stress (Proctor & Carpenter, 2007). Rohleder, Wolf, Maldonado and Kirschbaum, (2006) suggest that levels of alpha amylase are independent of saliva flow rate; however, Beltzer et al. (2010) argues that the enzyme is flow rate dependent. On this basis, in the present thesis, sAA levels are corrected for saliva flow rate, and both sAA concentration and sAA output (saliva flow rate corrected) are considered, in line with guidance provided by assay supplier Salimetics.

7.2.2.4.1 Passive drool procedure

Participants were instructed to tilt their head forward, allowing saliva to pool in the bottom of their mouth, and then to pass the saliva through the SalivaBio Collection Aid (SCA) into a 4ml polypropylene vial continuously for 2 minutes per sample. Samples were taken at baseline, pre-stressor, immediately post stressor, 10 min and 20 min post stressor. As alpha amylase has a diurnal profile in which levels are lower in the morning and steadily rise throughout the day (O'Donnell, Kammerer, O'Reilly, Taylor & Glover, 2009), testing for this study always took place in the afternoon (2pm).

Each saliva sample was labelled accordingly by participant number and time point. Samples were firstly stored at -20C and transferred to -80C within 24 hours. Samples underwent a single freeze-thaw cycle prior to assay. Stability of sAA has been observed for up to five freeze-thaw cycles, so this did not diminish the enzyme concentration (O'Donnell et al., 2009). On day of assay, the saliva samples were completely thawed and centrifuged at 1500 × g for 15 minutes to remove mucins.

7.2.2.4.2. Estimation of volume and saliva flow rate

Sample weights (g; grams) were computed by subtracting the empty cryovial weight from the observed total weight (cryovial + sample). Sample weights (g) were converted to estimated volumes (mL). As sAA is theorised to be saliva flow dependent, saliva flow rate

(mL/min) was computed by dividing the sample volume by the sample collection time (2 minutes). To compute sAA output in U/min after assay, untransformed sAA concentration (U/mL) was multiplied by flow rate (mL/min).

7.2.2.4.3 Determination of salivary alpha amylase

Assays were conducted in-house by a trained technician using a kinetic enzyme assay kit provided by Salimetrics. This method utilised a chromogenic substrate, 2-chloro-p-nitrophenol, linked to maltotriose. The enzymatic action of sAA on this substrate yields 2-chloro-p-nitrophenol, which can be spectrophotometrically measured at 405 nm using a standard laboratory plate reader. The amount of sAA present in the sample was directly proportional to the increase (over a 2-min period) in absorbance at 405 nm. The reaction was read in a 96-well microliter plate with controls provided and sAA concentration (U/mL) was computed using the following formula: [Absorbance difference per minute × total assay volume (0.328 mL) × dilution factor (200)]/ [millimolar absorptivity of 2-chloro-p-nitrophenol (12.9) × sample volume (.008 mL) × light path (.97)].

All samples were assayed in duplicate. Intra-assay variation (CV) was computed for the mean of duplicate samples and those with a CV above 15% excluded from analyses. This resulted in 56 participants providing full sAA data (31 non-Type D, 25 Type D). In all analyses involving sAA concentration (U/mL) and sAA output (U/min), data was log-transformed due to positive skew.

7.2.3 Cardiovascular Measurements

7.2.3.1 Physiological monitoring equipment

Continuous ambulatory measurements of cardiovascular function were recorded using the FMS Portapres and BeatScope 1.0 software (TNO, Biomedical Instrumentation Research Unit, Amsterdam, the Netherlands). The Portapres is a portable Finometer which enables non-invasive blood pressure measurement and beat to beat haemodynamic monitoring. The technique uses a finger cuff which the participant wears on the third finger of the non-

dominant hand, which continuously monitors finger arterial pressure throughout the session. The BeatScope software package is used to analyse arterial pressure waveforms and enables the application of filters to correct for pressure wave distortion when the arterial pressure is acquired from a peripheral site (i.e. a finger artery).

7.2.3.2 Cardiovascular parameters

Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) total peripheral resistance (TPR), cardiac output (CO), stroke volume (SV), interbeat interval (IBI), and ejection fraction (EJT) were measured throughout the testing session. Heart rate variability (HRV) was calculated from the standard deviations of inter beat intervals (IBI), and haemodynamic profile was examined by looking specifically at the reciprocal relationship between TPR and CO. This could enable identification of response patterns as: 'myocardial' (an increase in CO with an insufficient decrease in TPR), 'vascular' (an increase in TPR with an insufficient decrease in CO) or 'mixed' (synergistic increase or decrease in CO and TPR).

7.2.3.3 Time blocks and averaging data

Cardiovascular measurements were recorded for approximately 45 minutes per participant. Recording of continuous beat to beat data points for all 8 outputs began once the beat interval between physiological calibrations (physiocals) reached 30; indicating adequate reliability of data. Throughout the testing session event markers were entered into BeatScope to signify the following time blocks; baseline (10 minutes), task-practice (2 minutes), stressor (20 minutes) and recovery (10 minutes). After testing the data was then split by time block using a script written in Python 2.7 applying the Panda Data Frame module.

7.2.4 Blood samples

7.2.4.1 Phlebotomy technique

Intravenous blood samples were obtained by a trained phlebotomist using a 5.0 mL gold topped vacutainer (serum separator tube). After collection the tube was inverted 5 times before the blood was allowed to clot at room temperature for 30 minutes. This was then centrifuged

for 15 minutes. Serum was then extracted, transferred to a 1.0mL polyethene tube, labelled by participant number and stored at –80°C in anticipation of further processing. Samples were fully thawed to room temperature on day of assay. Blood samples were obtained from 55 out of the 75 participants (30 non-Type D, 25 Type D).

7.2.4.2 Determination of C - reactive protein

Assays were conducted by an in-house technician using Abcam's C-reactive protein (CRP) SimpleStep Enzyme-Linked Immunosorbent Assay (ELISA) kit. This Kit is a sandwich assay for the determination of CRP in serum. The method employed a specific antibody complex which firstly immunocaptured the CRP in solution. A Tetramethylbenzidine (TMB) substrate was then added and during incubation was catalysed by Horseradish Peroxidase **(**HRP), generating a blue colour. An acidic stopping solution was then added to convert the colour to yellow. The colour intensity of the yellow is directly proportional to the concentration of CRP in the sample. A dose response curve of the concentration vs. absorbance (at 450 nm) unit was produced and the amount of CRP present in the sample (mg/L), was determined from the calibration curve. Values below sensitivity (detection level) were raised to sensitivity value.

7.2.5 Procedure

The study received full ethical approval from the institutional ethics committee. Participants attended a single 2 hour testing session in the laboratory. On arrival at the laboratory participants were taken to a quiet private room to relax in a comfortable arm chair for a total of 30 minutes in order to achieve a true resting state. In this time the psychological questionnaires were completed. The Portapres finger cuff was then attached to participants and baseline cardiovascular measures were taken. Participants received verbal task instructions and given a 2-minute practice of the MTF before completing the task for 20 minutes. Throughout the task the participant received negative verbal feedback on aspects of their speed and performance. Once the task was completed a further 10 minutes of measurement were taken. Throughout the testing session, 5 saliva samples were obtained as

follows: baseline, pre-task, post-task, + 10 minutes (post-task) and +20minutes (post-task). The participant was then accompanied to the clinical centre where a trained phlebotomist took an intravenous blood sample. The blood sample was obtained after stress exposure so that the stress of obtaining a sample did not interfere with the other stress reactivity parameters under investigation. The full protocol is demonstrated in figure 7.2.

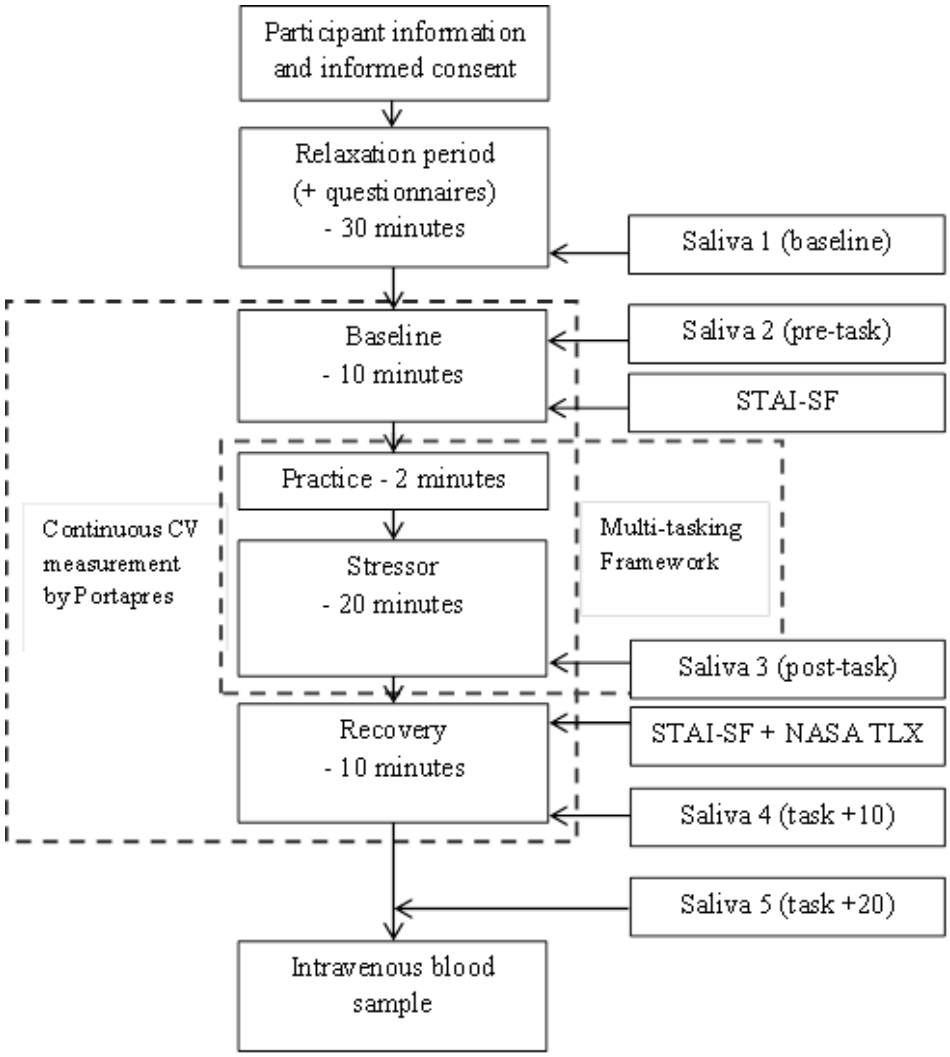

Figure 7.2. Full experimental protocol.

7.2.6 Treatment of data

Using the categorical approach, independent samples t-tests were conducted to assess differences between Type D and non-Type D individuals on self-reported physical symptoms, perceived stress, perceived stress reactivity, anxiety, depression, perceived workload and pre and post task state anxiety. Correlations were also conducted between the continuous Type D

scores and these outcomes.

Utilising the categorical approach, a number of 2 × 4 mixed factorial ANOVAs were run to assess the changes in each of the cardiovascular parameters across the testing period. The within subjects factor was timeframe (baseline, practice, stressor, and recovery) and Type D category was the between subjects factor. To assess cardiovascular function using the dimensional approach to Type D, a reactivity score (peak value minus baseline value) was calculated for each parameter and hierarchical multiple regression analyses were conducted to examine the predictive contribution of NA, SI (step 1) and Type D (step 2) to these outcomes.

Heart rate variability (HRV) was calculated by taking the standard deviations of each interbeat interval within each timeframe (which indicated the variability of the space in between heart beats) for each participant. A 2 × 4 ANOVA was conducted on these data to assess for any changes in HRV across the testing session.

Haemodynamic profile (HP; the relationship between CO and TPR) and compensation deficit (CD) scores were computed for each participant using the equations proposed by Gregg et al., (2002) and James et al., (2012). Firstly, singular HP and CD scores were computed with regards to the changes (log transformed) in mean TPR and CO scores between the baseline and task phases. The equation used to calculate HP was log(CO)r + log(TPR)r = log(MAP)r, (r = ratio of task to baseline values) and the orthogonal relationship between HP and CD was achieved by a 45º rotation of the two-dimensional space formed by the CO and TPR reactivity dimensions (see figure 6.5). Independent samples T-tests were conducted to assess the differences between Type D and non-Type D individuals on these HP and CD scores. Hierarchical multiple regression analyses were also undertaken to assess the dimensional predictive value of NA, SI and Type D on these scores.

A further number of HP and CD scores were also computed for the haemodynamic (TPR and CO) changes between baseline and practice (T1), baseline and task (T2) and baseline and recovery (T3). Two 2 × 3 mixed factorial ANOVAs were then conducted to assess the change in these HP and CD scores across the phases of the testing session, between Type D categories. The within subjects factors were the HP and CD scores with three levels; T1, T2

and T3, and the between subjects factor was Type D category with two levels; Type D and Non Type D. *p*=

For the salivary alpha amylase data, two 2 × 5 mixed factorial ANOVAs were conducted for sAA concentration, and sAA output. Again, the time point (baseline, pre-stress, post stress, +10minutes, and + 20 minutes) was the within subjects factor, and Type D category was the between subjects factor. A reactivity score was also calculated (individual peak stress value minus baseline value) for sAA output and sAA concentration and hierarchical regression analyses were conducted to assess the influence of NA, SI and Type D as previously described (See Chapter 2 for further details).

For the CRP analyses, an independent samples t-test was conducted to assess whether levels of CRP differed between Type Ds and non-Type Ds. Correlational analyses were also conducted between Type D score and CRP levels, and finally hierarchical regression analyses were also conducted in order to control for NA and SI (as above).

7.3 Results

7.3.1 Self-report measures

As can be observed in table 1, independent samples t tests indicated significant differences between Type D and non- Type D individuals on self-report measures of anxiety, depression, perceived stress, perceived stress reactivity, reactivity to social evaluation (RSE), reactivity to failure (RFa), and prolonged reactivity (PrR) in the expected directions. A significant difference was observed only on the metabolic symptoms on the CHIPS with Type Ds reporting more metabolic symptoms. A significant difference was also observed between Type D and non-Type Ds on pre-task state anxiety, and perceived physical workload. No significant differences were observed between groups on any of the other measures. T-test values and group means are reported in table 7.1 and 7.2.

Table 7.1. Independent t-tests showing differences in symptoms, anxiety, depression, perceived stress and stress reactivity between Type D groups (Mean [±SD]).

Measures	Non-Type D	Type D	*t*	*df*	*p*	*d*
Physical symptoms	12.95[14.41]	13.70[10.42]	-.250	73	.803	.058
Cardiac/sympathetic	1.14[2.70]	1.73[2.86]	-.906	73	.368	.212
Muscular	2.07[3.98]	2.03[2.28]	.053	73	.958	.012
Metabolic	3.62[2.85]	5.27[3.74]	-2.177	73	.033	.509
Gastrointestinal	1.62[2.84]	1.12[1.88]	.839	73	.404	.196
Vasovagal	0.90[1.83]	0.94[1.74]	-.088	73	.930	.021
Cold	1.95[1.99]	1.36[1.75]	1.342	73	.184	.314
Headache	0.86[1.20]	1.09[1.52]	-.742	73	.460	.174
Haemorrhagic	0.33[0.85]	0.48[8.85]	-.760	73	.450	.178
Anxiety	5.71 [3.07]	8.97 [3.92]	-4.036	73	<.001	.945
Depression	2.02 [1.51]	4.18 [3.12]	-3.944	73	.001	.923
Perceived Stress	14.38 [5.55]	19.15 [7.72]	-3.110	73	.003	.728
Perceived Stress Reactivity	41.62 [9.08]	48.03[8.75]	-3.098	73	.003	.725
Prolonged Reactivity	6.50[1.74]	7.45[2.00]	-2.206	73	.031	.516
Reactivity Social Conflict	10.00 [2.49]	11.36[2.95]	-1.855	72	.068	.437
Reactivity Failure	7.67 [2.07]	8.94[2.19]	-2.295	72	.025	.541
Reactivity Social Evaluation	8.69 [2.43]	10.36 [2.41]	-2.968	73	.004	.695
Reactivity Work Overload	8.76 [2.66]	9.91[2.72]	-1.837	73	.070	.430

Table 7.2. Independent t-tests showing differences in perceived workload and pre and post state anxiety between Type D groups (Mean [±SD]).

Measures	Non-Type D	Type D	t	df	p	*d*
Workload	326.55[76.76]	326.45[71.87]	.005	73	.996	.001
Mental	62.93[23.61]	65.73[21.88]	-.526	73	.600	.123
Physical	27.83[20.40]	18.79[15.61]	2.107	73	.039	.493
Temporal	68.29[17.77]	68.03[20.98]	.057	73	.955	.013
Effort	64.76[21.02]	66.48[21.16]	-.351	73	.726	.082
Performance	57.14[20.31]	58.94[19.06]	-.391	73	.697	.092
Frustration	45.60[23.45]	48.48[25.04]	-.514	73	.609	.120
Pre-Anxiety	8.36[2.09]	9.94[2.42]	-2.769	72	.007	.653
Post-Anxiety	11.43(2.55)	13.12[3.93]	-1.931	72	.057	.455

A 2x2 repeated measures ANOVA was also conducted on the state anxiety measures. Wilk's lambda indicated a significant main effect of time; $F(1,71) = 114.41$, $p<.001$, $\eta p^2=.617$, indicating that post task anxiety was significantly greater than pre-task state anxiety levels. A significant main effect of Type D was also observed; $F(1,71)=6.028$, $p=.017$, $\eta p^2=.078$, but no significant interaction effect ; $F(1,71) = .296$, $p=.588$, $\eta p^2=.004$.

Correlational analyses partially supported the categorical findings. Weak to moderate positive correlations were observed between Type D (NA × SI) score and scores for anxiety, depression, perceived stress, perceived stress reactivity (including all subscales of the PSRS), and both pre and post state anxiety. Positive correlations were also observed between Type D and metabolic symptoms, in addition to cardiac/sympathetic symptoms and headaches. Correlation coefficients and mean scores for each measure from the full sample can be observed in table 7.3.

Table 7.3. Correlations between total Type D scores and the self-report outcome measures.

Measures	Type D	*M*	*SD*
Physical symptoms	.180	13.28	12.73
Cardiac/sympathetic	.288*	1.40	2.77
Muscular	.053	2.05	3.32
Metabolic	.370**	4.35	3.35
Gastrointestinal	.010	1.40	2.55
Vasovagal	.169	0.92	1.67
Cold	-.073	1.69	1.90
Headache	.299**	0.96	1.35
Haemorrhagic	.085	0.40	0.85
Anxiety	.661**	7.15	3.81
Depression	.595**	2.97	2.57
Perceived Stress	.621**	16.48	6.96
Perceived Stress Reactivity	.556**	44.47	9.49
Prolonged Reactivity	.383**	6.92	1.91
Reactivity Social Conflict	.375**	10.47	2.55
Reactivity Failure	.391**	8.14	2.07
Reactivity Social Evaluation	.518**	9.43	2.55
Reactivity Work Overload	.407**	9.27	2.73
Workload	.040	326.51	74.15
Pre-Anxiety	.424**	8.95	2.19
Post-Anxiety	.367**	11.95	2.69

*p<0.05; **p<0.01

Hierarchical multiple regression analyses were also conducted for each of the self-reported outcomes. Type D personality was found to significantly predict depression when added to the final model at step 2 (β=.747, p=.036, R^2=.376, ΔR^2=.041*) and gastrointestinal symptoms (β=.953, p=.028, R^2=.085, ΔR^2=.066*).

NA was the only significant predictor in the final model (step 2), for the following outcomes; anxiety (β=.438, p=.022, R^2=.499), metabolic symptoms (β=.609, p=.009 R^2=.264), perceived stress (β=.415, p=.037, R^2=.451), perceived stress reactivity (β=.592, p=.006, R^2=.347).

However, the models for the remaining outcomes (CHIPS, cardiac symptoms, vasovagal symptoms, cold symptoms, muscular symptoms, headache, haemorrhagic symptoms, pre and post state anxiety and workload) were not significant at either step 1 or step 2.

7.3.2 Cardiovascular function.

7.3.2.1 Systolic Blood Pressure

In analyses of SBP (mmHg), Mauchly's test indicated that the assumptions of sphericity had been violated X^2 (5) = 15.56, p=.008 and ε >.75, therefore Greenhouse Geisser corrected values were used. Firstly, a main effect of time; F (2.665, 189.210) = 43.891, p<.001, ηp^2=.382, was observed, and pairwise comparisons with Bonferroni corrections indicated that the SBP values at each time point differed significantly from one another (p <.05) with the exception of practice and recovery (p =1.00). A main effect of Type D; F (1,71) = 4.171, p=.045, ηp^2=.055, on SBP was also observed. Type D individuals exhibited overall lower SBP than non-Type D individuals. There was no significant interaction effect evident; F (2.665, 189.210) = 0.584, p=.606, ηp^2=.008. Figure 7.3 indicates the changes in SBP measures over the time points for Type D and non- Type D participants.

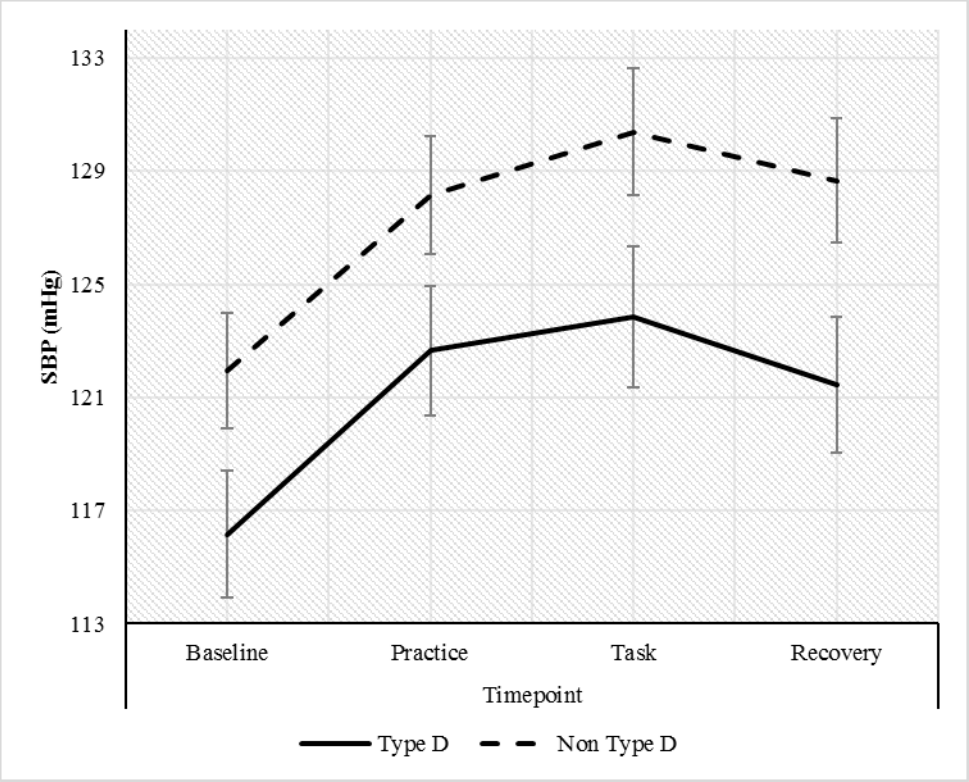

Figure 7.3. Pattern of systolic BP (mmHg) in response to acute stress for Type D and Non Type D individuals (error bars represent standard error).

The regression model for SBP reactivity was non-significant at both step 1 (p=.968) and step 2 (p=.795).

7.3.2.2 Diastolic blood pressure

In analyses of DBP (mmHg), Mauchly's test indicated that the assumptions of sphericity had been violated X^2 (5) = 21.168, p=.001 and ε >.75, therefore Greenhouse Geisser corrected values were used. Firstly, a main effect of time F (2.477,175.835) = 47.867, p<.001, ηp^2=.403, was observed. Pairwise comparisons indicated that the DBP values at each time point differed significantly from one another (p< .05) with the exception of practice and recovery (p =1.00) and; task and recovery (p =.204). A main effect of Type D category; F (1,71) = 7.212, p=.009, ηp^2=.092, on DBP was also observed. Type D individuals also exhibited lower overall DBP in comparison to non-Type D individuals, however, no significant interaction effect was evident; F (2.477,175.835) = 1.862, p=.148, ηp^2=.026. Figure 7.4 demonstrates the changes in DBP over the time points for Type D and non-Type Ds.

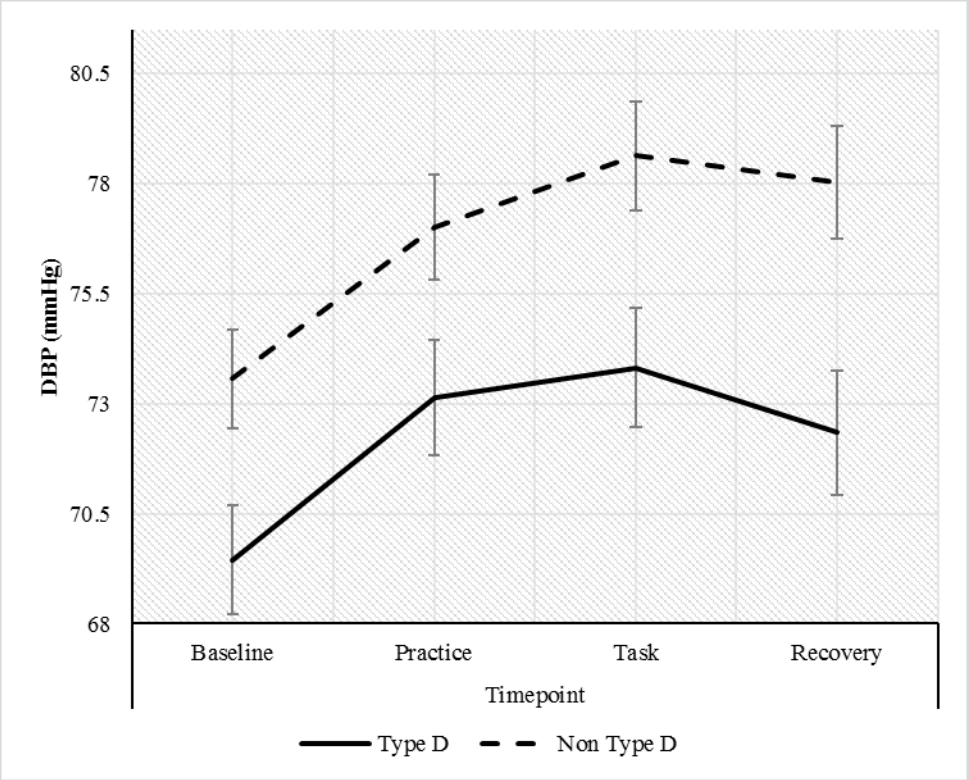

Figure 7.4. Pattern of diastolic BP (mmHg) in response to stress for Type D and non-Type D individuals (error bars represent standard error).

The regression model for peak DBP reactivity was also non-significant at step 1 (p=.503) and step 2 (p=.663).

7.3.2.3 Heart Rate

Analyses for HR (bpm), Mauchly's test indicated that the assumptions of sphericity had been violated X^2 (5) = 41.984, p<.001 and ε >.75, therefore Greenhouse Geisser corrected values were used. The mixed ANOVA showed there were no significant main effects of either time; F (2.208,161.167) = 1.407, p=.247, ηp^2=.019 or Type D; F (1,73) = 0.344, p=.559, ηp^2=.005 on HR. There was also no significant interaction effect; F (2.208,161.167) = 1.036, p=.363, ηp^2=.014.

The regression model at step 1 was found to be significant; F (2,69) =3.240, p=.045. However, NA and SI were found to not significantly predict HR reactivity at either step 1 or step 2, and Type D did not contribute the model at step 2.

7.3.2.4 Cardiac Output

In the analyses for CO (L/min), Mauchly's test indicated that the assumptions of sphericity were violated X^2 (5) = 41.947, p<.001 and ε >.75, therefore Greenhouse Geisser corrected values were used. The mixed ANOVA showed there was a significant main effect of time; F (2.371,173.12) = 4.497, p=.008, ηp^2=.058.

which can be observed in figure 7.5. Pairwise comparisons with Bonferroni corrections indicated that the only significant difference was between task and recovery (p=.012). The main effect of Type D category on CO was not significant; F (1,73) = 0.126, p=.723, ηp^2=.002, and there was no significant interaction effect; F (2.371,173.12) = 2.000, p=.130, ηp^2=.027.

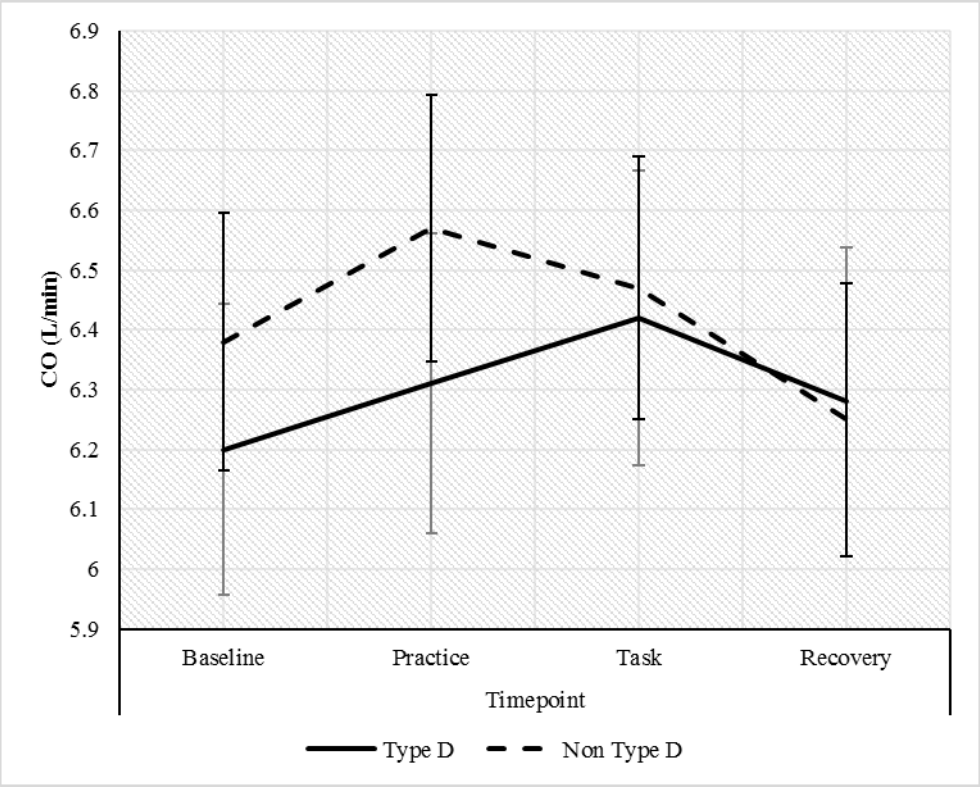

Figure 7.5. Patterns of CO (L/min) in response to acute stress in Type D and non-Type D individuals (error bars represent standard error).

The regression model containing peak CO reactivity was non-significant at both step 1 (p=.200) and step 2 (p=.357).

7.3.2.5 Interbeat interval

In the analysis of IBI (s), Mauchly's test indicated that the assumptions of sphericity

had been violated; X^2 (5) = 50.294, p<.001 and ε <.75, therefore Huynh-Feldt corrected values were used. There was no significant main effect of time; F (2.121,154.8) = 2.003, p=.136, ηp^2=.027, or Type D category; F (1,73) = 0.190, p=.664, ηp^2=.003 or interaction effect; F (2.121,154.8) = 0.823, p=.447, ηp^2=.011.

The regression model for IBI reactivity was again non-significant again at both step1 (p=.141) and step2 (p=.245).

7.3.2.6 Total Peripheral Resistance

For TPR (mmHg/min/mL-1), Mauchly's test indicated that the assumptions of sphericity had been violated X^2 (5) = 58.861, p<.001 and ε <.75, therefore Huynh-Feldt corrected values were used. The mixed ANOVA showed there was a significant main effect of time; F (1.988, 139.168) = 8.463, p<.001, ηp^2=.108. Pairwise comparisons with Bonferroni corrections showed that the differences lay between baseline and stressor (p=.003) and; baseline and recovery (p=.002).

There was no significant main effect of Type D on TPR; F (1,73) = 0.001, p=.977, ηp^2<.001 However, there was a significant interaction effect; F (1.988, 139.168) = 8.463, p=.044, ηp^2=.044. To assess the underlying differences driving the interaction, separate one-way repeated measures ANOVAs were conducted for i) the non-Type D group, and ii) the Type D group.

