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Type D personality and cardiovascular reactivity to acute stress:

The mediating effects of social support and negative social relationships

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Abstract

Type D personality has been consistently associated with adverse cardiovascular health with atypical cardiovascular reactions to psychological stress one potential underlying mechanism. As Type D individuals have been noted to report lower social support and greater perceptions of negativity in social interactions, this study examined if the association between Type D personality and cardiovascular reactivity was mediated by these social relationships. A sample of 195 undergraduate students (138 female) participated in this observational study, where they completed measures assessing Type D personality (DS14), social support and perceptions of negative social relationships (NIH social relationship scales), before undergoing a traditional cardiovascular reactivity protocol. Systolic and diastolic blood pressure (SBP; DBP), heart rate (HR), cardiac output (CO) and total peripheral resistance (TPR) were monitored throughout. ANCOVAs and regressions indicated that Type D personality was associated with lower cardiovascular reactivity to a mental arithmetic stressor. Furthermore, mediation analyses (process macro) indicated that the relationship between Type D personality and cardiovascular reactivity was mediated via increased perceptions of negative social relationships, as well as lower levels of social support. Apart from a significant association between Type D personality and increased HR reactivity, all results failed to withstand adjustment for the individual effects of negative affect and social inhibition in controlled analyses. Overall, these findings suggest that the predictive utility of Type D personality on cardiovascular reactivity above and beyond the individual effects of negative affect and social inhibition is limited, and may vary depending on the cardiovascular parameter of focus.

Key Words: Type D personality, Cardiovascular reactivity, Social support, Negative social relationships, Stress

1. Introduction

Type D (distressed) personality is characterised by increased levels of both negative affectivity (NA) and social inhibition (SI). The negative affectivity facet of Type D refers to the tendency to experience an array of negative emotions across time, while the social inhibition facet refers to the tendency to inhibit the expression of these negative emotions during social interactions (Denollet, 2005). Over the past two decades, Type D personality has been consistently associated with adverse health-related outcomes amongst cardiac patients, including poor prognosis, as well as cardiac and all-cause mortality (Denollet, Pedersen, Vrints, & Conraads, 2006; Denollet et al., 1996; Denollet et al., 2018; Leu et al., 2019; Martens, Mols, Burg, & Denollet, 2010; Schiffer, Smith, Pedersen, Widdershoven, & Denollet, 2010). In fact, a meta-analysis has found that Type D personality confers a 2-fold increased risk of hard endpoints (such as death and reoccurrence of cardiac events) in cardiac populations (Grande, Romppel, & Barth, 2012). More recently, Type D personality has been included in the European Cardiovascular Prevention Guidelines as a potential risk factor to assess (Piepoli et al., 2016).

Several mechanisms have been posited to facilitate the relationship between Type D personality and adverse cardiovascular health. Indirect mechanisms have primarily propounded the engagement in negative health behaviours such as unhealthy eating, physical inactivity and smoking (Booth & Williams, 2015; Ginting, van de Ven, Becker, & Näring, 2016; Williams, Abbott, & Kerr, 2016). Additionally, direct mechanisms have accentuated the influence of physiological processes such as increased diurnal cortisol output (Molloy, Perkins-Porras, Strike, & Steptoe, 2008; Whitehead, Perkins-Porras, Strike, Magid, & Steptoe, 2007), and pro-inflammatory activity (Denollet et al., 2009; Denollet, Vrints, & Conraads, 2008; Jandackova, Koenig, Jarczok, Fischer, & Thayer, 2017). One physiological mechanism that has received considerable support is cardiovascular reactivity to acute stress,

with Type D individuals continually found to exhibit atypical cardiovascular responses to stress (Allen, Wetherell, & Smith, 2019a; Bibbey, Carroll, Ginty, & Phillips, 2015; Gramer, Haar, & Mitteregger, 2018; Howard, Hughes, & James, 2011; Kelly-Hughes, Wetherell, & Smith, 2014; O'Leary, Howard, Hughes, & James, 2013; O'Riordan, Howard, & Gallagher, 2019; Williams, O'Carroll, & O'Connor, 2009).

This physiological mechanism is premised on the cardiovascular reactivity hypothesis, which postulates that prolonged or exaggerated cardiovascular responses to acute psychological stress promotes the development of cardiovascular diseases (Obrist, 2012). This hypothesis has received considerable support, with heightened cardiovascular reactions to stress continually associated with adverse cardiovascular outcomes including hypertension (Carroll et al., 2012b; Markovitz, Raczynski, Wallace, Chettur, & Chesney, 1998), atherosclerosis (Barnett, Spence, Manuck, & Jennings, 1997; Matthews et al., 1998), and cardiovascular disease mortality (Carroll et al., 2012a). More recently, atypically low or “blunted” cardiovascular reactions to stress have also been associated with a range of adverse health-related outcomes (Carroll, Ginty, Whittaker, Lovallo, & de Rooij, 2017), and have been similarly linked to negative cardiovascular outcomes including increased carotid intima-media thickness (Ginty et al., 2016), and all-cause mortality among heart failure patients (Kupper, Denollet, Widdershoven, & Kop, 2015). Thus, it has now been posited that both exaggerated and blunted cardiovascular responses to psychological stress implies a homeostatic dysfunction and psychosomatic disease vulnerability (Lovallo, 2011).

Although the majority of studies have found Type D individuals to exhibit blunted cardiovascular reactions (Howard et al., 2011; Kelly-Hughes et al., 2014; O'Leary et al., 2013), others have linked Type D personality with exaggerated (Kupper, Pelle, & Denollet, 2013; Williams et al., 2009) and mixed cardiovascular responses (Allen et al., 2019a).

However, these disparate findings may be explained on examination of potential moderating

variables, including gender and the type of stress task (Bibbey et al., 2015; Gramer et al., 2018; O’Riordan et al., 2019).

