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TYPE D PERSONALITY AND HEMODYNAMIC REACTIVITY TO LABORATORY STRESS IN WOMEN

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Abstract

The Type D personality (identified by high levels of both negative affectivity and social inhibition) has been associated with negative health consequences in cardiac patients. However, few studies have explored whether the Type D personality is associated with particular patterns of cardiovascular responses to stress. In the present study, cardiovascular reactivity to psychological stress (CVR) was examined as a possible mediating mechanism by which Type D personality may affect cardiovascular health, with specific focus on hemodynamic profile. Eighty-nine female university students completed a mental arithmetic stressor while undergoing hemodynamic monitoring. Blood pressure, heart rate, cardiac output, and total peripheral resistance in response to the stressor were examined. Type D personality was assessed using the 16-item Type D scale. Results indicated that there were no between-group differences in magnitude of blood pressure increase, with both Type D and non-Type D individuals demonstrating myocardial response profiles. However, Type D individuals were less "myocardial" than non-Type D individuals. This indicates that a weak myocardial response to an active stressor in Type D individuals may be indicative of hemodynamic maladaptation to stress, implicating CVR as a possible mechanism involved in Type D-cardiovascular health associations.

Keywords: Type D personality; cardiovascular reactivity; hemodynamic profile; myocardial response

1.0. Introduction

Characterized by high scores for both negative affectivity (NA; the propensity to experience negative emotions) and social inhibition (SI; the tendency to inhibit emotions in social situations), the so-called "distressed" or Type D personality has been shown to be statistically associated with health outcomes in coronary patients. Studies have linked Type D personality with cardiac and all-cause mortality (e.g., Denollet et al., 1996; Denollet et al., 2000) and increased cardiac morbidity (Denollet and Brutsaert, 1998). Furthermore, the Type D personality has been shown to be a predictor of patient adjustment to disease interventions (e.g., Pedersen et al., 2007; Pedersen et al., 2007). While Type D cannot be said to encompass all psychological risk factors involved in coronary heart disease (CHD; Denollet and Van Heck, 2001), its association with negative clinical outcomes appears to be on par with established biomedical risk factors such as smoking, older age, and poor exercise tolerance (e.g., Denollet et al., 1996; Denollet and Brutsaert, 1998; Denollet et al., 2000).

The precise mechanism by which Type D may influence health-related outcomes is unclear. Although indirect mechanisms have been proposed (e.g., Thomas et al., 2006; Brostrom et al., 2007; Schiffer et al., 2007; Williams et al., 2008; Hausteiner et al., 2010), it is possible that psychophysiological pathways involving Type D's affect dimensions might be implicated in Type D-health relationships. The separate components of the Type D personality have shown associations with biological indices of health; NA has been linked to higher levels of cortisol during the day (Van Eck et al., 1996; Miller et al., 1999) and cortisol reactivity to laboratory tasks (Phillips et al., 2005; Sher, 2005), while behavioral inhibition has been associated with a larger cortisol awakening response and larger response to stress in young children (Kagan et al., 1987) and with mechanisms of behavioral inhibition in a small sample of healthy female adults (Tops and Boksem, in press). In terms of an NA-SI synergy (i.e., Type D), Whitehead, Perkins-Porras, Strike, Magid, and Steptoe (2007) found that the

interaction term of NA and SI (computed by multiplying NA scores with SI scores, thereby representing Type D as a continuous measure) was positively related to the magnitude of cortisol awakening response in a sample of 72 patients with acute coronary syndrome, adjusting for age, gender, and body mass index (BMI). In addition, there have been independent reports of a link between Type D personality and elevated levels of cytokines implicated in CHD progression (e.g., Denollet et al., 2003; Conraads et al., 2006). Together, these studies point to the existence of a direct psychosomatic pathway implicated in Type D-health associations.

Given the nature of the links between cardiac disease outcomes and the Type D personality, the cardiovascular system appears to be a likely source of a direct psychosomatic pathway. Exaggerated cardiovascular reactivity to psychological stress (CVR) as measured in healthy adults is believed to lead to an increased risk of eventual cardiac disease (Kamarck and Lovallo, 2003) through a number of physiological mechanisms (Lovallo, 2005). However, as yet, very few studies have explored whether Type D personality is associated with particular patterns of cardiovascular responses to stress. In one laboratory study involving 173 college students, Habra et al. (2003) found both SI and NA subscores to be separately associated with differences in CVR among males, but found no effect for females or for the unified Type D personality itself (i.e., SI and NA scores in combination). However, a socially salient laboratory stressor was used and this may explain the observed tendency for high SI participants to show increased CVR; it may be the case that the pattern of responses exhibited by Type D individuals differs depending on the social context of the task and it would be interesting to note if Type D individuals demonstrate altered physiological response profiles to an *asocial* laboratory stressor.