In the non-Type D group, Wilks' lambda indicated that there was a significant TPR change across the four time points; F (3,36) = 4.782, p=.007, ηp^2=.285. Pairwise comparisons with Bonferroni corrections showed the differences lay between; baseline and recovery (p=.018) and; practice and recovery (p=.027). In the Type D group, Wilks' lambda indicated that TPR change across the four time points was also significant; F (3,30) = 3.164, p=.039, ηp^2=.240. Pairwise comparisons with Bonferroni corrections showed the differences lay between the baseline and recovery (p=.048) time points in isolation.

Further, independent samples t-tests were also conducted between Type D and non-Type Ds TPR measures at each time point (p values were adjusted to account for multiple

groups), however no significant differences were observed (ps<.05). The patterns of TPR across the four time points in Type D and non-Type D individuals can be observed in figure 7.6.

The regression analysis indicated that the model for TPR reactivity was not significant at either step 1 (p=.470) or step 2 (p=497).

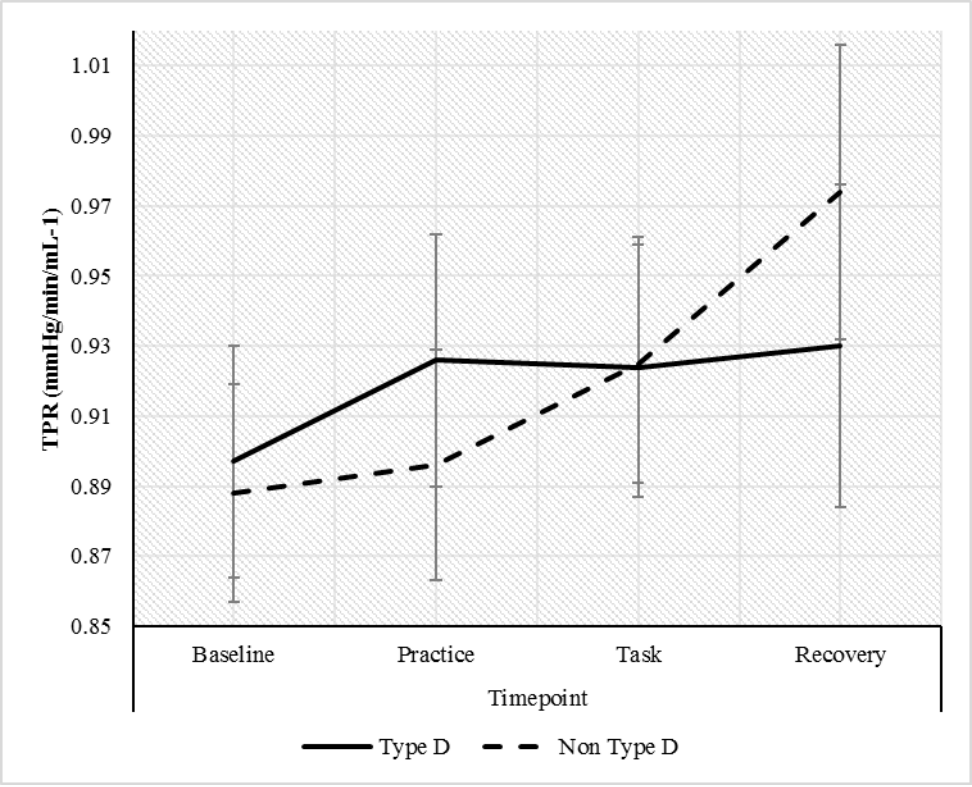

Figure 7.6. Patterns of TPR (mmHg g/min/mL) in response to acute stress in Type D and non-Type D individuals (error bars represent standard error).

7.3.2.7 Stroke volume

For SV(mL), Mauchly's test indicated that the assumptions of sphericity had been violated X^2 (5) = 43.521, p<.001 and ε >.75, therefore Greenhouse Geisser corrected values were used. The mixed ANOVA showed there was a significant main effect of time; F (2.281,166.547) = 8.739, p<.001, ηp^2=.107. Pairwise comparisons with Bonferroni corrections indicated the differences lay between every time point, with the exception of; baseline and recovery (p=1.00) and; practice and stressor (p=1.00). There was no significant main effect of

Type D; F (1,73) = 0.003, p=.955, ηp^2<.001 on SV. However, there was a significant interaction effect; F (2.281,166.547) = 3.915, p=.017, ηp^2=.051. To assess the underlying differences driving this interaction, separate one-way repeated measures ANOVAs were conducted for i) the non-Type D group, and ii) the Type D group.

In the non-Type D group, Wilks' lambda indicated that there was a significant change in SV across the four time points; F (3,36) = 7.192, p=.001, ηp^2=.356. Pairwise comparisons with Bonferroni corrections showed the differences lay between; baseline and practice (p=.008); practice and recovery (p=.006); and stressor and recovery (p=.038).

In the Type D group, Wilks' lambda indicated that SV also significantly changed across the four time points; F (3,30) = 5.685, p=.003, ηp^2=.362. Pairwise comparisons with Bonferroni corrections showed the differences lay between baseline and practice (p= .007), and baseline and stressor (p=.001).

Further, independent samples t-tests were also conducted between Type D and non-Type Ds SV measures at each time point (p values were adjusted to account for multiple groups), however no significant differences were observed (ps>.05).

The change in SV over the four time points for Type D and Non-type participants can be observed in figure 7.7.

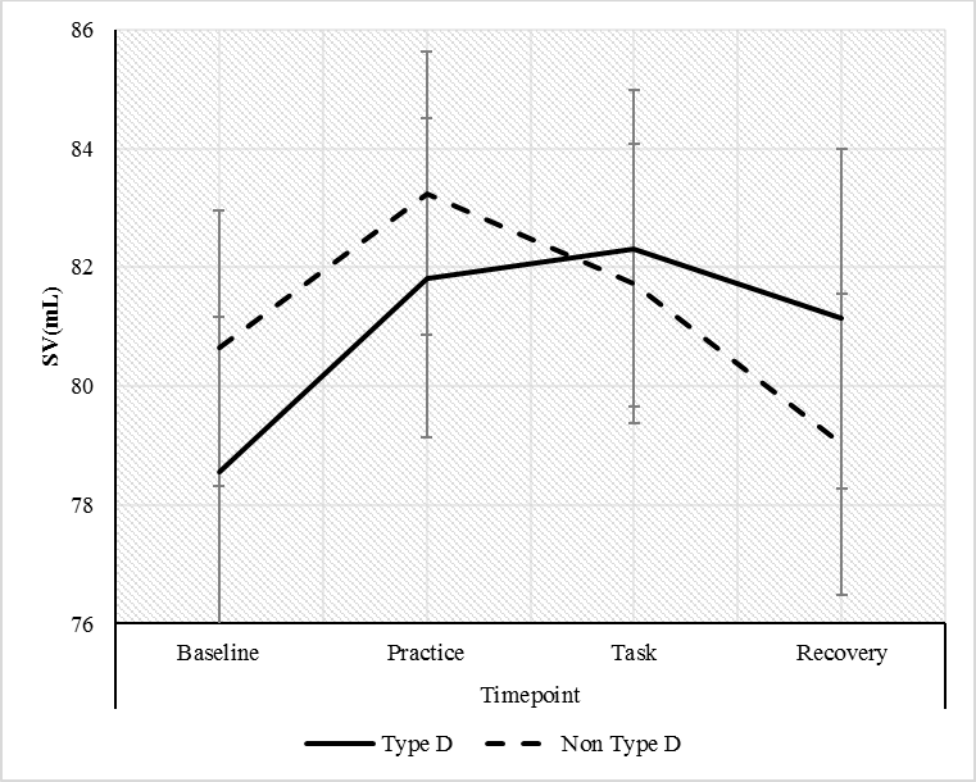

Figure 7.7. Patterns of SV (mL) in response to acute stress in Type D and non-Type D individuals (error bars represent standard error)

The regression model indicated that SI was a significant predictor of SV reactivity at Step 1 with the final model predicting a significant 9.8% of the variance; F (2, 69) = 3.732, p =.029. The overall regression model was also significant at step 2; F (3, 68) =4.220, p=.009 and the Type D interaction term significantly predicted an additional 5.9% of variance in SV reactivity. Regression coefficients, R square and R Square Change for the SV reactivity model are shown in table 7.4.

Table 7.4. Hierarchical regression analyses predicting SV reactivity

		B	SE b	B	t	p	R^2 (ΔR^2)
Step 1	SI	.266	.112	.318	2.386	.020	
	NA	-.009	.111	-.010	-.078	.938	.098*
Step 2	SI	-.187	.234	-.223	-.800	.426	
	NA	-.392	.206	-.470	-1.903	.061	
	Type D	.035	.016	.904	2.188	.032	.157(.059*)

*p<0.05; **p<0.01

7.3.2.8 Ejection Fraction

In the analyses for EJT (s), Mauchly's test indicated that the assumptions of sphericity had been violated; X^2 (5) = 30.673, p<.001 and ε >.75, therefore Greenhouse Geisser corrected values were used. The mixed ANOVA showed there was no significant main effect of time; F (2.402, 175.356) = 2.626, p=.065, ηp^2=.035 or Type D; F (1,73) = 0.803, p=.373, ηp^2=.011 on EJT. There was also no evidence of a significant interaction effect; F (2.402, 175.356) =0.400, p=.709, ηp^2=.005.

The regression analysis indicated that the model was not significant at either step 1 (p=.880) or step 2 (p=.940) for EJT reactivity.

7.3.3 Heart rate variability

In the analyses for HRV (standard deviations of interbeat intervals), Mauchly's test indicated that the assumptions of sphericity had been violated; X^2 (5) = 30.852, p<.001 and ε >.75, therefore Greenhouse Geisser corrected values were used. The mixed ANOVA showed there was a significant main effect of time; F (2.271,154.408) = 60.709, p<.001 ηp^2=.472. Pairwise comparisons with Bonferroni corrections demonstrated that all time points significantly differed from each other (ps<.05).

There was no significant main effect of Type D; F (1,68) = 1.770, p=.188, ηp^2=.025 on HRV and the interaction between Type D and time on HRV was non-significant; F (2.271,154.408) =0.712, p=.509, ηp^2=.010.

The regression model containing HRV reactivity indicated that NA and SI were not significant predictors at either step 1 or step 2 and Type D was not a significant predictor in the final model. The change in HRV over time for Type Ds and non-Type Ds can be observed in figure 7.8.

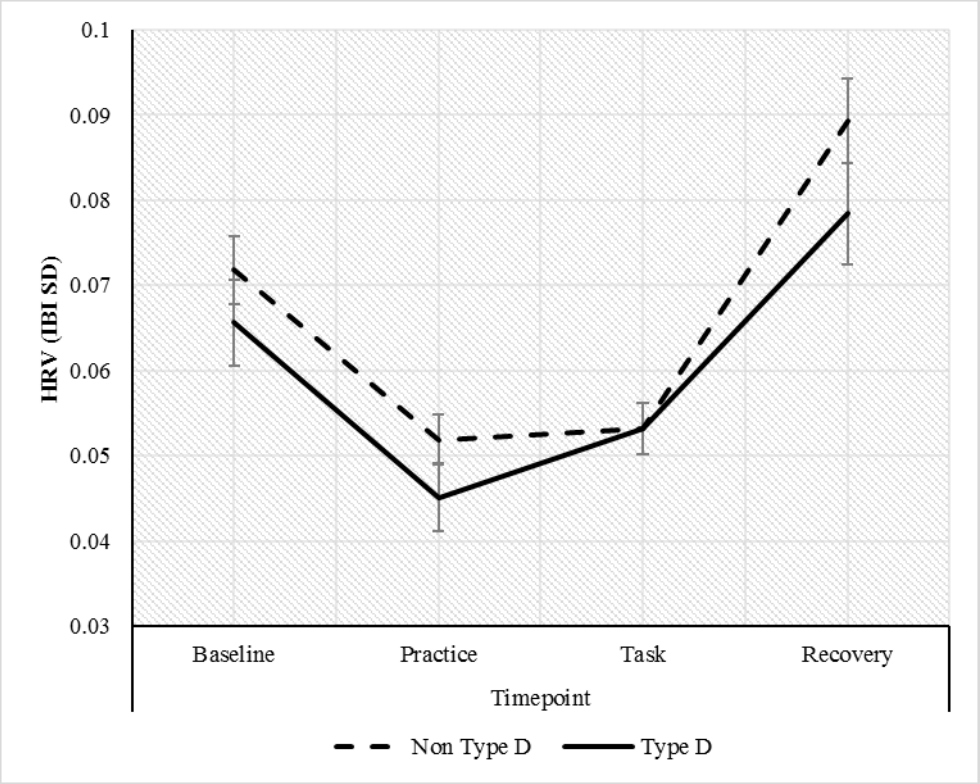

Figure 7.8. Decreases of HRV (IBI SDs) in response to acute stress in Type D and non-Type D individuals (error bars represent standard error).

7.3.4 Haemodynamic Profile and Compensation Deficit

Independent samples t-tests indicated that there were no significant differences between Type D and non-Type D individual's HP (t (70) =.688, p=.494, *d*=.164), and CD (t (70) =-.270, p=.788, *d*=.064) scores. The regression models containing HP and CD indicated that NA and SI were not significant predictors at either step 1 or step 2 and Type D was not a significant predictor of either HP or CD in the final models.

7.3.4.1 Haemodynamic Profile

In the mixed factorial ANOVA analyses for the changes in HP across the session, Mauchly's test indicated that the assumptions of sphericity had been violated X^2 (2) = 29.489, p<.001 and ε >.75, therefore Greenhouse Geisser corrected values were used. There was a significant main effect of time; F (1.484,103.874) = 5.780, p=.009, ηp^2=.063. Pairwise comparisons with Bonferroni corrections indicated the differences lay between T1 (practice-

baseline) and T3 (recovery-baseline) (p=.037), and between T2 (task-baseline) and T3 (p=.022). There was no significant main effect of Type D; F (1,70) = .480, p=.491, ηp^2=.006 on HP. A significant interaction effect was however evident; F (1.484,103.874) = 4.097, p=.030, ηp^2=.055. To assess the underlying differences driving this interaction, separate one-way repeated measures ANOVAs were conducted for i) the non-Type D group, and ii) the Type D group.

In the non-Type D group, Wilks' lambda indicated that there was a significant change in HP scores across the 3 time points; F (2,37) = 4.329, p=.020, ηp^2=.186. Pairwise comparisons with Bonferroni corrections indicated the differences lay between T1 and T3 (p=.015), and between T2 and T3 (p=.047). In the Type D group, Wilks' lambda indicated that HP did not significantly change across the three time points; F (2,31) = 1.496, p=.240, ηp^2=.028. The change in HP scores over the three time points for Type D and Non-type participants can be observed in figure 7.9.

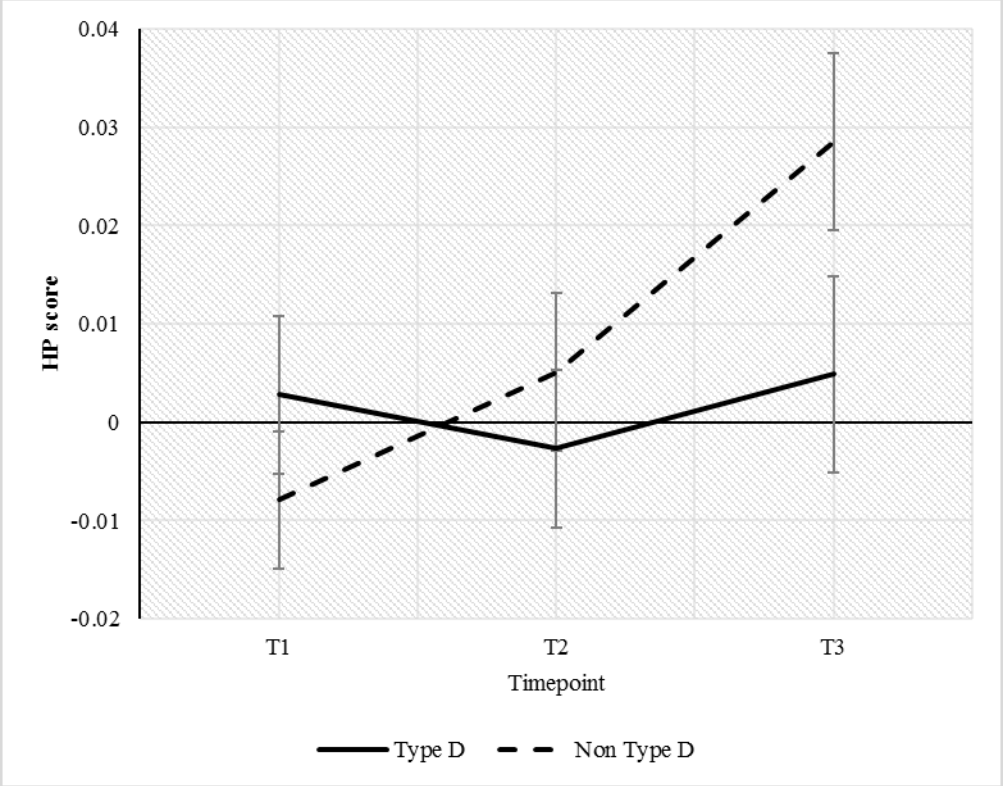

Figure 7.9. Changes in Haemodynamic Profile over testing session in Type D and non-Type D individuals (error bars represent standard error).

7.3.4.2 Compensation Deficit

In the ANOVA analyses for CD Mauchly's test also indicated that the assumptions of sphericity had been violated; X^2 (2) = 16.917, p<.001 and ε >.75, therefore Greenhouse Geisser corrected values were used. Analyses indicated there was a significant main effect of time; F (1.643,114.996) = 3.468, p=.042, ηp^2=.033, and pairwise comparisons with Bonferroni corrections demonstrated that the only significantly different time points were T1 and T2 (p=.002). The change in CD over the time points can be observed in figure 7.10.

There was no significant main effect of Type D; F (1,70) = .018, p=.895, ηp^2=. on CD and the interaction between Type D and time point on CD scores was also non-significant; F (1.643,114.996) =1.910, p=.160, ηp^2=.026.

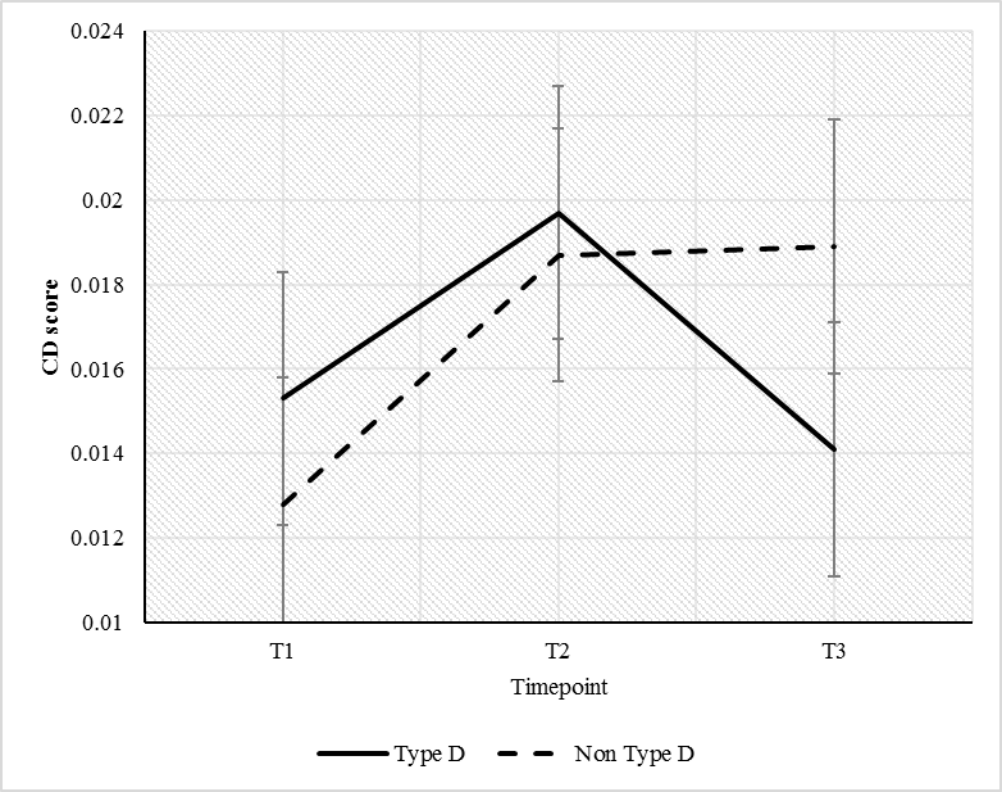

Figure 7.10. Changes in Compensation deficit over testing session in Type D and non-Type D individuals (error bars represent standard error).

7.3.5 Salivary Alpha Amylase

7.3.5.1 sAA concentration

In the analyses for sAA concentration (log -10) Mauchly's test indicated that the assumptions

of sphericity had not been violated; X^2 (9) = 10.921, p=.281, therefore Wilks' Lambda values were used. The mixed ANOVA showed there was a significant main effect of time point; F (4,51) = 5.092, p=.002, ηp^2=.285, and pairwise comparisons with Bonferroni corrections demonstrated that the differences lay between baseline and post-task (p=.005), and between baseline and +20 minutes (p=.014). All other comparisons were not significant.

There was no significant main effect of Type D on sAA concentration; F (1,54) = .539, p=.466, ηp^2=.010 and the interaction effect was also non-significant; F (4, 51) =0.977, p=.428, ηp^2=.071. Figure 7.11 demonstrates the change in sAA concentration over the acute stress period for Type D and Non Type D participants.

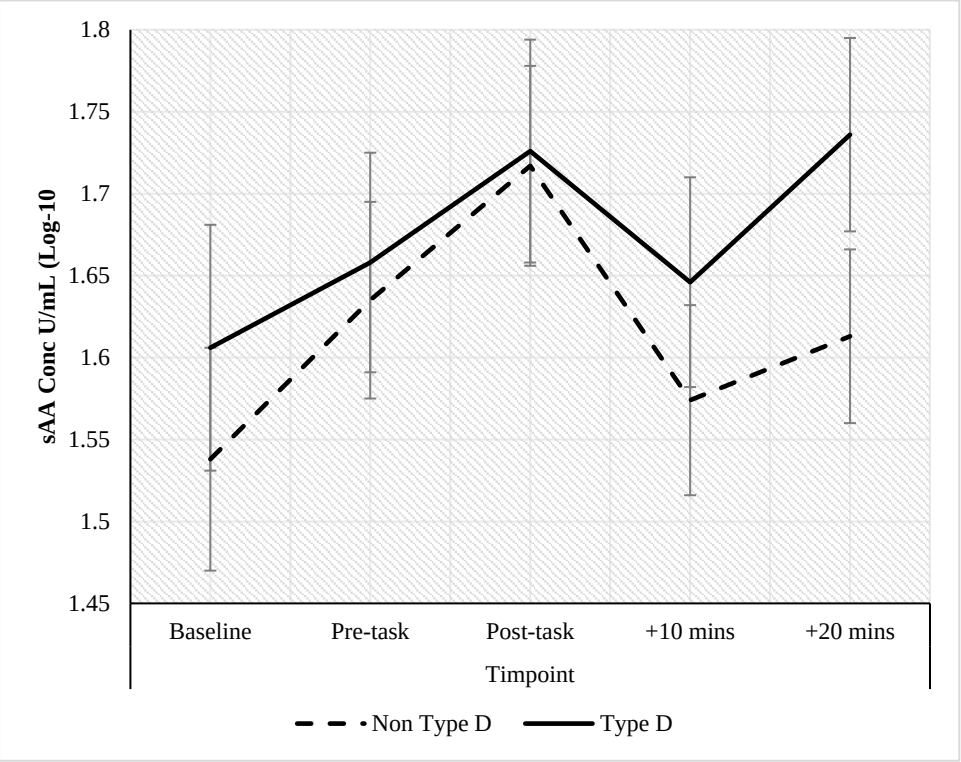

Figure 7.11. Patterns of sAA concentration (U/mL/Log-10) in response to acute stress in Type D and non-Type D individuals (error bars represent standard error).

The regression model containing peak sAA concentration reactivity was non-significant at step1 (p=.150) and step 2 (p=.278).

7.3.6 sAA output

In the analyses for sAA output (log -10) Mauchly's test indicated that the assumptions of sphericity had been violated; X^2 (9) = 30.852, p=.015 and ε >.75, therefore Greenhouse

Geisser corrected values were used. The mixed ANOVA showed there was a significant main effect of time point; F (3.260,176.047) = 20.006, p<.001, ηp^2=.270, and pairwise comparisons with Bonferroni corrections demonstrated that the differences lay between baseline and the all four other time points (ps<.05), between pre-task and post task (p<.001), and between post-task and +10 minutes (p<.001).

Figure 7.12 demonstrates the change in sAA output over the session for Type D and Non Type D participants.

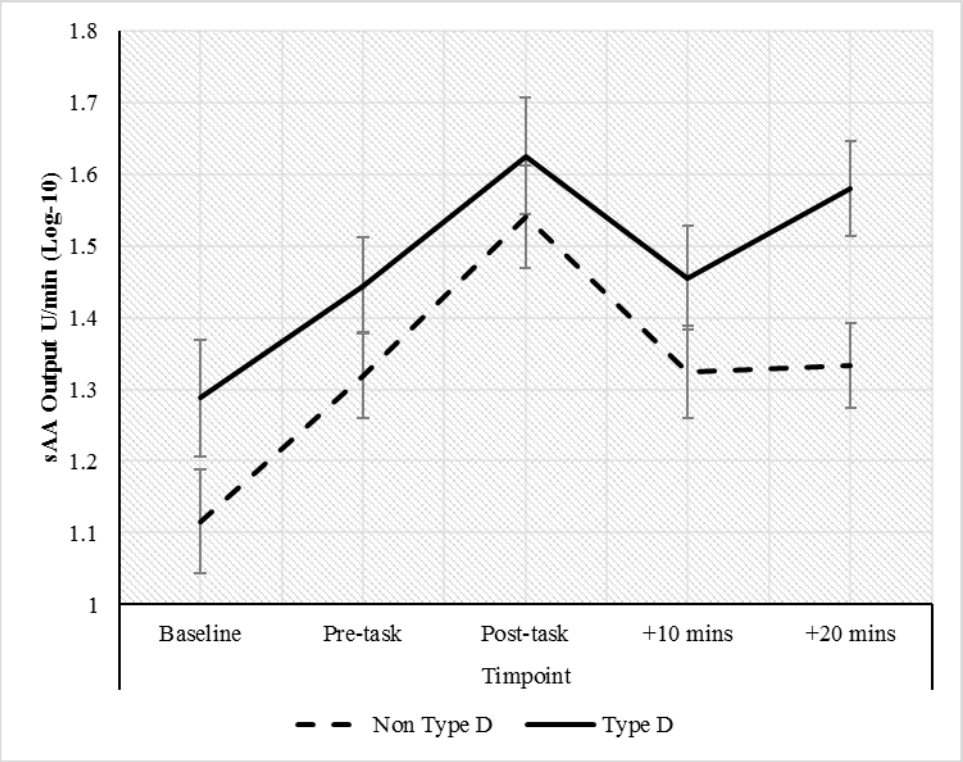

Figure 7.12. Changes in sAA output (U/Min/Log-10) in response to acute stress in Type D and non-Type D individuals (error bars represent standard error).

The main effect of Type D on sAA output was approaching significance; F (1,54) = 3.416, p=.070, ηp^2=.059 and pairwise comparisons with Bonferroni corrections showed that the difference between Type D and non-Type Ds at +20minutes was significant (p=.040). The interaction effect was however, non-significant; F (3.260,176.047) =0.982, p=.407, ηp^2=.018.

The regression model for sAA output reactivity was also non-significant at both steps (step 1 p= .100; step2 p= .120).

7.3.7 C- Reactive Protein

An independent samples t test indicated there was no significant difference between non-Type D (M= -0.985, SD=1.356) and Type D (M= -1.482, SD= 1.441) individuals on measures of CRP (log10); t (53) = 1.316, p=.194, *d*=.362. Correlational analyses also showed there were no significant relationships between CRP and the continuous Type D (SI × NA) scores (r (53) =-.143, p=.340), or the separate NA (r (53) =-.139, p=.313) and SI scores (r (53) =-.163, p=.234). Furthermore, the regression analyses for CRP were not significant at step 1 (p=.651) or step 2 (p=.778).

7.4 Discussion

The primary aim of the current chapter was to examine the relationship between Type D personality and patterns of SAM activity in response to an acute stress. A number of different biomarkers of sympathetic activation were explored; salivary alpha amylase and aspects of cardiovascular function including blood pressure, heart rate, and other haemodynamic parameters. These parameters were also used to examine haemodynamic profile, and a proxy measure of heart rate variability. A secondary aim was to investigate levels of serum CRP as a marker of low level inflammation. Again, analyses were conducted utilising both the categorical and dimensional approach to the construct where appropriate with the aim of elucidating potential psychobiological pathways that may explain the link between Type D personality and the physical symptom clusters identified in Chapters 3 and 4.

7.4.1 Findings

7.4.1.1 Self- report measures

Findings indicated that Type D individuals reported greater frequencies of metabolic symptoms, in comparison to non-Type D individuals; however, there were no other differences in the number of symptoms reported for other symptom clusters. This finding was supported by correlational analyses showing a weak correlation between the continuous Type

D scores and metabolic symptoms. However, there were also weak correlations evident between Type D and cardiac/sympathetic symptoms and headaches. This supports the findings of the initial cross sectional questionnaire based study documented in Chapter 4, however the lack of differences between means may be due to the younger (18-45), more restricted sample (mainly students from the North East) and tighter exclusion criteria employed within this study. The regression analyses did not find that Type D was a significant predictor of any symptom clusters when NA and SI scores were entered into the model, however, the smaller sample size in the current study is potentially insufficient for a reliable regression analyses (Tabachnick & Fidell, 2007).

In terms of the psychological outcome measures, the findings are similar to those in Chapter 4, as Type D individuals reported higher levels of anxiety, depression, perceived stress, perceived stress reactivity (with the exception of RSC and RWO), and state anxiety. These were also supported by the continuous correlational analyses. These findings support previous evidence that Type D personality is linked to increased psychological distress (e.g. Tully & Baker, 2010) and further maintain the theory that increased stress and maladaptive stress reactivity may be involved in the pathway through which Type D individuals are at risk for negative health outcomes.

7.4.1.2 Cardiovascular function

In line with the well documented effects of acute stress on blood pressure (e.g. Vrijkotte, van Doornen & de Geus, 2000; Wetherell & Carter, 2014) in the current study, the Multitasking Framework evoked a similar blood pressure response for all participants. Both SBP and DBP measures increased from baseline to practice and task and then began to reduce in the recovery phase. This was, however, not demonstrated for HR as no differences were observed in this parameters across the session. Interestingly, Type D individuals exhibited lower levels of both SBP and DBP (but not HR) throughout the testing session. This is similar to the findings of Kelly-Hughes et al., (2014) who observed an inverse relationship between Type D personality and blood pressure reactivity to a stressor. However, blood pressure

reactivity did not differ between Type D and non-Type D individuals in the current study. Using the dimensional approach, Type D status did not significantly predict levels of SBP, DBP or HR reactivity as assessed by their peak minus baseline measures, nor did the categorical approach indicate between group differences in reactivity.

A response in SV to the Multitasking Framework was also observed. In all participants SV increased from baseline to practice; however, although SV reduced between the stress phases and recovery in non-Type D individuals, no significant reduction in SV was observed in the Type D group. SV reactivity was also the only parameter significantly predicted by continuous Type D personality in the regression models when SI and NA were controlled for, suggesting that this particular haemodynamic parameter may be directly influenced by Type D personality. However, this finding requires testing in a larger more representative sample. Nevertheless, this does provide evidence of a potential cardiovascular stress maladaptation in Type D individuals which may underpin the link between the personality construct and the physical symptoms (particularly cardiac symptoms) demonstrated in Chapter 4. Furthermore, increased SV has been found to contribute to high blood pressure (McEniery et al., 2005) and Type D has been deemed a risk factor for hypertension (Denollet, 2000). In comparison to the other haemodynamic parameters, SV has not received the same level of attention within the cardiovascular stress literature. Nevertheless, contrary to the findings of the present study, acute stress has been found to lead to decreases rather than increases in SV (Matthews, Salomon, Brady & Allen, 2003). However, this may be reflected in the decrease in SV observed in non-Type D individuals after the practice phase, suggesting that the Type D response observed may be representative of underlying cardiovascular dysregulation.