Traditionally, Type D personality was analysed as a dichotomous typology, with participants scoring above the established cut-off point (≥ 10) on both subcomponents (NA and SI) classified as Type D and the remaining as non-Type D (Denollet, 2005). However, research has suggested that Type D may be better represented as a continuous variable based on the product of the SI and NA subscales, than as a dichotomous variable (Ferguson et al., 2009). Furthermore, whilst some of the aforementioned studies have noted a relationship between Type D personality and cardiovascular reactivity when solely using the traditional dichotomous Type D construct (Bibbey et al., 2015; Williams et al., 2009), others have noted effects using the continuous (Kelly-Hughes et al., 2014; O’Riordan et al., 2019) or both constructs (Allen et al., 2019a; Gramer et al., 2018; Howard et al., 2011; Kupper et al., 2013; O’Leary et al., 2013). Thus, all analyses in the current study will be initially conducted using the traditional Type D dichotomy and will be subsequently replicated using the continuous Type D interaction term (NA \times SI).

Moreover, traditionally in cardiovascular reactivity research, the focus has been on individual cardiovascular indices, i.e. SBP, CO. However, blood pressure reactivity to stress is regulated by the reciprocal relationship between CO and TPR, referred to as hemodynamic profile. Changes in CO can be compensated by inverse changes in TPR and vice versa. Thus, a greater compensatory deficit between CO and TPR results in greater increases in blood pressure. Blood pressure responses of similar magnitude may occur as a result of discrete patterns of change in CO and TPR. Changes in blood pressure may be due to an increase in CO accompanied by an insufficient decrease in TPR (myocardial response), an increase in TPR accompanied by an insufficient decrease CO (vascular response), or an increase in both physiological parameters (mixed response) (Gregg, Matyas, & James, 2002; James, Gregg,

Matyas, Hughes, & Howard, 2012). It is posited that differential hemodynamic profiles may engender cardiovascular disease risk via discrete pathways (Gregg et al., 2002; Gregg, Matyas, & James, 2005). Examination of this profile will be investigated here, albeit research examining the hemodynamic profile exhibited by Type D individuals have yielded mixed findings, with prior research linking Type D personality to myocardial (Howard et al., 2011), and mixed (Allen et al., 2019a; O'Leary et al., 2013) hemodynamic profiles during active stress tasks. Thus, further research is warranted.

While Type D personality has been consistently associated with abnormal cardiovascular reactivity to stress, research has not yet elucidated the mediating factors that may facilitate this association. Considering the socially inhibited nature of Type D personality, factors that are likely to mediate this association may pertain to social relationships. In fact, Type D individuals have been consistently found to report lower perceptions of social support (Ginting et al., 2016; Polman, Borkoles, & Nicholls, 2010; Sararoudi, Sanei, & Baghbanian, 2011; Shao, Yin, & Wan, 2017; Staniute et al., 2015; Williams et al., 2008). Perceived social support is often not representative of the actual social support received by an individual, and is dependent on the appraisal and beliefs of the recipient regarding the quality and accessibility of social support (Eagle, Hybels, & Proeschold-Bell, 2019; Uchino, 2009; Uchino, Carlisle, Birmingham, & Vaughn, 2011). Given that Type D individuals are posited to feel tense, socially inhibited, and insecure when in the presence of other people (Denollet, 2005), this perception of lower social support is likely to be due to a cognitive bias of interpersonal interpretation amongst Type D individuals. Furthermore, this cognitive bias has been found to promote increased perceptions of negativity (perceived threat, anticipated distress and difficulty forming verbal responses) during hypothetical social interactions amongst Type D individuals (Grynberg, Gidron, Denollet, & Luminet, 2012; Howard, O'Riordan, & Nolan, 2018). Additionally, prior studies have found this cognitive bias of

interpersonal interpretation to influence patterns of physiological arousal amongst Type D individuals (Howard et al., 2018). Given that social support (stress buffering) and negative social relationships (stress exacerbation/social aggravation) are propounded to impact health outcomes by influencing stress appraisal and coping (Birmingham & Holt-Lunstad, 2018; Cohen & Wills, 1985; Cranford, 2004; Rook, 1984), it is likely that these types of social relationships are important mediating factors engendering the aberrant physiological reactions to stress for Type D individuals.

Albeit a myriad of research findings accentuating the negative health effects of Type D personality, some have proffered criticisms of the Type D construct (Coyne & de Voogd, 2012; Coyne et al., 2011; Smith, 2011). One common criticism of Type D personality pertains to the predictive utility of Type D personality above and beyond the independent effects of NA and SI, as well as the conceptualization of Type D personality as a dichotomous rather than a continuous variable. Type D personality is posited to consist of more than the presence of NA and SI and is suggested to represent a synergistic interactional effect of both constructs combined (Denollet, 2005; Kupper & Denollet, 2007, 2014). Thus, Type D personality should predict outcomes above and beyond the effects of NA and SI independently. Analyses controlling for NA and SI separately, is therefore the most appropriate analytical method of determining the predictive utility of Type D personality. Previous research that has begun to control for the individual effects of NA and SI have reported null-effects of Type D personality on a range of self-reported and objective health outcomes (Akram et al., 2018; Coyne et al., 2011; Grande et al., 2011; O'Riordan, Howard, & Gallagher, 2020; Stevenson & Williams, 2014; Williams, O'Connor, Grubb, & O'Carroll, 2012). However, others have reported small, but independent effects of Type D on health outcomes after controlling for NA and SI (Allen, Wetherell, & Smith, 2019b). 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 et al., 2013; O'Riordan et al., 2019; Williams et al., 2009), a small number of studies have found Type D to predict aberrant physiological responses after controlling for the individual Type D subcomponents (Allen et al., 2019a; Howard et al., 2011; Kelly-Hughes et al., 2014; Kupper et al., 2013).

Considering the above evidence, the present study has three key aims. Firstly, the current study will examine the association between Type D personality and cardiovascular reactivity to acute psychological stress, as well as the hemodynamic profile underlying these cardiovascular responses. Secondly, the current study aims to examine if the association between Type D personality and cardiovascular reactivity to acute stress is mediated via perceptions of social support and of negative social relationships. Finally, the current study will examine if Type D personality has predictive utility when treated as both a dichotomous and dimensional variable, and when controlling for the independent main effects of NA and SI.