In a subsequent study, Williams, O'Carroll, and O'Connor (2009) exposed 90 college students to a cognitive stressor in the laboratory (again, with a social dimension; participants

returned solutions verbally to the experimenter) and found that Type D personality was associated with exaggerated cardiac output (CO) reactivity. Like Habra et al. (2003), however, effects were confined to male participants, with no associations between Type D and systolic blood pressure (SBP), diastolic blood pressure (DBP), or heart rate (HR) reactivity in either male or female participants.

A noteworthy aspect of the previously published findings is that Type D effects have been observed only in relation to particular underlying hemodynamic determinants of blood pressure. It is known that changes in blood pressure reflect underlying dynamics including changes in CO, total peripheral resistance (TPR), or both (Turner, 1994). There is a reciprocal relationship between CO and TPR such that an increase in one parameter tends to be accompanied by a proportional decrease in the other (e.g., Guyton, 1987). As such, little or no change in blood pressure level can sometimes disguise more vigorous changes in underlying physiological determinants. The dynamic relationship between CO and TPR, or hemodynamic profile, may be further characterized as myocardial (changes in CO exceed proportional changes in TPR) or vascular (changes in TPR exceed proportional changes in CO). There is evidence that certain characteristics of hemodynamic profile indicate potentially harmful disruption of the inherent homeostasis between CO and TPR (Obrist, 1981; Eliot et al., 1982; Kasprowicz et al., 1990). Thus, examination of the physiological determinants of blood pressure, and in particular, hemodynamic profile (i.e., relative change in CO and TPR), may help to clarify whether Type D personality influences physiological reactivity to stress, which in turn could have long-term consequences for physical health.

Examination of hemodynamic profile can be quantified using trigonometric rotation, as proposed by Gregg et al. (2002). Composite scores are computed, allowing representation of hemodynamic profile (HP) and compensation deficit (CD) as continuous variables. This offers possible improvements on earlier methods for characterizing hemodynamic profile,

which relied on categorization into CO-dominant, TPR-dominant, and mixed-response groups (e.g., Eliot et al., 1982; Girdler et al., 1990; Sherwood et al., 1990). Such categorization not only involves the use of arbitrary criteria for determining group membership, but also involves loss of information due to the reduction of continuous variables to a few categories. By computing both HP and CD, the individual can be described in terms of their response profile (HP) and the extent of reactivity (CD).

The present study sought to examine the association between Type D personality and laboratory measures of CVR, focusing on blood pressure, HR, and hemodynamic profile. In a sample of female college students, a standardized asocial laboratory stressor was employed to assess CVR. Although women tend to demonstrate lower resting blood pressure and higher SBP response to stress than men (Turner, 1994), males were not recruited for this study mainly due to the highly imbalanced gender distribution within the sampling population (undergraduate psychology students), which impeded our ability to include sufficient numbers of biometrically comparable males. However, as previous findings examining Type D personality and CVR to laboratory in stress in healthy individuals reported effects confined to males, the inclusion of a female-only sample allowed investigation of the association between Type D personality and CVR in females. An asocial cognitive task was chosen in order to examine if Type D personality was associated with an altered cardiovascular response profile when there was no social dimension to the laboratory task. It was predicted that Type D personality would be associated with a maladaptive cardiovascular response, in particular, influencing hemodynamic profile in response to the mental arithmetic stressor.

2.0. Materials and Methods

2.1. Participants

Participants were 89 female college students (age 18 to 29 years; M = 19.70, SD = 1.87

years) with normal body mass index (M = 22.67, SD = 3.33). All participants were normotensive (resting blood pressure <140/90 mmHg), physically healthy, and reported no history of heart disease. Students were recruited through class announcements and received course credit for participation. Participation was voluntary and participants were free to withdraw at any time. All participants signed a consent form prior to participation.