The measurement of blood pressure and other cardiovascular parameters alone however, reveals little about key underlying processes (James et al., 2012). Blood pressure measures may appear unchanged; however, the underlying haemodynamic parameters (TPR and CO) can be dynamic. For example; patterns of TPR and CO may change from a myocardial HP (CO predominates) to a vascular profile (TPR predominates), or vice versa; however, the compensation deficit (reciprocal relationship between TPR and CO) may not

change and would be exemplified by what would appear to be stable blood pressure. Therefore, despite these null findings regarding SBP and DBP reactivity, as blood pressure is underpinned by more complex haemodynamic parameters and homeostatic regulation, these do not necessarily indicate an absence of differences in cardiovascular stress reactivity in relation to Type D.

When examining the parameters that underpin haemodynamic profile, cardiac output showed a similar acute stress response to that of the blood pressure measures. A rise in CO was observed from the baseline to stress (practice and task) phases then a decrease to the recovery phase. However, only the practice and recovery phases significantly differed from each other. No differences were observed between the two Type D groups on measures of CO across the session. This finding partially opposes Williams, O'Carroll, and O'Connor, (2009) who found that Type D males (but not females) exhibited a larger CO response to an acute stressor. However, two thirds of the current sample were female which may explain the converse findings observed here. As the investigation of gender differences is beyond the scope of this study, these are not further explored here; however, it could be recommended that separate analyses be undertaken in larger samples of males and females in future similar studies.

TPR also increased in the stress phases relative to baseline; however, did not decline during the recovery phase. Type D and non-Type D individuals again did not differ on TPR across the testing session. However, there was a significant interaction effect observed, suggesting that TPR reactivity may differ between Type D groups. TPR increased in response to the stressor (albeit during the practice phase only) in non-Type Ds but not in Type Ds. However, in both groups TPR was higher in the recovery phase in comparison to baseline. These findings could suggest that the Multitasking Framework initially evokes a mixed haemodynamic response (CO and TPR both increase or remain the same), which then changes to a vascular profile between the stress and recovery phases (increase in TPR and decrease in CO) (Gregg et al., 1999). It has been suggested that the relationship between Type D personality and cardiovascular function may be seen at the underlying haemodynamic

determinant level. Therefore, examination of the HP-CD model (James et al., 2012) of blood pressure reactivity aimed to explore this further. Utilising this technique, recent research has shown that Type D personality in females is associated with a maladaptive pattern of cardiovascular responding; where Type D individuals were less "myocardial" than non-Type D individuals, (Howard et al., 2011).

Contrary to the previous findings of Howard, Hughes and James, (2011), in the current study no significant differences were observed between the singular HP calculations for Type D and non-Type D individuals. However, an interaction effect was observed showing that HP scores did not differ between practice, task or recovery in the Type D group, but increased between practice and recovery, and between task and recovery, in the non-Type D group. Further, similar to the study by Howard et al., (2011), compensation deficit scores did not differ between Type D and non-Type D individuals. Positive CD scores indicate an increase in blood pressure reactivity (James et al., 2012) and CD was found to follow a similar pattern of reactivity to that demonstrated by SBP and DBP, across the testing session. As there was a change in HP for non-Type Ds but not Type Ds, this may suggest that Type Ds exhibit a maladaptive pattern of change in haemodynamic profile in response to an acute stressor. Therefore, according to the HP-CD model proposed by James et al., (2012) it could be suggested that the blood pressure increases observed for Type Ds were due to a 'mixed' (HP scores close to 0) profile, whereas for non-Type D the increase in blood pressure observed was due to an initial 'myocardial' (HP score below 0) to 'vascular' (i.e. increased HP scores) profiles. A myocardial profile (i.e. CO predominates), is demonstrated by an increase in CO together with a decrease in TPR, whereas, a decrease (or no change) in CO, accompanied by an increase in TPR, is classed as a vascular profile (i.e. TPR predominates). A vascular profile is seen as a typically more healthy response (Eliot et al., 1982).

This finding demonstrates that two similar patterns of BP reactivity could be underpinned by two different patterns of HP, and further supports the theory that BP measures can mask underlying cardiovascular changes (Gregg et al., 2002). These findings appear to mirror the findings of Kupper, Pelle, and Denollet, (2013) who found that Type D individuals

exhibited an exaggerated haemodynamic profile to the cold pressure task; however research has shown that more 'passive' tasks such as the cold pressure evoke different haemodynamic response compared with more 'active' cognitive stressors (Gregg et al., 1999) such as the one employed in the current study.

In agreement with Howard et al., (2011), it could therefore be argued that a maladaptive (blunted) haemodynamic profile in Type D individuals may underpin the link with poor physical health. This is particularly relevant given that Type D has been found to predict cardiac/sympathetic symptoms in previous studies within this project, in addition to other symptom clusters which have a stress-related link (See Chapter 4).

Finally, in regards to the other haemodynamic parameters measured within the present study, interbeat intervals and ejection fraction measures were not influenced by either Type D category or the stressor. However, dimensional analyses of Type D indicated that IBI reactivity (peak minus baseline) was significantly predicted by NA scores. All other cardiovascular parameters (with the exception of SV) in the current study were not predicted by the continuous NA, SI or Type D scores.

7.4.1.3 Heart rate variability

HRV (as measured by the standard deviations of interbeat intervals) decreased in response to the stressor and increased again in the recovery phase of the testing session. This supports the literature that increases in stress can reduce HRV. Reduced HRV can lead to negative health consequences including diabetic neuropathy, tetraplegia, myocardial infarction, and other cardiac events (Malik, Bigger, Camm & Kleiger, 1996), therefore this finding therefore may support the large body of literature that demonstrates that stress can lead to poorer health (Thayer, Åhs & Fredrikson, 2012; Thayer, Yamamoto & Brosschot, 2010). In the present study, HRV did not appear to differ between Type D and Non Type D groups. This was unexpected, given the findings of Martin et al., (2010) who observed unhealthy HRV patterns in Type D individuals in response to a stressful experience, however, this was observed in a larger sample of individuals. Future research may therefore wish to further assess

HRV in relation to Type D, in a larger sample.

7.4.1.4 Salivary alpha amylase

The Multitasking Framework also induced a response in both sAA concentration and sAA output measures across the testing period. Following the observed pattern of blood pressure responses, both sAA measures increased in the stress phases and began to decrease after 10 minutes' recovery. It also appears that sAA levels began to increase again 20 minutes after the stressor ceased. However, this may be evidence of an anticipatory increase in sympathetic arousal due to the blood draw at the end of the study.

Nevertheless, the findings regarding the sAA levels corroborate the literature that has demonstrated increases in sAA activity in response to acute stress (Nater et al., 2006; Rohleder et al., 2004, 2006). The present study is the first to test and observe sAA reactivity to multitasking stress. As the pattern of response is similar to the blood pressure responses, this finding provides further evidence that sAA can be a reliable biomarker of sympathetic activation and adds to the evidence that multitasking stress activates the SAM axis. However, in the current study no differences in sAA reactivity were observed in relation to Type D personality. Therefore, no evidence was found to suggest that maladaptive sympathetic activation in response to cognitive stress, as measured by sAA, may be a mechanism underpinning the relationship between Type D and ill health.

7.4.1.5 C- Reactive protein

Levels of serum C-Reactive protein; a biomarker of inflammation; were not found to be related to Type D personality via categorical or dimensional analyses. This is contrary to the findings of a number of studies that have demonstrated increased inflammation as exemplified by a number of biomarkers including CRP (Mommersteeg et al., 2012) and other cytokines in relation to Type D personality. However, the current study comprised a sample of healthy young adults, whereas the majority of previous studies have utilised clinical populations and older participants. It could therefore be suggested that levels of CRP are more likely to be a consequence of the cardiac health problems documented in the literature, and

may manifest later in the progression of cardiac related illnesses.

7.4.2 Strengths and limitations

The current study benefits from a number of methodological strengths. Firstly, the use of both subjective and objective measures increases the robustness of findings, particularly as many studies of of Type D rely upon subjective measures in isolation. Furthermore, it could be suggested that distressed individuals may report more physical symptoms in line with the symptom perception hypothesis (Gijsbers van Wijk & Kolk, 1997), which could influence interpretation of the findings relating to physical symptoms throughout this programme. This will be discussed further in Chapter 9.

In terms of the experimental aspect of the study, the stressor that was employed (the Multitasking Framework) is an ecologically valid acute stress paradigm that reflects the kinds of multitasking demands that an individual may incur and be potentially stressed by in the real world (Kelly-Hughes et al., 2014; Wetherell & Carter, 2014). The additional negative feedback also strengthens the validity of the stressor as critical evaluation has been postulated as an important additional factor involved in inducing a reliable stress response (Wetherell et al., 2017). However, despite these strengths, it must be considered that the results presented here may not be wholly representative of a real-world stress response in Type D individuals, due to the use of a laboratory-based paradigm. Given Type D individuals socially inhibited nature, it may be suggested that a real-world socially salient stressor may induce a differential stress response in these individuals. Therefore, as previously discussed in Chapter 6, the use of a public speaking task such as the TSST (Kirschbaum et al., 1993) may help overcome this potential limitation in this group, due to the high levels of social evaluative threat, which has been shown to be an important factor in Type D stress research (Bibbey et al., 2015).

The use of the Portapres and BeatScope software to assess cardiovascular function is a further strength of this study as they provided a comprehensive range of beat-to-beat haemodynamic data. The Portapres is deemed a reliable, non-invasive measure of haemodynamic parameters including CO and TPR (Marshall, Diesch & Hainsworth, 2004).

However, as Portapres measurements are estimated using arterial waveform pressure, it is not entirely representative of true cardiovascular function, as finger arterial mean pressure (as measured by Finopres) has been found to be 5 to 10 mmHg lower than intra-arterial pressure in the brachial artery (Langewouters, Settels, Roelandt & Wesseling, 1998). Nevertheless, as the present study aims to assess changes in these parameters rather than the values themselves, this limitation will have had little influence on these results. Further, the use of a proxy measure of HRV in the current study could be improved by accurate high and low frequency HRV assessment by means of EKG and HRV Analysis Software (e.g. Martin et al., 2010).

In line with previous similar studies (O'Leary et al., 2013), participants were given 20 minutes to acclimatize to the laboratory before the Portapres finger cuffwas fitted and cardiovascular readings commenced. This was to ensure that baseline measures were representative of true resting state.

Salivary alpha amylase was collected using the unstimulated passive drool technique which is considered the gold standard in saliva sampling as it enables a large sample volume to be collected, which maybe be assayed for multiple biomarkers while minimizing the influence of other substances (DeCaro, 2008). This procedure takes precedence over cotton swab and salivette collection, particularly for sAA collection due to the potential issues with saliva flow rate (Beltzer et al., 2010). Storage of the specimens was in line with recommendations (DeCaro, 2008) as samples were frozen promptly after collection and stored at -80C until assaying took place. To ensure further robustness, all samples were assayed in duplicate to assess intra and inter variability and samples with large variations (CVs above 15%) were removed from analyses. As discussed in Chapter 6 sAA has been found to be a reliable biomarker of SAM activity. However, to enhance our knowledge of SAM activity in relation to Type D, future studies may wish to assess levels of catecholamines in plasma as a more direct measure of SAM activity whilst considering the additional logistical issues that this would entail.

A number of further limitations of the current study must also be acknowledged. The most important being the inclusion of the potentially stress-inducing blood draw procedure at

the end of the study session. This was necessary to gain assessment of CRP levels in participants. This element was not conducted at the beginning of the study to limit the influence of any stress in relation to the blood draw on the remainder of the protocol. However, anticipation of blood draws has been found to evoke various psychobiological responses to stress (Mills & Krantz, 1979), and therefore may have confounded the results in any case. However, as the purpose of the study was to examine any differences in stress responses between the two Type D groups (or in the dimensional case, in relation to Type D personality), this would have had limited effect on the findings of prominent interest. Alternatively, this element may have actually evoked an additional 'real-life' stress aspect to the study. Although CRP levels can also be measured in saliva, previous research has found that salivary CRP levels do not correlate with the levels found in blood (Dillon et al., 2010). Consequentially, instead it may be recommended that in stress studies wishing to also assess biomarkers in blood, it would be beneficial for the blood draw to be conducted on another day so as not to interfere with the results.

7.4.3 Summary and key findings

The present study has provided a number of interesting insights into the relationship between stress reactivity and Type D personality. A number of cardiovascular measures appeared to be associated with Type D personality including lower basal SBP and DBP, blunted TPR reactivity, and reduced recovery of increased SV in response to stress. Furthermore, Type D appears to be related to maladaptive changes in haemodynamic profile which may be viewed as unhealthy (less vascular than non-Type Ds). These abnormalities can be postulated as evidence of a cardiovascular maladaptation mechanism which may underpin the link between the Type D personality and increased stress-related physical symptoms.

Contrary to expectation, neither levels of sAA or heart rate variability were related to Type D personality. However, they did exhibit the anticipated responses to the ecologically valid Multitasking Framework. Consequentially, these findings do not provide evidence to suggest that sympathetic activation abnormalities may be present in Type D individuals.

Nevertheless, given the cardiovascular findings, it is proposed that relationship between Type D and poor health could be due to factors specifically underpinning the haemodynamic profile and cardiovascular health rather than the activation of the SAM system as a whole.

In summary, it is suggested that the link between Type D and physical health complaints can be attributed to specific components of cardiovascular stress reactivity. Given these findings it it would be beneficial to develop and implement an intervention to attenuate the experience of physical symptoms in Type D individuals. This proposal will be explored further and tested in Chapter 8.

Chapter 8: Examination of the efficacy of an online positive writing intervention for enhancing well-being in Type D individuals.

This chapter will document a study which was conducted to ascertain whether the influence of Type D personality on physical health complaints and aspects of psychological distress could be reduced by a positive writing intervention. Positive writing interventions have been found to reduce negative affect in the general population, however the aim of this study was to gauge whether individuals who are also socially inhibited (i.e. Type D individuals) could benefit from this intervention. A secondary aim of the study was to assess a range of language dimensions used by each group within the writing task to ascertain whether Type D individual's language use may differ from non-Type Ds. Findings indicate that positive writing can reduce cold/flu related symptoms in Type D individuals. Assessment of the language dimensions appeared to indicate that aspects of writing use differed between the conditions as expected, indicating that participants appropriately engaged with the task. Type D individuals were found to use less positive emotions than Non Type D individuals and more swear words, although this difference did not reach significance. The utility of a positive writing intervention in Type D individuals for the reduction in cold/flu symptoms is clear, however its use for other health issues are yet to be evidenced.

8.1 Introduction

8.1.1 Background

As previously discussed, Type D personality has been found to influence both physical and psychological health in a negative way. Previous research has indicated links with increased physical symptoms (Williams & Wingate, 2012), psychological distress (Pedersen et al., 2004), poor health behaviours (Williams et al., 2008), coping styles (Booth & Williams, 2015), and increases in perceived stress (Aquarius et al., 2005). In short, the studies documented within this thesis have supported previous work and extended knowledge in the area. Type D has been found to be related to specific clusters of physical symptoms, and

longitudinally to predict specific symptom clusters over a one-year period. Further, these relationships appear to be mediated by both anxiety and experiences of stress (see Chapter 5). Moreover, Chapter 7 indicated that Type D personality was related to cardiovascular stress reactivity and sympathetic activation. Throughout the previous chapters, findings have also consistently demonstrated relationships between Type D and increases in anxiety, depression, and perceived stress, in addition to stressful life events and perceived stress reactivity.

Given that Type D appears to adversely affect health, the next logical step would be to explore potential ways in which to reduce the adverse effects of Type D personality, and to potentially improve health in these individuals. The literature regarding the implementation of interventions in Type D individuals is sparse. However, in a recent RCT to examine if an 8-week mindfulness-based stress reduction program could lead to a reduction in Type D personality characteristics, individuals in the intervention group showed larger reductions in both NA and SI (Nyklíček, Beugen & Denollet, 2013). Type D personality has been found to be a relatively stable construct (Dannemann et al., 2010; Denollet, 2005) so it could be suggested that interventions aimed at reducing levels of its underlying components (i.e. SI and NA) may not be beneficial in the long term. Conversely, it is therefore proposed that focussing on ameliorating or buffering the negative effects associated with Type D may be more conducive to longer term benefits for the individual.

Research has indicated that individuals who experience positive emotions can rebound from negative experiences, and show faster cardiovascular recovery after experiencing negative emotions such as stressful events (Tugade & Fredrickson, 2004). Therefore, interventions that aim to increase positive emotions have been implemented previously with the aim of improving both psychological and physical health (Tugade, Fredrickson & Feldman Barret, 2004). Given that this project has shown that Type D individuals appear to endure a higher number of negative stressful experiences (Chapter 5) and also demonstrate slower cardiovascular recovery to an acute stressor (Chapter 7), it appears likely that implementing an intervention which is aimed at enabling individuals to experience positive emotions may be of benefit in this group.

Psychological interventions can both promote positive wellbeing in the general population and reduce the prevalence of many common physical and psychological illnesses (Joseph, 2015). The principle underlying positive psychological interventions is that they aim to indirectly overcome negative consequences such as mental and physical health issues, associated with certain behaviours. Therefore, it could be suggested that an intervention could attempt to overcome the issues associated with the repression of negative emotions (i.e. the combination of NA and SI) characteristic of Type D individuals. Positive psychological interventions focus on enhancing well-being, by reducing the influence of negative events and encouraging individuals to consider and evaluate positive aspects of their lives (Parks & Layous, 2016). Such interventions were originally used as a buffer of negative emotions in the general population, but have also been successfully applied in clinical populations (Parks & Layous, 2016). A meta-analytic review of the evidence revealed that positive psychology interventions significantly improve well-being and ameliorate depressive symptoms (Sin & Lyubomirsky, 2009). Further, positive psychological interventions boast a strong evidence base for their efficacy and ease of implementation (Bolier et al., 2013; Sin & Lyubomirsky, 2009).

The efficacy of positive psychological interventions is supported by a number of studies which have shown an improvement in health outcomes across a range of healthy and clinical groups. For example, a positive motivational interviewing intervention was found to improve self-management and quality of life in diabetes patients (Chen, Creedy, Lin & Wollin, 2012), and a number of positive psychological interventions have showed promise in improving psychological and physical health in cardiac patients (Huffman et al., 2011; Huffman et al., 2016). In the general population, positive interventions have demonstrated reductions in stress, increases in positive emotions, and less reporting of symptoms.

Mechanisms underlying the relationship between positive emotions and physical health are not entirely clear. However, optimism and other positive emotions have been associated with superior cardiovascular outcomes (Huffman et al., 2011). Further, a recent longitudinal study has demonstrated experimental evidence that increasing positive emotions lead to

increases in vagal tone (Kok et al., 2013). Vagal tone is a core component of the parasympathetic nervous system, and governs heart rate in response to stressful events (Porges, 1992). Lower vagal tone is associated with increased inflammation, in addition to poor cardiac health (Thayer et al., 2010). Furthermore Kok et al., (2013) found that the relationship between increased positive emotions and increased vagal tone as an indicator of health, was mediated by perceptions of social connections. It therefore seems feasible that a positive intervention could have a positive effect on health in Type D individuals.

Positive psychology interventions can incorporate various strategies including: thinking about positive life experiences (Lyubomirsky, Sousa & Dickerhoof, 2006); writing about positive experiences (Burton & King, 2004); writing about best possible selves (King, 2001; Layous, Katherine Nelson & Lyubomirsky, 2013); solution-focused coaching (Grant, 2012); and happiness, optimism and gratitude enhancing strategies (Boehm, Lyubomirsky & Sheldon, 2011). While the majority of thesestrategies have their strengths, emotional writing studies have been particularly praised for their ease of implementation and positive results (Layous et al., 2013).

A plethora of research relating to the efficacy of the emotional writing paradigm has suggested that expressing emotional events into words is associated with a range of social, psychological, and health improvements (Niederhoffer & Pennebaker, 2009). While earlier research in this area proposed that writing about traumatic life experiences can lead to improvements in health and well-being (Baikie & Wilhelm, 2005; Pennebaker, 1997; Smyth, 1998), further research indicated that it was not necessarily the expression of negative emotions in the writing which lead to the health benefits (Pennebaker, Mayne & Francis, 1997; Pennebaker & Seagal, 1999). In recent years, a more a positive psychological approach has been taken, with the development of interventions which encourage participants to write about positive experiences. The benefits of such positive writing interventions have been demonstrated (Burton & King, 2004). King and Miner, (2000) found that writing about the positive aspects of a distressing experience was linked to health benefits similar to those associated with writing about trauma. Writing about intensely positive experiences has also

been associated with increases in positive mood and fewer healthcare visits (Burton & King, 2004), and writing about one's best possible future self was found to benefit physical health and enhance psychological well-being (King, 2001).

Indeed, research has begun to explore a variety of writing topics that may lead to health benefits. It is proposed that writing itself may be related to enhanced self-regulation and greater understanding of the individual's needs, priorities and feelings which may in turn work to facilitate physical health (Burton & King, 2004; King & Miner, 2000; King, 2001). Therefore, the tone of the writing may not necessarily need to be negative as suggested in earlier studies (Niederhoffer & Pennebaker, 2009). As positive writing has been shown to enhance positive emotions, reduce negative emotions, and improve physical health, which would all be beneficial to Type D individuals, examining the efficacy of a positive writing intervention in this population seems promising.

Although previous studies have used writing diaries to assess the effects of positive writing on well-being, the current study will employ a relatively novel approach, by using an online platform to deliver the intervention. This approach aims to attract more participants due to increased accessibility and remote participation. A recent paper examined the utility of a positive psychological intervention implemented online and deemed it an appropriate and effective method (Layous et al., 2013). Therefore, the current study will further examine the efficacy of using an online approach.

As discussed in Chapter 1, the Type D construct has been criticised for simply being another measure of neuroticism. It has been suggested that the detrimental effects of Type D personality are solely due to the heightened levels of NA in this group (Coyne et al., 2011; Ferguson et al., 2009a). This critique is at odds with the idea that it is the combination of the two traits that is so detrimental to health (Denollet, 2006). Research shows that high levels of NA and general psychological distress have been associated with increased physical symptoms (Cohen et al., 1993), often irrespective of SI. Although in previous studies social inhibition has been identified as a risk factor for poor health outcomes (e.g. Wallston, Alagna, DeVellis & DeVellis, 1983), NA is considered a greater predictor of negative health outcomes

(Brown & Moskowitz, 1997). Given that positive psychology interventions are aimed at ameliorating the effects of negative affect and increasing well-being (e.g. Sin & Lyubomirsky, 2009), it is therefore important to assess whether the intervention has a positive influence on socially inhibited individuals, in comparison to non–inhibited individuals. Therefore, the current study will assess the utility of a positive writing intervention in high NA individuals, comparing a high SI group (Type D), with a group low in SI (non-Type D).

8.1.2 Aims and hypotheses

The primary aims of the current study are to investigate whether a three-day positive writing intervention can i) reduce physical symptoms, anxiety, depression, perceived stress, and stress reactivity, relative to a neutral control writing task one-month post intervention, and ii) if so, whether the magnitude of change will be greater for socially inhibited individuals with high levels of negative affectivity (Type D) relative to individuals with low levels of social inhibition (Non – Type D).

It is hypothesised that a greater reduction in physical symptoms, anxiety, depression, perceived stress, and stress reactivity will be observed in the intervention condition, relative to the control condition. Additionally, given Type D individuals higher propensity for distress, it is also hypothesised that the magnitude of reduction in these variables will be greater in the Type D group.

A secondary aim of the study is to examine to use of language used by participants, to assess whether any language dimensions are used to a greater degree i) in the intervention condition relative to the control condition and ii) by Type D individuals in comparison to non-Type D individuals. It is anticipated that participants in the intervention condition will use more complex and emotional language than those in the control condition due to the consideration of positive emotional experiences. It may also be suggested that due to Type D individuals increased propensity for distress, they may use more negative language and less positive emotions than the non-Type D individuals, and given their socially inhibited nature may use less social-related words. These findings are anticipated to indicate the efficacy of

implementing a positive writing intervention in Type D individuals, and further expand our knowledge regarding language use and the expression of written emotions in a distressed population as a novel exploration of aspects of Type D behaviour, which has not been considered previously.

8.2 Method

8.2.1 Participants

Initially, participants were recruited to the pre-screen using a variety of recommended online platforms (Branley, Covey & Hardey, 2014) which included dedicated participation sites (e.g. callforparticipants.com), social media (e.g. Facebook, Twitter, Reddit, and LinkedIn), university and research group mailing lists, and student participation pools. Snowball sampling was also used to maximise recruitment to the pre-screen by encouraging participants to refer the link to friends and family friends, and/or share on social media. The study was also advertised via the distribution of posters and leaflets within Northumbria University.

Participants first completed the pre-screen which comprised of the DS14 to determine self-reported levels of negative affect (NA) and social inhibition (SI). Participants scoring below 10 on the NA scale did not take any further part in the study. Participants that did score above 10 on the NA scale were split in to low (<10) SI scorers (non-Type D group), or high (>10) SI scorers (Type D group). These participants were randomly allocated to either the control or intervention condition and sent an email invitation to take part in the full study.

A total of 278 participants completed the pre-screen questionnaire and a subsequent 150 were invited to take part in the full study. A total of 45 Type D individuals and 30 Non-Type D individuals were allocated to the intervention group, and 47 Type D individuals and 27 non-Type Ds were allocated to the control group. Of these 150 participants a total of 73 (mean age = 28.51, 86.3% female, 98.6% UK residents) completed the full study: 20 in the Type D intervention group, 20 in the non-Type D intervention group, 17 in the Type D control group and 16 in the non-Type D control group. Of those participants who did not complete the full

study; 17 completed the pre-task questions only, 13 completed one or two days of the writing task and 2 did not complete the follow up questionnaires. The remaining 45 individuals did not continue after the initial pre-screen phase.

Participants were reimbursed £10 paid via bank transfer upon full completion of the study. Undergraduate psychology students also received course credit for their participation in both the pre-screen and the full study. Testing took place over a six-month period from October 2016 to March 2017.

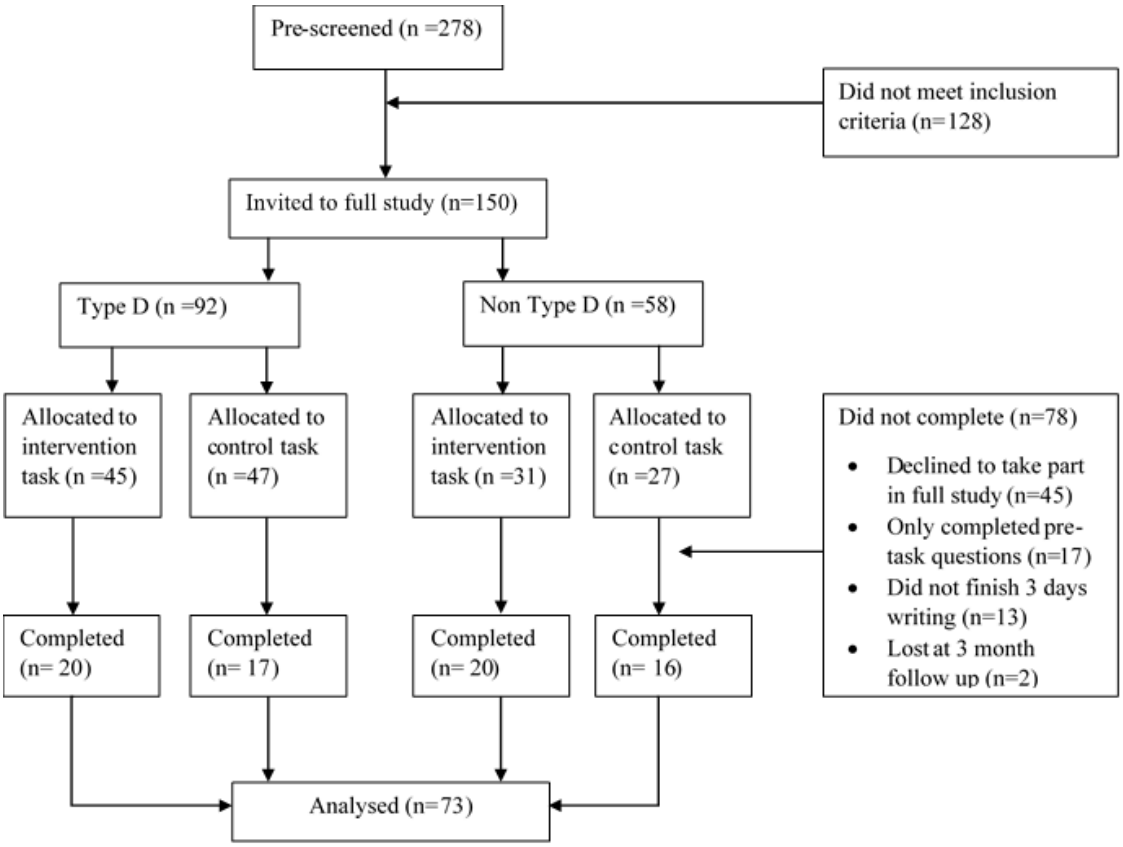

Figure 8.1. Consort diagram showing participation and attrition rates

8.2.2 Design

The study utilised a 2 × 2 intervention design in which the first factor was Type D group (Non- Type D or Type D) and the second factor was task condition (intervention or control task). Measures of physical symptoms, anxiety, depression, perceived stress, and stress reactivity were assessed before the intervention, and again one month later. Dependent

variables were the change in scores on these measures from pre to post intervention. Post-task state anxiety was also assessed each day and a 2x2x3 ANOVA was conducted to assess for any changes over the three days. A number of language dimensions were also examined as additional dependent variables.

8.2.3 Materials and procedure

The full procedure comprised a pre-screen phase followed by 5 study phases which were completed over a 5-week period. Each phase was completed online via the survey platform tool Qualtrics. The study comprised of the following phases 1) pre-screen, 2) pre-task questionnaires, 3) writing task and 4) follow up questionnaires. Each phase is described below:

8.2.3.1 Pre-screen

The purpose of the pre-screen phase was to determine participants' Type D personality scores, and therefore their eligibility to take part in the full study. Participants were able to follow an anonymous link to Qualtrics where the full study information was provided. Participants were required to provide informed consent, their email address and a unique code in order to be contacted and identified in the follow up phases. Participants then completed a number of short demographic questions (Appendix A), and the DS14 (Denollet, 2005) (see Chapter 2 for full details of measures) which took approximately 5 minutes.

8.2.3.2 Pre task Questionnaires

Participants chosen from the pre-screen were sent the link to the pre-task questionnaires via email. The pre-task questionnaires comprised a number of self-report questionnaires; the CHIPS (Cohen & Hoberman, 1983), HADS (Zigmond & Snaith, 1983), PSS (Cohen et al., 1983) and PSRS (Schlotz et al., 2011). This phase took take approximately 10-15 minutes to complete. Once participants completed the questionnaires they were instructed to follow a subsequent URL to complete to the writing task on the following three consecutive days.

8.2.3.3 Writing task

On the three consecutive days following completion of the pre-task questionnaires, the participants were required to complete the writing task. They were provided with specific instructions on what to write about depending on their allocated condition and given 20 minutes (timed by Qualtrics) to complete the task. Following the protocol of Burton and King, (2004) participants in the intervention condition were instructed to write about one of their most positive, happiest experiences on each of the three days and were given the following instructions:

'Think of the most wonderful experience or experiences in your life, happiest moments, ecstatic moments, moments of rapture, perhaps from being in love, or from listening to music, or suddenly ''being hit'' by a book or painting or from some great creative moment. Choose one such experience or moment. Try to imagine yourself at that moment, including all the feelings and emotions associated with the experience. Now write about the experience in as much detail as possible trying to include the feelings, thoughts, and emotions that were present at the time. Please try your best to re-experience the emotions involved' (Burton & King, 2004).

In the control condition participants were given the following instructions; 'In as much detail as possible, write about your plans for the rest of the day from when you finish writing for this study to when you go to bed tonight. (Day 1) Today, write a description of the shoes you are wearing. Be as detailed as possible. (Day 2) Today, write a detailed description of your bedroom. (Day 3). Each day the writing task was completed, all participants completed the STAI-SF (Marteau & Bekker, 1992).

8.2.3.4 Follow-up Questionnaires

One-month post completion of the writing task, participants received a link to the follow up questionnaires via email. The follow-up questionnaires comprised the same measures as in the pre-task questionnaire phase. Once completed, participants were presented with the full study debrief.

