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### Type D personality and cardiac output in response to stress

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## **Type D personality and cardiac output in response to stress**

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Type D personality is predictive of adverse clinical outcome and psychological distress in cardiac patients. However, the mechanisms by which Type D affects health are largely unknown. This study (1) investigated the relationship between Type D and cardiovascular reactivity to experimentally induced stress and (2) tested the influence of Type D on subjective feelings of stress. Eighty four healthy young adults (50% males, mean (SD) age 22 (6.84) years), completed measures of Type D personality, stress arousal and a stress-inducing procedure involving a taxing mental arithmetic task. Cardiovascular measures were recorded throughout the experiment. Mixed measures ANOVA showed a significant main effect of Type D and a significant group by time effect of Type D on cardiac output in male participants. Type D males exhibit significantly higher cardiac output during the stressor phase compared to non-Type D males. However, there was no relationship between Type D and cardiovascular reactivity in females. In addition, Type D individuals exhibited significantly higher feelings of subjective stress compared to non-Type D's. These findings provide new evidence on Type D and suggest that Type D may affect health through increased cardiac output and higher subjective feelings of stress following acute stress.

**Keywords:** cardiovascular disease; negative affect; reactivity; social inhibition; Type D personality

### **Introduction**

A number of psychological risk factors for cardiovascular disease (CVD) have been identified, such as depression (Frasure-Smith, Lesperance, & Talajic, 1995), low social support (Lett et al., 2005) and hostility (Shekelle, Gale, Ostfeld, & Paul 1983). One emerging risk factor for CVD is Type D personality (or distressed personality) which describes individuals who simultaneously experience high levels of negative affectivity (NA) and social inhibition (SI) (Denollet, 2005). Type D is therefore characterised as the tendency to experience negative emotions, paired with the non-expression of these emotions in social interactions.

Accumulating evidence from a number of patient groups, including patients suffering from chronic heart failure (Schiffer et al., 2005), peripheral arterial disease (Aquarius, Denollet, Hamming, & De Vries 2005) and hypertension (Denollet, 2005) indicates that Type D is associated with increased psychological distress, including symptoms of depression and social alienation (Denollet, Sys, & Brutsaert, 1995; Pedersen, van Domburg,

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Theuns, Jordaens, Erdman, 2004), anger (Denollet & Brutsaert, 1998), anxiety (Pedersen et al., 2004) and vital exhaustion (Pedersen & Middel, 2001), poorer quality of life (Schiffer et al., 2005; Al-Ruzzeq et al., 2005) and adverse clinical outcome (Denollet & Brutsaert, 1998; Denollet et al., 1996; Denollet, Vaes, & Brutsaert, 2000; Pedersen et al., 2004). Indeed, Type D incurs a risk on par with established biomedical risk factors, with Type D patients experiencing a 4-fold increased risk of adverse clinical outcome (including death and myocardial infarction (MI)) (Denollet & Brutsaert, 1998; Denollet et al., 1996; Denollet et al., 2000).

Although Type D has been deemed by some authors as simply another measure of negative affect (Lesperance & Frasur-Smith, 1996), it is more than that, as it also includes a measure of the ways in which people deal with this negative affect. Indeed, the combination of NA and SI, rather than negative emotions alone was a significantly stronger predictor of death and repeat MI in a population of cardiac patients (Denollet et al., 2006).

While the studies cited above are suggestive of a potential causal link between Type D and CVD, little is known about the specific pathways which may explain the relationship. Pedersen and Denollet (2006) suggest that Type D may have its effect through either psychophysiological or behavioural pathways. Indeed, previous research which was sought to uncover specific psychophysiological routes has met with some success. Evidence from two studies (Denollet et al., 2003; Conraads et al., 2006) now suggests that Type D is related to immune activation, with Type D patients being found to have increased circulating levels of soluble tumour necrosis factor (sTMF)- $\alpha$ , a powerful predictor of mortality in patients with chronic heart failure.

Recently, Whitehead, Perkins-Porras, Strike, Magid, and Steptoe (2007) investigated a further biological pathway by which Type D may affect prognosis. They assessed cortisol output in 72 patients who had recently suffered from acute coronary syndrome (ACS). Patients with Type D personality showed significantly higher cortisol awakening responses, independent of demographic and clinical factors and depression. This finding suggests that Type D may be associated with disruption of HPA axis function.

