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**Type D personality is associated with lower cardiovascular reactivity
to stress in women**

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Abstract

Objective: This study examines if Type D personality is (1) associated with cardiovascular reactivity to acute stress in a healthy sample, and (2) has predictive utility for cardiovascular reactivity above its individual subcomponents (negative affect; NA, social inhibition; SI), as well as anxiety and depression.

Design: Undergraduate students ($n = 173$) completed a standardised cardiovascular reactivity experimental protocol consisting of resting baseline and stressor phase (mental arithmetic), with systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) monitored throughout.

Main Outcome Measures: The main outcome measures were cardiovascular reactivity to the stressor, which was operationalised as the difference between resting baseline and the stressor phase for SBP, DBP and HR.

Results: The continuous Type D interaction term (NA \times SI) significantly predicted lower SBP reactivity to the mental arithmetic stressor amongst women, independent of NA, SI and confounding variables. Moreover, this remained significant after adjustment for anxiety and depressive symptoms. Depression, NA and SI were also significant independent predictors of SBP reactivity amongst women.

Conclusion: Type D personality is associated with lower SBP reactivity to acute stress in women, which may be indicative of blunted cardiovascular reactivity. This association was independent of NA, SI, Anxiety and Depression.

Key Words: Type D personality, cardiovascular reactivity, depression, anxiety, acute stress

Introduction

Type D personality is characterised by increased levels of both negative affectivity (NA) and social inhibition (SI). Negative affectivity refers to the tendency to experience an array of negative emotions across both time and situations, and is propounded to encapsulate feelings of dysphoria, anxiety, and irritability. Social inhibition refers to the conscious tendency to inhibit the expression of emotions during social interactions in order to avoid disapproval from others. High-SI individuals tend to feel tense, insecure, and inhibited in social situations (Denollet, 2005). Type D personality is suggested to represent more than the mere presence of NA and SI alone, with SI moderating the effect of NA on clinical outcomes (Kupper & Denollet, 2007). Thus, Type D personality is posited to reflect a synergistic interactional effect of NA and SI combined (Kupper & Denollet, 2007, 2014).

Type D personality has received considerable research attention due its association with prospective cardiovascular health. In cardiac populations, Type D personality has been consistently found to predict poorer prognosis (e.g., cardiovascular revascularization, reoccurring myocardial infarction), as well as cardiac and all-cause mortality (Denollet et al., 2006, 2013; Denollet et al., 1996; Denollet et al., 2000; Martens et al., 2010; Schiffer et al., 2008). In fact, meta-analyses suggest that Type D personality confers approximately a 2-fold increased risk of adverse cardiovascular health in cardiac patients (Grande et al., 2012; O'Dell et al., 2011; Reich & Schatzberg, 2010). Consequently, Type D personality has recently been included in the European Guidelines on cardiovascular disease prevention in clinical practice as a risk factor to assess (Piepoli et al., 2016).

A further body of research has aimed to elucidate the underlying mechanisms that facilitate the association between Type D personality and adverse cardiovascular health. Indirect mechanisms have primarily accentuated negative health behaviours including unhealthy eating, physical inactivity, and smoking (Booth & Williams, 2015; Ginting et al.,

2016; Williams et al., 2016). Direct mechanisms have implicated the role of physiological processes including pro-inflammatory activity (Denollet et al., 2009; Denollet et al., 2008) and increased diurnal cortisol output (Molloy et al., 2008; Whitehead et al., 2007). One physiological mechanism that has received substantial support is cardiovascular reactivity to acute psychological stress. Here, several experimental studies have found individuals with Type D personality to exhibit atypical cardiovascular responses to acute stress, in comparison to their non-Type D counterparts (Allen et al., 2019; Bibbey et al., 2015; Gramer et al., 2018; Howard et al., 2011; Kelly-Hughes et al., 2014; O'Leary É et al., 2013; O'Riordan, Howard, Brown, et al., 2020; O'Riordan et al., 2019; Williams et al., 2009).

The cardiovascular reactivity hypothesis propounds that exaggerated or prolonged cardiovascular reactions to psychological stress promotes the development of cardiovascular diseases (Obrist, 2012). This hypothesis has been substantiated by an array of prospective studies, whereby exaggerated cardiovascular reactivity to psychological stress has been linked to future hypertension (Carroll, Ginty, Painter, et al., 2012; Carroll et al., 2003; Carroll et al., 1995), atherosclerosis (Barnett et al., 1997; Matthews et al., 1998), and cardiovascular disease mortality (Carroll, Ginty, Der, et al., 2012). Additionally, more recent evidence has suggested that atypically low or “blunted” cardiovascular reactivity is also linked to a range of adverse health outcomes (Carroll et al., 2017; Phillips et al., 2013). In fact, blunted cardiovascular reactivity has also been linked to poorer cardiovascular health outcomes including increased intima-media thickness of the carotid artery amongst healthy participants (Ginty et al., 2016), as well as cardiovascular hospitalization and mortality in cardiac samples (Kupper et al., 2015; Sherwood et al., 2017). Thus, it is now postulated that both exaggerated and blunted cardiovascular responses to psychological stress are indicative of a bias in homeostatic regulation, and therefore, vulnerability to psychosomatic diseases (Lovallo, 2011).