8.2.4 Treatment of data

Data were downloaded from Qualtrics, scored, and assessed for missing data accordingly. Change scores were calculated for each outcome measure by subtracting the pre-task scores from the post task scores. A number of 2 × 2 ANOVAs were then performed with condition as the first factor, with the two levels; intervention and control task, and Type D group as the second factor with the two levels; Type D and Non Type D.

Linguistic Inquiry and Word Count (LIWC 2015) software (Pennebaker, Boyd, Jordan & Blackburn, 2015) was implemented to analyse the text of each written script for every participant who fully completed the 3 day task. LIWC is a computerized text analysis program which calculates the degree to which individuals use different categories of words within their writing with a focus on content, style and emotional tone (Niederhoffer & Pennebaker, 2009). The writing scripts were analysed for 96 language dimensions including: 4 summary language variables (analytical thinking, clout, authenticity, and emotional tone); 3 general descriptor categories (words per sentence, percent of target words captured by the dictionary, and percent of words in the text that are longer than six letters); 21 standard linguistic dimensions (e.g., percentage of words in the text that are pronouns, articles, auxiliary verbs, etc.); 41 word categories tapping psychological constructs (e.g., affect, cognition, biological processes, drives); 6 personal concern categories (e.g., work, home, leisure activities); 5 informal language markers (assents, fillers, swear words, netspeak); and 12 punctuation categories (periods, commas, etc) (Pennebaker et al., 2015). Each script was analysed individually and the scores for each language dimension were averaged across each of the three days for each participant.

On the resulting scores, a 2x 2 ANOVA was conducted to assess whether Type D group, condition or the interaction between the two had an effect on the language dimensions assessed. Only significant effects are reported for the language dimensions.

8.3 Results

8.3.1 Self-reported physical symptoms

For all changes in physical symptoms, no significant main effects of Type D were observed. A significant main effect of condition was observed for cold/flu symptoms (F=

4.045 (1,68) p=.048, $\eta p^{2=}$.056), indicating the intervention task (M= -0.84, ±2.20) evoked a significantly larger reduction in cold/flu symptoms than the control task (M= 0.00, ±1.55). The cold and flu symptom change scores for both Type D and non-Type D individuals in both conditions can be observed in figure 8.2. For all other symptom change scores, no significant main effect of condition and no significant interaction effects (between Type D group and writing task condition) were evident.

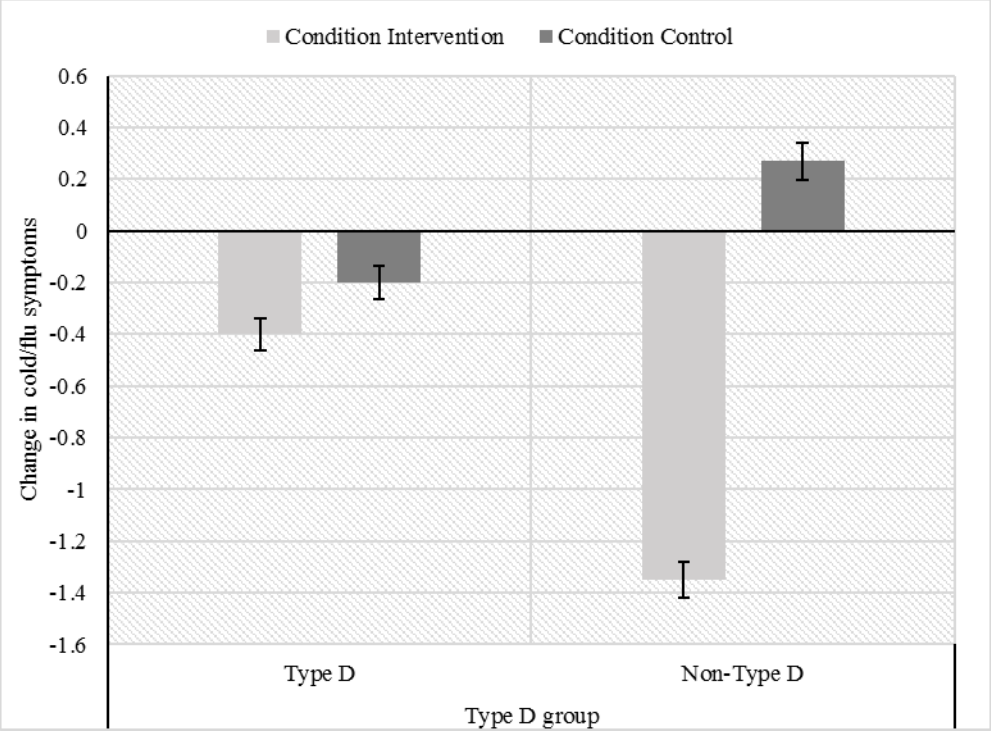

Figure 8.2. The difference in the change in cold and flu symptoms from pre to post intervention (error bars represent standard error).

8.3.2 Psychological and stress-related outcomes

No significant main effects of either Type D group or condition were observed in the change scores for the measures of anxiety, depression, perceived stress, and stress reactivity. A significant interaction effect (between Type D group and writing task condition) was evident for the change in depression scores (F (1,68) = 4.798, p=.032, ηp^2 =.066). However, post hoc t-tests (values doubled to correct for multiple groups) indicated no significant differences between the groups or conditions (p>.05).

A significant interaction effect was also evident for the reactivity to work overload

(RWO) change scores (F (1,68) =5.880, p=.018, ηp^2=.080). Post hoc t-tests indicated that the change in RWO scores from pre to post task in the intervention group was poorer (positive) for Type D (M=0.30 SD=1.66) than non-Type D individuals (M= -1.24, SD= 2.19) (p=.042). All other post hoc t tests were non-significant (p>.05). Changes in RWO scores across groups can be observed in figure 8.3.

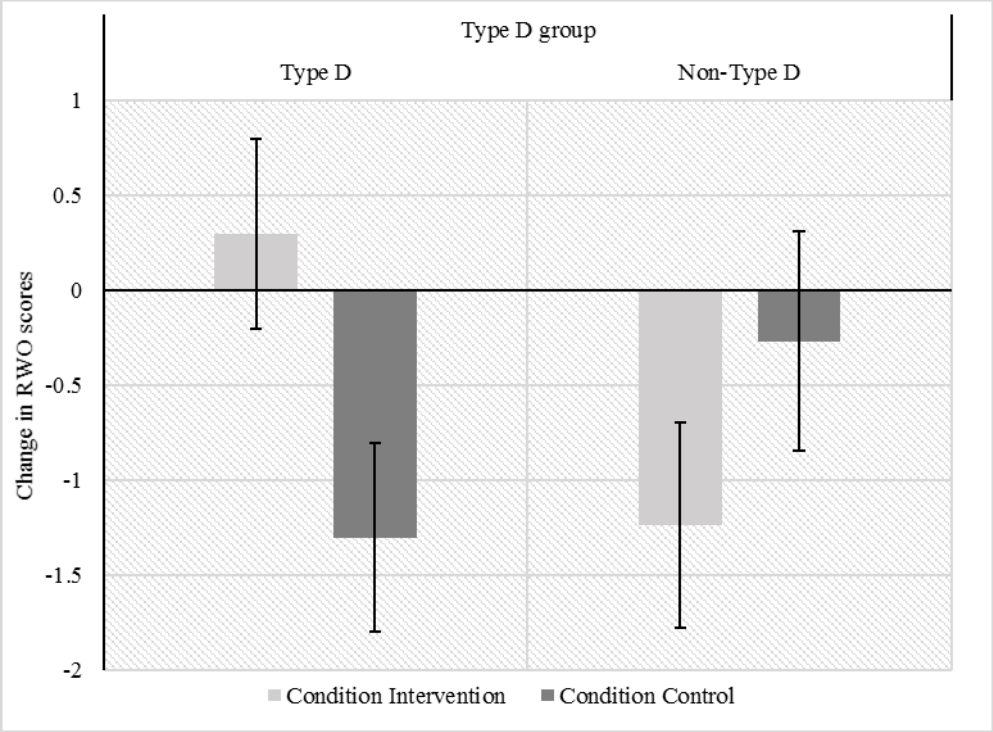

Figure 8.3. The difference in RWO change scores for Type D and non-Type D individuals between the intervention and control conditions (error bars represent standard error).

8.3.3 State anxiety

Results of the 2 × 2 × 3 mixed ANOVA indicated no significant main effects of day, condition or Type D on levels of post-task state anxiety. No significant interaction effects were observed.

8.3.4 Language Dimensions

8.3.4.1 Manipulation check

Analyses revealed no significant main effect of condition (F (1,68) =2.093, p=.153, ηp^2

=.030) on the number of words participants used in the task indicating no difference in compliance with the writing procedure between conditions.

With respect to first person singular pronouns (e.g. 'I', 'me', 'mine'), and second person pronouns (e.g. 'you', 'your', 'thou') there were also no significant main effects of condition. For first person plural pronouns (e.g. 'we', 'us', 'our') there was a significant main effect of condition ($F(1,68) = 29.986$, $p < .001$, $\eta p^2 = .306$) in addition to both third person singular pronouns (e.g. 'she', 'him'), ($F(1,68) = 19.702$, $p < .001$ $\eta p^2 = .306$), and third person plural pronouns (e.g. they, their, they'd) ($F(1,68) = 37.570$, $p < .001$ $\eta p^2 = .356$). Greater use of first person plural pronouns was observed in the intervention condition (M= 1.59, SD=1.15), than in the control condition (M=0.40, SD=0.56). Similarly, third person singular pronouns were also used more in the intervention condition (M=1.38, SD=1.12) than in the control condition (M=0.29, SD=0.36), whereas greater use of third person plural pronouns was observed in the control condition (M=1.54, SD=0.75) in comparison to the intervention condition (M=0.58, SD= 0.63).

Results also indicated there was a significant main effect of condition on emotional tone ($F(1,68) = 86.692$, $p < .001$, $\eta p^2 = .560$). The essays in the intervention condition demonstrated greater expression of emotional tone (M=80.86, SD= 15.41) than those in the control condition (M=51.44, SD=11.55). There was a significant main effect of task condition for use of positive emotion words (e.g. 'love', 'nice', 'sweet') found in the essays ($F(1,68) = 169.009$, $p < .001$, $\eta p^2 = .713$). Individuals in the intervention condition (M=5.09, SD=1.24) showed greater use of positive emotion words than those in the control condition (M=2.06, SD=3.62). With respect to negative emotion words (e.g. 'hurt', 'ugly', 'nasty'), a significant main effect of task condition was also found ($F(1,68) = 21.236$, $p < .001$, $\eta p^2 = .238$). Individuals in the intervention condition (M=1.40, SD=0.72) also used a greater proportion of negative emotion words in comparison to those in the control condition (M=0.64, SD=0.33). Thus indicating participants complied with task instructions.

A significant main effect of condition was observed for levels of past focus (e.g. 'ago', 'did, 'talked') in the essays, ($F(1,68) = 354.391$, $p < .001$, $\eta p^2 = .839$). Individuals in the

intervention condition (M= 9.81, SD= 2.27) demonstrated more incidences of past focus words in their writing than those in the control condition (M=2.39, SD= 0.78). With respect to present focus (e.g. 'today', 'is', 'now') observed in the essays, there was a significant main effect of condition, (F (1,68) = 164.404, p<.001, ηp^2=.707). Individuals in the control condition (M= 11.68, SD=1.67) demonstrated more incidences of present focus words in their writing than those in the intervention condition (M=5.44, SD=2.41). A significant main effect of condition was also observed for levels of future focus (e.g. 'may', 'will', 'soon'), (F (1,68) = 41.455, p<.001, ηp^2=.379). Individuals in the control condition (M= 2.34, SD=1.30) demonstrated more incidences of future focus words in their writing than those in the intervention condition (M=0.86, SD=0.40). Given the different conditions this again indicates participants complied with the task instructions.

No significant main effect of condition on the use of swear words (e.g. 'fuck', 'damn', 'shit') was observed.

8.3.4.2 Type D differences in language use

There was no significant main effect of Type D group (F (1,68) =3.065 p=.085 ηp^2=.043) or interaction effect (F (1,68) =2.266, p=.137, ηp^2 =.032) on the number of words used.

With respect to first person singular pronouns (e.g. 'I', 'me', 'mine'), first person plural pronouns (e.g. 'we', 'us', 'our'), second person pronouns (e.g. 'you', 'your', 'thou'), third person plural pronouns and third person singular pronouns, the main effects of Type D were non-significant. Results for first person singular pronouns initially indicted a significant interaction effect (F (1,68) =5.702, p=.020, ηp^2=.077); however, corrected post hoc t-tests indicated no significant differences between groups or conditions (p>.05). The interaction effect for third person singular pronouns was also significant (F (1,68) =3.136, p=.032, ηp^2=.066), however, post hoc t-tests indicated that the significant differences lay only between the task conditions for both groups. No significant interaction effects were observed for the use of first person plural pronouns (e.g. 'we', 'us', 'our'), second person pronouns (e.g. 'you',

'your', 'thou') or third person plural pronouns.

There was a marginally significant main effect of Type D group on emotional tone (F (1,68) =3.981, p=.050, ηp^2=.055). Type D individuals (M=70.65, SD=20.86) exhibited less emotional tone in their writing in comparison to non-Type D individuals (M=63.29, SD=19.09). Differences in emotional tone across groups can be observed in figure 8.4. The interaction effect was non-significant.

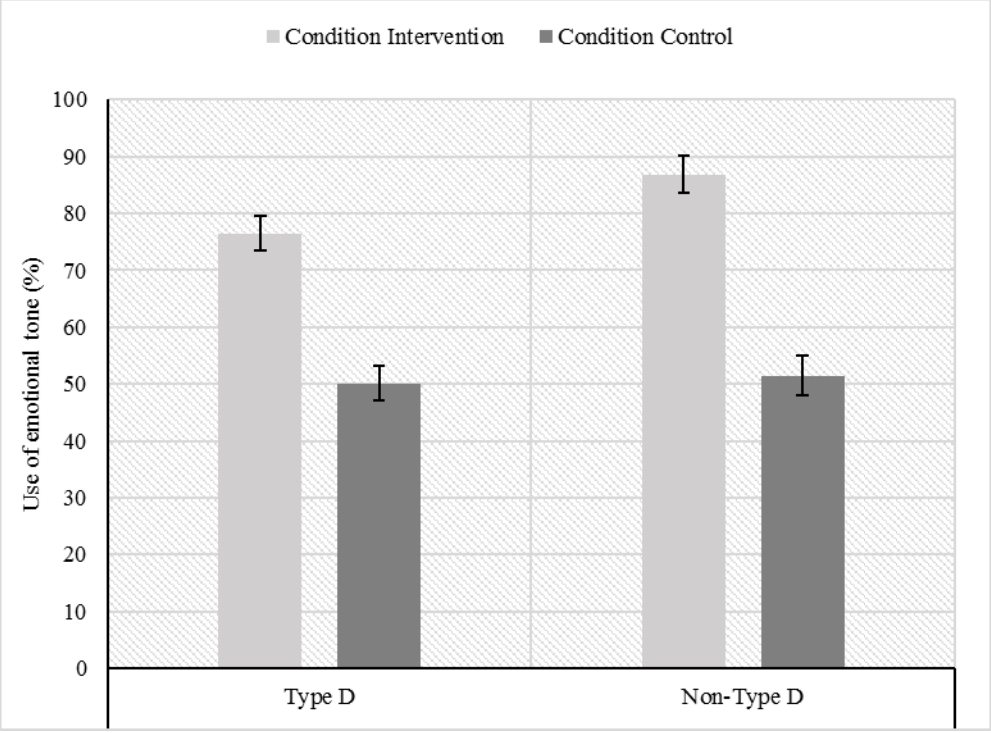

Figure 8.4. The difference in emotional tone (%) by Type D and non-Type D individuals between the intervention and control conditions (error bars represent standard error).

A significant main effect of Type D was also observed for use of positive emotion words (F (1,68) = 6.737, p=.012, ηp^2=.090). Type D individuals had a greater usage of positive emotion words (M=3.30, SD=1.58) than non-Type D individuals (M=4.01, SD=2.08). No significant interaction effect was observed. These differences are presented in figure 8.5. The main effects of Type D group and the interaction effect on the use of negative emotion words were both non-significant.

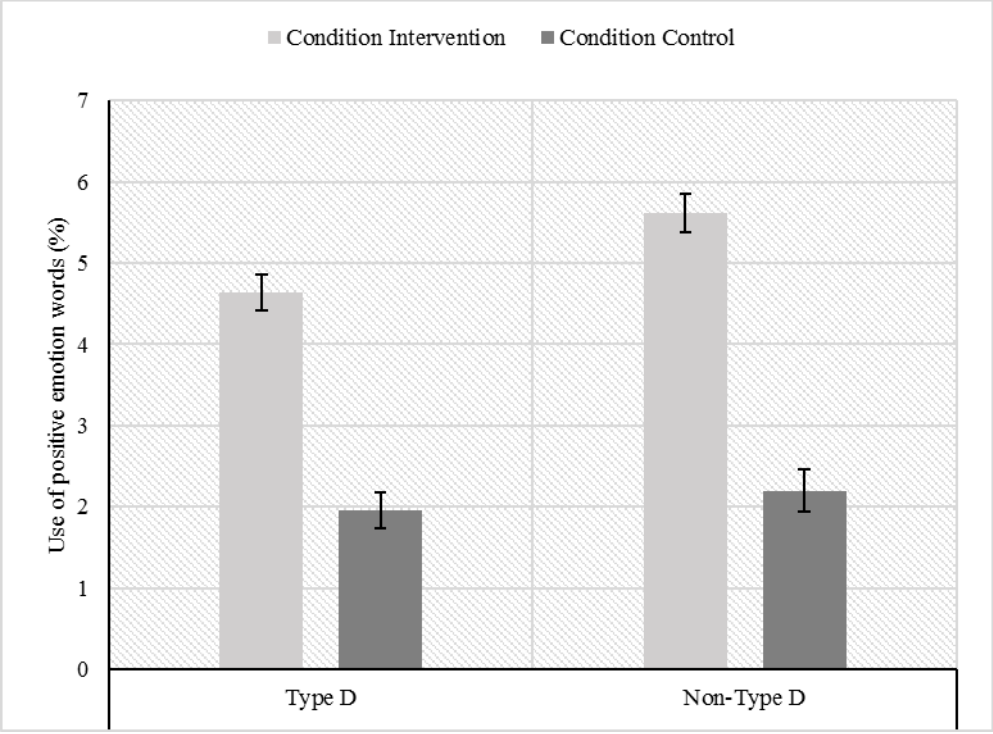

Figure 8.5. The difference in the use of positive emotions (%) by Type D and non-Type D individuals between the intervention and control conditions (error bars represent standard error).

No significant main effects of Type D group or significant interaction effects were observed for use of past, present or future focus within the essays.

Results indicated that for the use of swear words (e.g. 'fuck', 'damn', 'shit') the main effect of Type D was approaching significance ($F(1,68) = 3.804$, $p=.055$, $\eta p^2=.053$). It was revealed that Type D individuals ($M=0.035$, $SD=.085$) used more swear words in their essays than the non-Type D individuals ($M=.006$, $SD=0.019$). The difference in the use of swear words across groups and conditions is presented in figure 8.6. No significant interaction effect was observed.

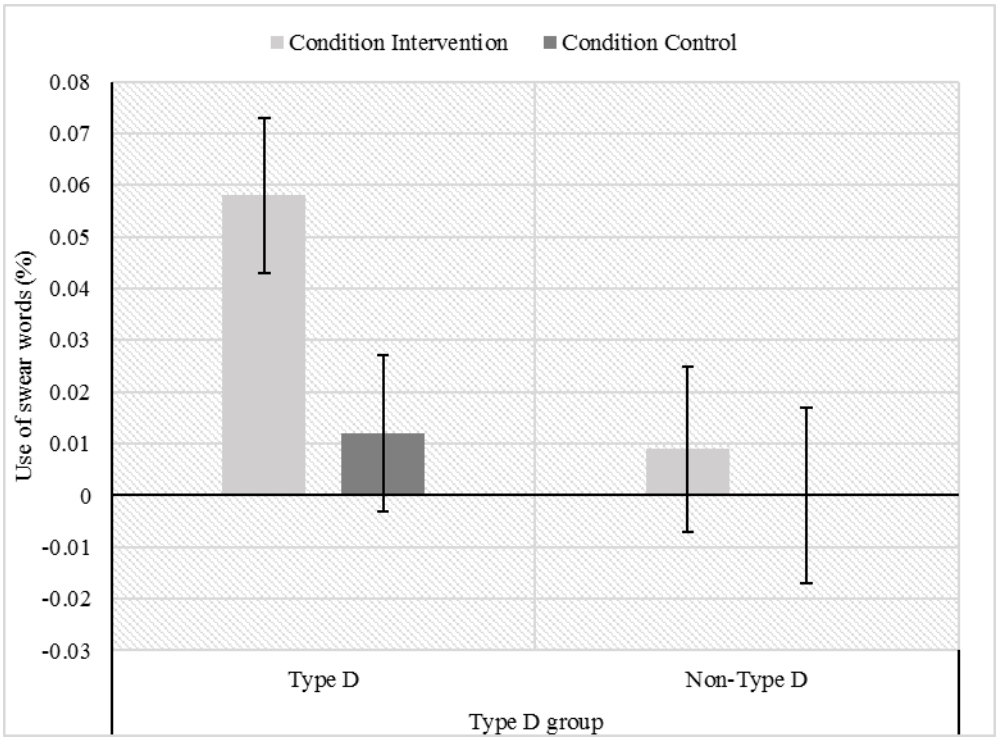

Figure 8.6. The difference in the use of swear words (%) by Type D and non-Type D individuals between the intervention and control conditions (error bars represent standard error).

8.4 Discussion

The aims of the current study were to investigate the efficacy of a positive writing intervention in Type D and Non Type D individuals in reducing physical symptoms and self-reported psychological and stress related outcomes. Given that positive psychology interventions are aimed at ameliorating the effects of negative affect and increasing well-being (e.g. Sin & Lyubomirsky, 2009), and particularly health related outcomes, all individuals in the current study were chosen based on their high level of negative affectivity; as defined by the DS14 (Denollet, 2005). Type D and non-Type D individuals were therefore compared based on their levels of social inhibition. The characteristics of these groups were also selected in light of i) the theory that the effects of Type D are mainly driven by levels of NA and ii) findings within this project (Chapters 4, 5 and 7) indicating that levels of NA have a higher predictive value for physical symptoms. A secondary aim of the current study was to explore the language dimensions used within the writing tasks, to assess any differences between

language used between Type D and non-Type D individuals and the separate writing task conditions (control and intervention).

8.4.1 Physical symptoms

The only cluster of physical symptoms that changed from pre to post intervention as a function of condition was cold and flu symptoms. Individuals in the intervention group reported a greater reduction in the number of cold and flu symptoms than those allocated to the control condition. This suggests that positive writing may be able to reduce the incidence of cold and flu related illness. Psychosocial stress and negative life events are associated with increases in common cold symptomology (Cohen et al., 1993), therefore as the positive writing task is intended to increase positive emotions, it may work to ameliorate stressful feelings and therefore lead to a reduction in these symptoms. This finding also supports the literature that pertains to positive writing leading to improvements in health in general (Burton & King, 2004; Tugade, et al., 2004). Furthermore, positive emotions have been found to improve immune function (Trama & Kaur, 2009) which underpins the development of cold and flu symptoms. It is suggested that writing about topics that enable the consideration of priorities and emotional reactions may facilitate health, regardless of the emotional tone. Therefore, a crucial mechanism underlying the benefits of writing is the process of creating a coherent narrative and gaining understanding of experiences (Burton & King, 2004).

Although there were no between-group differences for the change in cold or flu symptoms, the efficacy of positive writing for reducing cold and flu symptoms in Type D individuals is supported, given that there was a reduction in these symptoms across both groups. Contrary to expectation however, the positive writing intervention failed to demonstrate any significant reduction in symptoms on the remainder of the symptom clusters in comparison to the control task. In the previous studies within this programme, Type D personality has been linked to various stress-related symptom clusters (Chapter 4 and 5) as well as aspects of maladaptive cardiovascular reactivity (Chapter 7). Therefore, these findings suggest that the clinical utility of positive writing may not be particularly effective for the

health detriments specific to Type D personality, despite research which suggests that positive psychology interventions are beneficial for improving cardiac health (Kok et al., 2013). However, as the participants in the current study were all 'healthy' individuals from the general population, it is suggested that testing the efficacy of the intervention in a 'non-healthy' clinical population of Type D individuals is warranted.

8.4.2 Psychological and stress related outcomes

Results indicated that the change in anxiety, depression, perceived stress reactivity, and perceived stress from pre- to post intervention did not differ between either the task conditions or Type D groups. This is contrary to previous studies which have found positive writing to enhance mental well-being (Sin & Lyubomirsky, 2009). An interaction between Type D group and the task condition was however, evident for changes in perceived stress reactivity to work overload. Type D individuals in the intervention group reported a slight increase in reactivity to work overload, whereas non-Type Ds reported a decrease over the month period. This could suggest that translating positive experiences into writing may reduce feelings of stress for non-Type D individuals but not for Type D individuals. Reactivity to work overload was measured by the RWO subscale of the PSRS, which includes questions about dealing with demands, and fulfilling tasks and duties (Schlotz et al., 2011). Given these findings, it is suggested that Type D individuals found that the intervention task was in fact demanding, potentially due to their inhibited nature manifesting in a possible reluctance to write about positive emotional experiences.

Overall, these findings potentially demonstrate that positive writing interventions are not entirely beneficial in terms of enhancing particular aspects of well-being for everyone. Individual differences such as personality must be taken into account if positive psychological interventions are to be implemented in treatment plans, particularly in distressed individuals. Furthermore, an abundance of research proposes that the acceptance of negative emotions and experiences, is in fact more advantageous (e.g. Alberts, Schneider & Martijn, 2012) in comparison to the attempted amendment of negative feelings via shifting focus onto positive

ones. Therefore, interventions centered around acceptance (e.g. forgiveness training, mindfulness-based stress reduction programs, and acceptance and commitment therapy) (Harris & Thoresen, 2006; Veehof, Oskam, Schreurs & Bohlmeijer, 2011), may need to be explored in Type D individuals, to examine whether these type of interventions may lower perceptions of stress reactivity and lead to more favourable outcomes in these individuals.

It may also be the case that the requirement to write about experiences rather than talking about them may also have played a part in the differences observed for the changes in stress reactivity between participants. Lyubomirsky et al., (2006) for example, found that participants who wrote about their happiest moments reported smaller reductions in well-being when compared to participants who talked about or replayed their positive experiences. Perhaps an intervention in which Type D individuals talked about or replayed an emotional experience in isolation, (i.e. with no audience or lasting record of their account, to reduce the social aspect which may be distressing for Type D individuals), may be more favourable.

8.4.3 Language dimensions

Contrary to the findings of Burton and King (2004) there were no differences in the word count across the three days of writing between the control and intervention conditions, indicating that engagement with the task was comparable regardless of group or condition. Furthermore, the intervention condition evoked greater use of language with a past focus, whereas the essays from the control condition demonstrated more present and future focus. Given the different instructions given to participants in each task condition, this provides further evidence that participants adhered to protocol. Greater use of both positive emotional words and negative emotional words was observed in the intervention condition in comparison to the control condition, indicating that the intervention encouraged the use of more emotional words, regardless of the tone. This may be unexpected as the positive writing instructions were to write about a happy experience; however, one suggestion is that the participants may have discussed their positive experiences in comparison to times in which they were less happy.

Furthermore, Type D individuals used less positive words than non-Type D individuals,

thus suggesting that poorer psychological wellbeing typically reported by Type D individuals (Denollet, 2005) may also manifest in language use. However, there was no difference between groups on the use of negative emotion words, perhaps suggesting that even if Type D individuals experience more negative emotions, they do not express them, potentially due to their increased levels of social inhibition.

Participants also demonstrated greater use of words pertaining to social processes in the intervention condition. This was found in both Type D and non- Type D individuals, suggesting that the intervention successfully encourage Type D individuals to re-experience and describe positive emotions associated with social experiences, despite their socially inhibited nature. This may indicate a degree of utility of a positive writing intervention in Type Ds, or conversely; may explain the findings regarding stress reactivity. As aspects of social anxiety and social inhibition are linked to increased distress (e.g. Beesdo et al., 2007), it is plausible that inadvertently encouraging socially inhibited individuals to write about social experiences may not beneficial in terms of reducing stress.

Furthermore, greater use of first person plural pronouns (e.g. 'we', 'us', 'our') were found in the intervention essays; as these words indicate inclusion in a group, this supports the findings regarding social processes discussed above. However, with respect to the use of third person pronouns, some seemingly opposing findings were observed. Greater use of third person singular pronouns (e.g. 'she', 'her', 'him') was evident in the intervention condition, whereas greater use of third person plural pronouns (e.g. they, their, they'd) was observed in the control condition. However, this difference may be due to the control task writing instructions and the interchangeable semantic nature of the plural pronouns such as 'they', which are often used in the description of items such as shoes (as required by the second day of the control task). With respect to third person singular pronouns, Type D individuals demonstrated a greater difference in usage of these words in the intervention condition compared to the control condition. The same was observed for non-Type D individuals but to a lesser extent (the difference was only marginally significant between conditions). The increased use of personal pronouns has been associated with improved psychological and

physical health with a particular focus on how people view their social relationships (Campbell & Pennebaker, 2003), which could explain the health benefits associated with positive writing, especially given that the intervention encouraged the discussion of social experiences. The data pertaining to pronoun use provides further evidence that participants adhered to the instructions of the writing tasks.

Interestingly, the difference in the percentage of swear words (e.g. 'fuck', 'damn', 'shit') used by Type D and non-Type D individuals approached significance, with Type Ds' showing greater use of swear words in their essays in comparison to non-Type Ds'. Swearing is a linguistic tool to convey the expression of strong emotions and is often viewed as a type of coping mechanism (Vingerhoets, Bylsma & de Vlam, 2013). Swearing is hypothesized to produce a catharsis-effect and has been linked to dealing with stress and pain (Stephens & Umland, 2011; Wang, 2013). Therefore, it may not be surprising given Type D individuals' propensity to distress, that they may utilise this language to greater extent. This may be evidence of a further protective mechanism invoked by Type Ds in attempt to reduce their levels of distress.

8.4.4 Strengths and limitations

A particular strength of the current study is the use of an already established positive writing paradigm which has reliably demonstrated positive results in terms of physical health benefits (Burton & King, 2004). The use of LIWC software is a second strength of the current study. LIWC has been used in a number of previous writing studies and is regarded as the gold standard in language analysis software (Tausczik & Pennebaker, 2010). However, as with all text analysis programmes, LIWC is still a relatively crude tool and errors in identifying and counting individual words are possible. Furthermore, language concepts such as irony, sarcasm, or metaphor are not understood. However, it is claimed that these errors seldom affect the conclusions that can be drawn from the results as they are compensated by the way in which words are most commonly used (Pennebaker et al., 2015).Nevertheless, findings should be interpreted with these limitations in mind.

Several limitations relating to design, data collection, and analyses should also be mentioned. All health and psychological outcomes were measured via self-report, and therefore may risk social desirability and response biases. This will be discussed further in Chapter 9. Future research would be strengthened by using other means of health assessment, including physiological measures (such as those implemented in chapter 7), and questions about general health and healthcare utilisation (similar to the retrospective health questions included in chapter 5). These findings should also be considered in relation to the relatively small sample and the generalisability of the results due to the large proportion of student participants.

The conceptualisation of the Type D and non-Type D groups in the current study (i.e. based on their levels of social inhibition) could also be viewed as a limitation. As previously explained, this grouping was implemented as positive writing interventions tend to be aimed at ameliorating the effects of NA. Therefore, it is plausible that the inclusion of individuals in the non-Type D group, who were low in NA, may have masked any influences of the intervention between the groups. However, as this conceptualisation was not inclusive of all individuals (i.e. those low in NA) it may have also contributed to the lack of differences between Type D and non-Type D individuals on the outcome measures in the current study. Furthermore, due to the design of the study, assessment using a dimensional approach to Type D in order to strengthen the analyses was not possible. In future studies, in which categorical analyses are required, it would be beneficial to examine differences between four groups based on their SI and NA scores; with a Type D group comprising of those high in both NA and SI, and three non-Type D groups including i) low scorers on both SI and NA ii) low SI, high NA scorers and iii) high SI and low NA scorers. This would enable a comprehensive examination of the effects of the intervention on individuals who exhibit these traits, but would also have significant implications in terms of the number of participants required. The ways in which Type D personality can be conceptualised and analysed will be discussed further in chapter 9.