2. Method

2.1 Design

The current study employed a between-subjects design. The main predictor variable was Type D personality. Mediating variables included two measures of social support (instrumental and emotional) and two measures of negative social relationships (perceived rejection and perceived hostility). The main outcome variables included measures of cardiovascular reactivity including SBP, DBP, HR, CO and TPR. In line with previous research, reactivity scores were computed as the difference between mean baseline and mean task value for each cardiovascular parameter (Gallagher, O'Riordan, McMahon, & Creaven, 2018; Phillips, Gallagher, & Carroll, 2009). All analyses were initially conducted using the traditional Type D dichotomy and were subsequently replicated using the continuous Type

D interaction term ($NA \times SI$). A total of 75 participants were classified as Type D using the cut-off of ≥ 10 on both the NA and SI subscales (Denollet, 2005).

2.2 Participants

One hundred and ninety-five undergraduate students (70.8% female) participated in this study. Participants were recruited using the University's online research participation system and were provided with 3 course credits in exchange for their participation. The study was advertised on the university's research participation website and students who wished to participate signed up for the study and were allocated a time slot to attend the laboratory. Participants ranged in age from 18-53 years ($M = 20.95$, $SD = 4.58$). In order to minimise the potential influence of confounding variables, participants were excluded from the study if they reported taking medication that may influence cardiovascular measures or if they had a diagnosis of a cardiovascular condition. Furthermore, due to the subsequent change in blood pressure following smoking (Cruickshank, Neil-Dwyer, Dorrance, Hayes, & Patel, 1989) and consuming caffeine (Hartley et al., 2000; James & Richardson, 1991; Savoca et al., 2005), all participants were instructed to refrain from consuming caffeine and smoking for at least 2 hours before attending the testing session. In addition, in order to eliminate the influence of exercise (Somers, Conway, Coats, Isea, & Sleight, 1991) and alcohol intake (Potter, Watson, Skan, & Beevers, 1986) on cardiovascular functioning, participants were asked to refrain from engaging in vigorous exercise and consuming alcohol for at least 12 hours prior to attending the laboratory session. A total of 30 participants (15.38%) were missing data on one or more study variables. Missing data was excluded using excluded cases pairwise (Pallant, 2013). Furthermore, a G-power analysis indicated that a sample of $N \geq 138$ was required to detect medium effects ($p = .05$, $f^2 = 0.15$) with a power of .95.

2.3 Measures

2.3.1 Type D Measure

The DS14 was used to assess Type D personality (Denollet, 2005). The DS14 is a 14-item scale, measuring both social inhibition (SI; 7 items) and negative affectivity (NA; 7 items). Participants were required to respond to each item on a 5-point Likert scale ranging from 0 (*False*) to 5 (*True*). Examples of items measuring SI include ‘I am a closed kind of person’ and ‘I would rather keep other people at a distance’ while NA is assessed using items such as ‘I am often down in the dumps’ and ‘I am often in a bad mood’. Both scales were found to display strong internal consistency, with a Cronbach’s α of .86 and .85 for the NA and SI scales respectively. Scores on both subscales can range from 0-28, with individuals scoring ≥ 10 on both subscales classified as having Type D personality. Additionally, prior research has demonstrated that Type D may be more accurately 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 SI and NA subscales (Howard & Hughes, 2013; Howard et al., 2011; Howard et al., 2018). All analyses were initially conducted using the traditional Type D dichotomy and were subsequently replicated using the continuous Type D interaction term ($NA \times SI$).

2.3.2 Social Support

Instrumental and emotional social support were assessed using the two independent 8-items scales from the NIH (National Institute of Health) social relationship questionnaire (Cyranowski et al., 2013). Participants were required to rate on a 5-point Likert scale ranging from 1 (*Never*) to 5 (*Always*), how often they experienced each item over the past month. The emotional support scale includes items such as ‘I have someone who will listen to me when I need to talk’ and ‘I have someone I trust to talk with about my feelings’. The instrumental support scale includes items such as ‘I have someone to take me to the doctor if I need it’ and

‘I have someone to help me if I’m sick in bed’. Both scales were found to display strong internal consistency, with a Cronbach’s α of .93 and .91 for the instrumental support and emotional support scales respectively.

2.3.3 Negative Social Relationships

Perceptions of hostility and rejection from others were assessed using the social distress scales from the NIH adult social relationship questionnaire. The perceived hostility scale assesses perception of ridicule, criticism and hostility from others and the perceived rejection scale assesses perception of neglect and rejection from others. Items measuring perceived hostility include ‘Yell at me’ and ‘Act nasty to me’ and items measuring perceived rejection include ‘Don’t listen when I ask for help’ and ‘Act like they don’t have time for me’. Both scales were answered on a 5 point Likert scale ranging from 1 (*Never*) to 5 (*Always*). Both social distress subscales were also found to display strong internal consistency with a Cronbach’s α of .91 and .90 for the perceived rejection and perceived hostility scales respectively.

2.3.4 Cardiovascular Measurement

Cardiovascular parameters including systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), cardiac output (CO) and total peripheral resistance (TPR) were assessed using a Finometer Pro hemodynamic cardiovascular monitor (Finapres Medical Systems BV, BT Arnhem, The Netherlands). The Finometer takes continuous beat-to-beat non-invasive measures from one’s finger arterial pressure using the volume clamp method (Penaz, 1973). A finger cuff is attached to the participants’ middle finger on their non-dominant hand and an arm cuff is attached to the participants’ upper arm to calibrate reconstructions of the intrabrachial pressure derived from the finger cuff. The Finometer also uses a hydrostatic height correction system to correct participant’s hand height to heart level. The Finometer has been extensively used in previous cardiovascular psychophysiology

studies (Gallagher et al., 2018; O'Súilleabháin, Howard, & Hughes, 2018; Soye & O'Súilleabháin, 2019) and has been continually found to provide an accurate measure of blood pressure (Guelen et al., 2003; Schutte, Huisman, van Rooyen, Malan, & Schutte, 2004; Schutte, Huisman, Van Rooyen, Oosthuizen, & Jerling, 2003). Beat-to-beat data for each cardiovascular parameter was averaged across resting baseline (10-minutes), the maths task (6-minutes) and the speech task (7-minutes) using the BeatScope programme for downloading Finometer data.

2.3.5 Stress Task Measures

Immediately before and after the stress tasks participants were required to indicate how stressful they *expected* to find each task and how stressful they *found* each task. Participants were required to report the expected stressfulness and perceived stressfulness of both tasks on a 7-point Likert scale ranging from 0 (*not at all stressful*) to 6 (*extremely stressful*).