However, prior studies linking Type D personality to cardiovascular reactivity have yielded inconsistent findings, with some showing Type D individuals to exhibit blunted cardiovascular reactivity (Howard et al., 2011; Kelly-Hughes et al., 2014; O'Leary É et al., 2013), and others showing Type D individuals to exhibit exaggerated cardiovascular reactivity (O'Riordan, Howard, Brown, et al., 2020; Williams et al., 2009). One likely explanation for these antithetical findings is the moderating effect of **gender** and the social salience of the stress task (Bibbey et al., 2015; Gramer et al., 2018; O'Riordan et al., 2019). Type D personality is primarily associated with lower cardiovascular reactivity amongst women (Gramer et al., 2018; Howard et al., 2011; O'Leary É et al., 2013), and in response to stressors of low social salience (Kelly-Hughes et al., 2014). In contrast, Type D personality appears to be associated with exaggerated cardiovascular reactivity amongst men (Gramer et al., 2018; O'Riordan et al., 2019; Williams et al., 2009), and in response to stressors of high social salience (Allen et al., 2019; Bibbey et al., 2015; O'Riordan, Howard, Brown, et al., 2020). It has been consistently proffered that the differential responses exhibited by Type D men and women is likely owing to societal norms pertaining to social inhibition (Habra et al., 2003; Howard & Hughes, 2013; O'Riordan et al., 2019), whereby it is less acceptable and more difficult for men to engage in socially inhibited behaviour (e.g. social withdrawal). Furthermore blunted cardiovascular reactivity is suggested to represent a motivational dysregulation, resulting in withdrawal and disengagement with the stressor (Carroll et al., 2017; Phillips et al., 2013). Thus, it is likely that Type D women are more readily able to withdraw from the stress task, exhibiting blunted cardiovascular reactivity. In contrast, Type D men are likely to maintain engagement, particularly during stressors of greater social salience, which they find specifically more stressful. Therefore, in line with prior studies (Williams et al., 2009), analyses will be conducted separately for men and woman in the current study.

Amid the myriad of research findings accentuating the negative health effects of Type D personality, it is important to highlight the many criticisms of the Type D construct (Coyne & de Voogd, 2012; Coyne et al., 2011; Smith, 2011). Firstly, while Type D personality was traditionally operationalised as a dichotomous construct (Denollet, 2005), this approach is posited to reduce the sensitivity of statistical tests, and increase the probability of “lucky cuts” that produce spurious findings (Lodder, 2020; Royston et al., 2006; Smith, 2011). Additionally, the operationalisation of Type D personality as a dichotomous construct may also result in false synergistic Type D effects, which are instead produced by the main effect of NA and SI (Coyne & de Voogd, 2012; Smith, 2011). Thus, it is suggested that many Type D findings in the literature may represent the main effects of either NA or SI, rather than a synergistic interactional effect of both constructs combined. Therefore, analyses using a continuous Type D interaction term ($NA \times SI$), whilst controlling for the main effects of NA and SI, is the most appropriate analytical method of determining the predictive utility of Type D personality, whilst maintaining statistical power and reducing bias (Lodder, 2020; Smith, 2011). While the majority of the cardiovascular reactivity literature has not controlled for the individual effects of NA and SI (Bibbey et al., 2015; Gramer et al., 2018; O’Leary É et al., 2013; O’Riordan et al., 2019; Williams et al., 2009), others have found Type D to predict abnormal physiological responses after controlling for the individual Type D subcomponents (Allen et al., 2019; Howard et al., 2011; Kelly-Hughes et al., 2014; O’Riordan, Howard, Brown, et al., 2020).

Others have questioned the predictive utility of Type D personality above other closely related negative affect variables, particularly anxiety and depression (Coyne & de Voogd, 2012; Coyne et al., 2011; Smith, 2011). In fact, Coyne et al. (2011) postulates that Type D personality is a likely proxy for clinical depression. This criticism emerged after prospective studies reported null effects of Type D on clinical outcomes in analyses adjusting for

depression and anxiety (Coyne et al., 2011; Grande et al., 2011; Volz et al., 2011). However, others have provided evidence refuting this criticism and have reported significant effects of Type D on adverse cardiovascular health and poor cardiac prognosis after controlling for depression and anxiety (Denollet & Pedersen, 2008; Martens et al., 2010; Schiffer et al., 2010). Although depression and anxiety have been consistently associated with cardiovascular reactivity to acute psychological stress (Brindle et al., 2013; Carroll et al., 2007; Schwerdtfeger & Rosenkaimer, 2011; York et al., 2007; Yuenyongchaiwat et al., 2017), little research to date has examined if Type D personality has predictive utility for cardiovascular reactivity above these closely related constructs. While Bibbey et al. (2015) found Type D to predict cardiovascular reactivity in analyses adjusting for depression, they did not concurrently adjust for the main effect of NA and SI. More specifically, depression and anxiety have been associated with blunted cardiovascular reactivity to acute psychological stress (Brindle et al., 2013; Carroll et al., 2007; Schwerdtfeger & Rosenkaimer, 2011; York et al., 2007; Yuenyongchaiwat et al., 2017). Thus, it is questionable if the association between Type D personality and blunted cardiovascular reactivity, particularly exhibited in response to stressors of lower social salience (e.g. mental arithmetic), withstands adjustment for these constructs.

Considering the above evidence, the current study has three main aims. Firstly, we aim to examine the association between Type D personality and cardiovascular reactivity to a mental arithmetic stress task. Secondly, we aim to investigate if Type D personality has predictive utility for cardiovascular reactivity above its individual subcomponents (NA and SI), as well as depression and anxiety. Finally, we aim to examine if the effects observed using the continuous Type D interaction term (NA \times SI) could be replicated using the traditional Type D dichotomy. Here, we hypothesise that Type D personality will be associated with lower cardiovascular reactivity, particularly amongst women. Additionally, we also hypothesise that

Type D personality will have predictive utility above its individual subcomponents (NA and SI), depression and anxiety, and will have greater power to detect significant effects when treated as a continuous interaction term in comparison to a dichotomy.

Materials and Methods

Design

The present study employed a between subject correlational design. The main predictor variable was Type D personality, as well as its individual constructs, NA and SI. Additional predictor variables including anxiety and depression. The main outcome variables were SBP, DBP and HR reactivity.