The strengths and limitations of the use of an online platform to implement this intervention must also be considered. This novel online approach increased ease of use for

participants, enabled remote participation, and was easily accessible through a URL, which could be shared via social media, email, blogs, websites etc. This afforded a number of advantages for the researcher: this approach easily enabled the pre-screen of a large number of individuals; it provided easy immediate access to the writing data (i.e. no requirement for transcription); and provided objective data regarding protocol adherence (i.e. to check participants completed the tasks on the correct days). The efficacy of implementing a writing study online has therefore indicated substantial promise, particularly for hard-to reach populations.

However, this approach does have its pitfalls, for example it limits participants to an online population, a limitation which will be discussed further in chapter 9, but is slightly more problematic in this study due to the requirement for a significant amount of writing. Essentially, potential participants who were not confident in using computers may have declined to take part due to the amount of typing required. Furthermore, many writing studies suggest that it is the potential therapeutic nature of writing itself which is the key to the health benefits observed (Niederhoffer & Pennebaker, 2009), which may be responsible for some of the null findings in the current study. On the other hand, the lack of effects could be due to the control condition comprising of a writing task, and therefore any benefits from writing may have been experienced by all participants. Although, in a recent study, no differences were found between participants who completed a positive writing activity online in comparison to in-person (Layous et al., 2013) suggesting that online implementation of the intervention is similarly effective.

A further limitation is that state anxiety was only measured at the end of the task each day to assess whether state anxiety changed over the three days. It would; however, have been beneficial to assess state anxiety both before and after the writing task each day to examine the immediate effects that the writing task may have had on participant's mood. Previous studies have found that the process of writing about an important emotive topic can lower anxiety levels and improve mood in the short term. It would therefore have been interesting to examine whether this occurred to a different extent between the Type D groups, particularly

in light of the mediating effects of anxiety highlighted in Chapter 5.

Finally, the length of the intervention could also be viewed as a limitation in the current study, as the task was only completed for three consecutive days. Although this was in line with the protocol of previous writing studies (Burton & King, 2004), and was intended to limit burden on participants, it would be interesting to assess how positive writing on a more regular basis may influence the outcomes assessed within this study.

8.4.5 Future directions

As this is the first study to investigate the benefits of a simple and easily implemented writing intervention in Type D individuals, there are a number of recommendations for future studies. Given that the intervention did not appear to benefit Type D individuals' psychological well-being, alternative interventions may warrant testing in this population. For example; writing about traumatic experience has been found to be beneficial (e.g. King & Miner, 2000) and writing about one's best possible self (e.g. Layous et al., 2013) may also be useful, particularly as this may bypass the social aspects of positive writing, which potentially exacerbate the stress experienced by Type D individuals.

Furthermore, previous research has found that flexibility in writing style is consistently associated with health improvements (Campbell & Pennebaker, 2003). Therefore, some assessment of the similarities of the essays over the three days may have been beneficial to provide a clearer picture of the mechanisms involved in positive writing and how this may be affected by Type D personality. It may also be beneficial to test an emotional disclosure intervention, where participants are required to write about a personal traumatic event, which may encourage Type D individuals to disclose their distressing experiences and accept their negative emotions.

Finally, all outcome measures included in the study comprised negative aspects of well-being (i.e. physical symptoms, anxiety, depression, stress reactivity). Therefore the inclusion of a scale in which to quantify positive and negative affect on a continuum, such as the depression-happiness scale (McGreal & Joseph, 1993), in addition to the objective and

retrospective health measures previously mentioned, may be interesting to further examine the extent to which this intervention could be beneficial to participants.

8.4.6 Conclusions

In summary, the positive writing intervention appeared to be minimally beneficial for participants' health and well-being. Physical symptoms associated with Type D earlier in this programme (i.e. cardiac/sympathetic, metabolic, vasovagal symptoms, headaches) remained unaffected by the positive writing intervention; however, both groups reported reductions in cold and flu symptoms. This indicates the efficacy of positive writing for these types of symptoms in Type D individuals. However, the psychological outcomes were not significantly improved by the intervention task. Reactivity to work overload appeared to reduce only for non-Type D individuals in the intervention condition, suggesting that the positive writing task may have maintained, or even supplemented demands experienced by Type D individuals.

Assessment of language dimensions indicated similar task engagement and adherence to writing instructions across both task conditions. The intervention task evoked increased expression of emotional tone and greater use of words pertaining to social processes, first person personal pronouns, and third person singular pronouns, whereas more third person plural pronouns were demonstrated by those in the control condition. Type D individuals demonstrated differences in the use of language across conditions in comparison to non- Type Ds. Most notably, the Type D group exhibited reduced positive emotional word use and greater swear word use relative to the non-Type D group, suggesting that aspects of distress characteristic of Type D personality may be reflected in written language use.

Chapter 9: General discussion of project findings

This chapter aims to summarise the findings of the four studies comprising this PhD thesis with respect to the i) the relationships between Type D personality and physical health and ii) the underlying psychobiological mechanisms. How this project has made an original contribution to knowledge will be explained, and suggestions for the direction of future research based on these findings are discussed. Finally, the project as whole is evaluated and strengths and limitations of methodological and analytical aspects implemented throughout the project will be discussed, including conclusions drawn regarding the conceptualisation of the Type D personality construct and the potential clinical implications of this research.

9.1 Recap of project aims

The aims of the current project were to comprehensively investigate the mechanisms underpinning the link between Type D personality and physical health within the general population. The specific objectives of the programme were firstly to establish the nature of the relationships between Type D personality and physical symptoms, in addition to a variety of psychological and behavioural factors. In addition to this initial cross-sectional aspect, the project aimed to extend previous work and investigate these associations prospectively, to examine the development and possible dynamic nature of the Type D-health relationship. The programme then went on to explore the potential psychobiological mechanisms experimentally. This comprised the objective measurement of numerous markers of sympathetic arousal in response to acute stress, in addition to levels of inflammation/ immune function. Finally, the programme aimed to assess the efficacy of a positive writing intervention in Type D individuals, in attempt to attenuate the negative effects of Type D identified within the preceding studies.

9.2 Summary of findings

In preparation for the investigation of Type D in relation to physical symptoms, the first empirical study (Chapter 3) aimed to conceptualise the measurement of physical symptoms

within this project. Different 'types' of symptoms tend to be associated with distinct illnesses, causes and health consequences (Eriksen et al., 1999), and it would be unlikely that Type D personality would influence distinct symptoms via the same mechanism. The determination of how physical symptoms may cluster was an important first step in the examination of the relationship between Type D personality and physical health. The factor structure of the CHIPS tool was therefore identified, and eight logical and internally consistent (with the exception of haemorrhagic symptoms) symptom clusters were extracted as follows: cardiac, muscular, gastrointestinal, metabolic, muscular, vasovagal, headaches and haemorrhagic symptoms (Allen et al., 2017). These physical symptom clusters were then assessed in relation to Type D in the subsequent empirical studies.

The aim of Chapter 4 was to examine the cross-sectional relationships between Type D and the symptom clusters, in addition to a number of psychological and behavioural factors. It was demonstrated that Type D individuals reported more physical symptoms than non-Type D individuals regardless of the 'type' of symptom, supporting previous research in the area (Stevenson & Williams, 2014; Williams & Wingate, 2012). Type D individuals also reported increased subjective stress, stress reactivity, anxiety, depression, and pain sensitivity, lower levels of social support, higher use of maladaptive coping strategies, and engagement in poorer health behaviours, which also supports existing findings (e.g. Dooren et al., 2015; Gilmour & Williams, 2012; Williams & Wingate, 2012). However, more stringent regression analyses identified that Type D personality was only associated with increased cardiac/sympathetic, vasovagal, metabolic, muscular symptoms, and headaches when the separate effects of NA and SI were controlled. This finding extended previous work by providing a more in depth assessment of the aspects of health that may be associated with Type D in 'apparently healthy' individuals. It was also theorised that the relationships with these particular symptom clusters were underpinned by stress related mechanisms.

As cross-sectional findings cannot reliably infer cause and effect, Chapter 5 extended previous research (e.g. Stevenson & Williams, 2014; Williams & Wingate, 2012) by

prospectively assessing the relationships between Type D and physical symptoms. Longitudinal relationships were identified between Type D and metabolic, gastrointestinal, and cold/flu symptoms. Bootstrapped mediation analyses also indicated that anxiety fully mediated the relationship between Type D and metabolic symptoms and stressful life events played a partial mediating role in the link between Type D and both metabolic and gastrointestinal symptoms but not cold symptoms. Analyses of additional retrospective health questions also indicated that Type D individuals were more likely to report worse health status, higher frequency of illnesses, work absences and a reduced likelihood of seeking alternative medical information, however no differences in actual health care utilisation were observed.

Given the findings of these first two empirical studies, it is suggested that Type D personality may be associated with certain stress-related complaints such as cardiac symptoms in the short term, but may only influence the deterioration of metabolic, gastrointestinal and cold/flu symptoms over time. This suggests that the symptoms influenced by Type D personality may manifest differently over longer periods of time. Alternatively, these particular symptom clusters may be a characteristic of the development of a more serious underlying health problem. In light of the links between metabolic and gastrointestinal symptoms with autonomic dysregulation (e.g. Drossman, Camilleri, Mayer & Whitehead, 2002) and distress (e.g. Haug, Mykletun & Dahl, 2002), it seemed likely that maladaptive sympathetic arousal due to autonomic dysregulation could be a mechanism underpinning the Type D-health relationship.

Subsequently, the aim of Chapter 7 was to examine sympathetic activation in response to acute stress in relation to Type D by measuring a range of beat-to-beat cardiovascular parameters and levels of salivary alpha amylase. C-Reactive protein levels were also assessed as a measure of inflammation, and both heart rate variability and haemodynamic profile were calculated from the CV measures in order to extend previous Type D research. Interestingly, Type D individuals exhibited lower levels of both SBP and DBP in response to a cognitively demanding stressor (the multitasking framework), and this supported the findings of Kelly-

Hughes et al., (2014). Conversely, Type Ds demonstrated heightened stroke volume reactivity. More specifically, SV in Type Ds appeared not to reduce after the task was complete, which may be evidence of an early warning sign for the development of hypertension (McEniery et al., 2005). Clearly these seemingly opposing findings are tentative; however, as BP is governed by complex underlying processes including HP and CD (James et al., 2012), these processes were also explored. Findings suggest the existence of different HP patterns between Type Ds and non-Type Ds. It appeared that non-Type Ds response was initially myocardial (which is normal given the cognitively demanding stress task) and then becomes more vascular, which is viewed as a healthy response. In contrast, the Type Ds exhibited a mixed response, and were 'less' myocardial during the stress task; supporting the findings of Howard et al. (2011), changing to less 'vascular' in the recovery phase. This may represent a less 'healthy' response (Eliot et al., 1982).

Levels of sAA were not significantly influenced by Type D personality; however, the interaction effect on sAA output approached significance, suggesting further investigation may be warranted, particularly given the SV and HP findings. All things considered, these findings implicate the role of maladaptive sympathetic arousal in the relationship between Type D and the stress-related symptoms, particularly cardiac/sympathetic, vasovagal, gastrointestinal, and metabolic symptom clusters (as identified in Chapter 4 and chapter 5). Levels of serum C-Reactive protein; a biomarker of inflammation; were not related to Type D personality, suggesting the inflammatory effects observed in previous research (e.g. Mommersteeg et al., 2012) may ensue later in disease progression.

The final empirical study within this programme of research examined the efficacy of a positive writing intervention in Type D individuals in order to assess whether the negative health effects identified in Type D individuals could be attenuated. It was demonstrated that positive writing reduced cold and flu symptoms in Type D and non-Type D individuals, thus indicating some promise regarding the utility of the intervention in this population. However,

other symptoms in addition to levels of subjective stress, anxiety, and depression were not influenced by the intervention in Type D individuals.

9.3 Original contribution to knowledge

This project has extended our knowledge of the relationships between Type D personality and physical health symptoms, as well as the psychobiological mechanisms involved. It can be concluded that Type D personality is a prospective risk factor for increased physical symptoms in the general population. This project has however, indicated that the relationships are not clear cut; the outcomes depend on numerous factors including the experience of stressful life events, and the type of symptoms influenced can be dynamic (i.e. may change over time) as some cross-sectional links between Type D and symptom clusters were also demonstrated prospectively (e.g. metabolic symptoms), whereas some were not (e.g. cardiac/sympathetic symptoms). Previous studies have neglected to consider the prospective influence of Type D on physical symptoms in the general population, thus the present work is novel in being able to draw definitive conclusions regarding the dynamic nature of the relationships between Type D and physical symptoms. This project is also the first to consider a wide range of potential psychological and behavioural mediators, which has shed new light on the mechanistic pathways by which Type D personality predicts symptoms of ill-health in the general population. Furthermore, this project has provided further evidence that the experience of, and response to stress, may increase allostatic load (McEwen, 2000) in individuals who score highly on Type D, and this is likely to be due to aspects of autonomic dysregulation (Juster et al., 2010). This programme was also the first to examine sympathetic arousal in Type D individuals by examining salivary alpha amylase, a reliable biomarker of sympathetic stress reactivity. This project also comprised one of the first Type D personality intervention studies; specifically, the final study attempted to attenuate the adverse effects of Type D on health. While the efficacy of the intervention which was evaluated here, was equivocal, the findings provide some promise that intervention to attenuate the adverse effects of Type D personality may be possible.

9.4 Future directions

This project has provided evidence of links between Type D and physical health complaints. With the respect to the psychobiological mechanisms involved, it has provided further knowledge regarding the influence of stress reactivity, particularly maladaptive sympathetic arousal. The findings are not entirely conclusive, but are promising enough to warrant aspects of further research.

As previously mentioned, in light of the interaction between Type D and sAA reactivity approaching significance, it is certainly warranted to conduct further investigations of sAA levels in relation to Type D. Firstly it seems logical to use a larger sample to increase the power of the study and reduce the likelihood of a type II error occurring. It would also seem appropriate to explore the effects of other stress paradigms, particularly given the previous findings of Bibbey, Carroll, Ginty, and Phillips, (2015) that demonstrated differences in stress reactivity were evident only in terms of social evaluative threat. Although a social evaluative aspect was included in the protocol of the experimental study, it only comprised negative verbal feedback to the task performance provided by a researcher seated behind the participant, and potentially did not evoke sufficient levels of threat required for differences in stress reactivity between Type Ds and non-Type Ds to be observed.

The nature of critical evaluation and multitasking is a premise which has been explored by Wetherell et al., (2017) who have suggested that the response format and method of evaluation are important factors influencing the salience of social evaluation. It is suggested that tasks may wish to involve the additional vulnerability associated face-to-face contact, and verbal delivery; such as a public speaking task. Public speaking is a well-established acute stressor involving the risk of embarrassment and humiliation, that can provoke significant subjective and physiological responses (Garcia-Leal, Graeff & Del-Ben, 2014). Therefore, given the socially inhibited nature of Type D individuals and their propensity to experience negative feelings, the experience of completing a public speaking task may result in more Type D related SAM-axis differences becoming evident. Furthermore, the gold standard of

stress paradigms; the TSST (Kirschbaum, Pirke & Hellhammer, 1993) has been repeatedly shown to evoke sAA responses, in addition to HPA activation. The TSST also includes a particular stress-inducing social evaluative component, which could be particularly interesting to use in future research examining potential differences in Type D personality.

Although this programme has identified relationships between Type D and both physical symptoms and aspects of maladaptive sympathetic arousal, it cannot be fully concluded that i) Type D personality can predict atypical sympathetic arousal (due to the cross-sectional design) and ii) whether sympathetic arousal mediates the identified relationships. Therefore, future research examining Type D and sympathetic arousal in larger samples (required for mediation analyses) would benefit from conducting mediation analyses to see whether aspects of cardiovascular function, haemodynamic profile, or sAA levels may contribute to the pathways by which Type D is related to particular physical symptoms. On a similar note, it is also warranted to prospectively assess the relationship between Type D personality and sympathetic activation, to fully elucidate the direction of effect in the association between Type D and the aspects of stress reactivity demonstrated.

Finally, with respect to the final phase of this project which aimed to investigate how the effects of Type D on physical health may be attenuated, future research in this area is certainly warranted. As mentioned in Chapter 8, there are a number of different interventions (e.g. those aimed at accepting negative emotions) which may be deemed useful in Type D individuals. Given the findings that demonstrate the likelihood of a stress-related mechanism underpinning the Type D-health relationship, interventions aimed at stress-reduction may be particularly useful. For example, Mindfulness-based stress reduction (MBSR) interventions have been shown to exhibit particular health benefits (Grossman, Niemann, Schmidt & Walach, 2004). A previous study has examined MBSR in Type D individuals (Nyklíček et al., 2013); however it would be worth extending this research to incorporate a wider range of outcomes including the symptom clusters identified in this programme, in addition to both subjective and objective measures of distress.

9.5 Programme strengths and limitations

The programme as a whole boasts a number of strengths, the most notable being the consideration of physical symptoms as distinct clusters which has been lacking in previous Type D research. This enabled the relationship between Type D and physical symptoms to be examined in closer detail, and provided insights into the ways in which Type D could influence health outcomes. Furthermore, this project has conducted analyses using both the categorical and dimensional approach to Type D conceptualisation as recommended by Coyne and de Voogd, (2012) and Ferguson et al. (2009). Previous studies suggest that Type D should be treated in terms of the continuous additive effect of SI and NA due to the absence of interaction effects between the subscales (Horwood et al., 2016). However, the findings of this project have provided support for the prognostic value of the interactive Type D personality construct, and the more stringent regression analyses used to assess the dimensional relationships have indicated relationships with a specific set of symptoms and aspects of sympathetic arousal (i.e. stroke volume). This would suggest that Type D may be an independent risk factor for health via these outcomes, over and above the separate contributions of SI and NA. These findings therefore suggest that Type D personality does still deserve some attention in the stress and health literature.

The way in which Type D is conceptualised is however, an on-going contentious issue. As previously mentioned, the categorical approach is heavily criticised for being generated from high-low, 2 × 2 crossings of continuous SI and NA scores, and for the use of arbitrary cut-offs (Coyne et al., 2011). However, although it does not accurately represent how the combination of SI and NA synergistically interact, the Type D versus non-Type D concept is a useful and easily implemented way to assess the effect of Type D on outcomes, particularly in smaller samples in which regression is not possible. It must be acknowledged; however, that findings utilising the taxonomy should be interpreted with caution and the dimensional approach should be implemented to support these analyses where possible to fully elucidate the effects that can be solely attributed to Type D.

Furthermore, as briefly mentioned in Chapter 8, when it is necessary to assess Type D as a categorical variable it would be beneficial to examine differences between groups separated by both their NA and SI groupings. The traditional categorical approach labels those high in SI and NA as Type D and all others as Non-Type D. Within the non-Type D group however, it is clear that there is a considerable variability. Therefore, it is suggested that comparison of three non-Type D groups; low SI and low NA; low SI and high NA; high SI and low NA, with the Type D (High NA and high SI) would provide a more comprehensive assessment of the influences of NA and SI, and particularly how they interact.

Furthermore, it is suggested that when categorically conceptualising Type D versus non Type D individuals, it is advised to avoid risk of misclassification. For example, by taking participants SI and NA scores, multiplying them, and then conducting a tertiary split and removing the participants scoring in the middle. This would enable comparison between those high in Type D with those low in Type D, and remove the risk of borderline cases. This is similar to the method used by Bibbey et al., (2015) who used more stringent cut-offs of the DS14 to classify Type D and non– Type D. They used the cut-offs of ≥14 and ≤8 on the SI and NA subscales respectively, based on the upper and lower quartiles of their sample.

In terms of methodological aspects of the programme, with the exception of the experimental study described in Chapter 7, the studies were implemented online via the survey platform Qualtrics. As previously mentioned this was particularly useful for recruiting large numbers of participants in the first online study, and enabled the easy recruitment of a substantial proportion of those for the follow up. Furthermore, given the social inhibitory nature of Type Ds, online recruitment was particularly beneficial for recruiting individuals from this population. The large percentage of Type D participants recruited in the online studies of this project could also be attributed to this factor. Online studies increase perceptions of privacy, and encourage participants to disclose more information (Branley et al., 2014). The online questionnaire format is easily accessible and usable, enabling participants to complete the study remotely in their own time, and therefore potentially increasing

participation rates. Online research methods are also economical both financially and with regards to time. Data collection via this method also diminishes the risk of human error (Lefever, Dal & Matthíasdóttir, 2007) in data entry as Qualtrics provides the raw data, and scores, the questionnaires. There are of course a number of limitations associated with use of online studies, including only being accessible to those who have access to the internet. Online studies often attract larger proportions of females and younger participants (Lefever et al., 2007). Furthermore, previous studies have found that respondents complain about the length and time-consuming nature of online surveys, which may implicate a loss of concentration (Lefever et al., 2007). Therefore, the number of questionnaires, particularly in the follow up study (Chapter 5) was chosen with the intention of not over burdening participants.

Nevertheless, the reliance on self-report data throughout this project may be viewed as a limitation. As with all subjective measures, the data risks social desirability bias. This may have been a particular issue for measurement of Type D itself, given that some individuals may not wish to consider themselves as distressed or uncomfortable in social situations. This may also be true for assessments of anxiety and depression, particularly given the stigma surrounding mental health issues (Bharadwaj, Pai & Suziedelyte, 2015). Similarly, for the physical symptoms questionnaires, some of the symptoms listed may be embarrassing (e.g. constipation) and people may not wish to disclose that information. Furthermore, the health behaviours and retrospective health questions may also suffer social desirability as people may indicate more favourable answers (Sallis & Saelens, 2000). Nevertheless, self-report methods remain the easiest way to obtain data about individual's thoughts and feelings, and therefore this approach retains merit.

The documented benefits of self-report measures are their ability to collect data from a large sample of individuals at low cost (Sallis & Saelens, 2000). In order to assess a large sample of participants within this programme (particularly in the initial online study and follow up), self-report measures were the most effective way to gather the large amounts of data required, and as previously mentioned, the online method of data collection should have

reduced the risk of social desirability bias. Furthermore, the self-report of variables does not alter the behaviour or psychological construct under study, and with respect to physical health it is the easiest way possible to assess all the dimensions necessary, particularly as the experience of symptoms is entirely subjective. Self-report measures can be used in a range of ages, and can be adapted to particular populations (e.g. older adults or children), however as this project examined 'healthy individuals' from the general population aged 18-65, this was not necessary.

The objective data collection methods used in the experimental study (Chapter 7) also represent the consideration of the most reliable and relevant indices, whilst limiting burden and discomfort for participants. The use of the multitasking framework as an acute stressor, was selected for its ecologically valid characteristics, and together with the additional negative verbal feedback provided a cognitively demanding task which mimicked real-life situations, with an element of both uncontrollability and socially evaluative threat as recommended by Dickerson and Kemeny (2004). The use of the highly regarded portapres (Langewouters et al., 1998) to measure the cardiovascular parameters, and passive drool technique; the gold standard in saliva sampling (Williamson, Munro, Pickler, Grap & Elswick, 2012), to assess sAA levels, are both further examples of strengths of the experimental study.

A number of limitations must however also be acknowledged. Firstly, the assessment of physical symptoms in the general population brings a number of complications. Data regarding the frequency and severity of physical symptoms tend not to follow a normal distribution and be positively skewed (Eriksen, et al., 1999), as often symptoms or clusters of symptoms are not present and therefore a score of zero is recorded. This can pose problems for statistical analyses of such data, as even logarithmic transformation of the data will not improve the normality of the data set. Furthermore, the physical symptoms data in the current study was not subjected to outlier removal as the technique employed (+/- 2SDs from the mean) was also not appropriate given the large proportion of zero scores. Therefore, removal of such outliers would almost certainly cause the loss of important information. Although no

strategy has been developed or implemented to overcome this issue with physical symptom data, this must be considered as a limitation in interpreting the findings of the studies within this project.

Further, the treatment of outliers in data concerning the other psychological and objective biological variables measured within the current project (as described in chapter 2) must also be mentioned. The approach (+/- 2SDs from the mean) was reasonably conservative in order to retain as much data as possible, particularly given the small sample size in the experimental study. There is no consensus as to the best way to treat outliers; however, it is highly recommended that they be identified and dealt with appropriately (Osborne & Overbay, 2004). In light of this, it must therefore be considered that extreme data points have been removed, when interpreting these results.

The uneven gender representation (female bias) throughout the studies documented in this programme must also be considered, particularly in the interpretation of the findings of Chapter 7. Although some existing studies in this area have featured gender disparities in relation to stress reactivity (Habra et al., 2003; Howard et al., 2011), as the literature pertaining to the relationships between Type D and physical symptoms have not identified any such gender differences (e.g. Williams & Wingate, 2012), the analyses in the current studies were conducted on the combined gender samples. However, future research aiming to examine sympathetic stress reactivity as a mediating factor statistically, may wish to consider the influence of gender and run separate analyses.

Finally, from a critical psychology perspective, the concept of a particular personality 'type' influencing psychological and health outcomes may be viewed as limiting human behaviour to groups, and it must therefore be reiterated that the findings of this programme do not advocate that the effects attributed to Type D personality apply to all Type D individuals. Consideration of personality variables is often absent in predictive health models; however, increasing evidence from Type D research suggests it may be warranted (Horwood et al.,

2016). The current research does potentially advocate the requirement for personality and individual assessment in clinical practice. This would be particularly useful for identifying individuals who fit the Type D criteria and suffer particular psychological and physical symptoms, as these individuals are potentially at risk for negative health consequences in the future. This may lead to measures to provide further support or alternative personalised treatment plans for such individuals. However, more research is necessary before this could be realistically implemented.

9.6 Conclusions

Overall, this programme contributes to research on Type D and adverse health in a number of ways. Essentially, the most salient findings identified from this programme of work are as follows:

i) Type D research has been extended to the investigation of previously unexamined physical symptom clusters, retrospective health, and aspects of sympathetic stress reactivity, in apparently healthy individuals. It has been demonstrated that the interactive Type D construct has some predictive value for particular health and stress-related outcomes.

ii) Further evidence that sympathetic dysregulation underpins the relationship between Type D and relatively poor health in the general population has also been provided.

iii) The potential utility of a short positive psychology intervention has opened up the field for future research aiming to intervene with respect to the effects of Type D on psychological and physical health.

These findings as a whole contribute to a greater understanding of the Type D construct, and may assist in the development of both empirical models conceptualising the links between personality and health, and interventions for use in clinical practice.

References

Al'Absi, M., Bongard, S., Buchanan, T., Pincomb, G. A., Licinio, J. & Lovallo, W. R. (1997). Cardiovascular and neuroendocrine adjustment to public speaking and mental arithmetic stressors. *Psychophysiology 34*(3) 266-275. doi:10.1111/j.1469-8986.1997.tb02397.x

Alberts, H. J. E. M., Schneider, F. & Martijn, C. (2012). Dealing efficiently with emotions: acceptance-based coping with negative emotions requires fewer resources than suppression. *Cognition & Emotion, 26*(5), 863–70. doi:10.1080/02699931.2011.625402

Allen, S. F., Wetherell, M. A. & Smith, M. A. (2017). The Cohen–Hoberman inventory of physical symptoms: Factor structure, and preliminary tests of reliability and validity in the general population. *Psychology & Health, 32*(5), 1–21. doi:10.1080/08870446.2017.1290237

Allgower, A., Wardle, J. & Steptoe, A. (2001). Depressive Symptoms, Social Support, and Personal Health Behaviors in Young Men and Women. *Health Psychology, 20*(3), 223–227. doi:10.1037//0278-6133.20.3.223

Andrews, B. & Wilding, J. M. (2004). The relation of depression and anxiety to life-stress and achievement in students. *British Journal of Psychology, 95*(4), 509–521. doi:10.1348/0007126042369802

Aquarius, A. E., Denollet, J., Hamming, J. F. & De Vries, J. (2005). Role of disease status and Type D personality in outcomes in patients with peripheral arterial disease. *The American Journal of Cardiology, 96*(7), 996–1001. doi:10.1016/j.amjcard.2005.05.059

Armon, G. (2014). Type D personality and job burnout: The moderating role of physical activity. *Personality and Individual Differences, 58*, 112–115. doi:10.1016/j.paid.2013.10.020

Arunodaya, G. R. & Taly, A. B. (1995). Sympathetic skin response: a decade later. *Journal of the Neurological Sciences, 129*(2), 81–89. doi:10.1016/0022-510X(94)00265

Baikie, K. a. & Wilhelm, K. (2005). Emotional and physical health benefits of expressive writing. *Advances in Psychiatric Treatment, 11*(5), 338–346. doi:10.1192/apt.11.5.338

Barnett, M. D., Ledoux, T., Garcini, L. M. & Baker, J. (2009). Type D personality and chronic pain: construct and concurrent validity of the DS14. *Journal of Clinical Psychology in Medical Settings, 16*(2), 194–9. doi:10.1007/s10880-009-9152-0

Barone, S., Bacon, S. L., Campbell, T. S., Labrecque, M., Ditto, B. & Lavoie, K. L. (2008). The association between anxiety sensitivity and atopy in adult asthmatics. *Journal of Behavioral Medicine, 31*(4), 331–339. doi:10.1007/s10865-008-9164-5

Bartels, H., Middel, B., Pedersen, S. S., Staal, M. J. & Albers, F. W. J. (2010). The Distressed (Type D) Personality Is Independently Associated with Tinnitus: A Case–Control Study. *Psychosomatics, 51*(1), 29–38. doi:10.1016/S0033-3182(10)70656-3

Bartels, H., Pedersen, S. S., van der Laan, B. F. A. M., Staal, M. J., Albers, F. W. J. & Middel, B. (2010). The impact of Type D personality on health-related quality of life in tinnitus patients is mainly mediated by anxiety and depression. *Otology & Neurotology, 31*(1), 11–18. doi:10.1097/MAO.0b013e3181bc3dd1

Barth, J., Schumacher, M. & Herrmann-Lingen, C. (2004). Depression as a Risk Factor for Mortality in Patients with Coronary Heart Disease: A Meta-analysis. *Psychosomatic Medicine, 66*(6), 802–813. doi:10.1097/01.psy.0000146332.53619.b2

Beesdo, K., Bittner, A., Pine, D. S., Stein, M. B., Höfler, M., Lieb, R. & Wittchen, H.-U. (2007). Incidence of social anxiety disorder and the consistent risk for secondary depression in the first three decades of life. *Archives of General Psychiatry, 64*(8), 903–912. doi:10.1001/archpsyc.64.8.903

Bellingrath, S., Weigl, T. & Kudielka, B. M. (2009). Chronic work stress and exhaustion is associated with higher allostastic load in female school teachers. *Stress, 12*(1), 37–48. doi:10.1080/10253890802042041

Beltzer, E. K., Fortunato, C. K., Guaderrama, M. M., Peckins, M. K., Garramone, B. M. & Granger, D. a. (2010). Salivary flow and alpha-amylase: Collection technique, duration, and oral fluid type. *Physiology and Behavior, 101*(2), 289–296. doi:10.1016/j.physbeh.2010.05.016

Benham, G. (2006). The Highly Sensitive Person: Stress and physical symptom reports. Personality and *Individual Differences, 40*(7), 1433–1440. doi:10.1016/ j.paid.2005.11.021

Berkman, L. F., Leo-Summers, L. & Horwitz, R. I. (1992). Emotional support and survival after myocardial infarction: A prospective, population-based study of the elderly. *Annals of Internal Medicine, 117*(12), 1003–1009.

Berkman, L. F. & Syme, L. S. (1979). Social networks, host resistance, and mortality: A nine-year follow-up study of alameda county residents. *American Journal of Epidemiology, 109*(2), 186–204. doi:10.1093/aje/kwx103

Bharadwaj, P., Pai, M. & Suziedelyte, A. (2015). Mental Health Stigma. National Bureau of Economic Research. Cambridge: Cambridge University Press. doi:10.1128/AAC.03728-14

Bibbey, A., Carroll, D., Ginty, A. T. & Phillips, A. C. (2015). Cardiovascular and Cortisol Reactions to Acute Psychological Stress Under Conditions of High Versus Low Social Evaluative Threat: Associations with the Type D Personality Construct. *Psychosomatic Medicine, 77*(5), 599–608. doi:10.1097/PSY.0000000000000194

Björling, E. A. (2009). The momentary relationship between stress and headaches in adolescent girls. *Headache, 49*(8), 1186–1197. doi:10.1111/j.1526-4610.2009.01406.

Blascovich, J. J. & Katkin, E. S. (1993). *Cardiovascular reactivity to psychological stress & disease*. Washington, DC: American Psychological Association.