2.2. Materials and Apparatus

Participants first underwent psychometric testing to establish Type D status using the 16-item Type D scale (DS16; Denollet, 1998) immediately prior to the laboratory session. The DS16 consists of two 8-item scales measuring NA and SI, each producing subscores ranging from 0 to 32. Conventionally, a median split on both scales is used to identify Type D (e.g., Denollet, 1998; Denollet et al., 2000; Pedersen and Middel, 2001), and good internal reliability has been reported (Denollet, 1998). In the present sample, Cronbach's α for the NA and SI scales was .87 and .75, respectively, indicating acceptable internal consistency. Participants scoring above the median on both subscales (i.e., ≥10 on the NA scale and ≥12 on the SI scale) were identified as Type D. This resulted in 33 individuals identified as Type D and 56 as non-Type D. As the convention of classifying Type D by cross-tabulated double-dichotomies of subscores is reliant on median splits, there is a particular risk of misclassification error with regard to borderline cases (Veiel, 1988). Although the use of median splits to create dichotomies in psychometrics is generally advised against (Veiel, 1988), the use of a cross-tabulated double-dichotomy to create quadrants remains common in personality and social psychology research (cf, Berry, 1970; Karasek, 1979; Dworkin, 1990; Jamner et al., 1991; Ries and Miller, 1992; Everson et al., 1997; Derakshan et al., 2007). Given the possibility that Type D may represent a continuum rather than a taxon (Ferguson et al., 2009), it is important that researchers consider how they might compute continuous Type

D variables (such as might be achieved by combining NA and SI scores arithmetically) in order to assess whether such measures offer statistically stronger tests of Type D-related hypotheses. As such, a continuous score representing Type D personality tendencies was computed as the product of raw NA and SI subscores (i.e., NA × SI; cf Whitehead et al., 2007), and all analyses reported below were duplicated using this score as a continuous independent variable in place of categorical Type D classifications.

Participants also completed the state form of the state-trait anxiety inventory (STAI; Spielberger et al., 1983) immediately prior to the laboratory session. The STAI has been used extensively in research and clinical practice. The state anxiety scale consists of 20 questions which evaluate how the person feels *right now*. Responses range on a four-point Likert scale from *not at all* to *very much so*. Alpha coefficients of over .90 have been reported for the state anxiety scale and validity has been demonstrated (Spielberger et al., 1983). In the present sample, Cronbach's α for the state anxiety scale was .90.

Beat-to-beat blood pressure and HR were measured non-invasively using a Finometer hemodynamic cardiovascular monitor (Finapres Medical Systems BV, BT Arnhem, The Netherlands). The Finometer is the successor to the TNO Finapres-model-5 and of the Ohmeda Finapres 2300e which have been used in previous research (e.g., Beckham et al., 2002; Gregg et al., 2002; van Rooyen et al., 2004; Philippsen et al., 2007). The Finometer is based on the volume-clamp method first developed by Peňaz (1973). An appropriate-sized finger cuff is attached to the participant's middle finger which inflates to keep the arterial walls at a set diameter. In-built into this finger cuff is an infrared photo-plethysmograph which detects changes in the diameter of the arterial wall. When the volume clamp is active at the proper unloaded diameter, intra-arterial pressure equals that of the finger cuff pressure. Measures of arterial pressure CO are provided based on the previously validated Modelflow modeling method (Wesseling et al., 1993; Wesseling et al., 1995). The Finometer has been

shown to accurately assess absolute blood pressure in young participants (Schutte et al., 2003) and in cardiac patients (Guelen et al., 2003). According to these studies, the validation criteria of the Association for the Advancement of Medical Instrumentation and the revised protocol of the British Hypertension Society are satisfied by the Finometer.

To minimize the impact of variations in environmental cues on reactivity (Christenfeld et al., 1998), all testing took place in the same laboratory. Participants who were smokers (n = 15) were instructed to abstain from smoking for 1 hour before arriving at the laboratory. This timeframe allowed for the subsidence of acute cardiovascular effects of smoking prior to testing (Silvestrini et al., 1996; Monfrecola et al., 1998; Terborg et al., 2002; Domino et al., 2004), while avoiding the cardiovascular effects of prolonged smoking abstinence (Tsuda et al., 1996; Primatesta et al., 2001). Although present in the room throughout the procedure, the researcher was separated from the participant by an opaque screen.