A further study, undertaken by Habra, Linden, Anderson, and Weinberg (2003), investigated the relationship between Type D personality and cardiovascular reactivity to acute stress. Research on cardiovascular reactivity (CVR) is based on the premise that individuals showing exaggerated cardiovascular responses to stressful conditions may be more at risk for the development of cardiovascular syndromes, such as hypertension or coronary heart disease, than those exhibiting relatively smaller responses. The findings from Habra et al. (2003) provide some limited support for the association between Type D and CVR. Although no associations were found between the global Type D construct and reactivity, the individual NA and SI components were significantly linked to blood pressure reactivity and cortisol reactivity. More specifically, SI was associated with heightened systolic and diastolic reactivity in the male participants. In addition, both NA and SI were found to be related to greater cortisol reactivity, again only in the male participants. The authors propose that their findings suggest a potential pathophysiological role for the Type D construct in the development of cardiac disease, particularly in men.

The aim of the current study is to extend the research on Type D and CVR carried out by Habra et al. (2003). First, it is important to further investigate whether the global Type D construct (in addition to the separate components as established by Habra et al.) is associated with cardiovascular reactivity. Second, this study will utilise more comprehensive measures of psychophysiological response, by measuring cardiac output and peripheral resistance in addition to blood pressure and heart rate. Third, the current

study will also include a recovery phase post-stressor in order to examine the role of Type D on recovery from stress, extending previous research which relied solely on responses during the stressor.

The current study has four key research questions: (1) Do Type D individuals exhibit a differential cardiovascular response to stressful stimuli compared to non-Type D's? (2) Are the components of Type D (NA and SI) related to cardiovascular responses to stress, in line with previous research carried out by Habra et al. (2003)? (3) Is the cardiovascular response to stress more evident in Type D males, as reported by Habra et al. (2003)? and (4) Do Type D individuals demonstrate higher subjective feelings of stress following a stressor compared to non-Type D individuals? It is known that Type D individuals experience higher levels of distress than non-Type D individuals (Denollet et al., 1995; Pedersen et al., 2004), which may be because they perceive situations as being more stressful than non-Type D's.

## Method

### *Participants*

Four hundred and fifteen healthy young adults (339 females) completed a screening questionnaire. Following this, a 2-stage selection process was carried out. First, participants were classified as Type D and non-Type D using the established cut off scores (Denollet, 2005). Second, in order to maximise the chances of detecting significant effects (i.e. statistical power) in this exploratory experimental comparison, we selected those participants who scored in the upper and lower quartiles of the NA and SI subscales of the Type D Scale, for the laboratory study (i.e. for the top quartile scoring  $\geq 13$  on both the NA and SI subscales, and for the lower quartile scoring  $\leq 9$  on both subscales). Through this process 166 participants (120 females) were identified and invited to take part in the experimental phase and from this 84 participants took part, representing a 50% recruitment rate. The respondents and non-respondents did not differ significantly in terms of age ( $t(1, 164) = 1.32$ , ns) or gender ( $\chi^2(1, N = 166) = 6.34$ , ns). All participants were university students who took part in the study for course credit.

Participants in the experimental phase were 84 healthy young adults (42 females, age  $M = 22.0$  yrs,  $SD = 6.8$  yrs). The men (mean = 21.0,  $SD = 4.6$ ) and women (mean = 22.9,  $SD = 8.5$ ) did not differ significantly in terms of age,  $t(1, 82) = 1.26$ , ns. There were 24 Type D females and 21 Type D males who scored  $\geq 13$  on both the NA and SI subscales of the DS14 Type D scale (Denollet, 2005). In addition, there were 18 non-Type D females, and 21 non-Type D males who scored  $\leq 9$  on both subscales. Traditionally, Type D personality is classified based on Denollet's (2005) cut-off points of  $\geq 10$ . Of course, participants in our study would also be classified as Type D or non-Type D using Denollet's recommended cut-off points. No participants reported either being in poor health or taking any medication that might influence the cardiovascular measurements.

### *Physiological measures*

Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), cardiac output (CO) and total peripheral resistance (TPR) were recorded throughout the experiment. These measurements were recorded using the FMS Portapres blood pressure monitor and Beatscope 1.0 (TNO, Biomedical Instrumentation Research Unit, Amsterdam, The Netherlands), which takes non-invasive blood pressure measurement

and beat to beat haemodynamic monitoring. The instrument has been used extensively in cardiovascular reactivity research (e.g. Chafin, Roy, Gerins, & Christenfeld 2004; Wright, O'Donnell, Brydon, Wardle, & Steptoe 2007). This technique uses a finger cuff which the participant wears on the third finger of the left hand, which monitors finger arterial pressure continuously throughout the experimental session. Beatscope is a software package used for the analysis of arterial pressure waveforms. It enables the application of filters that correct for pressure wave distortion when the arterial pressure is obtained from a peripheral site, such as a finger artery.