Boehm, J. K., Lyubomirsky, S. & Sheldon, K. (2011). A Longitudinal Experimental Study Comparing the Effectiveness of Happiness-Enhancing Strategies in Anglo Americans and Asian Americans. *Cognition and Emotion, 25*(7), 1263–1272. doi:10.1016 /j.drudis.2013.07.001.

Bogg, T. & Roberts, B. (2013). The case for conscientiousness: Evidence and implications for a personality trait marker of health and longevity. *Annals of Behavioral Medicine, 45*(3), 278–288.

Bolier, L., Haverman, M., Westerhof, G. J., Riper, H., Smit, F. & Bohlmeijer, E. (2013). Positive psychology interventions: a meta-analysis of randomized controlled studies. *BMC Public Health, 13*(1), 119. doi:10.1186/1471-2458-13-119

Bonnet, M. H. (1985). Effect of sleep disruption on sleep, performance, and mood. *Sleep: Journal of Sleep Research & Sleep Medicine, 8*(1), 11–19. doi:10.1016/0031-9384(89)90236-9

Booth, L. & Williams, L. (2015). Type D personality and dietary intake: The mediating effects of coping style. *Journal of Health Psychology, 20*(6), 921–927. doi:10.1177/1359105315573433

Borkoles, E., Polman, R. & Levy, A. (2010). Type-D personality and body image in men: the role of exercise status. *Body Image, 7*(1), 39–45. doi:10.1016/j.bodyim.2009.10.005

Borkoles, E., Polman, R., Ski, C. F. & Thompson, D. R. (2012). Type D personality and cardiovascular outcomes. *Annals of Behavioral Medicine, 44*(1), 139–40. doi:10.1007/s12160-012-9373-6

Borkoles, E., Reynolds, N., Ski, C. F., Stojanovska, L., Thompson, D. R. & Polman, R. C. J. (2015). Relationship between Type-D personality, physical activity behaviour and climacteric symptoms. *BMC Women's Health, 15*(1), 18. doi:10.1186/s12905-015-0176-3

Braithwaite, E. C., Ramchandani, P. G., Lane, T. A. & Murphy, S. E. (2015). Symptoms of prenatal depression are associated with raised salivary alpha-amylase levels. *Psychoneuroendocrinology, 60*, 163–172. doi:10.1016/j.psyneuen.2015.06.013

Branley, D., Covey, J. & Hardey, M. (2014). *Online Surveys: Investigating Social Media Use and Online Risk*. London: SAGE. doi:10.4135/9781446273050135146666

Brown, K. W. & Moskowitz, D. S. (1997). Does unhappiness make you sick? The role of affect and neuroticism in the experience of common physical symptoms. *Journal of Personality and Social Psychology, 72*(4), 907–917. doi:10.1037/0022-3514.72.4.907

Bruce, G., Curren, C. & Williams, L. (2013). Type D personality, alcohol dependence, and drinking motives in the general population. *Journal of Studies on Alcohol and Drugs, 74*(1), 120–124.

Buckley, T. M. & Schatzberg, A. F. (2005). On the interactions of the hypothalamic-pituitary-adrenal (HPA) axis and sleep: normal HPA axis activity and circadian rhythm, exemplary sleep disorders. *The Journal of Clinical Endocrinology and Metabolism, 90*(5), 3106–14. doi:10.1210/jc.2004-1056

Burton, C. M. & King, L. A. (2004). The health benefits of writing about intensely positive experiences. Journal of Research in Personality, 38(2), 150–163. doi:10.1016/S0092-6566(03)00058-8

Buske-Kirschbaum, A., Geiben, A., Höllig, H., Morschhäuser, E. & Hellhammer, D. (2002). Altered responsiveness of the hypothalamus-pituitary-adrenal axis and the sympathetic adrenomedullary system to stress in patients with atopic dermatitis. *Journal of Clinical Endocrinology and Metabolism, 87*(9), 4245–4251. doi:10.1210/jc.2001-010872

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R. & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*(2), 193–213. doi:10.1016/0165-1781(89)90047-4

Cacioppo, J. T. & Hawkley, L. C. (2003). Social isolation and health, with an emphasis on underlying mechanisms. Perspectives in Biology and Medicine, 46(3), 39–52. doi:10.1353/pbm.2003.0063

Campbell, R. S. & Pennebaker, J. W. (2003). The secret life of pronouns: Flexibility in writing stryle and physical health. *Psychological Science, 14*(1), 60–65. doi:10.1111/1467-9280.01419

Cannon, W. B. (1932). The Wisdom of the Body. New York: Norton.

Carney, R., Freedland, K. & Veith, R. (2005). Depression, the autonomic nervous system, and coronary heart disease. *Psychosomatic Medicine, 67*(1), 29–33.

Carroll, D., Ginty, A. T., Der, G., Hunt, K., Benzeval, M. & Phillips, A. C. (2012). Increased blood pressure reactions to acute mental stress are associated with 16-year cardiovascular disease mortality. *Psychophysiology, 49*(10), 1444–8. doi:10.1111/j.1469-8986.2012.01463.x

Carroll, D., Lovallo, W. R. & Phillips, A. C. (2009). Are Large Physiological Reactions to Acute Psychological Stress Always Bad for Health? *Social and Personality Compass, 3*(5), 725–743. doi:10.1111/j.1751-9004.2009.00205.x

Carstensen, L. L., Turan, B., Scheibe, S., Ram, N., Ersner-Hershfield, H., Samanez-Larkin, G. R., Nesselroade, J. R. (2011). Emotional experience improves with age: Evidence based on over 10 years of experince sampling. *Psychology of Aging, 26*(1), 21–33. doi:10.1037/a0021285.

Carver, C. S. (1997). You want to measure coping but your protocol's too long: consider the brief COPE. *International Journal of Behavioral Medicine, 4*(1), 92–100. doi:10.1207/s15327558ijbm0401_6

Carver, C. S., Scheier, M. F. & Weintraub, J. K. (1989). Assessing Coping Strategies: A Theoretically Based Approach. *Journal of Personality and Social Psychology, 56*(2), 267–283. doi:10.1037/0022-3514.56.2.267

Chapman, B. P., Duberstein, P. R. & Lyness, J. M. (2007). The Distressed personality type: replicability and general health associations. *European Journal of Personality, 21*(7), 911–929. doi:10.1002/per.645

Chatterton, R. T., Vogelsong, K. M., Lu, Y., Ellman, A. B. & Hudgens, G. A. (1996). Salivary Alpha Amylase as a measure of Endongenous Adrenergic Activity. *Clinical Physiology and Functional Imaging, 16*(4), 43–448.

Chen, S. M., Creedy, D., Lin, H. S. & Wollin, J. (2012). Effects of motivational interviewing intervention on self-management, psychological and glycemic outcomes in type 2 diabetes: A randomized controlled trial. *International Journal of Nursing Studies, 49*(6), 637–644. doi:10.1016/j.ijnurstu.2011.11.011

Chrousos, G. P. (2009). Stress and disorders of the stress system. *Nature Reviews Endocrinology, 5*(7), 374–81.

Chrousos, G. P. & Gold, P. W. (1992). The Concepts of Stress and Stress System Disorders Overview of Physical and Behavioral Homeostasis. *Jama, 267*(9), 1244–52. doi:10.1001/jama.267.9.1244

Ciprandi, G., Cirillo, I., Klersy, C., Marseglia, G. L., Caimmi, D. & Vizzaccaro, A. (2005). Nasal obstruction is the key symptom in hay fever patients. *Otolaryngology - Head and Neck Surgery, 133*(3), 429–435. doi:10.1016/j.otohns.2005.05.049

Clow, A., Thorn, L., Evans, P. & Hucklebridge, F. (2004). The Awakening Cortisol Response: Methodological Issues and Significance. *Stress, 7*(1), 29–37. doi:10.1080/10253890410001667205

Cohen, H., Benjamin, J., Geva, A. B., Matar, M. A., Kaplan, Z. & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: Application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry Research, 96*(1), 1–13. doi:10.1016/S0165-1781(00)00195-5

Cohen, S., Doyle, W. J., Skoner, D. P., Gwaltney, Jr., J. M. & Newsom, J. T. (1995). State and trait negative affect as predictors of objective and subjective symptoms of respiratory viral infections. *Journal of Personality and Social Psychology*,*68*(1),159-169.

Cohen, S. & Hoberman, H. M. (1983). Positive Events and Social Supports as Buffers of Life Change Stress. *Journal of Applied Social Psychology, 13*(2), 99–125.

Cohen, S., Janicki-Deverts, D. & Miller, G. E. (2007). Psychological Stress and Disease. *Jama, 298*(14), 1685–1687. doi:10.1001/jama.298.14.1685

Cohen, S., Kamarck, T. W. & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*(4), 385–396.

Cohen, S., Tyrrell, D. A. & Smith, A. P. (1993). Negative life events, perceived stress, negative affect, and susceptibility to the common cold. *Journal of Personality and Social Psychology, 64*(1), 131–140. doi:10.1037/0022-3514.64.1.131

Cohen, S. & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin, 98*(2), 310–357. doi:10.1037/0033-2909.98.2.310

Compas, B. E. (2006). Psychobiological processes of stress and coping: Implications for resilience in children and adolescents. *Annals of the New York Academy of Sciences, 1094*(1), 226–234. doi:10.1196/annals.1376.024

Condén, E., Ekselius, L. & Aslund, C. (2013). Type D personality is associated with sleep problems in adolescents. Results from a population-based cohort study of Swedish adolescents. *Journal of Psychosomatic Research, 74*(4), 290–295. doi:10.1016/j.jpsychores.2012.11.011

Condén, E., Leppert, J., Ekselius, L. & Åslund, C. (2013). Type D personality is a risk factor for psychosomatic symptoms and musculoskeletal pain among adolescents: a cross-sectional study of a large population-based cohort of Swedish adolescents. *BMC Pediatrics, 13*(1), 11. doi:10.1186/1471-2431-13-11

Conraads, V. M., Denollet, J., De Clerck, L. S., Stevens, W. J., Bridts, C. & Vrints, C. J. (2006). Type D personality is associated with increased levels of tumour necrosis factor (TNF)-alpha and TNF-alpha receptors in chronic heart failure. *International Journal of Cardiology, 113*(1), 34–8. doi:10.1016/j.ijcard.2005.10.013

Cornwell, E. Y. & Waite, L. J. (2009). Social disconnectedness, perceived isolation, and health among older adults. *Journal of Health and Social Behavior, 50*(1), 31–48. doi:10.1177/002214650905000103

Costa, P. T. & McCrae, R. R. (1980). Influence of extraversion and neuroticism on subjective well-being: happy and unhappy people. *Journal of Personality and Social Psychology, 38*(4), 668–678. doi:10.1037/0022-3514.38.4.668

Coyne, J. C. & de Voogd, J. N. (2012). Are we witnessing the decline effect in the Type D personality literature? What can be learned? *Journal of Psychosomatic Research, 73*(6), 401–407. doi:10.1016/j.jpsychores.2012.09.016

Coyne, J. C., Jaarsma, T., Luttik, M.-L., van Sonderen, E., van Veldhuisen, D. J. & Sanderman, R. (2011). Lack of prognostic value of Type D personality for mortality in a large sample of heart failure patients. *Psychosomatic Medicine*, *73*(7), 557–62. doi:10.1097/PSY.0b013e318227ac75

Cronbach, L. J. (1951). Coefficient alpha and the internal structure of tests. *Psychometrika, 16*(3), 297–334. doi:10.1007/BF02310555

Dalgard, O. S., Bjork, S. & Tambs, K. (1995). Social support, negative life events and mental health. *The British Journal of Psychiatry*, *166*(1), 29–34. doi:10.1192/bjp.166.1.29

Dannemann, S., Matschke, K., Einsle, F., Smucker, M. R., Zimmermann, K., Joraschky, P., Köllner, V. (2010). Is Type-D a stable construct? An examination of Type-D personality in patients before and after cardiac surgery. *Journal of Psychosomatic Research, 69*(2), 101–9. doi:10.1016/j.jpsychores.2010.02.008

De Voogd, J. N., Wempe, J. B., Postema, K., Van Sonderen, E., Ranchor, A. V, Coyne, J. C. & Sanderman, R. (2009). More evidence that depressive symptoms predict mortality in COPD patients: Is Type D personality an alternative explanation? *Annals of Behavioral Medicine*, *38*(2), 86–93. doi:10.1007/s12160-009-9105-8

DeCaro, J. A. (2008). Methodological considerations in the use of salivary ??-amylase as a stress marker in field research. *American Journal of Human Biology, 20*(5), 617–619. doi:10.1002/ajhb.20795

Dembroski, T. M., MacDougall, J. M., Herd, A. J. & Shields, J. L. (1979). Effects of Types of Challenge on Pressor and Heart Rate Responses in Type A and B Subjects. J*ournal of Applied Social Psychology, 9*(3), 209–228. doi:10.1111/j.1469-8986.1981.tb01532.x

Denollet, J. (1993). Emotional distress and fatigue in coronary heart disease: the Global Mood Scale (GMS). *Psychological Medicine, 23*(1), 111–121.

Denollet, J. (1994). Health complaints and outcome assessment in coronary heart disease. *Psychosomatic Medicine, 56*(5), 463–474.

Denollet, J. (1998a). Personality and coronary heart disease: The Type D Scale-16 (DS16). *Annals of Behavioural Medicine, 20*(3), 209–215.

Denollet, J. (1998b). Personality and risk of cancer in men with coronary heart disease. *Psychological Medicine, 28*(4), 991–995. doi:10.1017/S0033291797006442

Denollet, J. (2000). Type D personality: A potential risk factor refined. *Journal of Psychosomatic Research, 49*(4), 255–266. doi:10.1016/S0022-3999(00)00177-X

Denollet, J. (2005). DS14: standard assessment of negative affectivity, social inhibition, and Type D personality. *Psychosomatic Medicine, 67*(1), 89–97. doi:10.1097/01.psy.0000149256.81953.49

Denollet, J., Conraads, V. M., Brutsaert, D. L., De Clerck, L. S., Stevens, W. J. & Vrints, C. J. (2003). Cytokines and immune activation in systolic heart failure: the role of Type D personality. *Brain, Behavior, and Immunity, 17*(4), 304–309. doi:10.1016/S0889-1591(03)00060-6

Denollet, J., de Jonge, P., Kuyper, A., Schene, A. H., van Melle, J. P., Ormel, J. & Honig, A. (2009). Depression and Type D personality represent different forms of distress in the Myocardial INfarction and Depression–Intervention Trial (MIND-IT). *Psychological Medicine, 39*(5), 749–756.

Denollet, J. & Kupper, N. (2007). Type-D personality, depression, and cardiac prognosis: cortisol dysregulation as a mediating mechanism. *Journal of Psychosomatic Research, 62*(6), 607–9. doi:10.1016/j.jpsychores.2007.04.008

Denollet, J., Pedersen, S. S., Vrints, C. J. & Conraads, V. M. (2006). Usefulness of Type D personality in predicting five-year cardiac events above and beyond concurrent symptoms of stress in patients with coronary heart disease. *The American Journal of Cardiology, 97*(7), 970–3. doi:10.1016/j.amjcard.2005.10.035

Denollet, J., Rombouts, H., Gillebert, T., Brutsaert, D. L., Sys, S. & Stroobant, N. (1996). Personality as independent predictor of long-term mortality in patients with coronary heart disease. *The Lancet, 347*(8999), 417–421. doi:10.1016/S0140-6736(96)90007-0

Denollet, J., Schiffer, A. A., Kwaijtaal, M., Hooijkaas, H., Hendriks, E. H., Widdershoven, J. W. & Kupper, N. (2009). Usefulness of Type D personality and kidney dysfunction as predictors of interpatient variability in inflammatory activation in chronic heart failure. *The American Journal of Cardiology, 103*(3), 399–404. doi:10.1016/j.amjcard.2008.09.096

Denollet, J., Sys, S. & Brutsaert, D. L. (1995). Personality and mortality after myocardial infarction. *Psychosomatic Medicine, 57*(6) 582-591.

Denollet, J., Vaes, J. & Brutsaert, D. L. (2000). Inadequate response to treatment in coronary heart disease: adverse effects of Type D personality and younger age on 5-year prognosis and quality of life. *Circulation, 102*(6), 630–635. doi:10.1161/01.CIR.102.6.630

Denollet, J., Vrints, C. J. & Conraads, V. M. (2008). Comparing Type D personality and older age as correlates of tumor necrosis factor-alpha dysregulation in chronic heart failure. *Brain, Behavior, and Immunity, 22*(5), 736–43. doi:10.1016/j.bbi.2007.10.015

Desborough, J. P. (2000). The stress response to trauma and surgery. *British Journal of Anaesthesia, 85*(1), 109–117. doi:10.1093/bja/85.1.109

Dickerson, S. S. & Kemeny, M. E. (2004). Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*(3), 355–391. doi:10.1037/0033-2909.130.3.355

Dickerson, S. S., Mycek, P. J. & Zaldivar, F. (2008). Negative social evaluation, but not mere social presence, elicits cortisol responses to a laboratory stressor task. *Health Psychology, 27*(1), 116–21. doi:10.1037/0278-6133.27.1.116

Dillon, M. C., Opris, D. C., Kopanczyk, R., Lickliter, J., Cornwell, H. N., Bridges, E. G., Bridges, K. G. (2010). Detection of homocysteine and C-reactive protein in the saliva of healthy adults: Comparison with blood levels. *Biomarker Insights, 5*, 57–61. doi:10.4137/BMI.S5305

Dishman, R. K., Nakamura, Y., Garcia, M. E., Thompson, R. W., Dunn, A. L. & Blair, S. N. (2000). Heart rate variability, trait anxiety, and percieved stress among physically fit men and women. *International Journal of Psychophysiology, 37*, 121–133.

Dooren, F. E. P., Verhey, F. R. J., Pouwer, F., Schalkwijk, C. G., Sep, S. J. S., Stehouwer, C. D. A., Denollet, J. (2015). Association of Type D personality with increased vulnerability to depression_ Is there a role for inflammation or endothelial dysfunction? – The Maastricht Study. *Journal of Affective Disorders, 189*(2016), 118–125. doi: 10.1016/j.jad.2015.09.028

Drossman, D. A., Camilleri, M., Mayer, E. A. & Whitehead, W. E. (2002). AGA technical review on irritable bowel syndrome. *Gastroenterology, 123*(6), 2108–2131. doi:10.1053/gast.2002.37095

Dua, J. K. (1994). Comparative predictive value of attributional style, negative affect, and positive affect in predicting self-reported physical health and psychological health. *Journal of Psychosomatic Research, 38*(7), 669–680.

Dulfer, K., Hazemeijer, B. A. F., Van Dijk, M. R., Van Geuns, R. J. M., Daemen, J., Van Domburg, R. T. & Utens, E. M. W. J. (2015). Prognostic value of Type D personality for 10-year mortality and subjective health status in patients treated with percutaneous coronary intervention. *Journal of Psychosomatic Research, 79*(3), 214–221. doi:10.1016/j.jpsychores.2015.05.014

Ehrström, S., Kornfeld, D. & Rylander, E. (2007). Perceived stress in women with recurrent vulvovaginal candidiasis. *Journal of Psychosomatic Obstetrics and Gynaecology, 28*(3), 169–176. doi:10.1080/01674820601168176

Einvik, G., Dammen, T., Hrubos-Strom, H., Namtvedt, S. K., Randby, A., Kristiansen, H. A., Omland, T. (2011). Prevalence of cardiovascular risk factors and concentration of C-reactive protein in Type D personality persons without cardiovascular disease. *European Journal of Cardiovascular Prevention and Rehabilitation, 18*(3), 504–509. doi:1741826710389383

Einvik, G., Dammen, T., Namtvedt, S. K., Hrubos-Strom, H., Randby, A., Kristiansen, H. A., Omland, T. (2014). Type D personality is associated with increased prevalence of ventricular arrhythmias in community-residing persons without coronary heart disease. *European Journal of Preventive Cardiology, 21*(5), 592–600.

Elder, G. J., Wetherell, M. A., Barclay, N. L. & Ellis, J. G. (2014). The cortisol awakening response--applications and implications for sleep medicine. *Sleep Medicine Reviews, 18*(3), 215–24. doi:10.1016/j.smrv.2013.05.001

Eliot, R. S., Buell, J. C. & Dembroski, T. M. (1982). Bio-behavioral perspectives on coronary heart disease, hypertension and sudden cardiac death. *Acta Medica Scandinavica, 606*, 203–219.

Emons, W. H. M., Meijer, R. R. & Denollet, J. (2007). Negative affectivity and social inhibition in cardiovascular disease: evaluating Type-D personality and its assessment using item response theory. *Journal of Psychosomatic Research, 63*(1), 27–39.

Eriksen, H., Ihlebæk, C. & Ursin, H. (1999). A scoring system for subjective health complaints (SHC). *Scandinavian Journal of Public Health, 1*, 63–72.

Everson, S. A., Lynch, J. W., Kaplan, G. A., Lakka, T. A., Sivenius, J., Salonen, J. T. & Matthews, K. A. (2001). Stress-Induced Blood Pressure Reactivity and Incident Stroke in Middle-Aged Men. *Stroke, 32*(6), 1263–1270. doi:10.1161/01.STR.32.6.1263

Ewing, D. J., Neilson, J. M. & Travis, P. (1984). New method for assessing cardiac parasympathetic activity using 24 hour electrocardiograms. *British Heart Journal, 52*(4), 396–402. doi:10.1136/hrt.52.4.396

Eysenck, H. J. & Eysenck, S. B. (1976). *Eysenck personality questionnaire*. Educational and industrial testing service.

Farmer, M. & Ferraro, K. (1997). Distress and perceived health: mechanisms of health decline. *Journal of Health and Social Behavior, 38*(3),298–311.

Farrington, D. P. (1991). Longitudinal Research Strategies: Advantages, Problems, and Prospects. *Journal of the American Academy of Child & Adolescent Psychiatry*, *30*(3), 369–374. doi:10.1097/00004583-199105000-00003

Ferguson, E., Williams, L., O'Connor, R. C., Howard, S., Hughes, B. M., Johnston, D. W., O'Carroll, R. E. (2009). A taxometric analysis of Type D personality. *Psychosomatic Medicine, 71*(9), 981–986.

Fitzmaurice, G. M., Laird, N. M. & Ware, J. (2012). *Applied Longitudinal Analysis*. John Wiley & Sons.

Fries, J. F., Spitz, P. R., Kraines, G. & Holman, H. R. (1980). Measurement of Patient Outcome in Arthritis. *Arthritis & Rheumatism, 23*(2), 137–145.

Fruyt, F. De, Denollet, J., De Fruyt, F. & Denollet, J. (2002). Type D personality: A five-factor model perspective. *Psychology and Health, 17*(5), 671–683. doi:10.1080/08870440290025858

Garcia-Leal, C., Graeff, F. G. & Del-Ben, C. M. (2014). Experimental public speaking: Contributions to the understanding of the serotonergic modulation of fear. *Neuroscience and Biobehavioral Reviews, 46*(3), 407–417. doi:10.1016/j.neubiorev.2014.09.011

Garnefski, N., Legerstee, J., Kraaij, V., Van Den Kommer, T. & Teerds, J. (2002). Cognitive coping strategies and symptoms of depression and anxiety: a comparison between adolescents and adults. *Journal of Adolescence, 25*(6), 603–611. doi:10.1006/jado.2002.0507

Geuens, N., Braspenning, M., Van Bogaert, P. & Franck, E. (2015). Individual vulnerability to burnout in nurses: The role of Type D personality within different nursing specialty areas. *Burnout Research, 2*(2), 80–86. doi:10.1016/j.burn.2015.05.003

Gijsbers Van Wijk, C. M. T. & Kolk, A. M. (1997). Sex differences in physical symptoms: The contribution of symptom perception theory. *Social Science and Medicine, 45*(2), 231–246. doi:10.1016/S0277-9536(96)00340-1

Gilmour, J. & Williams, L. (2012). Type D personality is associated with maladaptive health-related behaviours. *Journal of Health Psychology, 17*(4), 471–8. doi:10.1177/1359105311423117

Ginty, A. T., Phillips, A. C., Higgs, S., Heaney, J. L. J. & Carroll, D. (2012). Disordered eating behaviour is associated with blunted cortisol and cardiovascular reactions to acute psychological stress. *Psychoneuroendocrinology, 37*(5), 715–724. doi:10.1016/j.psyneuen.2011.09.004

Glaser, R. & Kiecolt-Glaser, J. K. (2005). Science and society: Stress-induced immune dysfunction: implications for health. *Nature Reviews Immunology, 5*(3), 243–251. doi:10.1038/nri1571

Goodin, B. R., Smith, M. T., Quinn, N. B., King, C. D. & McGuire, L. (2012). Poor sleep quality and exaggerated salivary cortisol reactivity to the cold pressor task predict greater acute pain severity in a non-clinical sample. *Biological Psychology, 91*(1), 36–41. doi:10.1016/j.biopsycho.2012.02.020

Gordis, E. B., Granger, D. A., Susman, E. J. & Trickett, P. K. (2008). Salivary alpha amylase-cortisol asymmetry in maltreated youth. *Hormones and Behavior, 53*(1), 96–103. doi:10.1016/j.yhbeh.2007.09.002

Gorman, J. M. & Sloan, R. P. (2000). Heart rate variability in depressive and anxiety disorders. *American Heart Journal, 140*(4), 77–83. doi:10.1067/mhj.2000.109981

Grande, G., Glaesmer, H. & Roth, M. (2010). The construct validity of social inhibition and the Type-D taxonomy. *Journal of Health Psychology, 15*(7), 1103–12. doi:10.1177/1359105310369186

Grande, G. & Romppel, M. (2011). Type D personality and all-cause mortality in cardiac patients-data from a German cohort study. *Psychosomatic Medicine, 73*(7), 548–556.

Grande, G., Romppel, M. & Barth, J. (2012). Association between Type D personality and prognosis in patients with cardiovascular diseases: a systematic review and meta-analysis. *Annals of Behavioral Medicine, 43*(3), 299–310. doi:10.1007/s12160-011-9339-0

Grant, A. M. (2012). Making Positive Change: A Randomized Study Comparing Solution-Focused vs. Problem-Focused Coaching Questions. *Journal of Systemic Therapies, 31*(2), 21–35. doi:10.1521/jsyt.2012.31.2.21

Gregg, M. E., James, J. E., Matyas, T. A. & Thorsteinsson, E. B. (1999). Haemodynamic profile of stress-induced anticipation and recovery. *International Journal of Psychophysiology, 34*(2), 147–162. doi:10.1016/S0167-8760(99)00074-4

Gregg, M. E., Matyas, T. A & James, J. E. (2002). A new model of individual differences in haemodynamic profile and blood pressure reactivity. *Psychophysiology, 39*(1), 64–72. doi:10.1017/S0048577202001154

Gregory, A. M. & O'Connor, T. G. (2002). Sleep Problems in Childhood: A Longitudinal Study of Developmental Change and Association with Behavioral Problems. *Journal of the American Academy of Child & Adolescent Psychiatry, 41*(8), 964–971. doi:10.1097/00004583-200208000-00015

Grippo, A. J. & Johnson, A. K. (2002). Biological mechanisms in the relationship between depression and heart disease. *Neuroscience and Biobehavioral Reviews, 26*(8), 941–962. doi:10.1016/S0149-7634(03)00003-4

Grossman, P., Niemann, L., Schmidt, S. & Walach, H. (2004). Mindfulness-based stress reduction and health benefits: A meta-analysis. *Journal of Psychosomatic Research, 57*(1), 35–43. doi:10.1016/S0022-3999(03)00573-7

Habra, M. E., Linden, W., Anderson, J. C. & Weinberg, J. (2003). Type D personality is related to cardiovascular and neuroendocrine reactivity to acute stress. *Journal of Psychosomatic Research, 55*(3), 235–245. doi:10.1016/S0022-3999(02)00553-6

Hair, J. F., Anderson, R. E., Tatham, R. L. & Black, W. C. (1998). *Multivariate Data Analysis.* New Jersey, USA: Englewood Cliff.

Hall, J. M., Podawiltz, A., Mummert, D. I., Jones, H. & Mummert, M. E., (2012). Psychological stress and the cutaneous immune response: roles of the HPA axis and the sympathetic nervous system in atopic dermatitis and psoriasis. *Dematology Research and Practice*. doi:10.1155/2012/403908.

Hamer, M., Endrighi, R., Venuraju, S. M., Lahiri, A. & Steptoe, A. (2012). Cortisol responses to mental stress and the progression of coronary artery calcification in healthy men and women. *PloS One, 7*(2), 1-6. doi:10.1371/journal.pone.0031356

Hamer, M., O'Donnell, K., Lahiri, A. & Steptoe, A. (2010). Salivary cortisol responses to mental stress are associated with coronary artery calcification in healthy men and women. *European Heart Journal, 31*(4), 424–9. doi:10.1093/eurheartj/ehp386

Hanebuth, D., Meinel, M. & Fischer, J. E. (2006). Health-related quality of life, psychosocial work conditions, and absenteeism in an industrial sample of blue- and white-collar employees: a comparison of potential predictors. *Journal of Occupational and Environmental Medicine, 48*(1), 28–37. doi:10.1097/01.jom.0000195319.24750.f8

Hansel, S. L., Umar, S. B., Lunsford, T. N., Harris, L. A., Dibaise, J. K. & Crowell, M. D. (2010). Personality Traits and Impaired Health-Related Quality of Life in Patients with Functional Gastrointestinal Disorders. *Clinical Gastroenterology and Hepatology, 8*(2), 220–222. doi: 10.1016/J.Cgh.2009.10.008

Harris, A. H. & Thoresen, C. E. (2006). Extending the influence of positive psychology interventions into health care settings: Lessons from self-efficacy and forgiveness. *The Journal of Positive Psychology, 1*(1), 27–36. doi:10.1080/17439760500380930

Harris, K. F. & Matthews, K. (2004). Interactions between autonomic nervous system activity and endothelial function: a model for the development of cardiovascular disease. *Psychosomatic Medicine, 66*(2),153-164.

Harris, M. A., Brett, C. E., Johnson, W. & Deary, I. J. (2016). Personality stability from age 14 to age 77 years. *Psychology and Aging, 31*(8), 862–874. doi:10.1037/pag0000133

Hart, S. G. & Staveland, L. E. (1988). Development of NASA-TLX (Task Load Index): Results of Empirical and Theoretical Research. *Advances in Psychology, 52*, 139–183. doi:10.1016/S0166-4115(08)62386-9

Haug, T. T., Mykletun, A. & Dahl, A. A. (2002). Are anxiety and depression related to gastrointestinal symptoms in the general population? *Scandinavian Journal of Gastroenterology, 37*(3), 294–8. doi:10.1080/003655202317284192

Haug, T. T., Mykletun, A. & Dahl, A. A. (2004). The Association Between Anxiety, Depression, and Somatic Symptoms in a Large Population: The HUNT-II Study. *Psychosomatic Medicine, 66*(6), 845–851. doi:10.1097/01.psy.0000145823.85658.0c

Hausteiner, C., Klupsch, D., Emeny, R., Baumert, J. & Ladwig, K.-H. (2010). Clustering of negative affectivity and social inhibition in the community: prevalence of Type D personality as a cardiovascular risk marker. *Psychosomatic Medicine, 72*(2), 163–71. doi:10.1097/PSY.0b013e3181cb8bae

Hinkin, T. R. (1995). A Review of Scale Development Practices in the Study of Organizations. *Journal of Management, 21*(5), 967–988. doi:10.1177/014920639502100509

Hinkin, T. R. (1998). A Brief Tutorial on the Development of Measures for Use in Survey Questionnaires. *Organizational Research methods 1*(1), 104-121. doi:10.1017/CBO-9781107415324.004

Holmes, T. H. & Rahe, R. H. (1967). The social readjustment rating scale. *Journal of Psychosomatic Medicine, 11*(2), 213–218.

Horwood, S., Anglim, J. & Tooley, G. (2016). Statistically Modelling the Relationships between Type D Personality and Social Support, Health Behaviors and Symptom Severity in Chronic Illness Groups. *Psychology & Health, 31*(9) 1047-1063. doi:10.1080/08870446.2016.1167209

Horwood, S., Chamravi, D. & Tooley, G. (2015). Examining the Prevalence of Type-D Personality in an Australian Population. *Australian Psychologist, 50*(3),212-218.