2.3. Procedure

All procedures were approved by the Institutional Research Ethics Committee. Participants were greeted by the (female) researcher and seated at a computer desk in a comfortable chair with an arm support. A personal computer was situated on the desk. The Finometer cuff was attached to the participant's middle finger of their non-dominant hand. Participants were given 30 minutes to acclimatize to the laboratory situation during which the psychometric measures (including the DS16 and the state form of the STAI) were completed. Reading material was also supplied in order to facilitate relaxation and the establishment of cardiovascular baselines, by offsetting the risk of rumination-related arousal (Jennings et al., 1992). Following this acclimatization period, participants were given verbal instructions about the procedure. Participants were told that they would be required to solve subtraction problems appearing on-screen, inputting their answers using the computer keypad. The level

of difficulty varied according to the answers given; the problems became more difficult if correct answers were returned, or became easier if incorrect answers were returned. This ensured the task controlled for individual mathematical ability, employing the principle of standardized flexibility previously recommended for CVR assessment (Turner et al., 1986; Turner, 1994; Hughes, 2001). Participants had 15 seconds to return a solution, otherwise it was coded as a "timeout". After the initial 30-minute acclimatization period, participants were instructed to relax quietly for 10 minutes. Resting measures were obtained during this time period. Following this baseline period, participants were asked to perform the five-minute mental arithmetic task. Cardiovascular parameters were measured non-invasively using the Finometer throughout the procedure. Participants completed the state form of the STAI after completing the laboratory task, as well as some short Likert scales where the participant rated how difficult and enjoyable they found the task. Participants were debriefed following completion of the laboratory session.

3.0. Results

3.1. Overview of Analyses

To identify if there were any Type D differences in task engagement, a series of independent *t*-tests were conducted to identify differences in the number of problems attempted or in the number timeouts (where the participant did not return a solution within the allotted 15 seconds). In addition, independent *t*-tests were used to identify if there were group differences in state anxiety or how enjoyable and difficult the task was rated. Pearson's product moment correlation coefficient analyses were used to confirm these group-based analyses (where the categorical representation of Type D was used), using a continuous representation of the Type D construct (i.e., NA × SI).

Mean levels of SBP, DBP, HR, CO, and TPR were computed for both phases of the

experiment, namely, baseline and task. Excellent internal reliability consistency for each measure was observed (Cronbach's $\alpha > .98$ for each SBP, DBP, HR, and CO mean; $\alpha > .89$ for each TPR mean).

In order to examine the association between Type D classification and CVR, a mixed factorial 2 × 2 ANOVA was conducted for each cardiovascular parameter (SBP, DBP, HR, CO, and TPR). The within-subjects factor (time) comprised two levels; baseline and task. The between-subjects factor (personality) had two levels: Type D and non-Type D. To confirm findings based on Type D classification with effects that might be observed for a continuous measure of Type D, a series of stepwise multiple regressions for each cardiovascular parameter was conducted, with the two DS16 subscores and their product (i.e., NA, SI, and NA × SI) entered as predictors.

For measures of TPR, a Kolmogorov-Smirnov test indicated that there had been a violation of the assumption of normal distribution. After examination of the outlying scores ($n = 9$) it was decided to remove these participants from the data set when examining TPR (i.e., four participants were removed from the non-Type D group and five participants were removed from the Type D group). This left a sample size of $N = 80$ when examining TPR only, resulting in a reduction of degrees of freedom for these analyses. As Kolmogorov-Smirnov tests confirmed that all other cardiovascular parameters were normally distributed for both phases of the experiment, the full sample of $N = 89$ was used for those variables.

Examination of the hemodynamic changes underlying observed blood pressure responses was supplemented by a quantification of hemodynamic profile as proposed by Gregg et al. (2002). That is, trigonometric rotation was used to compute composite scores representing HP and CD; the former indicating the degree to which blood pressure changes are mainly attributable to increases in either CO or TPR, and the latter indicating the magnitude of homeostatic compensation between CO and TPR. If a blood pressure response

is mainly attributable to increased CO it is considered myocardial, and if it is mainly attributable to increased TPR it is considered vascular. In addition, the computation of a score for HP allows reciprocal changes in CO and TPR to be measured and tested for statistical significance.

Effect sizes are presented as partial η^2 for ANOVA effects, with values of .04, .25, and .64, being taken as representing small, medium, and large effect sizes, respectively (Cohen, 1988; Cohen, 1992). Partial η^2, rather than simple η^2, is recommended for ANOVA designs with multiple independent variables, as simple η^2 contains systematic variance attributable to other effects and interactions (Tabachnick and Fidell, 1989). For independent *t*-tests and correlation analyses, effect sizes are presented as *r*, with values of .10, .25, and .37 being taken as representing small, medium, and large effect sizes respectively (Cohen, 1988; Cohen 1992).