Participants

A total of 182 participants took part in the experimental protocol. Complete questionnaire data were missing for 2 participants. Additionally, 6 participants reported taking medication (not including contraceptive medication) or suffering from a medical condition that influences cardiovascular measurements. Finally one participant failed to complete questionnaires pertaining to medication use. After the removal of these participants ($n = 9$), the final sample size consisted of 173 participants. Of the 173 participants, missing data was observed for smoking status (2 participants), SI/Type D (1 participant), depression (1 participant) and pre-stress task perceived stressfulness (6 participants). Excluded cases pairwise was used for treatment of missing data, whereby participants were included in analyses for which data was available. A G-power analysis indicated that a minimum sample size of $N \geq 118$ was required to detect medium effects ($p = .05$, $f^2 = 0.15$) with a power of .80. See table 1 for descriptive and demographic information. All Participants were recruited using the University's online research participation system (SONA) and provided with course credits in exchange for participating in the study. The study was advertised on the

University's research participation website and students who wished to participate signed up for the study and selected an appropriate time slot to attend the health and psychophysiology laboratory. Due to the subsequent change in blood pressure following caffeine consumption (Hartley et al., 2000; James & Richardson, 1991; Savoca et al., 2005) and smoking (Cruickshank et al., 1989), all participants were instructed to refrain from both consuming caffeine and smoking for at least 2 hours before attending the laboratory for their testing session. In order to eliminate the influence of exercise (Somers et al., 1991) and alcohol intake (Potter et al., 1986) on cardiovascular measures, participants were asked to refrain from engaging in vigorous exercise and consuming alcohol for at least 12 hours prior to attending the laboratory session. This study was approved by the university's research ethics committee. All participants provided written informed consent prior to participating and were debriefed following the testing session.

Measures

Type D personality

The 14-item DS14 scale was used to assess Type D personality (Denollet, 2005). This scale measures both SI (7-items) and NA (7-items). Participants are required to answer each item on a 5-point Likert scale ranging from 0 (False) to 5 (true). Examples of items that are used to assess the NA subcomponent include "I am often in a bad mood" and "I am often down in the dumps". Social inhibition is assessed using items such as "I am a closed kind of person" and "I would rather keep other people at a distance". Scores on both the NA and SI subscales can range from 0-28, with individuals scoring ≥ 10 on both subscales classified as having Type D personality. Cronbach's α in the present sample was .87 and .85, for the NA and SI subscales respectively, indicating strong internal consistency.

Although Type D personality is traditionally operationalised as a categorical construct, evidence suggests that Type D personality may be better represented as a continuous construct (Ferguson et al., 2009). Thus, in line with prior Type D studies, a continuous Type D construct was computed as the product of the NA and SI subscales ($NA \times SI$) (e.g. O'Riordan, Howard, Brown, et al., 2020; Whitehead et al., 2007). All analyses were initially conducted using the traditional Type D categorical construct (Type D dichotomy) and were subsequently replicated using the continuous Type D interaction term ($NA \times SI$).

Depression and Anxiety

Anxiety and depressive symptoms were assessed using the Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983). The HADS is a 14-item scale, with 7 items measuring depression and 7 items measuring anxiety. Examples of items used to assess depression include “I feel as if I am slowed down” and “I have lost interest in my appearance”, while items used to assess anxiety include “I feel tense or wound up” and “I get a sort of frightening feeling as if something awful is about to happen”. Each item is scored on a 4-point scale ranging from 0-3, with higher scores on each scale indicating greater levels of anxiety. Both scales showed adequate internal consistency with a Cronbach's α of .67 and .83, for the depression and anxiety subscales respectively.

Cardiovascular Assessment

Cardiovascular parameters including SBP, DBP, and HR were assessed non-invasively using GE Dinamap Pro 300 series electronic oscillometric blood pressure monitor (GE Medical Systems, Freiburg, Germany). Using oscillometric principals, the Dinamap measures mean blood pressure and subsequently estimates SBP and DBP (Stergiou et al., 2012). The Dinamap has been recommended for clinical use (Reinders et al., 2006), and is frequently used in cardiovascular psychophysiology research (Keogh et al., 2021). The arm cuff was attached to the participants' upper arm, over the brachial artery on their non-dominant hand.

A total of 8 measurements were taken during the testing session; four during baseline (0,3,7,9 minutes) and three during the stress task (11,13,15 minutes). The measurements for each phase were averaged to yield a mean baseline and mean stress task value for each participant on SBP, DBP and HR.

Stress Task Measures

Immediately before and after the stress task, participants were asked to rate how stressful they *expected* to find the stress task (pre-task measure), and how stressful they *found* the stress task to be (post-task measure). Participants rated their expected and perceived stressfulness on a 7-point Likert scale ranging from 0 (not at all stressful) to 6 (extremely stressful). These items were included to examine if the stress task was perceived as psychologically stressful.

Demographic and Anthropometric Variables

A weighing scales and stadiometer were used to measure weight and height, which in turn were used to compute BMI. Socio-demographic information including age, sex, smoking status, nationality, and marital status were assessed using a standardised demographic questionnaire.

Stress Task

Participants completed the mental arithmetic stressor from the Trier Social Stress Task (TSST) (Kirschbaum et al., 1993). During this 6-minute task, participants were instructed to begin with the number 1022 and continually subtract 13 as quickly as possible. Participants were instructed to call their answer aloud until the experimenter told them to stop. When the participant made a mistake, they were instructed by the experimenter to restart from 1022. The experimenter sat behind an opaque screen during the stressor phase and was out of the participant's direct line of sight. Cardiovascular measures were recorded at the end of the first, third and fifth minute of the stress task.