2.4 Stress Task

The stress task was an adapted version of the Trier Social Stress Task (TSST) (Kirschbaum, Pirke, & Hellhammer, 1993), which included both a maths task (6-minutes) and a speech task (7-minutes). The paced auditory serial addition test (PASAT) (Gronwall, 1977) was used as our mental arithmetic task. During this task, participants listened to an audio track in which single digit numbers were played aloud. The digits were played at a speed of 2.4 seconds during the first minute of the task, with the speed increasing by .4 seconds each minute throughout the task. Participants were required to retain the digit presented and add it to the subsequent digit. During the speech task, participants were instructed to give a speech in which they were required to describe 3 of their best and worst characteristics, with the use of real life examples (Bosch et al., 2009). Participants were instructed to continually speak for the entire task without any cessation. If the participants stopped speaking at any point throughout the task, they were immediately instructed to continue speaking by the

experimenter. However, unlike the original TSST, there was no panel present during the stress tasks and participants were not voice or video recorded. Further, only one experimenter was present during the study. These tasks have been previously used in cardiovascular reactivity studies and have been found to successfully perturb cardiovascular activity (Gallagher et al., 2018). Given, that the relationship between Type D and cardiovascular reactivity has been found to vary across stress tasks (Bibbey et al., 2015; Gramer et al., 2018), we examined reactivity to both stress tasks separately.

2.5 Procedure

Prior to arriving at the laboratory, all participants were presented with an information sheet detailing relevant information about the study and the study restrictions. Students who volunteered to take part were invited to attend a 1 hour testing session. From the moment of arrival at the laboratory, participants were given 20 minutes to acclimatise to the laboratory environment. During this period, participants were firstly provided with an information sheet and the researcher went through a short checklist of exclusion criteria. Any questions participants had regarding the study were then answered by the researcher. Once participants signed the consent form, they completed a demographic questionnaire and then had their height and weight assessed in order to calculate body mass index (BMI). Participants were then asked to take a seat at a desk on which a laptop and lamp were placed. The Finometer was then attached to the participant. Participants remained seated and were provided with reading material for the remainder of the acclimatisation period. Following acclimatisation, resting cardiovascular function was assessed for a 10-minute period. Immediately before the stress task began, the experimenter provided participants with the pre-stress task measure and switched off the main lights in the laboratory. Participants completed the task under the spotlight of the lamp. 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. Immediately after the stress tasks participants completed the post-stress task questionnaire. Following the post-task 15-minute recovery period, the Finometer was detached and participants were provided with a debriefing sheet. Although a recovery period was included in the experimental procedure, an a priori decision was made to solely examine cardiovascular reactivity and the mediation pathways.

2.6 Data analyses

All analyses were initially conducted using the traditional Type D dichotomy and were subsequently replicated using the continuous Type D interaction term ($NA \times SI$). Correlations ($NA \times SI$ interaction term) and independent sample t-tests (Type D dichotomy) were used to examine the association between Type D personality and social relationship variables. In order to investigate if the stress task successfully perturbed cardiovascular activity a series of repeated measures (baseline, task) ANOVAs were conducted on each cardiovascular parameter. Similarly, in order to determine if the stress tasks were perceived as psychologically stressful, repeated measures ANOVAs (pre and post task) were conducted on self-reported stress for both tasks.

Main effects of Type D personality on measures of cardiovascular reactivity were examined using ANCOVAs for the categorical Type D construct and hierarchical multiple regressions for the continuous Type D interaction term ($NA \times SI$). Type D was entered into ANCOVAs/regressions as the independent/predictor variable and measures of cardiovascular reactivity were entered as dependant/outcome variables. In order to control for potential confounding variables, age, sex, BMI, smoking status, task order and baseline cardiovascular measures were entered into ANCOVAs as covariates, and into step 1 of multiple regressions, with the interaction term ($NA \times SI$) entered at step 2.

Hemodynamic profile (HP) and compensation deficit (CD) scores were computed using the model proposed by Gregg et al. (2002); subsequently reviewed by James et al. (2012). As per previous studies (Howard et al., 2011; O'Leary et al., 2013), one sample t-tests were conducted to examine if HP and CD scores were significantly different from 0 for both Type D and non-Type D individuals. Correlations between the continuous Type D interaction terms ($NA \times SI$) and scores of HP and CD were then conducted.

Multiple parallel mediation analyses using model 4 of Hayes (2017) PROCESS module for SPSS was used to examine if the relationship between Type D personality and cardiovascular reactivity was mediated via social support and negative social relationships. Type D (categorical and continuous) was entered into the model as the predictor variable. All social relationship variables were entered simultaneously into the model as potential mediation variables and reactivity parameters were entered separately as outcome variables. 95% confidence levels for confidence intervals were estimated using bootstrapping samples of 5000. Ranges in confidence interval levels (lower to upper confidence intervals) for indirect effects that did not include 0 were used to identify significance. Partial or full mediation was determined by examining if direct effects were significant whilst mediation variables were included in the model. Mediation analyses were conducted whilst controlling for the aforementioned confounding variables (age, sex, BMI, smoking status, task order and baseline cardiovascular measures).

Subsequently, in order to test if Type D personality was associated with social relationship and cardiovascular reactivity variables after controlling for the individual Type D continuous subcomponents (NA and SI) hierarchical multiple regressions were conducted. For analyses on social relationship variables, the effects of NA and SI were entered independently in step 1 and the dichotomous Type D typology (dummy coded; non-Type D = 0, Type D = 1) was then entered into the model at step 2. For analyses on cardiovascular reactivity variables, the

aforementioned confounding variables were entered at step 1, the individual effects of NA and SI were entered at step 2, and dichotomous Type D typology was entered at step 3. These multiple regressions were replicated, with the continuous Type D interaction term ($NA \times SI$) entered into the models in place of the Type D dichotomy. Subsequent mediation analyses were conducted whereby the NA and SI subcomponents were entered into the model as additional covariates in order to investigate if mediation effects withstood adjustment for the Type D subcomponents.