3.2 Task engagement

Independent *t*-tests indicated that there were no Type D differences in the number of problems attempted or number of time-outs (all *p*s > .30). This was confirmed by a series of correlations which revealed no associations between the NA × SI interaction term and number of problems attempted or number of time-outs.

Likewise, independent *t*-tests and Pearson's r revealed no Type D differences or associations in ratings of how enjoyable or difficult the task was (all *p*s > .10). However, Type D individuals had higher state anxiety scores ($M = 41.72$, $SD = 10.57$) than non-Type D individuals ($M = 36.79$, $SD = 8.73$; $t[87] = 2.38$, $p = .019$). This was confirmed by Person's r which revealed a strong positive correlation between the NA × SI interaction term and state anxiety scores ($r = .38$, $p < .001$).

3.3. Confirmation of reactivity

Mean levels of all cardiovascular parameters during baseline and the stressor task are shown in Table 1. A main effect for time was observed for SBP, $F(1,87) = 117.55$, $p < .001$, partial $\eta^2 = .58$, DBP, $F(1,87) = 146.03$, $p < .001$, partial $\eta^2 = .63$, HR, $F(1,87) = 79.97$, $p < .001$, partial $\eta^2 = .47$, and for CO, $F(1,87) = 93.73$, $p < .001$, partial $\eta^2 = .52$. There was no main effect for time for TPR, $F(1,78) = 1.37$, $p = .253$. The significant main effects corresponded with increases in all parameters from baseline to task, indicating that reactivity was successfully elicited.

3.4. Type D personality and reactivity to stress

There was no significant main effect for personality on either SBP, $F(1,87) = .99$, $p = .323$, or DBP, $F(1,87) = .84$, $p = .363$. Similarly, the time × personality interaction was non-significant for both variables, $F(1,87) = .93$, $p = .337$ for SBP, $F(1,87) = .53$, $p = .47$, for DBP. These ANOVA results suggest that Type D personality had no influence on SBP and DBP level or reactivity.

A significant main effect for personality on HR, $F(1,87) = 4.86$, $p = .03$, partial $\eta^2 = .053$, indicated that Type D individuals showed relatively lower HR levels across the experiment (see Table 1). However, this main effect was qualified by a significant time × personality interaction, $F(1,87) = 4.44$, $p = .038$, partial $\eta^2 = .05$. While both Type D and non-Type D individuals showed HR reactivity, the extent of reactivity was greater in non-Type D individuals (see Figure 1).

The ANOVA for CO revealed a significant main effect for personality, $F(1,87) = 6.21$, $p = .015$, partial $\eta^2 = .07$, with Type D participants exhibiting lower CO levels across time than non-Type D participants. This main effect was qualified by a significant time × personality interaction, $F(1,87) = 6.28$, $p = .014$, partial $\eta^2 = .07$. CO reactivity from baseline

to task was more pronounced among non-Type D individuals. There was also a significant time × personality interaction for TPR, $F(1,78) = 4.02$, $p = .048$, $\eta^2 = .05$, again with greater reactivity (in terms of a decrease in TPR from baseline to task levels) seen in non-Type D participants.

Considering CO and TPR together, it can be seen that although Type D and non-Type D participants exhibited similar blood pressure reactivity, CO and TPR reactivity was distinctly different across the two groups. Non-Type D participants appeared to show greater changes in these hemodynamic variables when compared with Type D participants. For clarity, reactivity levels for CO and TPR are represented by change scores in Figure 2. Simple comparisons of means confirms that Type D individuals showed less reactivity on both CO, $t(87) = 2.51$, $p = .014$, and TPR, $t(78) = 2.01$, $p = .048$, with Type D individuals failing to show the expected compensatory TPR response to the increase in CO.

3.5. Consideration of Hemodynamic Profile

As proposed by Gregg et al. (2002), values for HP and CD were computed for each group. Values are returned on a scale of quasi-standard scores, with a hypothesized mean of 0 and SD close to 1. Type D participants were found to have a mean HP of -.03 (*SD* = .07) and a mean CD of .03 (*SD* = .03), with non-Type D participants showing means of -.06 (*SD* = .08) and .03 (*SD* = .05), respectively.