Procedure

Upon arrival at the laboratory, participants were greeted by the researcher who went through a brief checklist of exclusionary criterion. From the moment of arrival, participants were allocated a 20-min period to acclimatise to the laboratory setting. During this time participants read the information sheet, signed the consent form and had their height and weight measured for calculation of body mass index (BMI). Participants were seated at a small testing booth and were asked to place their feet into a basin under the table. This action was taken in order to control for unnecessary movements that may influence cardiovascular measures. The arm cuff was then attached to the participant's upper arm and a sample reading was taken to ensure that the blood pressure monitor was functioning correctly, and also to familiarise participants with having their blood pressure measured. For the remainder of the acclimatisation period, participants were provided with questionnaire booklets to complete. During the 10-minute resting baseline phase, participants completed additional questionnaire booklets and were provided with reading material for the baseline phase. Immediately before the stress task began, participants were asked to complete the pre-stress task questionnaire. The experimenter turned off the main lights in the laboratory, leaving participants in the spotlight of a table lamp. Furthermore, the experimenter wore a white laboratory coat throughout the entire experimental procedure and instructed participants to speak aloud whilst completing the stress tasks. These conditions were deliberately manufactured in order to ensure a psychological separation between the experimenter and the participant, and were adapted from prior cardiovascular reactivity studies (Gallagher et al., 2018). There were no other evaluative components to the task. Following the stress-task, the main lights were switched back on, and participants completed the post-task questionnaire. At the end of the experiment, the arm cuff was detached and participants were provided with a debriefing

sheet, thanked for their participation, and then left the laboratory. Details of experimental procedure are detailed in figure 1.

[Insert Figure 1 about here]

Data analyses

Cardiovascular reactivity scores were computed as the difference between mean baseline and mean task values for each cardiovascular parameter. One participant was missing scores on SI and Type D, and one participant was missing scores on depression. Excluded cases pairwise was used for treatment of missing data. All analyses were initially conducted using the traditional Type D dichotomy and were subsequently replicated using the continuous Type D interaction term (NA \times SI). The association between Type D personality and scores on both depression and anxiety were assessed using independent sample t-tests (Type D dichotomy) and correlations (NA \times SI interaction term). A total of 62 (36.04%) participants were classified as Type D, and 110 (63.95%) as non-Type D using the cut-off of ≥ 10 on both the NA and SI subscales (Denollet, 2005). The proportion of participants classified as Type D is similar to prior studies (O'Riordan, Howard, & Gallagher, 2020)

In order to investigate if the stress task successfully perturbed cardiovascular activity, a series of paired sample t-tests were used to test the difference between baseline and task values for each cardiovascular parameter. Similarly, in order to determine if the stress task was perceived as psychologically stressful, a paired samples t-test (pre and post task) were conducted on self-reported stress.

Hierarchical multiple regressions were used to examine the main effect of Type D personality on measures of cardiovascular reactivity. Here, potential confounding variables that are known to influence cardiovascular reactivity (age, gender, BMI, smoking status, baseline cardiovascular measures) were entered into the model at step 1, followed by the main effect of NA and SI at step 2. Type D (dummy coded; non-Type D = 0, Type D = 1)

was then entered into the model at step 3. Similarly, hierarchical multiple regressions were also used to examine the association between the continuous Type D interaction term (NA × SI) and measures of cardiovascular reactivity. Here, hierarchical multiple regressions were conducted with the continuous Type D interaction term entered in place of the Type D dichotomy at step 3 of the above models.

Given that many Type D effects on cardiovascular reactivity are observed in same sex/gender samples (e.g., all women samples) (Howard et al., 2011; Howard et al., 2018; O'Leary É et al., 2013), or in subgroup analyses of men and women (Kupper et al., 2013; O'Riordan et al., 2019; Williams et al., 2009), follow-up replication analyses were subsequently conducted for men ($n = 52$) and women ($n = 121$) separately (gender was removed as covariate in these models). Additionally, follow up analyses were conducted to examine if significant effects of Type D on cardiovascular reactivity remained significant following additional adjustment for anxiety and depressive symptoms. Here, continuous anxiety and depressive symptom scores were added to step 2 of the above models. Finally, a sensitivity moderation analyses using Hayes (2017) PROCESS module for SPSS was used to examine the interaction between Type D and gender on parameters of cardiovascular reactivity.

Results

Descriptive statistics

Descriptive statistics for study variables are reported in table 1, and correlations between all continuous variables are reported in table 2.

[Insert Table 1 and 2 about here]

Type D personality, Anxiety and Depression

Analyses using the categorical Type D construct revealed that Type D individuals reported significantly greater levels of depression $t(169) = 4.67, p < .001$, and anxiety, $t(170) = 9.00, p < .001$, in comparison to their non-Type D counterparts. Furthermore as seen in Table 2, correlational analyses using the continuous Type D construct (NA \times SI), confirmed the association between Type D personality and higher levels of both anxiety and depression. Similarly, both NA and SI were also positively associated with depression and anxiety scores.

Manipulation check

A series of paired samples t -tests (baseline, task) revealed that the stress task successfully perturbed cardiovascular activity: for SBP, $t(172) = 18.04, p < .001$, for DBP, $t(172) = 18.10, p < .001$, and for HR, $t(172) = 16.38, p < .001$. All results were in the expected direction, with a significant increase from resting baseline to the stress task for each cardiovascular parameter.

Furthermore, a paired samples t -test also confirmed a significant increase from pre-task to post-task ratings of self-reported stress, $t(164) = 3.82, p < .001$.

Cardiovascular reactivity

Categorical analyses

Hierarchical multiple regressions were conducted with potential confounding variables entered at step 1, the independent effects on NA and SI at step 2, and the Type D dichotomy (dummy coded; non-Type D = 0, Type D = 1) at step 3. The dichotomous Type D construct did not significantly predict SBP, $b = -.13, t = -1.09, p = .28$, DBP, $b = -.04, t = -.32, p = .753$, or HR reactivity, $b = -.15, t = -1.26, p = .21$. Similarly, no significant main effect emerged for analyses conducted for men or women separately (all $ps \geq .343$).