### ***Psychological measures***

#### *Type D personality*

This was assessed using the 14-item Type D Scale (DS14; Denollet, 2005). The DS14 comprises a 7-item subscale, which measures NA (e.g. 'I often feel unhappy') and a 7-item subscale measuring SI (e.g. 'I am a closed kind of person'). Respondents rate their personality on a 5-point Likert-type scale which ranges from 0 = false to 4 = true. The NA and SI scales can be scored as continuous variables (range 0–28). Traditionally, participants who score highly on both NA and SI using a cut-off point of  $\geq 10$  are classified as having a Type D personality. However, for the purposes of the current study we employed a more stringent cut-off criteria of  $\geq 13$  on both subscales for high Type D personality, and  $\leq 9$  on both subscales for low Type D personality. Cronbach's  $\alpha$  was 0.91 and 0.90 respectively for NA and SI, demonstrating excellent internal consistency in the current study.

### ***Affect measures***

#### *Stress arousal checklist*

Participants' subjective feelings of stress were measured by the stress arousal checklist (McKay, Cox, Burrows, & Lazzarini 1978). For the purposes of this experiment only the stress items were selected. Participants are required to respond to 19 words such as 'tense', 'worried' and 'jittery' on the following response categories 'definitely feel', 'slightly feel', 'cannot decide' and 'definitely do not feel' based on how they feel at the present moment. This gives each participant a score of 0–19. Participants completed the checklist three times during the experiment; baseline, stressor and recovery. Cronbach's  $\alpha$  was 0.92 in the current study, indicating excellent internal consistency.

An additional self-report measure of subjective feelings of stress was in the form of a visual analogue scale (VAS; 10 cm), labelled from low stress (0 cm) to high stress (10 cm) which was administered at baseline, stressor and recovery.

### ***Stressor task***

#### *Paced Auditory Serial Addition Test (PASAT)*

The PASAT mental arithmetic task was used as the experimental stressor (Gronwall, 1977). This task was selected as it has been shown in numerous studies to reliably perturb the cardiovascular system (Ring, Burns, & Carroll 2002; Winzer et al., 1999). It also demonstrates good test-retest reliability (e.g. Willemson et al., 1998). In this task, participants were required to add two sequentially presented single digit numbers together.

In order to do this, the participant is required to retain the latter number and add it to the next number presented, and so on. Numbers were presented using a tape recorder and answers given orally. If participants faltered, they were instructed to start again with the next numbered pair. After completing a practice trial, the participants completed the first task phase in which 60 numbers were presented at a rate of one every 4 s, then the second sequence of 60 numbers at one for every 2 s. In total, the task lasted for 6–4 min for the slower sequence and 2 min for the faster sequence. Participant performance was recorded by the experimenter.

### *Procedure*

Each participant arrived individually for the experiment. The experimenter explained that the participants' heart rate and blood pressure would be monitored during a mental arithmetic task. After the participant consented to take part they were seated at a table and the finger cuff of the blood pressure monitor was attached to them. Participants were then asked to complete the Type D Scale, the stress arousal checklist and visual analogue scale for the first time. The physiological measures taken during this initial period were discarded, as they would not represent a genuine resting state. Following this, recording began for the 5-min baseline period; during this time participants were asked to sit quietly and read a textbook.

After the 5-min baseline period was completed, the experimenter began the PASAT mental arithmetic task (stressor); participants were given a practice trial to ensure that they understood the instructions of the task. Following this, the stressor phase of the experiment began, starting with the presentation rate of one number every 4 s, immediately followed by a rate of one number every 2 s.

At the end of the mental arithmetic task, the experimenter turned off the Portapres, and removed the finger cuff from the participant (this was done to minimise discomfort for the participant in wearing the Portapres for a long period of time). At this point, participants were asked to complete the stress arousal checklist and visual analogue scale for the second time. After this participants were instructed to sit quietly during a 10-min rest period where they were again asked to read a textbook. Following this 10-min period, participants were re-attached to the Portapres for measurement during a 5-min recovery phase, where they were again asked to sit quietly and read a textbook. Upon completion of the recovery period, participants were asked to complete the stress arousal checklist and visual analogue scale for the final time. The participant was then debriefed before leaving the room.

### *Data reduction and statistical analyses*

Four cardiovascular measures were examined: SBP, DBP, HR and TPR. These measures were assessed across three time periods: baseline, stressor and recovery. The stressor phase recordings were taken during the 2-s presentation rate of the PASAT. The average value for the cardiovascular measures at each time point is used as the dependent variable. In order to investigate the effect of Type D on each of the physiological dependent variables, a series of mixed measures ANOVA were computed across the three time periods (baseline, stressor, recovery) while controlling for initial levels of the dependent variable in each case (in order to control for individual differences in baseline levels and their impact on subsequent magnitude of change). As the assumption of sphericity was violated in each case, the Greenhouse–Geisser correction was used. It is this value that

is reported in each of the analyses which follow. In addition, analyses were subsequently carried out separately for males and females. This was done because gender differences in reactivity studies are commonly observed (e.g. Habra et al., 2003). Furthermore, the effects of the components of Type D–NA and SI on each of the physiological measures was also investigated using mixed measures ANOVA. An  $\alpha$  level of 0.05 was used throughout. Statistical analyses were done using the Statistical Package for the Social Sciences, version 14 (SPSS Inc, Chicago, Illinois).