One-sample *t*-tests showed that both Type D and non-Type D individuals showed significant CD change [$t(32) = 5.81$, $p < .001$, for Type D individuals; $t(55) = 4.08$, $p < .001$, for non-Type D individuals] and HP change [$t(33) = -2.12$, $p = .041$, for Type D individuals; $t(55) = -5.74$, $p < .001$ for non-Type D individuals]. The negative *t*-value on HP scores, signaling a significant decrease from 0, is indicative of a myocardial response to the mental arithmetic stressor, in both Type D and non-Type D individuals. To examine if there were

between-group differences in HP and CD between Type D and non-Type D individuals, independent samples *t*-tests were conducted. As can be seen in Figure 3, while there were no differences in CD between Type D and non-Type D individuals, $t(87) = -.06$, $p > .05$, there were differences in HP, $t(88) = -2.04$, $p = .03$. That is, while Type D and non-Type D individuals demonstrated similar increases in blood pressure to mental arithmetic, the response for non-Type D individuals was significantly "more myocardial" than for Type D individuals.

3.6. Continuous Measure of Type D personality and CVR

For each cardiovascular parameter, a stepwise multiple regression was conducted with the two DS16 subscores (NA and SI) and their product (NA × SI) entered as predictors, and CVR (the change in a given parameter from baseline to task) entered as the criterion variable. The NA × SI product score represented a continuous composite measure of Type D personality. Individual subscores did not emerge as significant predictors of reactivity for any cardiovascular parameter. However, the NA × SI product score emerged as a significant predictor of HR reactivity, $F(1,87) = 4.56$, $p = .036$, $\Delta R^2 = .04$, CO reactivity, $F(1, 87) = 7.27$, $p = .008$, $\Delta R^2 = .07$, and TPR reactivity, $F(1,78) = 5.57$, $p = .02$, $\Delta R^2 = .06$. The direction of each β coefficient is consistent with the results found for categorical Type D categorization; namely, Type D personality was inversely associated with HR and CO, and was positively associated with TPR.

4.0. Discussion

The present study confirms that Type D personality affects cardiovascular response to psychological stress, highlighting the potential role of cardio-physiological reactivity as a mechanism in Type D-health associations. In addition, it was shown that Type D personality

is associated with stress responding at the level of hemodynamic determinants of blood pressure, rather than being directly associated with a maladaptive pattern of blood pressure response. Moreover, the results were confirmed when examining the Type D personality as a continuous, rather than a categorical variable, in that the continuous score underlying Type D classification was found to reveal similar effects.

Visual scrutiny of Figure 2 suggests pertinent between-group differences in hemodynamic patterning. It can be noted that for non-Type D individuals, large increases in CO were accompanied by large decreases in TPR. This pattern of change in both variables appears consistent with an adaptive homeostatic response to stress. As CO increases in response to stress, the pressure placed on the vasculature is offset by an accompanying decrease in TPR, thereby limiting the risk of shear-stress damage in the cardiovascular system as a whole (cf., Obrist, 1981; Eliot et al., 1982; Kasprowicz et al., 1990). However, in the Type D group, no such homeostatic relationship can be seen in that increases in CO are accompanied by small *increases* in TPR, which may indicate a maladaptive "mixed" response to stress (Kasprowicz et al., 1990).

The fact that Type D effects were observed only at the level of physiological determinants of blood pressure rather than actual blood pressure level is consistent with the findings of previous research. Habra et al. (2003) were unable to isolate full Type D effects for measures of blood pressure reactivity, although the fact that they identified individual relationships for NA and SI appeared to implicate Type D as having some role in the determination of CVR. As in the present study, Williams et al. (2009) found Type D classification to be associated with CO reactivity, although their effects were confined to male participants and were in the opposite direction to those observed for females in the present study. Williams et al. employed a different stressor task than that used in the present study, which might help explain the differences in CO responses between the two studies.