Continuous analyses

Hierarchical multiple regressions were conducted with potential confounding variables entered at step 1, the independent effects on NA and SI at step 2, and the Type D interaction term (NA \times SI) at step 3. Regression analyses revealed no significant association between the continuous Type D interaction term (NA \times SI) and SBP reactivity, $b = -.53$, $t = -1.93$, $p = .055$, DBP reactivity, $b = -.40$, $t = -1.46$, $p = .147$, or HR reactivity, $b = -.51$, $t = -1.86$, $p = .065$.

Regression analyses conducted separately for men and women revealed no significant association between the continuous Type D interaction term (NA \times SI) and cardiovascular reactivity for men (all p s $\geq .175$). However, amongst women, the continuous Type D interaction term (NA \times SI) was significantly associated with lower SBP reactivity. Furthermore, both NA and SI were also significant independent predictors of SBP reactivity in the same step of this model (see table 3, model 1).

[Insert Table 3 about here]

A follow-up analysis was conducted to examine if the association between the continuous Type D interaction terms (NA \times SI) and lower SBP reactivity amongst woman remained significant after controlling for depression and anxiety. Here, depression and anxiety scores were entered into step 2 of the model in addition to NA and SI. After controlling for depression and anxiety, the association between the continuous Type D interaction term (NA \times SI) and lower SBP reactivity remained significant at step 3. Furthermore, the inclusion of Type D (NA \times SI) at step 3 significantly increased the amount of variance explained in SBP reactivity by 6.9% (R^2 change = .069), $F(1, 108) = 9.04$, $p = .003$. Additionally, the overall model at step 3 was significant, $F(9, 108) = 2.53$, $p = .011$, and explained 17.4% of the variance in SBP reactivity. As can be seen in Table 3, NA, SI and depression, were all significant independent predictors of SBP reactivity in step 3 of the

model. (See table 3, model 2). The association between the continuous Type D interaction term ($NA \times SI$) and SBP reactivity is illustrated in figure 2.

[Insert Figure 2 about here]

Sensitivity analyses

Sensitivity moderation analyses revealed no significant interaction between Type D and gender on cardiovascular reactivity (all $ps > .25$). These non-significant interaction effects may indicate a lack of power to detect effect amongst males.

Discussion

While the current study found no significant association between Type D personality and cardiovascular reactivity amongst men, Type D was found to predict lower SBP reactivity amongst women. Similarly, prior studies that have employed analogous stress tasks (stressors of lower social salience) including mental arithmetic and multitasking stressors have also noted atypically lower cardiovascular responses amongst Type D individuals (Howard et al., 2011; Kelly-Hughes et al., 2014; O'Riordan, Howard, Brown, et al., 2020; O'Riordan et al., 2019), and particularly among women (Gramer et al., 2018; O'Leary É et al., 2013; O'Riordan et al., 2019). In contrast, prior evidence suggests that Type D is associated with exaggerated cardiovascular reactivity amongst men (Williams et al., 2009), particularly in response to highly social stressors (e.g. speech tasks) (Gramer et al., 2018; O'Riordan et al., 2019).

Variations in cardiovascular reactivity between Type D men and women may represent discrete processes leading to separate clusters of health outcomes. Exaggerated cardiovascular reactivity has been primarily associated with adverse cardiovascular health outcomes including cardiac death, hypertension and increased left ventricular mass (Barnett et al., 1997; Carroll, Ginty, Der, et al., 2012; Carroll, Ginty, Painter, et al., 2012; Carroll et

al., 2003; Carroll et al., 1995; Matthews et al., 1998). Furthermore, much of the research from which Type D personality has been found to predict adverse cardiovascular health have employed male dominated samples (Denollet et al., 2006, 2013; Denollet et al., 1996; Denollet et al., 2000). Thus, exaggerated cardiovascular reactivity may serve as a mechanism leading to adverse cardiovascular health, solely amongst Type D men or males. In contrast, many of the outcomes associated with blunted cardiovascular reactivity including obesity, depression, anxiety and eating disorders (e.g. anorexia nervosa, bulimia nervosa) (Carroll et al., 2017), are more prevalent amongst women (Ferrari et al., 2013; Garawi et al., 2014; Hudson et al., 2007; Kessler et al., 1993; McLean et al., 2011; Ng et al., 2014). Therefore, blunted cardiovascular reactivity may be a marker of several negative behavioural and psychological states amongst Type D women or females. Therefore, **gender** differences in cardiovascular reactivity to acute psychological stress amongst Type D individuals may represent different psychosomatic mechanisms, with exaggerated cardiovascular responses leading to adverse cardiovascular health amongst Type D men, and blunted cardiovascular reactivity signalling increased risk of negative psychological and behavioural outcomes (e.g., obesity, depression, anxiety, eating disorders) amongst Type D women.

A second explanation for these sex/gender differences in Type D personality relates to societal norms pertaining to social inhibition. It is suggested that certain behavioural characteristics associated with social inhibited (e.g. social withdrawal) are less acceptable amongst men, who are instead, expected to exhibit highly assertive behaviour (Habra et al., 2003). Furthermore, blunted reactivity is posited to reflect a motivational dysregulation, which engenders withdrawal and disengagement with the stressor (Carroll et al., 2017; Phillips et al., 2013). Therefore, in an effort to maintain social engagement and assertiveness, Type D men may *not* exhibit blunted cardiovascular reactions, as they do not withdraw or disengage with the stress task. In contrast, Type D women may more comfortably exhibit

socially inhibited behaviours (e.g. social withdrawal), and withdraw from the stress task, engendering blunted cardiovascular responses. However, research has yet to distinguish, or indeed even test, whether these differences are related to gender or sex.