3. Results

3.1 Descriptive Statistics

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

INSERT TABLE 1 ABOUT HERE

3.2 Type D Personality and Social Relationships

Analyses using the categorical Type D construct revealed that Type D individuals reported significantly lower levels of emotional, $t(186) = 4.83, p < .001$, and instrumental, $t(185) = 2.62, p = .01$, social support. Furthermore, Type D individuals reported significantly greater perceptions of hostility, $t(186) = 4.46, p < .001$, and rejection, $t(185) = 4.70, p < .001$, from others.

As seen in Table 2, all results were confirmed using the continuous Type D construct ($NA \times SI$). Similar results were observed for both Type D continuous subcomponents, with both NA and SI associated with lower social support and increased perceptions on negative social relationships.

INSERT TABLE 2 ABOUT HERE

3.3 Manipulation Check

A series of repeated measures (baseline, task) ANOVAs confirmed that both the maths task and the speech task successfully perturbed cardiovascular activity for all cardiovascular parameters (all $ps < .001$), with an increase from baseline to both stress tasks across all parameters. Further, repeated measures ANOVAs also revealed a significant increase from pre to post-task ratings of self-reported stress for the maths task, $F(1, 189) = 67.82, p < .001, \eta_p^2 = .26$, and the speech task $F(1, 191) = 69.73, p < .001, \eta_p^2 = .27$, indicating that both tasks were perceived as psychologically stressful.

3.4 Type D personality and cardiovascular reactivity

ANCOVA analyses using the categorical Type D construct revealed that there was no significant main effect for Type D on cardiovascular reactivity to the speech task (all $ps > .13$). However, in response to the maths task, there was a near significant main effect of Type D on SBP reactivity, $F(1, 162) = 3.88, p = .051, \eta_p^2 = .02$, and a significant effect of Type D on DBP reactivity, $F(1, 162) = 4.65, p = .03, \eta_p^2 = .03$. Type D individuals exhibited significantly lower blood pressure reactions to the maths task in comparison to non-Type D individuals.

Regression analyses using the continuous Type D construct ($NA \times SI$) also yielded a significant association between Type D personality and lower DBP reactivity to the maths task, $b = -.15, t = -2.02, p = .045$, but not SBP reactivity.

3.5 Consideration of hemodynamic profile

In order to examine the hemodynamic profile underlying these blood pressure responses, one sample t-tests were conducted on measures of HP and CD. Sample means for Type D and non-Type D individuals were compared against a hypothesised mean of 0. As expected, CD scores for both Type D individuals [$t(66) = 12.29, p < .001$, for the maths task; $t(66) = 14.83$,

$p < .001$, for the speech task] and non-Type D individuals [$t(108) = 16.63$ $p < .001$, for the maths task; $t(108) = 20.29$, $p < .001$, for the speech task] were significant. Additionally, HP scores for non-Type Ds were significant [$t(108) = 2.01$, $p = .047$, for the maths task; $t(108) = 3.21$ $p = .002$, for the speech task]. The positive t-scores indicate a significant increase from 0, suggesting a vascular response. HP stress task scores for Type Ds were non-significant [$t(66) = 1.06$, $p = .30$, for the maths task; $t(66) = 1.46$, $p = .15$, for the speech task], indicating a mixed hemodynamic response.

Similarly, there was no significant correlations between the continuous Type D interaction term ($NA \times SI$) and CD or HP scores (all $ps \geq .06$).

3.6 Mediation Analyses

There was a significant indirect effect of Type D personality on SBP, $B = -1.10$ $[-2.86, -.06]$, and DBP reactivity to the maths task, $B = -.63$ $[-1.57, -.05]$, through perceived hostility. Type D individuals reported increased perceptions of hostile social relationships, which resulted in lower cardiovascular responses to the maths task. Additionally, there was a significant indirect effect of Type D on CO reactivity via instrumental social support, $B = .05$ $[-.0004, .13]$, whereby Type D individuals reported lower levels of instrumental support, resulting in increased CO reactivity. No significant mediation effects on cardiovascular reactivity to the maths tasks were observed for perceived rejection or emotional social support. See figures 1-3 for significant mediation pathways on cardiovascular reactivity to the maths task.

INSERT FIGURES 1-3 ABOUT HERE

Although there was no significant main effect of Type D on cardiovascular reactivity to the speech task, several indirect effects were observed. There was a significant indirect effect of Type D on SBP, $B = -1.30$ $[-3.03, -.20]$, through perceived hostility. Here, Type D personality was associated with increased perceptions of hostile social relationships, which resulted in

lower cardiovascular responses. Additionally, emotional social support significantly mediated the association between Type D personality and DBP, $B = -.74 [-2.06, -.03]$, and TPR reactivity, $B = -.04 [-.08, -.01]$, whereby lower levels of self-reported emotional support amongst Type D individuals resulted in lower cardiovascular responses to the speech task. No significant mediation effects on cardiovascular reactivity to the speech task were observed for perceived rejection or instrumental social support. See figures 4-6 for significant mediation pathways on cardiovascular reactivity to the speech task.

INSERT FIGURES 4-6 ABOUT HERE

Further, apart from the indirect effect of Type D on TPR reactivity via emotional support, all significant mediation effects were confirmed using the continuous Type D construct (NA x SI). No direct effect of Type D personality on cardiovascular reactivity variables were observed in the aforementioned mediation models, indicating complete mediation.

3.7 Adjusted analyses controlling for negative affect and social inhibition

In multiple regression analyses examining the effects of Type D on social relationship variables, the effects of NA and SI were entered independently in step 1 and the dichotomous Type D typology was then entered into the model at step 2. After controlling for NA and SI, no significant effects of Type D personality on social relationship variables were observed. NA significantly predicted lower levels of emotional support and increased perceptions of hostility and rejection from others. Additionally, SI was associated with lower levels of social support (see table 3). Replication analyses with the continuous Type D interaction term (NA \times SI) entered into the model at step 2 in place of the Type D dichotomy also revealed no significant effects of the Type D construct on social relationship variables after controlling for NA and SI.