Howard, S. & Hughes, B. M. (2012). Construct, concurrent and discriminant validity of Type D personality in the general population: associations with anxiety, depression, stress and cardiac output. *Psychology & Health, 27*(2), 242–58. doi:10.1080/08870446.2011.603423

Howard, S. & Hughes, B. M. (2013). Type D personality is associated with a sensitized cardiovascular response to recurrent stress in men. *Biological Psychology, 94*(2), 450–5. doi:10.1016/j.biopsycho.2013.09.001

Howard, S., Hughes, B. M. & James, J. E. (2011). Type D personality and haemodynamic reactivity to laboratory stress in women. *International Journal of Psychophysiology, 80*(2), 96–102. doi:10.1016/j.ijpsycho.2011.02.006

Huffman, J. C., Mastromauro, C. a., Boehm, J. K., Seabrook, R., Fricchione, G. L., Denninger, J. W. & Lyubomirsky, S. (2011). Development of a positive psychology intervention for patients with acute cardiovascular disease. *Heart International, 6*(2), 47–54. doi:10.4081/hi.2011.e13

Huffman, J. C., Millstein, R. a., Mastromauro, C. A., Moore, S. V., Celano, C. M., Bedoya, C. A., Januzzi, J. L. (2016). A Positive Psychology Intervention for Patients with an Acute Coronary Syndrome: Treatment Development and Proof-of-Concept Trial. *Journal of Happiness Studies, 17*(5), 1985–2006. doi:10.1007/s10902-015-9681-1

Hunt, S. M., Mcewen, J. & Mckenna, M. S. R. (1985). Measuring health status: a new tool for clinicians and epidemiologists. *Journal of the Royal College of General Practitioners, 35*(273), 185–188. doi:10.1037/0021-9010.76.5.644

Hutcheson, G. D. & Sofroniou, N. (1999). *The Multivariate Social Scientist: Introductory Statistic Using Generalized Linear Models*. London: SAGE publications.

Ihlebaek, C., Eriksen, H. R. & Ursin, H. (2002). Prevalence of subjective health complaints (SHC) in Norway. *Scandinavian Journal of Public Health, 30*(1), 20–29. doi:10.1080/140349401753481547

Jacobsen, B. K. & Thelle, D. S. (1988). The Tromso Heart Study: responders and non-responders to a health questionnaire, do they differ? *Scandinavian Journal of Social Medicine, 16*(2), 101–104

James, J. E., Gregg, E. D. M., Matyas, T. A., Hughes, B. M. & Howard, S. (2012). Stress reactivity and the Haemodynamic Profile-Compensation Deficit (HP-CD) Model of blood pressure regulation. *Biological Psychology, 90*(2), 161–170. doi:10.1016/j.biopsycho.2012.02.021

Jarcho, M. R., Slavich, G. M., Tylova-Stein, H., Wolkowitz, O. M. & Burke, H. M. (2013). Dysregulated diurnal cortisol pattern is associated with glucocorticoid resistance in women with major depressive disorder. *Biological Psychology, 93*(1), 150–158. doi:10.1016/j.biopsycho.2013.01.018

Jellesma, F. C. (2008). Health in young people: social inhibition and negative affect and their relationship with self-reported somatic complaints. *Journal of Developmental and Behavioral Pediatrics, 29*(2), 94–100. doi:10.1097/DBP.0b013e31815f24e1

Johansson, J. A. & Rubenowitz, S. (1994). Risk indicators in the psychosocial and physical work environment for work-related neck, shoulder and low back symptoms: a study among blue- and white-collar workers in eight companies. *Scandinavian Journal of Rehabilitation Medicine, 26*(3), 131–142.

Jonge, P. de, Denollet, J. & Melle, J. van. (2007). Associations of Type-D personality and depression with somatic health in myocardial infarction patients. *Journal of Psychosomatic Research, 63*(5), 477–482.

Joseph, S. (2015). *Positive psychology in practice: Promoting human flourishing in work, health, education, and everyday life*. John Wiley & Sons.

Julius, S. (1988). Transition from high cardiac output to elevated vascular resistance in hypertension. *American Heart Journal, 116*(2), 600–606.

Juruena, M. F. (2014). Early-life stress and HPA axis trigger recurrent adulthood depression. *Epilepsy and Behavior, 38*, 148–159. doi:10.1016/j.yebeh.2013.10.020

Juster, R. P., McEwen, B. S. & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews, 35*(1), 2–16. doi:10.1016/j.neubiorev.2009.10.002

Kaiser, H. F. (1958). The varimax criterion for analytic rotation in factor analysis. *Psychometrika, 23*(3), 187–200. doi:10.1007/BF02289233

Kamarck, T. W., Everson, S. A., Kaplan, G. A., Manuck, S. B., Jennings, J. R., Salonen, R. & Salonen, J. T. (1997). Exaggerated blood pressure responses during mental stress are associated with enhanced carotid atherosclerosis in middle-aged Finnish men. *Circulation, 96*(11), 3842–3848.

Kamarck, T. W. & Lovallo, W. R. (2003). Cardiovascular Reactivity to Psychological Challenge: Conceptual and Measurement Considerations. *Psychosomatic Medicine, 65*(1), 9–21. doi:10.1097/01.PSY.0000030390.34416.3E

Kaplan, G. A., Wilson, T. W., Cohen, R. D., Kauhanen, J., Wu, M. & Salonen, J. T. (1994). Social Functioning and Overall Mortality: Prospective Evidence from the Kuopio Ischemic Heart Disease Risk Factor Study. *Epidemiology, 5*(5) 495-500.

Kaplan, R. M., Bush, J. W. & Berry, C. C. (1979). Health Status Index: Category Rating versus Magnitude Estimation for Measuring Levels of Well-Being. *Medical Care, 17*(5), 501–525.

Kelly, J. T. & Busse, W. W. (2008). Host immune responses to rhinovirus: Mechanisms in asthma. *Journal of Allergy and Clinical Immunology, 122*(4), 671–682. doi:10.1016/j.jaci.2008.08.013

Kelly-Hughes, D. H., Wetherell, M. A. & Smith, M. A. (2014). Type D personality and cardiovascular reactivity to an ecologically valid multitasking stressor. *Psychology & Health, 29*(10), 1156–75. doi:10.1080/08870446.2014.915970

Kiecolt-Glaser, J. K. & Glaser, R. (2002). Depression and immune function central pathways to morbidity and mortality. *Journal of Psychosomatic Research, 53*(4), 873–876. doi:10.1016/S0022-3999(02)00309-4

Kiecolt-Glaser, J. K., Glaser, R., Shuttleworth, E. C., Dyer, C. S., Ogrocki, P. & Speicher, C. E. (1987). Chronic stress and immunity in family caregivers of Alzheimer's disease victims. *Psychosomatic Medicine, 49*(5), 523–535. doi:10.1097/00006842-198709000-00008

Kim, E. J. & Dimsdale, J. E. (2007). The effect of psychosocial stress on sleep: a review of polysomnographic evidence. *Behavioral Sleep Medicine, 5*(4), 256–78. doi:10.1080/15402000701557383

King, L. A. (2001). The Health Benefits of Writing about Life Goals. *Personality and Social Psychology Bulletin, 27*(7), 798–807. doi:10.1177/0146167201277003

King, L. A. & Miner, K. N. (2000). Writing About the Perceived Benefits of Traumatic Events: Implications for Physical Health. *Personality and Social Psychology Bulletin, 26*(2), 220–230. doi:10.1177/0146167200264008

Kirschbaum, C. & Hellhammer, D. H. (1989). Salivary Cortisol in Psychobiological Research: An Overview. *Neuropsychobiology, 22*(3), 150–169.

Kirschbaum, C., Pirke, K. M. & Hellhammer, D. H. (1993). The "Trier Social Stress Test"-a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*(1-2), 76–81. doi: 10.1159/000119004

Kok, B. E., Coffey, K. a., Cohn, M. a., Catalino, L. I., Vacharkulksemsuk, T., Algoe, S. B., Fredrickson, B. L. (2013). How Positive Emotions Build Physical Health: Perceived Positive Social Connections Account for the Upward Spiral Between Positive Emotions and Vagal Tone. *Psychological Science, 24*(7), 1123–1132. doi:10.1177/0956797612470827

Korotkov, D. (2008). Does personality moderate the relationship between stress and health behavior? Expanding the nomological network of the five-factor model. *Journal of Research in Personality, 42*(6), 1418–1426. doi:10.1016/j.jrp.2008.06.003

Korotkov, D. & Hannah, T. E. (2004). The Five-Factor Model of Personality: strengths and limitations in predicting health status, sick-role and illness behaviour. *Personality and Individual Differences, 36*(1), 187–199. doi:10.1016/S0191-8869(03)00078-3

Kroenke, K. & Price, R. K. (1993). Symptoms in the Community: prevalence, classification, and psychiatric comorbidity. *Archives of Internal Medicine, 153*(21), 2474. doi:10.1001/archinte.1993.00410210102011

Kroenke, K. & Spitzer, R. L. (1998). Gender differences in the reporting of physical and somatoform symptoms. *Psychosomatic Medicine, 60*(2), 150–155. doi:10.1.1.330.5685

Kudielka, B. M., Hellhammer, D. H. & Wüst, S. (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology, 34*(1), 2–18. doi:10.1016/j.psyneuen.2008.10.004

Kudielka, B. M., Von Känel, R., Preckel, D., Zgraggen, L., Mischler, K. & Fischer, J. E. (2006). Exhaustion is associated with reduced habituation of free cortisol responses to repeated acute psychosocial stress. *Biological Psychology, 72*(2), 147–153. doi:10.1016/j.biopsycho.2005.09.001

Kunst, M. J. J., Bogaerts, S. & Winkel, F. W. (2009). Peer and inmate aggression, Type D-personality and post-traumatic stress among Dutch prison workers. *Stress and Health, 25*(5), 387–395. doi:10.1002/smi.1247

Kuo, H.-K., Yen, C.-J., Chang, C.-H., Kuo, C.-K., Chen, J.-H. & Sorond, F. (2005). Relation of C-reactive protein to stroke, cognitive disorders, and depression in the general population: systematic review and meta-analysis. *The Lancet Neurology, 4*(6), 371–80. doi:10.1016/S1474-4422(05)70099-5

Kupper, N. & Boomsma, D. (2011). Nine-year stability of Type D personality: contributions of genes and environment. *Psychosomatic Medicine, 73*(1), 75–82.

Kupper, N. & Denollet, J. (2007). Type D personality as a prognostic factor in heart disease: assessment and mediating mechanisms. *Journal of Personality Assessment, 89*(3), 265–76. doi:10.1080/00223890701629797

Kupper, N., Denollet, J., de Geus, E. J. C., Boomsma, D. & Willemsen, G. (2007). Heritability of type-D personality. *Psychosomatic Medicine, 69*(7), 675–81. doi:10.1097/PSY.0b013e318149f4a7

Kupper, N., Denollet, J., Widdershoven, J. & Kop, W. J. (2015). Cardiovascular reactivity to mental stress and mortality in patients with heart failure. *JACC: Heart Failure,* 3(5), 373–382. doi:10.1016/j.jchf.2014.12.016

Kupper, N., Pelle, A. & Denollet, J. (2013). Association of Type D personality with the autonomic and haemodynamic response to the cold pressor test. *Psychophysiology, 50*(12), 1194–1201. doi:10.1111/psyp.12133

Laederach-Hofmann, K., Mussgay, L., Büchel, B., Widler, P. & Rüddel, H. (2002). Patients with erythrophobia (fear of blushing) show abnormal autonomic regulation in mental stress conditions. *Psychosomatic Medicine, 64*(2), 358–65.

Landgraf, J. M., Abetz, L. & Ware, J. E. (1996). Child Health Questionnaire (CHQ): A user's manual. Boston: Health Institute, New England Medical Center.

Langewouters, G. J., Settels, J. J., Roelandt, R. & Wesseling, K. H. (1998). Why use Finapres rather than intra-arterial or intermittent techniques of blood pressure measurement? *Journal of Medical Engineering & Technology, 22*(1), 37–43. doi:10.3109/03091909809009997

Lasikiewicz, N., Hendrickx, H., Talbot, D. & Dye, L. (2008). Exploration of basal diurnal salivary cortisol profiles in middle-aged adults: Associations with sleep quality and metabolic parameters. *Psychoneuroendocrinology, 33*(2), 143–151. doi:10.1016/j.psyneuen.2007.10.013

Layous, K., Katherine Nelson, S. & Lyubomirsky, S. (2013). What Is the Optimal Way to Deliver a Positive Activity Intervention? The Case of Writing About One's Best Possible Selves. *Journal of Happiness Studies, 14*(2), 635–654. doi:10.1007/s10902-012-9346-2

Lee, Y. C., Chibnik, L. B., Lu, B., Wasan, A. D., Edwards, R. R., Fossel, A. H., Karlson, E. W. (2009). The relationship between disease activity, sleep, psychiatric distress and pain sensitivity in rheumatoid arthritis: a cross-sectional study. *Arthritis Research & Therapy, 11*(5), R160. doi:10.1186/ar2842

Lefever, S., Dal, M. & Matthíasdóttir, Á. (2007). Online data collection in academic research: Advantages and limitations. *British Journal of Educational Technology, 38*(4), 574–582. doi:10.1111/j.1467-8535.2006.00638.x

Lepore, S. J., Miles, H. J. & Levy, Jodi, S. (1997). Relation of Chronic and Episodic Stressors to Psychological Distress, Reactivity, and Health Problems. *International Journal of Behavioral Medicine, 4*(1), 39–59. doi:10.1207/s15327558ijbm0401_3

Leventhal, E. A., Hansell, S., Diefenbach, M., Leventhal, H. & Glass, D. C. (1996). Negative affect and self-report of physical symptoms: two longitudinal studies of older adults. *Health Psychology, 15*(3), 193–199. doi:10.1037/0278-6133.15.3.193

Licht, C. M. M., De Geus, E. J. C. & Penninx, B. W. J. H. (2013). Dysregulation of the autonomic nervous system predicts the development of the metabolic syndrome. *Journal of Clinical Endocrinology and Metabolism, 98*(6), 2484–2493. doi:10.1210/jc.2012-3104

Lindsay, G. M., Smith, L. N., Hanlon, P. & Wheatley, D. J. (2001). The influence of general health status and social support on symptomatic outcome following coronary artery bypass grafting. *Heart, 85*(1), 80–6. doi:10.1136/HEART.85.1.80

Lovallo, W. R., Farag, N. H., Sorocco, K. H., Acheson, A., Cohoon, A. J. & Vincent, A. S. (2013). Early life adversity contributes to impaired cognition and impulsive behavior: studies from the Oklahoma Family Health Patterns Project. *Alcoholism: clinical and experimental research, 37*(4), 616-623.

Lund, H. G., Reider, B. D., Whiting, A. B. & Prichard, J. R. (2010). Sleep Patterns and Predictors of Disturbed Sleep in a Large Population of College Students. *Journal of Adolescent Health, 46*(2), 124–132. doi:10.1016/j.jadohealth.2009.06.016

Lundgren-Nilsson, Å., Jonsdottir, I. H., Pallant, J. & Ahlborg, G. (2012). Internal construct validity of the Shirom-Melamed Burnout Questionnaire (SMBQ). *BMC Public Health, 12*(1), 1. doi:10.1186/1471-2458-12-1

Lyubomirsky, S., Sousa, L. & Dickerhoof, R. (2006). The costs and benefits of writing, talking, and thinking about life's triumphs and defeats. *Journal of Personality and Social Psychology, 90*(4), 692–708. doi:10.1037/0022-3514.90.4.692

Malik, M., Bigger, J., Camm, A. & Kleiger, R. (1996). Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *European Heart Journal, 17*, 354–381. doi:0195-668X/96/030354 + 28

Mancia, G., Bousquet, P., Elghozi, J. L., Esler, M., Grassi, G., Julius, S. & Van Zwieten, P. A. (2007). The sympathetic nervous system and the metabolic syndrome. Journal of hypertension, 25(5), 909-920.

Mansukhani, M. P., Kara, T., Caples, S. M. & Somers, V. K. (2014). Chemoreflexes, sleep apnea, and sympathetic dysregulation. *Current Hypertension Reports, 16*(9), 476. doi:10.1007/s11906-014-0476-2

Mantella, R. C., Butters, M. A., Amico, J. A., Mazumdar, S., Rollman, B. L., Begley, A. E., … Lenze, E. J. (2008). Salivary cortisol is associated with diagnosis and severity of late-life generalized anxiety disorder. *Psychoneuroendocrinology, 33*(6), 773–781. doi:10.1016/j.psyneuen.2008.03.002

Marshall, P., Diesch, J. P. & Hainsworth, R. (2004). Cardiac output by Portapres®. *Clinical Science, 106*(4), 407–412. doi:10.1042/CS20030279

Marteau, T. M. & Bekker, H. (1992). The development of a six-item short-form of the state scale of the Spielberger State-Trait Anxiety Inventory (STAI). *The British Journal of Clinical Psychology, 31*(3), 301–306. doi:10.1111/j.2044-8260.1992.tb00997.x

Martens, E. & Kupper, N. (2007). Type-D personality is a stable taxonomy in post-MI patients over an 18-month period. *Journal of Psychosomatic Research, 63*(5), 545–550.

Martin, L. A., Doster, J. A., Critelli, J. W., Lambert, P. L., Purdum, M., Powers, C. & Prazak, M. (2010). Ethnicity and Type D personality as predictors of heart rate variability. *International Journal of Psychophysiology, 76*(2), 118–21. doi:10.1016/j.ijpsycho.2010.03.001

Maslach, C., Schaufeli, W. B. & Leiter, M. P. (2001). Job Burnout. *Annual Reviews in Psychology, 52*, 397–422. doi:10.1146/annurev.psych.52.1.397

Matheson, E. M., Player, M. S., Mainous, A. G., King, D. E. & Everett, C. J. (2008). The association between hay fever and stroke in a cohort of middle aged and elderly adults. *Journal of the American Board of Family Medicine, 21*(3), 179–83. doi:10.3122/jabfm.2008.03.070273

Matsumoto, T., Ushiroyama, T., Kimura, T., Hayashi, T. & Moritani, T. (2007). Altered autonomic nervous system activity as a potential etiological factor of premenstrual syndrome and premenstrual dysphoric disorder. *Biopsychosocial Medicine, 1*(1), 24. doi:10.1186/1751-0759-1-24

Matthews, K. A., Salomon, K., Brady, S. S. & Allen, M. T. (2003). Cardiovascular Reactivity to Stress Predicts Future Blood Pressure in Adolescence. *Psychosomatic Medicine, 65*(3), 410–415. doi:10.1097/01.PSY.0000057612.94797.5F

Maxwell, S. E. & Cole, D. A. (2007). Bias in cross-sectional analyses of longitudinal mediation. *Psychological Methods, 12*(1), 23–44. doi:10.1037/1082-989x.12.1.23

Mazur, M., Furgała, A., Jabłonski, K., Mądroszkiewicz, D., Ciećko-Michalska, I., Bugajski, A. & Thor, P. J. (2007). Dysfunction of the autonomic nervous system activity is responsible for gastric myoelectric disturbances in the irritable bowel syndrome patients. *Journal of Physiology and Pharmacology, 58*(3), 131–139. doi:17901589

McCrae, R. R. & Costa, P. T. (1987). Validation of the five-factor model of personality across instruments and observers. *Journal of Personality and Social Psychology, 52*(1), 81–90. doi:10.1037/0022-3514.52.1.81

McEniery, C. M., Yasmin, Wallace, S., Maki-Petaja, K., McDonnell, B., Sharman, J. E., … Wilkinson, I. B. (2005). Increased stroke volume and aortic stiffness contribute to isolated systolic hypertension in young adults. *Hypertension, 46*(1), 221–226. doi:10.1161/01.HYP.0000165310.84801.e0

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *The New England Journal of Medicine, 338*(3), 171–9. doi:10.1056/NEJM199801153380307

McEwen, B. S. (2000). Allostasis and allostatic load: Implications for neuropsychopharmacology. *Neuropsychopharmacology 22*(2), 108-124. doi:10.1016/S0893-133X(99)00129-3

McGirr, A., Diaconu, G., Berlim, M. T., Pruessner, J. C., Sable, R., Cabot, S. & Turecki, G. (2010). Dysregulation of the sympathetic nervous system, hypothalamic–pituitary–adrenal axis and executive function in individuals at risk for suicide. *Journal of Psychiatry & Neuroscience, 35*(6), 399–408. doi:10.1503/jpn.090121

McGonigal, K. (2013). How to make stress your friend. TED Talk Video Online. Bit. ly/1z7kDLE.

McGreal, R. & Joseph, S. (1993). The Depression-Happiness Scale. *Psychological Reports, 73*(3), 1279–1282.

Meade, T. W., Roderick, P. J., Brennan, P. J., Wilkes, H. C. & Kelleher, C. C. (1992). Extra-cranial bleeding and other symptoms due to low dose aspirin and low intensity oral anticoagulation. *Thrombosis and Haemostasis, 68*(1), 1–6.

Meerlo, P., Sgoifo, A. & Suchecki, D. (2008). Restricted and disrupted sleep: Effects on autonomic function, neuroendocrine stress systems and stress responsivity. *Sleep Medicine Reviews, 12*(3), 197–210. doi:10.1016/j.smrv.2007.07.007

Melamed, S., Shirom, A., Toker, S., Berliner, S. & Shapira, I. (2006). Burnout and risk of cardiovascular disease: evidence, possible causal paths, and promising research directions. *Psychological Bulletin, 132*(3), 327–53. doi:10.1037/0033-2909.132.3.327

Meyer, T. J., Miller, M. L., Metzger, R. L. & Borkovec, T. D. (1990). Development and validation of the penn state worry questionnaire. *Behaviour Research and Therapy, 28*(6), 487–495. doi:10.1016/0005-7967(90)90135-6

Michal, M., Wiltink, J., Grande, G., Beutel, M. E. & Brähler, E. (2011). Type D personality is independently associated with major psychosocial stressors and increased health care utilization in the general population. *Journal of Affective Disorders, 134*(1-3), 396–403. doi:10.1016/j.jad.2011.05.033

Michal, M., Wiltink, J., Till, Y., Wild, P. S., Münzel, T., Blankenberg, S. & Beutel, M. E. (2010). Type-D personality and depersonalization are associated with suicidal ideation in the German general population aged 35-74: results from the Gutenberg Heart Study. *Journal of Affective Disorders, 125*(1-3), 227–33. doi:10.1016/j.jad.2010.02.108

Miilunpalo, S., Vuori, I., Oja, P., Pasanen, M. & Urponen, H. (1997). Self-rated health status as a health measure: The predictive value of self-reported health status on the use of physician services and on mortality in the working-age population. *Journal of Clinical Epidemiology, 50*(5), 517–528. doi:10.1016/S0895-4356(97)00045-0

Miller, G., Chen, E. & Cole, S. W. (2009). Health psychology: developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60*, 501–24. doi:10.1146/annurev.psych.60.110707.163551

Mills, P. J. & Ziegler, M. G. (2007). Sympathetic Hormones in Health Psychology Research. In *Handbook of Physiological Research Methods in Health Psychology* (75–94).

Mills, R. T. & Krantz, D. S. (1979). Information, choice, and reactions to stress: A field experiment in a blood bank with laboratory analogue. *Journal of Personality and Social Psychology, 37*(4), 608–620. doi:10.1037/0022-3514.37.4.608

Mischel, W. (1969). Continuity and change in personality. *American Psychologist, 24*(11), 1012–1018. doi:10.1037/h0028886

Molloy, G. J., Perkins-Porras, L., Strike, P. C. & Steptoe, A. (2008). Type-D personality and cortisol in survivors of acute coronary syndrome. *Psychosomatic Medicine*, *70*(18), 863–868. doi:10.1097/PSY.0b013e3181842e0c

Mols, F. & Denollet, J. (2010a). Type D personality among noncardiovascular patient populations: a systematic review. *General Hospital Psychiatry*, *32*(1), 66–72. doi:10.1016/j.genhosppsych.2009.09.010

Mols, F. & Denollet, J. (2010b). Type D personality in the general population: a systematic review of health status, mechanisms of disease, and work-related problems. *Health and Quality of Life Outcomes*, *8*(1), 9. doi:10.1186/1477-7525-8-9

Mols, F., Denollet, J., Kaptein, A. a, Reemst, P. H. M. & Thong, M. S. Y. (2012). The association between Type D personality and illness perceptions in colorectal cancer survivors: a study from the population-based PROFILES registry. *Journal of Psychosomatic Research*, *73*(3), 232–9. doi:10.1016/j.jpsychores.2012.07.004

Mols, F., Thong, M. S. Y., van de Poll-Franse, L. V, Roukema, J. A. & Denollet, J. (2012). Type D (distressed) personality is associated with poor quality of life and mental health among 3080 cancer survivors. *Journal of Affective Disorders*, *136*(1-2), 26–34. doi:10.1016/j.jad.2011.08.034

Mommersteeg, P. M. C., Herr, R., Bosch, J., Fischer, J. E. & Loerbroks, A. (2011). Type D personality and metabolic syndrome in a 7-year prospective occupational cohort. *Journal of Psychosomatic Research*, *71*(5), 357–63. doi:10.1016/j.jpsychores.2011.05.004

Mommersteeg, P. M. C., Kupper, N. & Denollet, J. (2010). Type D personality is associated with increased metabolic syndrome prevalence and an unhealthy lifestyle in a cross-sectional Dutch community sample. *BMC Public Health*, *10*(1), 714. doi:10.1186/1471-2458-10-714

Mommersteeg, P. M. C., Pelle, A. J., Ramakers, C., Szabó, B. M., Denollet, J. & Kupper, N. (2012). Type D personality and course of health status over 18 months in outpatients with heart failure: multiple mediating inflammatory biomarkers. *Brain, Behavior, and Immunity, 26*(2), 301–10. doi:10.1016/j.bbi.2011.09.010

Nater, U. M., La Marca, R., Florin, L., Moses, A., Langhans, W., Koller, M. M. & Ehlert, U. (2006). Stress-induced changes in human salivary alpha-amylase activity - associations with adrenergic activity. *Psychoneuroendocrinology, 31*(1), 49–58. doi:10.1016/j.psyneuen.2005.05.010

Nater, U. M. & Rohleder, N. (2009). Salivary alpha-amylase as a non-invasive biomarker for the sympathetic nervous system: current state of research. *Psychoneuroendocrinology, 34*(4), 486–96. doi:10.1016/j.psyneuen.2009.01.014

Nater, U. M., Rohleder, N., Schlotz, W., Ehlert, U. & Kirschbaum, C. (2007). Determinants of the diurnal course of salivary alpha-amylase. *Psychoneuroendocrinology, 32*(4), 392–401. doi:10.1016/j.psyneuen.2007.02.007

Nefs, G., Pouwer, F., Pop, V. & Denollet, J. (2012). Type D (distressed) personality in primary care patients with type 2 diabetes: validation and clinical correlates of the DS14 assessment. *Journal of Psychosomatic Research, 72*(4), 251–7. doi:10.1016/j.jpsychores.2012.01.006

Nefs, G., Speight, J., Pouwer, F., Pop, V., Bot, M. & Denollet, J. (2015). Type D personality, suboptimal health behaviors and emotional distress in adults with diabetes: Results from Diabetes MILES–The Netherlands. *Diabetes Research and Clinical Practice, 108*(1), 94–105. doi:10.1016/j.diabres.2015.01.015

Niederhoffer, K. G. & Pennebaker, J. W. (2009). Sharing one's story: On the benefits of writing about emotional experience. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology (2nd ed),* pp. 621–632). New York: Oxford University Press.

Nixon, A. E., Mazzola, J. J., Bauer, J., Krueger, J. R. & Spector, P. E. (2011). Can work make you sick? A meta-analysis of the relationships between job stressors and physical symptoms. *Work & Stress, 25*(1), 1–22. doi:10.1080/02678373.2011.569175

Norton, P. J., Burns, J. a., Hope, D. a. & Bauer, B. K. (2000). Generalization of social anxiety to sporting and athletic situations: gender, sports involvement, and parental pressure. *Depression and Anxiety, 12*(4), 193–202. doi:10.1002/1520-6394

Nyklíček, I., Van Beugen, S., Denollet, J., Beugen, S. van & Denollet, J. (2013). Effects of mindfulness-based stress reduction on distressed (Type D) personality traits: a randomized controlled trial. *Journal of Behavioral Medicine, 36*(4), 361–370. doi:10.1007/s10865-012-9431-3

Nyklíček, I., Vorselaars, A. & Denollet, J. (2011). Type D personality and cardiovascular function in daily life of people without documented cardiovascular disease. *International Journal of Psychophysiology, 80*(2), 139–42. doi:10.1016/j.ijpsycho.2011.02.014

O'Donnell, K., Kammerer, M., O'Reilly, R., Taylor, A. & Glover, V. (2009). Salivary alpha-amylase stability, diurnal profile and lack of response to the cold hand test in young women. *Stress, 12*(6), 549–554. doi:10.3109/10253890902822664

O'Leary, É. D., Howard, S., Hughes, B. M. & James, J. E. (2013). An experimental test of blunting using sleep-restriction as an acute stressor in Type D and non-Type D women. *International Journal of Psychophysiology, 90*(1), 37–43. doi:10.1016/j.ijpsycho.2013.02.006

Ogińska-Bulik, N. (2006). Occupational Stress and Its Consequences in Healthcare Professionals: The Role of Type D Personality. *International Journal of Occupational Medicine and Environmental Health, 19*(2), 113–122. doi:10.2478/v10001-006-0016-7

Orpana, H., Lemyre, L. & Gravel, R. (2009). Income and psychological distress: The role of the social environment. Statistics Canada, 20(82), 21–29.

Osborne, J. W. & Overbay, A. (2004). The power of outliers (and why researchers should always check for them). *Practical Assessment, Research & Evaluation, 9*(6), 1-12. doi:10.1017/CBO9781107415324.004

Paiva, T., Gaspar, T. & Matos, M. G. (2015). Sleep deprivation in adolescents: correlations with health complaints and health-related quality of life. *Sleep Medicine, 16*(4), 521–527. doi:10.1016/j.sleep.2014.10.010

Parks, A. C. & Layous, K. (2016). Positive psychological interventions. In J. C. Norcross, G. R. VandenBos, D. K. Freedheim & R. Krishnamurthy (Eds.), *APA handbook of clinical psychology: Applications and methods, Vol. 3* (pp. 439–449). Washington DC: American Psychological Society.

Pedersen, S. S. & Denollet, J. (2003). Type D personality, cardiac events, and impaired quality of life: a review. *European Journal of Cardiovascular Prevention & Rehabilitation, 10*(4), 241–248. doi:10.1097/01.hjr.0000085246.65733.06

Pedersen, S. S. & Denollet, J. (2004). Validity of the Type D personality construct in Danish post-MI patients and healthy controls. *Journal of Psychosomatic Research, 57*(3), 265–72. doi:10.1016/S0022-3999(03)00614-7

Pedersen, S. S. & Denollet, J. (2006). Is Type D personality here to stay? Emerging evidence across cardiovascular disease patient groups. *Current Cardiology Reviews, 6*(2), 205–213.

Pedersen, S. S. & Middel, B. (2001). Increased vital exhaustion among type-D patients with ischemic heart disease. *Journal of Psychosomatic Research, 51*(2), 443–449. doi:10.1016/S0022-3999(01)00203-3

Pedersen, S. S., Ong, A. T., Sonnenschein, K., Serruys, P. W., Erdman, R. A. & van Domburg, R. T. (2006). Type D personality and diabetes predict the onset of depressive symptoms in patients after percutaneous coronary intervention. *American Heart Journal, 151*(2), 367.

Pedersen, S. S., Theuns, D. A., Erdman, R. A. & Jordaens, L. (2008). Clustering of Device Related Concerns and Type D Personality Predicts Increased Distress in ICD Patients Independent of Shocks. *Pacing and Clinical Electrophysiology*, *31*(1), 20–27.

Pedersen, S. S., van Domburg, R. T., Theuns, D. A. M. J., Jordaens, L. & Erdman, R. A. M. (2004). Type D personality is associated with increased anxiety and depressive symptoms in patients with an implantable cardioverter defibrillator and their partners. *Psychosomatic Medicine*, *66*(5), 714–9. doi:10.1097/01.psy.0000132874.52202.21

Pennebaker, J. W. (1982). Perceptual processes II: schemas, selective search, and inference. In *The Psychology of Physical Symptoms*. New York: Springer US.

Pennebaker, J. W. (2012). Writing about emotional experiences as a therapeutic process. *Psychological Science*, *8*(3), 162–166. doi:10.1111/j.1467-9280.1997.tb00403.x

Pennebaker, J. W., Boyd, R. L., Jordan, K. & Blackburn, K. (2015). *The Development and Psychometric Properties of LIWC2015*. Austin TX: University of Texas at Austin. doi:10.1068/d010163

Pennebaker, J. W., Mayne, T. J. & Francis, M. E. (1997). Linguistic predictors of adaptive bereavement. *Journal of Personality and Social Psychology*, *72*(4), 863–871. doi:10.1037/00-223514.72.4.863

Pennebaker, J. W. & Seagal, J. D. (1999). Forming a Story: The Health Benefits of Narrative. *Journal of Clinical Psychology*, *55*(10), 1243–1254.