Research has also not differentiated between cardiovascular responses that are blunted, and responses that are simply low. Thus, it is also important to acknowledge that the lower cardiovascular responses exhibited amongst Type D women may not indicate blunted cardiovascular reactivity, but rather, a lower response that is healthful or benign. Women tend to be highly socially expressive in comparison to men (Brody & Hall, 2008; Gross & John, 1998; Kring & Gordon, 1998). It may be that the highly frequent expression of negative emotions results in ruminative thinking and focused attention on such negative emotions. Therefore, Type D personality may attenuate the over-expression of negative emotions amongst women, which may draw attention from these emotions and promote more healthful outcomes. However, without a designated threshold of blunted cardiovascular reactivity, we cannot definitively conclude if the responses reported here are benign (low) or pathogenic (blunted).

Type D personality is posited to represent a synergistic interactional effect of NA and SI combined, whereby Type D has predictive utility above the main effects of these individuals constructs (Kupper & Denollet, 2007, 2014; Lodder, 2020). However, prior studies have reported null-effects of Type D on various health outcomes in analyses adjusting for these individual subcomponents (Akram et al., 2018; Coyne et al., 2011; Grande et al., 2011; O'Riordan, Howard, & Gallagher, 2020; Stevenson & Williams, 2014; Williams et al., 2012). Furthermore, others have suggested that the effect observed for Type D are primarily driven by the NA subcomponent rather than a true synergistic interaction (Akram et al., 2018; O'Riordan, Howard, & Gallagher, 2020; Stevenson & Williams, 2014; Williams et al., 2012). However, in accordance with previous cardiovascular reactivity studies (Allen et al., 2019;

Howard et al., 2011; Kelly-Hughes et al., 2014; O'Riordan, Howard, Brown, et al., 2020), we found Type D personality to predict cardiovascular reactivity above and beyond NA and SI amongst women. In fact, NA, SI and Type D (NA \times SI) were found to independently predict SBP reactivity in the same model, with NA and SI predicting increased reactivity and Type D (NA \times SI) predicting diminished reactivity. This indicates that NA, SI and Type D (NA \times SI) are independent constructs, and accounts for discrete variance in cardiovascular reactivity.

Furthermore, others have questioned the predictive utility of Type D personality above other negative affect variables, particularly anxiety and depression (Coyne & de Voogd, 2012; Coyne et al., 2011; Steptoe & Molloy, 2007). In fact, it has been suggested that Type D personality may simply constitute a proxy measure of depression (Coyne et al., 2011), and claims pertaining to a prognostic value of Type D over depression should be treated with scepticism (Coyne & de Voogd, 2012). Smith (2011) suggests that controlling for depression and anxiety is an appropriate approach to determine the unique effect of Type D beyond the effect of potentially overlapping constructs. However, prior studies that have examined the effect of Type D on various health outcomes whilst controlling for anxiety and depression have yielded mixed findings. While some have reported significant effects of Type D in analyses controlling for anxiety and depression (Denollet & Pedersen, 2008; Martens et al., 2010; Mols et al., 2010; Schiffer et al., 2008), others have reported null-effects (Coyne et al., 2011; Grande et al., 2011; Volz et al., 2011). While depression and anxiety have been consistently noted to be a strong correlate of blunted cardiovascular reactivity (Brindle et al., 2013; Carroll et al., 2007; Schwerdtfeger & Rosenkaimer, 2011; York et al., 2007; Yuenyongchaiwat et al., 2017), little research to date has examined if Type D predicts cardiovascular reactivity in analyses controlling for anxiety and depression. Only one study to date has examined the effect of Type D on cardiovascular reactivity in analyses adjusting for depression (Bibbey et al., 2015). However, Bibbey et al. (2015) did not examine the effect

of the continuous Type D interaction term ($NA \times SI$), nor did they control for the main effects of NA or SI. Our findings suggest that the continuous Type D interaction term ($NA \times SI$) predicts cardiovascular reactivity above anxiety and depression, in addition to NA and SI, amongst women. In fact, both depression and Type D personality were independent predictors of blunted SBP reactivity. In line with these findings, Denollet and Conraads (2011) propounds that Type D personality and depression are distinct forms of distress, with clear conceptual differences, discrete consequences for cardiovascular health, and have independent pathways to disease outcomes.

Finally, while we found Type D personality to significantly predict cardiovascular reactivity when using the continuous interaction term ($NA \times SI$), these results could not be replicated using the more traditional Type D dichotomy. Not only has the operationalisation of Type D personality as a categorical construct been criticised as it increases the probability of “lucky cuts” that produce spurious findings (Coyne & de Voogd, 2012; Smith, 2011), but it also results in a loss of valuable statistical information, that reduces the statistical power to detect significant effects (Altman & Royston, 2006; Royston et al., 2006). Therefore, when significant effects of Type D exist, these may not be detected using the Type D dichotomy. In fact, prior evidence suggests that analyses employing the continuous Type D interaction term ($NA \times SI$) term are more adequately powered than analyses employing the Type D dichotomy (Lodder, 2020). Thus, it is not surprising that the significant effects reported here were observed when using the continuous Type D interaction term ($NA \times SI$), but not when using Type D dichotomy.