INSERT TABLE 3 ABOUT HERE

For adjusted regression analyses on cardiovascular reactivity, confounding variables were entered in step 1, the effects of NA and SI were entered independently in step 2 and the dichotomous Type D typology was then entered into the model at step 3. After controlling for NA and SI, regression models yielded no significant effect of Type D on cardiovascular reactivity to either the speech task or the maths task. Similarly, no significant effects emerged for either NA or SI at step 2 or step 3 of these models. Replication analyses with the continuous Type D interaction term ($NA \times SI$) entered into the model at step 3 in place of the Type D dichotomy also revealed that unadjusted results failed to withstand adjustment for NA and SI.

However, after controlling for NA and SI, the continuous Type D interaction term ($NA \times SI$) significantly predicted increased HR reactivity to the speech task, $b = .58, t = 2.20, p = .03$. Additionally, NA predicted reduced HR reactivity to the speech task in the same step of this model $b = -.39, t = -2.36, p = .02$. No other significant effects emerged.

For adjusted mediation analyses, NA and SI were entered into the mediation model as additional covariates. All mediation effects using both the categorical and continuous Type D constructs were non-significant when controlling for NA and SI.

4. Discussion

The present study had three key aims. Firstly, the current study aimed to examine the association between Type D personality and cardiovascular reactivity to acute psychological stress, as well as the hemodynamic profile underlying these cardiovascular responses.

Secondly, the current study aimed to examine if the association between Type D personality and cardiovascular reactivity to acute stress was mediated via perceptions of social support and of negative social relationships. Finally, the current study also aimed to examine if Type

D personality has predictive utility above and beyond the independent main effects of NA and SI.

Our unadjusted analyses for NA and SI showed that Type D individuals exhibited lower cardiovascular reactivity to acute psychological stress. These findings are consistent with the majority of previous Type D personality-cardiovascular reactivity studies (Howard et al., 2011; Kelly-Hughes et al., 2014; O'Leary et al., 2013), and may indicate blunted cardiovascular reactivity amongst Type D individuals. Additionally, while these unadjusted analyses revealed no significant difference between Type D and non-Type D individuals in response to the speech task, Type D individuals were found to exhibit significantly lower cardiovascular responses to the maths task. Type D individuals have previously been shown to exhibit divergent cardiovascular reactions to different stressors (Bibbey et al., 2015; Gramer et al., 2018; O'Riordan et al., 2019), with Type D individuals primarily exhibiting blunted reactions to stressors of lower social salience (Bibbey et al., 2015; O'Riordan et al., 2019). In fact, the majority of prior research reporting blunted reactions amongst Type D individuals have employed asocial stressors including maths tasks and multitasking stressors (Howard et al., 2011; Kelly-Hughes et al., 2014; O'Leary et al., 2013). Blunted cardiovascular reactivity to stress is suggested to reflect a motivational dysregulation, which engenders withdrawal and disengagement from the acute stressor (Carroll et al., 2017; Phillips, Ginty, & Hughes, 2013). Thus, given the social inhibition facet of Type D personality, it is likely that Type D individuals are more easily able to disengage and withdraw during stressors of lower social salience (e.g. maths tasks) in comparison to stressors of greater social salience (e.g. speech tasks), resulting in blunted physiological reactivity.

Furthermore, our unadjusted mediation analyses found that the association between Type D personality and cardiovascular reactivity was significantly mediated via social support

(instrumental and emotional) and perceptions of negative social relationships (perceived hostility). These findings are consistent with the stress buffering and stress exacerbation/social aggravation hypotheses, which propound that both supportive (stress buffering) and negative (stress exacerbation/social aggravation) social relationships impact health outcomes by influencing stress appraisal and coping (Birmingham & Holt-Lunstad, 2018; Cohen & Wills, 1985; Cranford, 2004; Rook, 1984). While, increased perceptions of hostile social relationships and lower levels of emotional support mediated the association between Type D personality and blunted blood pressure and TPR reactivity, lower levels of instrumental support resulted in increased CO reactivity for Type D individuals. These differential findings may pertain to the cardiovascular parameter of focus. While blunted cardiovascular reactions exhibited by Type D individuals have been primarily noted on cardiovascular parameters of blood pressure (Kelly-Hughes et al., 2014; O'Leary et al., 2013), greater reactions exhibited by Type D individuals have been mostly found on cardiovascular parameters central to sympathetic activation (Gordan, Gwathmey, & Xie, 2015), including HR and CO (O'Riordan et al., 2019; Williams et al., 2009). Nevertheless, bi-directional deviation from appropriate homeostatic adjustment in response to acute psychological stress is indicative of a homeostatic dysregulation and psychosomatic disease vulnerability (Lovallo, 2011).

Consistent with previous findings (Allen et al., 2019a; O'Leary et al., 2013), Type D individuals were found to exhibit a mixed hemodynamic profile in response to the stress tasks. A mixed hemodynamic profile is propounded to reflect a compromised blood pressure regulation, which may be indicative of a homeostatic dysfunction, as the compensatory reciprocal relationship between CO and TPR is not evident (Gregg et al., 2005). Additionally, non-Type D individuals were found to exhibit a vascular hemodynamic profile. While both mixed and vascular hemodynamic profiles are suggested to engender increased risk of

adverse cardiovascular outcomes (Gregg et al., 2005; Hejl, 1957; Palatini & Julius, 2009), it is somewhat unclear which hemodynamic profile is particularly toxic for cardiovascular health. Nevertheless, each hemodynamic profile is suggested to promote adverse cardiovascular health via discrete mechanisms (Gregg et al., 2002), which may further elucidate the process by which atypical cardiovascular reactivity may promote adverse cardiovascular health for Type D individuals.

After controlling for the individual effects of NA and SI, the aforementioned main and indirect mediation effects of Type D personality on cardiovascular reactivity did not remain significant. Type D personality is posited to consist of more than the mere presence of NA and SI and is suggested to be a synergistic effect of both constructs combined (Denollet, 2005; Kupper & Denollet, 2007, 2014). However, more recent evidence has reported null effects of Type D personality after controlling for the individual subcomponents and have suggested that effects observed for Type D are primarily driven by NA (Akram et al., 2018; O'Riordan et al., 2020; Stevenson & Williams, 2014; Williams et al., 2012). Similarly, our results for regression and correlational analyses indicate that of the two subcomponents, NA appeared to be the key subcomponent, driving the observed effects of Type D personality on social relationship and cardiovascular reactivity variables.