Penninx, B. W., Geerlings, S. W., Deeg, D. J., van Eijk, J. T., van Tilburg, W. & Beekman, A. T. (1999). Minor and major depression and the risk of death in older persons. *Archives of General Psychiatry*, *56*(10), 889–95. doi:10.1001/archpsyc.56.10.889

Peterson, R. A. & Heilbronner, R. L. (1987). The anxiety sensitivity index: Construct validity and factor analytic structure. *Journal of Anxiety Disorders*, *1*(2), 117–121. doi:10.1016/0887-6185(87)90002-8

Phillips, A. C. (2011). Blunted cardiovascular reactivity relates to depression, obesity, and self-reported health. *Biological Psychology*, *86*(2), 106–113. doi:10.1016/j.biopsycho.2010.03.016

Phillips, A. C., Ginty, A. T. & Hughes, B. M. (2013). The other side of the coin: blunted cardiovascular and cortisol reactivity are associated with negative health outcomes. *International Journal of Psychophysiology, 90*(1), 1–7. doi:10.1016/j.ijpsycho.2013.02.002

Piazza, J. R., Almeida, D. M., Dmitrieva, N. O. & Klein, L. C. (2010). Frontiers in the use of biomarkers of health in research on stress and aging. Journal of Gerontology: *Psychological Sciences, 65*(5), 513–25. doi:10.1093/geronb/gbq049

Polman, R., Borkoles, E. & Nicholls, A. R. (2010). Type D personality, stress, and symptoms of burnout: the influence of avoidance coping and social support. *British Journal of Health Psychology, 15*(3), 681–96. doi:10.1348/135910709X479069

Porges, S. W. (1992). Vagal tone: a physiologic marker of stress vulnerability. *Pediatrics, 90*(2), 498–504. doi:1513615

Pract, S. A. F. (2016). Stress-related IBS. *South African Family Practice, 58*(3), 23–28.

Proctor, G. B. & Carpenter, G. H. (2007). Regulation of salivary gland function by autonomic nerves. *Autonomic Neuroscience: Basic and Clinical, 133*(1), 3–18. doi:10.1016/j.autneu.2006.10.006

Pruessner, M., Hellhammer, D. H., Pruessner, J. C. & Lupien, S. J. (2003). Self-Reported Depressive Symptoms and Stress Levels in Healthy Young Men: Associations with the Cortisol Response to Awakening. *Psychosomatic Medicine, 65*(1), 92–99. doi:10.1097/01.PSY.0000040950-.22044.10

Putnam, S. P. (2011). Stability and instability of childhood traits: Implications for personality development of animals. *Developmental Psychobiology, 53*(6), 510–520. doi:10.1002/dev.20578

Rasmussen, H. N., Scheier, M. F. & Greenhouse, J. B. (2009). Optimism and physical health: a meta-analytic review. *Annals of Behavioral Medicine, 37*(3), 239–56. doi:10.1007/s12160-009-9111

Reiche, E. M. V., Morimoto, H. K. & Nunes, S. M. V. (2005). Stress and depression-induced immune dysfunction: Implications for the development and progression of cancer. *International Review of Psychiatry, 17*(6), 515–527. doi:10.1080/02646830500382102

Remme, W. (1998). The sympathetic nervous system and ischaemic heart disease. *European Heart Journal, 19*, 62-71

Rimmele, U., Seiler, R., Marti, B., Wirtz, P. H., Ehlert, U. & Heinrichs, M. (2009). The level of physical activity affects adrenal and cardiovascular reactivity to psychosocial stress. *Psychoneuroendocrinology, 34*(2), 190–198. doi:10.1016/j.psyneuen.2008.08.023

Rohleder, N., Nater, U. M., Wolf, J. M., Ehlert, U. & Kirschbaum, C. (2004). Psychosocial stress-induced activation of salivary alpha-amylase: an indicator of sympathetic activity? *Annals of the New York Academy of Sciences, 1032*(1), 258–63. doi:10.1196/annals.1314.033

Rohleder, N., Wolf, J. M., Maldonado, E. F. & Kirschbaum, C. (2006). The psychosocial stress-induced increase in salivary alpha-amylase is independent of saliva flow rate. *Psychophysiology, 43*(6), 645–652. doi:10.1111/j.1469-8986.2006.00457.

Roth, P. P. L. (1994). Missing Data: A Conceptual Review for Applied Psychologists. *Personnel Psychology, 47*(3), 537–560. doi:10.1111/j.1744-6570.1994.tb01736.

Ruiz-Robledillo, N., Bellosta-Batalla, M. & Moya-Albiol, L. (2015). Lower Cardiovascular Reactivity to Acute Stress in Informal Caregivers of People with Autism Spectrum Disorder than in Non-Caregivers: Implications for Health Outcomes. *International Journal of Psychophysiology*, *98*(1), 143-150. doi:10.1016/j.ijpsycho.2015.07.011

Ruscheweyh, R., Marziniak, M., Stumpenhorst, F., Reinholz, J. & Knecht, S. (2009). Pain sensitivity can be assessed by self-rating: Development and validation of the Pain Sensitivity Questionnaire. *Pain, 146*(1), 65–74. doi:10.1016/j.pain.2009.06.020

Sallis, J. F. & Saelens, B. E. (2000). Assessment of physical activity by self-report: status, limitations, and future directions. *Research Quarterly for Exercise and Sport, 71*(4), S1–S14. doi:10.1080/02701367.2000.11082780

Sapolsky, R. M., Romero, L. M. & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine reviews, 21*(1), 55-89.doi:10.1210/er.21.1.55

Sararoudi, R. B., Afshar, H., Peyman, A., Daghaghzadeh, H., Fallah, J. & Abotalebian, F. (2011). Type D personality and quality of life in patients with irritable bowel syndrome. *Journal of Research in Medical Science, 16*(8), 985–992. doi:10.3760/cma.j.issn.0366-6999.2011.17.007

Saucier, G. (1994). Mini-Markers: A brief version of Goldberg's unipolar Big-Five markers. *Journal of personality assessment, 63*(3), 506-516.doi:10.1207/s15327752jpa6303_8

Sausen, K. P., Lovallo, W. R., Pincomb, G. A. & Wilson, M. F. (1992). Cardiovascular responses to occupational stress in male medical students: A paradigm for ambulatory monitoring studies. *Health Psychology, 11*(1), 55–60. doi:10.1037/0278-6133.11.1.55

Schiffer, A. A. A. a., Pedersen, S. S., Widdershoven, J. W., Hendriks, E. H., Winter, J. B. & Denollet, J. (2005). The distressed (Type D) personality is independently associated with impaired health status and increased depressive symptoms in chronic heart failure. *European Journal of Cardiovascular Prevention & Rehabilitation, 12*(4), 341–346. doi:10.1097/01.hjr.-0000173107.76109.6c

Schiffer, A. A., Pedersen, S. S., Broers, H., Widdershoven, J. W. & Denollet, J. (2008). Type-D personality but not depression predicts severity of anxiety in heart failure patients at 1-year follow-up. *Journal of Affective Disorders, 106*(1), 73–81.

Schlotz, W., Yim, I. S., Zoccola, P. M., Jansen, L. & Schulz, P. (2011). The Perceived Stress Reactivity Scale: measurement invariance, stability, and validity in three countries. *Psychological Assessment, 23*(1), 80–94. doi:10.1037/a0021148

Schubert, M., Contreras, C., Franz, N. & Hellhammer, J. (2011). Milk-based phospholipids increase morning cortisol availability and improve memory in chronically stressed men. *Nutrition Research, 31*(6), 413–420. doi:10.1016/j.nutres.2011.05.012

Schulz, P., Kirschbaum, C., Prüßner, J. & Hellhammer, D. (1998). Increased free cortisol secretion after awakening in chronically stressed individuals due to work overload. *Stress Medicine, 14*(2), 91–97. doi:10.1002/1099-1700199804

Schutte, A. E., Huisman, H. W., Van Rooyen, J. M., Oosthuizen, W. & Jerling, J. C. (2003). Sensitivity of the Finometer device in detecting acute and medium-term changes in cardiovascular function. *Blood Pressure Monitoring, 8*(5), 195–201. doi:10.1097/01.0000103280.95050.f7

Schwab, K. O., Heubel, G. & Bartels, H. (1992). Free epinephrine, norepinephrine and dopamine in saliva and plasma of healthy adults. *European Journal of Chemistry and Clinical Biochemistry, 30*(9), 541–4.

Segerstrom, S. C. & O'Connor, D. B. (2012). Stress, health and illness: Four challenges for the future. *Psychology & Health, 27*(2), 128–140. doi:10.1080/08870446.2012.659516

Sephton, S. E. (2000). Diurnal Cortisol Rhythm as a Predictor of Breast Cancer Survival. *Journal of the National Cancer Institute, 92*(12), 994–1000. doi:10.1093/jnci/92.12.994

Serlachius, A., Hamer, M. & Wardle, J. (2007). Stress and weight change in university students in the United Kingdom. *Physiology & Behavior, 92*(4), 548–553. doi:10.1016/j.physbeh.2007.04.032

Sheikh, K. & Mattingly, S. (1981). Investigating non-response bias in mail surveys. *Journal of Epidemiology & Community Health, 35*(4), 293–296. doi:10.1136/jech.35.4.293

Sher, L. (2005). Type D personality: the heart, stress, and cortisol. *Quarterly Journal of Medicine, 98*(5), 323–9. doi:10.1093/qjmed/hci064

Shinoura, N. & Yamada, R. (2005). Head-down manoeuvre in patients with a high symptom score for orthostatic intolerance reveals impaired right brain frontal lobe vasoreactivity. *Clinical Neurophysiology, 116*(6), 1286–1290. doi:10.1016/j.clinph.2005.02.005

Shufflebotham, J., Wetherell, M. A., Hince, D., Hood, S., Lightman, S., Nutt, D., Potokar, J. (2009). Women with diarrhoea-predominant irritable bowel syndrome show an increased pressure response to 35% carbon dioxide stress challenge. *Stress, 12*(1), 30–36. doi:10.1080/10253890801976926

Simson, U., Nawarotzky, U., Porck, W., Friese, G., Schottenfeld-Naor, Y., Hahn, S., Kruse, J. (2008). Depression, Anxiety, Quality of Life, and Type D Pattern Among Inpatients Suffering from Diabetic Foot Syndrome. *Psychotherapy and Psychological Medicine, 58*(2), 44–50. doi:10.1055/s-2008-1077619

Sin, N. L. & Lyubomirsky, S. (2009). Enhancing well-being and Alleviating depressive symptoms with positive psych interventions: A practice friendly meta-analysis. *Journal of Clinical Psychology, 65*(5), 467–487. doi:10.1002/jclp

Smith, T. W. (2011). Toward a More Systematic, Cumulative, and Applicable Science of Personality and Health. *Psychosomatic Medicine, 73*(7), 528–532. doi:10.1097/PSY.0b013e31822e095e

Smith, T. W. & MacKenzie, J. (2006). Personality and risk of physical illness. *Annual Review of Clinical Psychology, 2*, 435–467. doi:10.1146/annurev.clinpsy.2.022305.095257

Smith, W. G. (2008). Does gender influence online survey participation? A record-linkage analysis of university faculty online survey response behavior. *Online Submission.*

Smolderen, K. G. E., Vingerhoets, A. J. J. M., Croon, M. A. & Denollet, J. (2007). Personality, psychological stress, and self-reported influenza symptomatology. *BMC Public Health, 7*(1), 339. doi:10.1186/1471-2458-7-339

Smyth, J. M. (1998). Written Emotional Expression: Effect Sizes, Outcome Types, and Moderating Variables. *American Psychological Association, 66*(1), 174–184. doi:10.1037/0022-006X.66.1.174

Spector, P. E., Dwyer, D. J. & Jex, S. M. (1988). Relation of job stressors to affective, health, and performance outcomes: a comparison of multiple data sources. *Journal of Applied Psychology, 73*(1), 11–19.

Spielberger, C. D., Gorsuch, R. L. & Lushene, R. E. (1970). Manual for the state-trait anxiety inventory.

Spindler, H., Pedersen, S. S., Serruys, P., Erdman, R. A. M. & van Domburg, R. T. (2007). Type-D personality predicts chronic anxiety following percutaneous coronary intervention in the drug-eluting stent era. *Journal of Affective Disorders, 99*(1-3), 173–179.

Steca, P., D'Addario, M., Magrin, M. E., Miglioretti, M., Monzani, D., Pancani, L., Greco, A. (2016). A type A and Type D combined personality typology in essential hypertension and acute coronary syndrome patients: Associations with demographic, psychological, clinical, and lifestyle indicators. *PLoS ONE, 11*(9), 1–28. doi:10.1371/journal.pone.0161840

Stephens, R. & Umland, C. (2011). Swearing as a response to pain - Effect of daily swearing frequency. *Journal of Pain, 12*(12), 1274–1281. doi:10.1016/j.jpain.2011.09.004

Steptoe, A. & Kivimäki, M. (2012). Stress and cardiovascular disease. *Nature Reviews Cardiology, 9*(6), 360–370. doi:10.1038/nrcardio.2012.45

Stevenson, C. & Williams, L. (2014). Type D personality, quality of life and physical symptoms in the general population: a dimensional analysis. *Psychology & Health, 29*(3), 365–73. doi:10.1080/08870446.2013.856433

Strik, J. J. M. H., Denollet, J., Lousberg, R. & Honig, A. (2003). Comparing Symptoms of Depression and Anxiety as Predictors of Cardiac Events and Increased Health Care Consumption after Myocardial Infarction. *Journal of the American College of Cardiology, 42*(10), 1801–1807. doi:10.1016/j.jacc.2003.07.007

Sullivan, M. D., LaCroix, A. Z., Russo, J. E. & Walker, E. A. (2001). Depression and Self-Reported Physical Health in Patients with Coronary Disease: Mediating and Moderating Factors. *Psychosomatic Medicine, 63*(2), 248–256.

Svansdottir, E., van den Broek, K. C., Karlsson, H. D., Gudnason, T. & Denollet, J. (2012). Type D personality is associated with impaired psychological status and unhealthy lifestyle in Icelandic cardiac patients: a cross-sectional study. *BMC Public Health, 12*(1), 42. doi:10.1186/1471-2458-12-42

Svansdottir, E., van den Broek, K. C., Karlsson, H. D., Olason, D. T., Thorgilsson, H. & Denollet, J. (2013). The distressed (Type D) and Five-Factor Models of personality in young, healthy adults and their association with emotional inhibition and distress. *Personality and Individual Differences, 55*(2), 123–128. doi:10.1016/j.paid.2013.02.008

Tabachnick, B. G. & Fidell, L. S. (2007). Using multivariate statistics. Pearson International, Ed.) *Using Multivariate Statistics* (5th Ed). Boston. doi:10.1037/022267

Taffe, J. & Dennerstein, L. (2000). Retrospective self-report compared with menstrual diary data prospectively kept during the menopausal transition. *Climacteric, 3*(3), 183–91. doi:10.1080/13697130008500099

Tausczik, Y. R. & Pennebaker, J. W. (2010). The Psychological Meaning of Words: LIWC and Computerized Text Analysis Methods. *Journal of Language and Social Psychology, 29*(1), 24–54. doi:10.1177/0261927X09351676

Taylor, D. J., Lichstein, K. L., Durrence, H. H., Reidel, B. W. & Bush, A. J. (2005). Epidemiology of insomnia, depression, and anxiety. *Sleep, 28*(11), 1457–1464. doi:10.1093/sleep/28.11.1457

Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. a & Updegraff, J. a. (2000). Biobehavioral responses to stress in females: tend-and-befriend, not fight-or-flight. *Psychological Review, 107*(3), 411–429. doi:10.1037/0033-295X.107.3.411

Tessler, R. & Mechanic, D. (1978). Psychological distress and perceived health status. *Journal of Health and Social Behavior, 19*(3) 254–262.

Thayer, J. F., Åhs, F., Fredrikson, M., Sollers, J. J. & Wager, T. D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: implications for heart rate variability as a marker of stress and health. *Neuroscience & Biobehavioral Reviews, 36*(2), 747-756.

Thayer, J. F., Yamamoto, S. S. & Brosschot, J. F. (2010). The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *International Journal of Cardiology, 141*(2), 122–31. doi:10.1016/j.ijcard.2009.09.543

Thomas, G., de Jong, F. I. C. R. S., Kooijman, P. G. C. & Cremers, C. W. R. J. (2006). Utility of the Type D Scale 16 and Voice Handicap Index to assist voice care in student teachers and teachers. *Folia Phoniatrica et Logopaedica, 58*(4), 250–63. doi:10.1159/000093182

Toker, S., Shirom, A., Shapira, I., Berliner, S. & Melamed, S. (2005). The association between burnout, depression, anxiety, and inflammation biomarkers: C-reactive protein and fibrinogen in men and women. *Journal of Occupational Health Psychology, 10*(4), 344–362. doi:10.1037/1076-8998.10.4.344

Toobert, D. J., Hampson, S. E. & Glasgow, R. E. (2000). The Summary of Diabetes Self-Care Activities Measure. *Diabetes Care, 23*(7), 943–950.

Trama, S. & Kaur, H. (2009). Effect of Positive Emotions on Health. *Journal of Indian Health, 4*(1), 13–23. doi:10.1089/act.2009.15706

Treiber, F. a, Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G. & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65*(1), 46–62.

Tugade, M. & Fredrickson, B. L. (2004). Resilient Individuals Use Positive Emotions to Bounce Back from Negative Emotional Experiences. *Journal of Personality and Social Psychology, 86*(2), 320–333. doi:10.1037/0022-3514.86.2.320

Tugade, M., Fredrickson, B. L., Feldman Barret, L., Tugade, M., Fredrickson, B. L., Feldman Barret, L., Feldman Barret. (2004). Psychological Resilience and Positive Emotional Granularity: Examining the Benefits of Positive Emotions on Coping and Health. *Journal of Personality, 72*(6), 1161–1190. doi:10.1038/jid.2014.371

Tully, P. & Baker, R. (2010). Depression, anxiety disorders and Type D personality as risk factors for delirium after cardiac surgery. *Australian and New Zealand Journal of Psychiatry, 44*(11), 1005–1011.

Uchino, B. N. (2006). Social support and health: A review of physiological processes potentially underlying links to disease outcomes. *Journal of Behavioral Medicine, 29*(4), 377–387. doi:10.1007/s10865-006-9056-5

Ursin, H. (1997). Sensitization, somatization, and subjective health complaints. *International Journal of Behavioral Medicine, 4*(2), 105–116. doi:10.1207/s15327558ijbm0402_1

van den Broek, K. C., Smolderen, K. G., Pedersen, S. S. & Denollet, J. (2010). Type D personality mediates the relationship between remembered parenting and perceived health. *Psychosomatics, 51*(3), 216–224. doi:10.1016/S0033-3182(10)70688-5

van den Tooren, M. & Rutte, C. (2015). Explaining Emotional Exhaustion and Work Engagement: The Role of Job Demands-Resources and Type D Personality. *International Journal of Stress Management, 23*(2) 147-167. doi:10.1037/a0039754

van Gestel, Y. R. B. M., Pedersen, S. S., van de Sande, M., de Jaegere, P. P. T., Serruys, P. W., Erdman, R. A. M., … Gestel, Y. van. (2007). Type-D personality and depressive symptoms predict anxiety 12??months post-percutaneous coronary intervention. *Journal of Affective Disorders, 103*(1), 197–203. doi:10.1016/j.jad.2007.01.030

van Stegeren, A., Rohleder, N., Everaerd, W. & Wolf, O. T. (2006). Salivary alpha amylase as marker for adrenergic activity during stress: effect of betablockade. *Psychoneuroendocrinology, 31*(1), 137–41. doi:10.1016/j.psyneuen.2005.05.012

Veehof, M. M., Oskam, M. J., Schreurs, K. M. G. & Bohlmeijer, E. T. (2011). Acceptance-based interventions for the treatment of chronic pain: A systematic review and meta-analysis. *Pain, 152*(3), 533–542. doi:10.1016/j.pain.2010.11.002

Vgontzas, A. N., Lin, H.-M., Papaliaga, M., Calhoun, S., Vela-Bueno, A., Chrousos, G. P. & Bixler, E. O. (2008). Short sleep duration and obesity: the role of emotional stress and sleep disturbances. *International Journal of Obesity, 32*(5), 801–9. doi:10.1038/ijo.2008.4

Vingerhoets, A. J. J. M., Bylsma, L. M. & de Vlam, C. (2013). Swearing: A biopsychosocial perspective. *Psihologijske Teme, 22*(2), 287–304.

Vrijkotte, T. G. M., van Doornen, L. J. P. & de Geus, E. J. C. (2000). Effects of Work Stress on Ambulatory Blood Pressure, Heart Rate, and Heart Rate Variability. *Hypertension, 35*(4), 880–886. doi:10.1161/01.HYP.35.4.880

Wallston, B. S., Alagna, S. W., DeVellis, B. M. & DeVellis, R. F. (1983). Social support and physical health. *Health Psychology, 2*(4), 367–391. doi:10.1037/0278-6133.2.4.367

Wang, N. (2013). An analysis of the pragmatic functions of "swearing" in interpersonal talk. En: Griffith Working Papers in Pragmatics and Intercultural Communication, 6, 71-79.

Ware, J. E. & Sherbourne, C. D. (1992). The MOS 36-Item Short-Form Health Survey (SF-36): I . Conceptual Framework and Item Selection. *Medical Care, 30*(6), 473–483.

Warr, P., Miles, A. & Platts, C. (2001). Age and personality in the British population between 16 and 64 years. *Journal of Occupational and Organizational Psychology, 74*(2), 165-199

Watson, D. (1988). Intraindividual and interindividual analysis of positive and negative affect: Their relation to health complaints, perceived stress, and daily activities. *Journal of Personality and Social Psychology, 54*(6), 1020–1030.

Watson, D. & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review, 96*(2), 234–254. doi:10.1037/0033-295X.96.2.234

Weiller, E., Bisserbe, J. C., Boyer, P., Lepine, J. P. & Lecrubier, Y. (1996). Social Phobia in General Health Care: An Unrecognised Undertreated Disabling Disorder. *British Journal of Psychiatry, 168*, 169–174. doi:10.1192/bjp.168.2.169

Weissbecker, I., Floyd, A., Dedert, E., Salmon, P. & Sephton, S. (2006). Childhood trauma and diurnal cortisol disruption in fibromyalgia syndrome. *Psychoneuroendocrinology, 31*(3), 312–324. doi:10.1016/j.psyneuen.2005.08.009

Wetherell, M. A., Atherton, K., Grainger, J., Brosnan, R. & Scholey, A. B. (2012). The effects of multitasking on psychological stress reactivity in recreational users of cannabis and MDMA. *Human Psychopharmacology, 27*(2), 167–176. doi:10.1002/hup.1261

Wetherell, M. A. & Carter, K. (2014). The multitasking framework: The effects of increasing workload on acute psychobiological stress reactivity. *Stress and Health, 30*(2), 103–109. doi:10.1002/smi.2496

Wetherell, M. A., Craw, O., Smith, K. & Smith, M. A. (2017). Psychobiological responses to critically evaluated multitasking. *Neurobiology of Stress, 7*, 68–73. doi:10.1016/j.ynstr.2017.05.002

Wetherell, M. A. & Sidgreaves, M. C. (2005). Short communication: Secretory immunoglobulin-A reactivity following increases in workload intensity using the Defined Intensity Stressor Simulation (DISS). *Stress and Health, 21*(2), 99–106. doi:10.1002/smi.1038

Whitehead, D. L., Perkins-Porras, L., Strike, P. C., Magid, K. & Steptoe, A. (2007). Cortisol awakening response is elevated in acute coronary syndrome patients with Type-D personality. *Journal of Psychosomatic Research, 62*(4), 419–25. doi:10.1016/j.jpsychores.2006.11.005

Whitehead, W. E. (1994). Assessing the effects of stress on physical symptoms. *Health Psychology, 13*(2), 99–102. doi:10.1037//0278-6133.13.2.99

Widiger, T. & Trull, T. (2007). Plate tectonics in the classification of personality disorder: shifting to a dimensional model. *American Psychologist, 62*(2), 71–83.

Williams, L., Abbott, C. & Kerr, R. (2015). Health behaviour mediates the relationship between Type D personality and subjective health in the general population. *Journal of Health Psychology, 21*(10), 2148-2155. doi:10.1177/1359105315571977

Williams, L., O'Carroll, R. E. & O'Connor, R. C. (2009). Type D personality and cardiac output in response to stress. *Psychology & Health, 24*(5), 489–500. doi:10.1080/08870440701885616

Williams, L., O'Connor, R. C., Grubb, N. R. & O'Carroll, R. (2011). Type D personality predicts poor medication adherence in myocardial infarction patients. *Psychology & Health, 26*(6), 703–12. doi:10.1080/08870446.2010.488265

Williams, L., O'Connor, R. C., Howard, S., Hughes, B. M., Johnston, D. W., Hay, J. L., … O'Carroll, R. E. (2008). Type-D personality mechanisms of effect: the role of health-related behavior and social support. *Journal of Psychosomatic Research, 64*(1), 63–9. doi:10.1016/j.jpsychores.2007.06.008

Williams, L. & Wingate, A. (2012). Type D personality, physical symptoms and subjective stress: the mediating effects of coping and social support. *Psychology & Health, 27*(9), 1075–85. doi:10.1080/08870446.2012.667098

Williamson, S., Munro, C., Pickler, R., Grap, M. J. & Elswick, R. K. (2012). Comparison of Biomarkers in Blood and Saliva in Healthy Adults. *Nursing Research and Practice, 2012*, 1-4. doi:10.1155/2012/246178

Zanstra, Y. J. & Johnston, D. W. (2011). Cardiovascular reactivity in real life settings: Measurement, mechanisms and meaning. *Biological Psychology, 86*(2), 98–105. doi: 10.1016/j.biopsycho-.2010.05.002

Zigmond, A. & Snaith, R. (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica, 67*(6), 361–370.

Zimet, G. D., Dahlem, N. W., Zimet, S. G. & Farley, G. K. (1988). The Multidimensional Scale of Perceived Social Support. *Journal of Personality Assessment, 52*(1), 30–41. doi:10.1207/s15327752jpa5503&4_17

APPENDIX A: Demographic Questions

PLEASE ANSWER THE FOLLOWING QUESTIONS ABOUT YOU AND YOUR HOUSEHOLD:

1) Your date of birth: -------/----------/----------
Day Month Year

2) Are you: Male □ or Female □

3) What is your country of birth? _____

4) What is your country of residence?_____

5) If you are female, would you describe yourself as: (please tick box)

Pre-menopausal □ Experiencing the menopause now □ Post-menopausal □

6) Which of the following best describes your ethnic background?

Please tick one of the following groups, if not applicable please specify your ethnic origin.

White □ Black – Other □ Bangladeshi □

Black – Caribbean □ Indian □ Chinese □

Black – African □ Pakistani □ Asian – Other □

Other– please specify □ _____

7) Which of the following best describes your marital status?

Please tick one of the following.

Single □ Widowed □ Married □ Divorced/Separated □

Living as married □ Other (please specify) □ _____

8) How tall are you? _____(feet) _____(inches) or _____(cm)

9) How much do you weigh? _____(stone) _____(pounds) or _____(kg)

10) Are you currently: (please tick as many boxes as apply)

In active paid work □ Unemployed and seeking work □ Retired □

unemployed due to illness or disability □ Doing voluntary work □ Full time student □

At home doing housework □7 Other (please specify) □8 _____

11) Please complete for present or last paid job (for retired or unemployed)

Job title: _____

12) Is / was this job: Full time □ Part time □

13) Major activities in job (e.g. tasks, level/grade, and qualifications):

_____

14) Are / were you an: Employee □ Employer/Manager □ Self-employed □

Supervisor/Foreman □

15) How many people do/did you supervise? _____

16) What was the total income (gross – before tax) of your household for the past 12 months?

(Please include salaries, wages, pension(s), benefits and allowances, share dividends etc. The final total should be the estimated combined wages etc. of everyone who lives in your household.)

<£5000 per year (< £100 per week) □ £25,000-£29,999/year(£500-£599/week) □

£5000-£9,999/year (£100-£199/week) □ £30,000-£34,999/year (£600-£699/week) □

£10,000-£14,999/year (£200-£299 / week) □ £35,000-£39,999/year (£700-£799/week) □

£15,000- £19,999/year(£300-399/week) □ £40,000+ /year (£800+ /week) □

£20,000-£24,999/year (£400-£499/week) □

17) How old were you when you left school? _____

18) Do you have any of these qualifications? (Please tick all that apply)

CSE / O levels / School Certificate / GCSE □ City and Guilds Certificate □

A levels / Highers / BTEC □ Recognised Trade Apprenticeship □

HND □ Clerical / Commercial Qualification □

First degree (BA, BSc etc) □ Higher Degree (MSc, PhD etc.) □

Medical / Nursing / Teaching qualifications □ Membership of Professional Institute □

Other (Please describe) _____

APPENDIX B: Health Behaviour Questions

1) Do you drink alcohol? Yes ☐ No ☐

If you answered yes:

2) On how many days in the last two weeks did you drink alcohol?

3) On how many of those occasions did you drink more than 2-3 units (women) or 3-4 units (men) of alcohol in one 'session'?

(Guide: 1 unit = ½ pint of beer or lager, 1 standard glass of wine, 1 single measure of spirits)

4) Do you smoke cigarettes? Yes ☐ No ☐ Previously☐

If you answered yes:

5) On how many days in the last two weeks have you smoked cigarettes (even just one)?

6) How many cigarettes do you typically smoke per week?

7) On how many days in the last week did you participate in at least 30 minutes of moderate physical activity (in bouts of at least 10 minutes, including walking)?

8) On how many days of the last week did you participate in a specific exercise session of more vigorous intensity such as swimming, running or cycling?

9) On how many days of the last week did you eat five or more servings of fruits and vegetables?

10) On how many days of the last week did you avoid eating fatty foods such as red meat or full-fat dairy products?

APPENDIX C: Retrospective Health Questions

1) How is your health in general right now, would you say it was:

- Very Good
- Good
- Fair
- Bad
- Very Bad

2) Over the last 12 months would you say your health has, on the whole, been ...:

- Very Good
- Good
- Fairly good
- Not very good
- Not good at all

3) Compared to one year ago, how would you say your health is now?

- much better now than 1 year ago
- somewhat better now
- about the same as 1 year ago
- somewhat worse now
- much worse now

4) How satisfied are you with your health in general?

- very satisfied
- satisfied
- neither satisfied nor dissatisfied
- dissatisfied
- very dissatisfied

5) During the last year, approximately how often have you felt generally ill, unwell or run down (due to tiredness, fatigue, stress etc.)?

- Never
- Rarely
- Sometimes
- Frequently
- Always

6) During the last year, approximately how often have you suffered a non-serious illness such as a cold, stomach bug or minor infection which has not required prescription medication?

- Never
- Once or twice
- Three or four times
- Five or six times
- More than six times

7) During the last year, approximately how often have you suffered a more serious illness which has required prescription medication (e.g. antibiotics, steroidal medication)?

- Never
- Once or twice
- Three or four times
- Five or six times
- More than six times

8) During the last year, approximately how frequently (per instance) have you had to take time off work due to illness?

- Never
- Once every 3-4 months
- Once every 2-3 months
- approximately once per month
- More than once per month
- Not applicable (if retired/un-employed/maternity leave etc)

9) During the last year, approximately how regularly have you visited your GP or other healthcare profession (e.g. district nurse, walk-in-centre) because of a health problem?

- Never
- Once every 3-4 months
- Once every 2-3 months
- approximately once per month
- More than once per month

10) During the last year, approximately how regularly have you sought medical information about a problem with your health without visiting a medical professional (e.g. via telephone, post or email, health leaflets, online health information websites)?

- Never
- Once every 3-4 months
- Once every 2-3 months
- approximately once per month
- More than once per month

11) During the last year, have you ever been admitted to hospital?

- ○ Never been in hospital
- ○ Yes, On 1 occasion I have been in hospital for treatment as a day-patient
- ○ Yes, On 1 occasion I have been in hospital as an inpatient and stayed overnight or longer
- ○ Yes, On multiple occasions I have been in hospital for treatment as a day-patient
- ○ Yes, On multiple occasions I have been in hospital as an inpatient and stayed overnight or longer