While the current study employed a relatively large sample size ($n = 173$), the sample consisted of undergraduate students mainly of a relatively young age, with specific sample characteristics. Additionally, while we attempted to recruit equal numbers of men and women to the study, we were unable to recruit an equal number of men, with our sample comprising

of 69.6% women. Therefore, it is questionable if the results are generalizable to other cohorts. Therefore, while our analyses were adequately powered for both the sample as a whole and amongst woman, analyses amongst men ($n = 52$) may have lacked adequate power to detect significant effects. This reduced power to detect effects amongst men may also explain why the interaction between Type D and gender on cardiovascular reactivity was non-significant in sensitivity moderation analyses. Thus, future studies should employ samples with a higher proportion of men to confirm the findings reported here. One strength of using healthy samples rather than clinical samples is that it avoids potential confounds associated with the occurrence of existing disease. Nevertheless, we recommend that future studies recruit different cohorts to confirm the results of the current study. Finally, while we interpreted the lower SBP responses exhibited by women scoring high on Type D as indicative of blunted cardiovascular reactivity, a precise threshold of blunted cardiovascular reactivity has yet to be determined. Therefore, the lower responses exhibited by Type D women may indicate a healthy or benign response. Thus, future research should distinguish between responses that are simply low (healthy) and responses that are blunted (unhealthy) in order to determine if the lower responses exhibited by Type D individuals can be considered pathogenic. Finally, while we used the HADS to assess anxiety and depressive symptoms, others have accentuated numerous structural, conceptual, and psychometric problems with this measure, resulting in inadequate sensitivity to identify clinical depression and anxiety based on psychiatric interviews (Cosco et al., 2012; James C Coyne & Eric van Sonderen, 2012; J. C. Coyne & E. van Sonderen, 2012; Mitchell et al., 2010). However, we employed continuous HADS scores in analyses, avoiding potential issues pertaining to the identification of depression and anxiety caseness based on cut-offs scores.

In conclusion, the current study examines the association between Type D personality and cardiovascular reactivity to a mental arithmetic stressor in a sample of healthy

undergraduate students. The continuous Type D interaction term (NA \times SI) was associated with blunted SBP reactivity amongst women, but not men. Furthermore, this result was significant in analyses adjusting for NA, SI, depression and anxiety, suggesting that Type D personality has predictive utility for cardiovascular reactivity above its individual subcomponents (NA and SI), as well as anxiety and depression.

Disclosure of interest statement

The authors report no conflict of interest.

Data availability statement

The data that support the findings of this study are available from the corresponding author, upon reasonable request.

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Table 1. Descriptive statistics of psychological, demographic and cardiovascular reactivity variables

Psychometric and Reactivity Variables	Type D (<i>N</i> = 62)	Non-Type D (<i>N</i> = 110)	Sample (<i>N</i> =173)
Mean (SD)			
Type D (Continuous)	239.68 (111.14)	59.18 (45.26)	124.24 (115.18)
Negative affect	15.92 (4.19)	8.18 (4.75)	10.98 (5.86)
Social inhibition	14.69 (4.46)	6.85 (3.87)	9.68 (5.56)
Depression	4.31 (3.00)	2.47 (2.12)	3.19 (2.72)
Anxiety	10.77 (3.96)	5.95 (3.00)	7.69 (4.08)
SBP Reactivity	11.80 (9.56)	12.85 (8.74)	12.41 (9.04)
DBP Reactivity	6.75 (5.45)	7.31 (5.02)	7.10 (5.16)
HR Reactivity	10.26 (7.79)	10.89 (9.02)	10.65 (8.55)
Age	20.31 (2.80)	20.79 (3.97)	20.61 (3.58)
BMI	23.09 (4.10)	23.73 (3.47)	23.52 (3.71)
<i>N</i> (% female)			
Sex	46 (74.2%)	74 (67.3%)	121 (69.9%)
<i>N</i> (%) Smoking status			
Smoker	10 (16.7%)	12 (10.9%)	22 (12.7%)
Non-Smoker	50 (83.3%)	98 (89.1%)	149 (86.1%)
<i>N</i> (%) Ethnicity			
White/Caucasian	60 (96.8%)	105 (95.5%)	165 (95.4 %)
Asian	1 (1.6%)	2 (1.8%)	3 (1.7%)
Black	0 (0%)	1 (.9%)	1 (.6%)
Latino	0 (0%)	1 (.9%)	1 (.6%)
Other	1 (1.6%)	1 (.9%)	3 (1.7%)

Table 2. Correlations between NA x SI, depression, anxiety and cardiovascular reactivity variables.

	1	2	3	4	5	6	7	8	9
1. Type D (Continuous)	-	.82**	.87**	.57**	.70**	-.05	-.06	-.002	.24**
2. Negative affect	-	-	.56**	.53**	.73**	-.06	-.01	.02	.41**
3. Social inhibition	-	-	-	.43**	.52**	.02	-.05	.05	.14
4. Depression	-	-	-	-	.56**	-.20**	-.05	-.14	.06
5. Anxiety	-	-	-	-	-	-.10	-.03	.04	.26**
6. SBP Reactivity	-	-	-	-	-	-	.43**	.53**	.02
7. DBP Reactivity	-	-	-	-	-	-	-	.37**	.17*
8. HR Reactivity	-	-	-	-	-	-	-	-	.13
9. Self-reported stress (post-task)	-	-	-	-	-	-	-	-	-

* = $P < 0.05$, ** $P < 0.01$

Table 3. Regression analyses for Type D personality (NA × SI) and SBP reactivity amongst females