However, it is noteworthy that the continuous Type D interaction term was associated with increased HR reactivity to the speech task after controlling for NA and SI. Here, Type D was associated with increased HR reactivity. This is consistent with previous research which has found Type D individuals to exhibit increased reactivity to stressors of greater social salience (Bibbey et al., 2015; O'Riordan et al., 2019). Prior research examining the predictive utility of Type D personality on cardiovascular reactivity above the individual Type D subcomponents have yielded mixed findings. These studies have primarily conducted unadjusted analyses using the dichotomous Type D construct and controlled analyses using

the continuous Type D interaction term (NA \times SI). While some have found Type D to predict aberrant cardiovascular reactivity after controlling for NA and SI (Allen et al., 2019a; Howard et al., 2011; Kelly-Hughes et al., 2014), others have reported null effects (Kupper et al., 2013). Our findings suggest that the predictive utility of Type D personality on cardiovascular reactivity above and beyond the individual effects of NA and SI is limited, and may vary depending on the cardiovascular parameter of focus. While effects on HR reactivity appear to be independent of NA and SI, effects on blood pressure appear to be primarily driven by the NA subcomponent.

One notable strength of the current study pertains to the sample size. In fact, our analyses employed one of the largest sample size examining the association between Type D and cardiovascular reactivity to date. However, the sample consisted of undergraduate students mainly of a relatively young age, with specific sample characteristics. Therefore, it is questionable if the results are generalizable to other cohorts. 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. Additionally, unlike the original TSST, there was no panel present during the stress tasks employed in the current study and participants were not voice or video recorded, limiting the social evaluative nature of the stressor. Given that Type D individuals are suggested to be more physiologically vulnerable to socially salient stressors (Bibbey et al., 2015; O’Riordan et al., 2019), it is likely that more effects may have been observed if the speech task included greater elements of social evaluation.

Type D has been consistently associated with depressive symptoms and anxiety (Al-Qezweny et al., 2016; De Fruyt & Denollet, 2002; Pedersen, van Domburg, Theuns, Jordaens, & Erdman, 2004; Van Den Broek, Smolderen, Pedersen, & Denollet, 2010). In fact, some have

questioned whether the Type D construct is sufficiently distinct from other negative affect variables, and if Type D can predict outcomes independent of depression (Coyne & de Voogd, 2012). Thus, future research would benefit from examining if the effects of Type D on cardiovascular reactivity are independent of anxiety and depression. Additionally, future research should extend the findings of the current study by examining if the provision of supportive and negative interactions during exposure to acute psychological stress moderate the association between Type D personality and cardiovascular reactivity. Perceived social support is posited to encapsulate how an individual appraises his/her situation, rather than a true reflection of how much support he/she receives (Eagle et al., 2019). In fact, the separability of these constructs is well documented (Uchino, 2009; Uchino et al., 2011). Thus, the manipulation of received support for Type D individuals may have yielded differential findings. However, despite the posited separability of received and perceived social support, prior research has found that the provision of social support during acute stress to those who report lower perceived network support may promote more healthful physiological responses (O'Donovan & Hughes, 2008). Thus, the receipt of support for Type D individuals may be beneficial in promoting more healthful cardiovascular responses.

In sum, the current study examined the relationship between Type D personality, social relationships and cardiovascular reactivity to acute psychological stress in a healthy sample, using both the traditional categorical approach and the more recent dimensional method of analysing Type D. Unadjusted analyses indicated that Type D individuals reported lower levels of social support, increased perceptions of negative social relationships and exhibited atypical cardiovascular reactivity to acute psychological stress. Furthermore, the association between Type D personality and cardiovascular reactivity was significantly mediated via increased perceptions of negative social relationships and lower levels of social support. However, apart from a significant association between Type D personality and increased HR

reactivity, all results failed to withstand adjustment for the individual effects of NA and SI in controlled analyses. Overall, these findings suggest that the predictive utility of Type D personality on cardiovascular reactivity above and beyond the individual effects of NA and SI is limited, and may vary depending on the cardiovascular parameter of focus.

Disclosure of interest

The authors report no conflict of interest.

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Table 1. Descriptive statistics of personality, social relationships, demographic and cardiovascular reactivity variables.

Psychometric and Reactivity Variables	Type D Mean (SD)	Non-Type D Mean (SD)	Test of difference	Sample Mean (SD)	Cronbach' α
Type D (Continuous)	242.15 (114.52)	67.03 (50.50)	$t(188) = 14.41, p < .001$	136.16 (118.48)	N/A
Negative affect	16.01 (3.90)	9.03 (4.86)	$t(188) = 10.45, p < .001$	11.85 (5.70)	.86
Social inhibition	14.67 (4.39)	7.43 (4.71)	$t(188) = 10.62, p < .001$	10.31 (5.78)	.85
Emotional Support	31.67 (5.19)	35.24 (4.79)	$t(186) = 4.83, p < .001$	33.82 (5.24)	.91
Instrumental Support	28.53 (6.96)	31.07 (6.13)	$t(185) = 2.62, p = .01$	30.03 (6.60)	.93
Perceived Hostility	17.38 (5.52)	14.22 (4.16)	$t(186) = 4.46, p < .001$	15.53 (5.16)	.90
Perceived Rejection	17.41 (5.52)	14.03 (4.29)	$t(185) = 4.70, p < .001$	15.41 (5.04)	.91
Maths SBP Reactivity	16.18 (11.62)	19.52 (13.38)	$F(1,162) = 3.88, p = .051, \eta_p^2 = .02$	17.86 (12.88)	N/A
Maths DBP Reactivity	10.37 (6.18)	12.79 (8.25)	$F(1,162) = 4.65, p = .03, \eta_p^2 = .03$	11.68 (7.58)	N/A
Maths HR Reactivity	3.54 (6.60)	4.11 (6.20)	$F(1,162) = .41, p = .52, \eta_p^2 = .003$	3.76 (6.37)	N/A
Maths CO Reactivity	.38 (.69)	.31 (.90)	$F(1,162) = .06, p = .82, \eta_p^2 = .000$.32 (.81)	N/A
Maths TPR Reactivity	.10 (.16)	.14 (.26)	$F(1,162) = .76, p = .39, \eta_p^2 = 0.01$.13 (.23)	N/A
Speech SBP Reactivity	18.78 (11.50)	20.64 (12.45)	$F(1,162) = 2.37, p = .13, \eta_p^2 = 0.01$	19.64 (12.15)	N/A
Speech DBP Reactivity	12.53(6.68)	13.67 (7.43)	$F(1,162) = 2.04, p = .16, \eta_p^2 = 0.01$	13.15 (7.20)	N/A
Speech HR Reactivity	6.21 (6.36)	5.69 (7.06)	$F(1,162) = .55, p = .46, \eta_p^2 = .003$	5.84 (6.85)	N/A
Speech CO Reactivity	.43 (.69)	.29 (.91)	$F(1,162) = .65, p = .42, \eta_p^2 = .004$.32 (.82)	N/A
Speech TPR Reactivity	.12 (.24)	.17 (.24)	$F(1,162) = 1.76, p = .19, \eta_p^2 = .01$.15 (.24)	N/A
Age	20.91 (4.50)	21.07 (4.73)	$t(188) = .24, p = .81$	20.95 (4.58)	N/A
Sex (% female)	66.7%	72.2%	$\chi(1) = .66, p = .42$	70.8%	N/A