The task employed involved serial subtraction where participants were required to maintain digits in short-term memory simultaneous to conducting mental arithmetic. As answers were returned verbally to the experimenter, the task employed by Williams et al. had some socially salient features not present in the present study, where participants performed an on-screen mental arithmetic task that made fewer demands on memory. Given that hemodynamic variables are particularly sensitive to task type, the fact that Type D personality predicted CO reactivity in a different way across the two studies might be related to qualitative differences in the two stressors. Mental arithmetic typically elicits a myocardial response profile, where changes in blood pressure are due to increased beta-receptor activation (Kasprowicz et al., 1990; Sherwood and Turner, 1993; Lawler et al., 1995). While the response profile for serial subtraction is not as established, previous work by Girdler, Turner, Sherwood, and Light (1990) showed that serial subtraction and mental arithmetic produced markedly different hemodynamic response profiles in males and females. Future Type D research might consider different task types (both vascular and myocardial) as well as social context (social versus asocial) when examining the hemodynamic response profiles in Type D and non-Type D individuals.

This study extended previous investigation of CVR in Type D individuals by examining specific response profiles. Individuals who typically react with increases in TPR that are not offset by decreases in CO may be at risk due to atherosclerotic changes arising from repeated or prolonged periods of increased vascular resistance (Gregg et al., 2002). In the present study, although both Type D and non-Type D individuals showed a myocardial response to the mental arithmetic stressor, Type D individuals showed a weaker myocardial profile. This poses an interesting notion that Type D individuals may exhibit an adaptive profile of response to certain stressors, perhaps offering a degree of emotional protection in the form of reduced myocardial reactivity. However, since a myocardial response is believed to be less

atherosclerotic than a vascular response, the weaker response profile exhibited by Type D individuals to a task known to invoke beta-receptor activation, could leave this sample more vulnerable to a vascular, and hence more atherosclerotic, response in situations that involve "mixed" (increases in both CO and TPR) or vascular stressors. This is intriguing and suggests the need for further studies of the response profile associated with differences in Type D personality, involving a range of stressors (myocardial, vascular, and mixed). Extending the research within this domain will help identify the nature (if any) of disease risk posed by the Type D personality.

There is a growing body of evidence to suggest that blunted, rather than exaggerated, physiological reactivity is associated with a range of health outcomes such as depression and obesity (Carroll et al., 2007; York et al., 2007; Carroll et al., 2008). Studies have shown that those who respond best to a vaccination challenge show greater cardiovascular and cortisol reactions to stress (e.g., Phillips et al., 2009), while young adolescents who spent a greater proportion of their lives in poverty show muted CVR to acute stress (Evans and Kim, 2007). These studies suggest that muted CVR to acute stress may be evidence of stress dysregulation, leading to (or a result of) poor physical health. In this context, the weak myocardial profile exhibited by persons who score highly on the Type D construct when responding to laboratory stress, paired with the muted HR reactivity, may be further evidence of a health-compromising physiological reaction to acute stress.

This study is the first to show that the Type D personality affects cardiovascular responses to stress in healthy female participants. Both previous CVR studies of Type D (Habra et al., 2003; Williams et al., 2009) failed to demonstrate effects for female participants, despite using mixed gender samples. Although the present study used a single-gender sample, thereby reducing its ecological validity, it succeeded in demonstrating that possible maladaptive effects of the Type D personality are not confined to males as could

previously have been surmised. This is important, as due to gender differences in base rates of cardiac disease, male participants far outweigh female participants in the clinical samples on which much Type D research has been conducted (e.g., Denollet et al., 1995; Denollet et al., 1996; Pedersen and Middel, 2001). If it were the case that only male Type D individuals showed a maladaptive cardiovascular response to stress, the context of the epidemiological findings would need to be re-examined. However, the present study demonstrated effects for women. In addition, Type D personality was associated with both CO and TPR reactivity; variables not included in Habra et al.'s study. Nevertheless, there remains the possibility that male gender exacerbates the negative effect of the Type D personality and further research is needed to clarify any possible gender differences in physiological reactivity associated with the Type D construct.

In eliciting CVR, the present study employed a stressor that was essentially asocial and non-emotional. Given the nature of the Type D personality, examination of response patterns arising from stressors with no affective dimension may be particularly warranted. The theory implicit in the Type D construct invokes both emotional and social cognition as psychosomatic processes, in which persons are put at risk by a combination of negative emotionality and a tendency to inhibit emotions in social settings. While the present study sought to isolate the impact of such a disposition on a participant's generalized reactivity to stress, the degree to which Type D is a socially embedded personality construct might best be examined by comparing reactivity to stressors of differing emotional and social dimensions. Nonetheless, the present study provides evidence implicating maladaptive cardiovascular responses to psychological stress as a possible physiological pathway linking Type D personality to health outcomes. In this way, a direct role for Type D personality in the etiology of cardiovascular ill-health is suggested.

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