	Model 1					Model 2				
	<i>F</i>	<i>R</i> ²	<i>β</i>	<i>t</i>	<i>p</i>	<i>F</i>	<i>R</i> ²	<i>β</i>	<i>T</i>	<i>p</i>
Step 1	1.54	.052	-	-	.196	1.54	.052	-	-	.196
Age			-.07	-.79	.429			-.07	-.79	.429
Smoking status			-.21	-2.28	.025			-.21	-2.28	.025
BMI			-.08	-.76	.450			-.08	-.76	.450
Resting SBP			-.01	-.15	.885			-.01	-.15	.885
Step 2	1.06	.054	-	-	.39	1.60	.105	-	-	.134
Age			-.08	-.88	.382			-.08	-.84	.405
Smoking status			-.21	-2.14	.035			-.17	-1.77	.079
BMI			-.08	-.76	.451			-.05	-.51	.612
Resting SBP			-.01	-.14	.886			.02	.24	.809
NA			-.04	-.40	.689			.06	.45	.657
SI			-.02	-.16	.872			.03	.31	.760
Anxiety			-	-	-			.02	.11	.912
Depression			-	-	-			-.28	-2.43	.017
Step 3	2.33	.129	-	-	.030	2.53	.174	-	-	.011
Age			.01	.07	.942			.01	.11	.912
Smoking status			-.17	-1.84	.068			-.14	-1.51	.134
BMI			-.07	-.70	.484			-.05	-.49	.627
Resting SBP			-.03	-.32	.753			.001	.01	.991
NA			.48	2.40	.018			.53	2.60	.011
SI			.66	2.72	.008			.68	2.84	.005
Anxiety			-	-	-			.08	.57	.568
Depression			-	-	-			-.27	-2.43	.017
Type D (NA × SI)			-1.06	-3.07	.003			-1.037	-3.01	.003

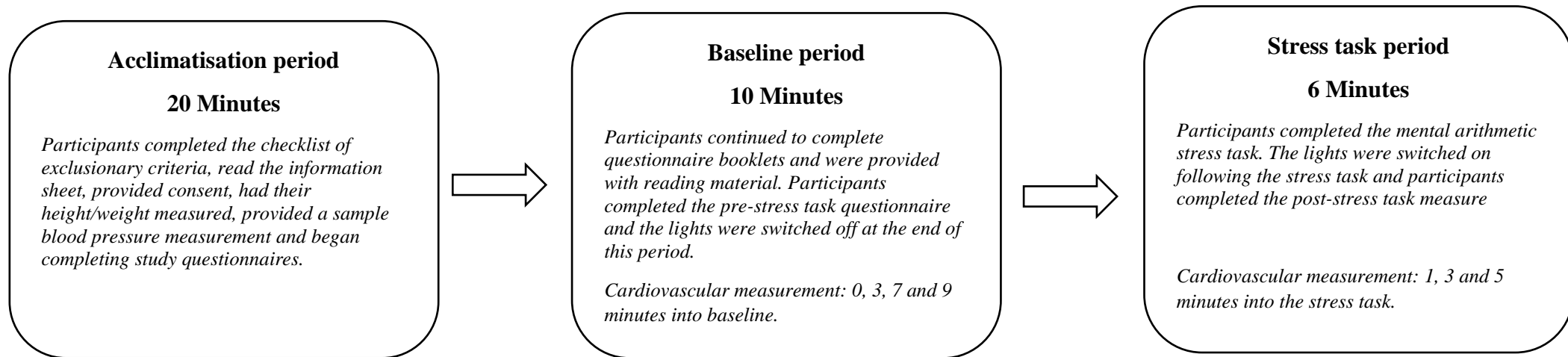


Figure 1. Flow diagram of the experimental procedure

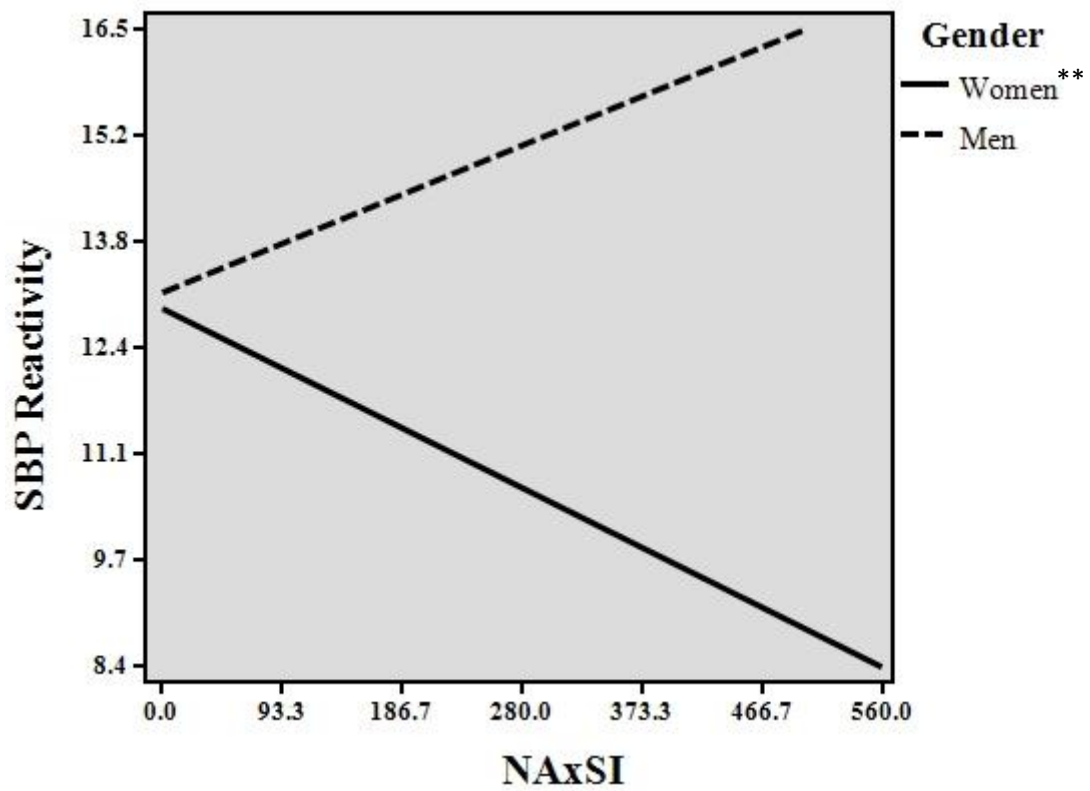


Figure 2. The association between Type D (NA \times SI) and SBP reactivity.

** = $P < 0.01$