Table 2. Correlations between NA x SI, social relationship variables and cardiovascular reactivity variables.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Type D (NA x SI)	-	.80**	.84**	-.47**	-.24**	.38**	.45**	-.13	-.15*	-.01	-.001	-.12	-.07	-.08	.05	.04	-.12
2. Negative affect	-	-	.46**	-.45**	-.15*	.47**	.49**	-.19*	-.18*	-.09	-.06	-.09	-.15	-.13	-.05	-.02	-.1
3. Social Inhibition	-	-	-	-.42**	-.23**	.24**	.31**	-.05	-.09	.02	.03	-.12	.02	.00	.05	.02	-.07
4. Emotional Support	-	-	-	-	.40**	-.41**	-.57**	.09	.12	.03	-.01	.05	.06	.08	-.01	-.03	.17*
5. Instrumental Support	-	-	-	-	-	-.07	-.27**	-.004	.04	-.04	-.17*	.13	-.15	-.14	-.12	-.16*	.07
6. Perceived Hostility	-	-	-	-	-	-	.48**	-.16*	-.16*	-.13	-.03	-.07	-.18*	-.16*	-.13	-.04	-.08
7. Perceived Rejection	-	-	-	-	-	-	-	-.07	-.07	-.07	-.01	-.02	-.02	-.003	-.01	-.02	-.04
8. (M) SBP Reactivity	-	-	-	-	-	-	-	-	.89**	.41**	.25**	.28**	.81**	.70**	.33**	.20**	.21**
9. (M) DBP Reactivity	-	-	-	-	-	-	-	-	-	.47**	-.05	.57**	.73**	.78**	.39**	.02	.41**
10. (M) HR Reactivity	-	-	-	-	-	-	-	-	-	-	.38**	-.04	.39**	.43**	.77**	.32**	.09
11. (M) CO Reactivity	-	-	-	-	-	-	-	-	-	-	-	-.67**	.22**	.01	.33**	.79**	-.49**
12. (M) TPR Reactivity	-	-	-	-	-	-	-	-	-	-	-	-	.19*	.39**	-.03	-.50**	.62**
13. (S) SBP Reactivity	-	-	-	-	-	-	-	-	-	-	-	-	-	.90**	.37**	.22**	.30**
14. (S) DBP Reactivity	-	-	-	-	-	-	-	-	-	-	-	-	-	-	.41**	-.02	.54**
15. (S) HR Reactivity	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	.52**	-.07
16. (S) CO Reactivity	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-.60**
17. (S) TPR Reactivity	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

** $p < 0.01$ level, * $p < 0.05$ level, S = Speech Task, M = Maths Task

Table 3. Regression analyses: Type D personality, Negative affect, Social inhibition and Social relationship variables

Variable	Emotional Support			Instrumental Support			Perceived Hostility			Perceived Rejection		
	β	t	p	β	T	p	β	t	p	β	t	p
Step 1												
NA	-.33	-4.72	< .001	-.08	-1.03	.31	.42	5.69	< .001	.43	6.03	< .001
SI	-.27	-3.78	< .001	-.19	-2.31	.02	.06	.78	.44	.12	1.63	.11
Step 2: Dichotomous Type D typology												
NA	-.36	-4.48	< .001	-.06	-.65	.52	.40	4.79	< .001	.44	5.38	< .001
SI	-.29	-3.64	< .001	-.16	-1.78	.08	.04	.45	.65	.12	1.49	.14
Type D	.06	.65	.52	-.06	-.55	.59	.05	.49	.62	-.01	-.12	.90
Step 2: Continuous Type D interaction term (NA \times SI)												
NA	-.44	-3.24	.001	.05	.32	.75	.43	3.04	.003	.39	2.84	.01
SI	-.38	-2.60	.01	-.04	-.23	.82	.07	.45	.65	.07	.47	.64
(NA \times SI)	.19	.90	.37	-.25	-1.01	.31	-.02	-.10	.92	.08	.33	.74

Figure 1. Unadjusted mediation path diagram: Indirect effects of Type D personality (categorical) on SBP reactivity to the maths task stress via the social relationship mediation variables. Significant effects are highlighted in bold text.

Figure 2. Unadjusted mediation path diagram: Indirect effects of Type D personality (categorical) on DBP reactivity to the maths task stress via the social relationship mediation variables. Significant effects are highlighted in bold text.

Figure 3. Unadjusted mediation path diagram: Indirect effects of Type D personality (categorical) on CO reactivity to the maths task stress via the social relationship mediation variables. Significant effects are highlighted in bold text.

Figure 4. Unadjusted mediation path diagram: Indirect effects of Type D personality (categorical) on SBP reactivity to the speech task stress via the social relationship mediation variables. Significant effects are highlighted in bold text.

Figure 5. Unadjusted mediation path diagram: Indirect effects of Type D personality (categorical) on DBP reactivity to the speech task stress via the social relationship mediation variables. Significant effects are highlighted in bold text.

Figure 6. Unadjusted mediation path diagram: Indirect effects of Type D personality (categorical) on TPR reactivity to the speech task stress via the social relationship mediation variables. Significant effects are highlighted in bold text.