## Results

### *Stress manipulation check*

In order to establish that the mental arithmetic task was an effective stressor in producing physiological change, a series of repeated measures ANOVA were run on each of the dependent variables (heart rate, blood pressure, cardiac output and peripheral resistance). For each outcome variable, a significant main effect of time was found. Mean scores for the baseline, stressor and recovery periods are presented in Table 1.

### *Type D personality and physiological data*

Analyses revealed that the separate components of Type D, i.e. NA and SI, were unrelated to the physiological variables. Therefore, all further analyses were conducted using the global Type D construct. Mixed measures ANOVA revealed a significant effect of time across each of the physiological variables, [HR: ( $F(2, 82) = 4.66, p < 0.001$ ), SBP: ( $F(2, 82) = 5.23, p < 0.01$ ), DBP: ( $F(2, 82) = 3.51, p < 0.05$ ), TPR: ( $F(2, 82) = 3.56, P < 0.05$ ) and CO: ( $F(2, 82) = 6.2, p < 0.001$ )]. However, there was no time  $\times$  Type D interaction for any of the physiological variables [HR: ( $F(2, 79) = 0.01, ns$ ); SBP: ( $F(2, 79) = 0.14, ns$ ); DBP: ( $F(2, 79) = 0.04, ns$ ); TPR: ( $F(2, 79) = 0.093, ns$ ); CO: ( $F(2, 79) = 2.31, ns$ )]. The non-significant interaction effects for time  $\times$  Type D indicate that Type D exerts a stable effect on outcome over time.

There was a significant group effect of Type D on cardiac output ( $F(2, 79) = 6.7, p < 0.03$ ), but not on any of the other physiological measures [HR: ( $F(2, 79) = 0.004, ns$ ); SBP: ( $F(2, 79) = 0.44, ns$ ); DBP: ( $F(2, 79) = 0.04, ns$ ); TPR: ( $F(2, 79) = 0.06, ns$ ); CO: ( $F(2, 79) = 6.7, p < 0.03$ )]. Given this significant group effect of Type D on CO, and the previous results from Habra et al. (2003) which revealed gender differences in Type D and

Table 1. Mean baseline and stressor values for the physiological data.

Measurement	Baseline	Stressor	Recovery
HR	79.46 (9.87)	85.55 (10.1)	76.03 (12.15)
SBP	122.83 (18.87)	142.34 (23.51)	117.99 (18.29)
DBP	70.14 (13.28)	81.92 (15.97)	66.48 (13.62)
CO	5.68 (1.26)	6.2 (1.26)	5.41 (1.32)
TPR	0.98 (0.3)	1.09 (0.59)	1.01 (0.48)

Notes: Values enclosed in parentheses are SD. HR = heart rate (bpm), SBP = systolic blood pressure (mm HG), DBP = diastolic blood pressure (mm HG), CO = cardiac output (lpm), TPR = total peripheral resistance ( $\text{dyn s cm}^{-5}$ ).

physiology, we conducted further analyses to investigate any gender differences in Type D and CO. A significant group  $\times$  time effect of Type D on CO was found for males [ $F(3,37)=3.4, p<0.05$ ] but not in the female participants [ $F(3,37)=0.78, ns$ ] as demonstrated in Figure 1, indicating that males high in Type D personality have a higher cardiac output compared to those low on Type D. ANCOVA (controlling for baseline cardiac output) revealed that the difference in cardiac output between Type D's and non-Type D's occurs during the stressor phase  $F(1, 82)=4.91, p<0.05$ . In the interest of completeness, it should be noted that no further gender differences were found on the remaining physiological measures.

### Type D personality and subjective stress

It is also important to examine the effect of Type D personality on the participants' self-ratings of stress on both the stress arousal checklist and the visual analogue scale. Mixed measures ANOVA were again conducted across the three time points; baseline, stressor and recovery while controlling for initial levels of the dependent variable.

There was a significant effect of time on subjective stress arousal as measured by the stress arousal checklist  $F(2, 82)=99.02, p<0.001$ , and a significant group effect of Type D on stress arousal  $F(1, 83)=6.43, p<0.03$ , as shown in Figure 2, indicating that

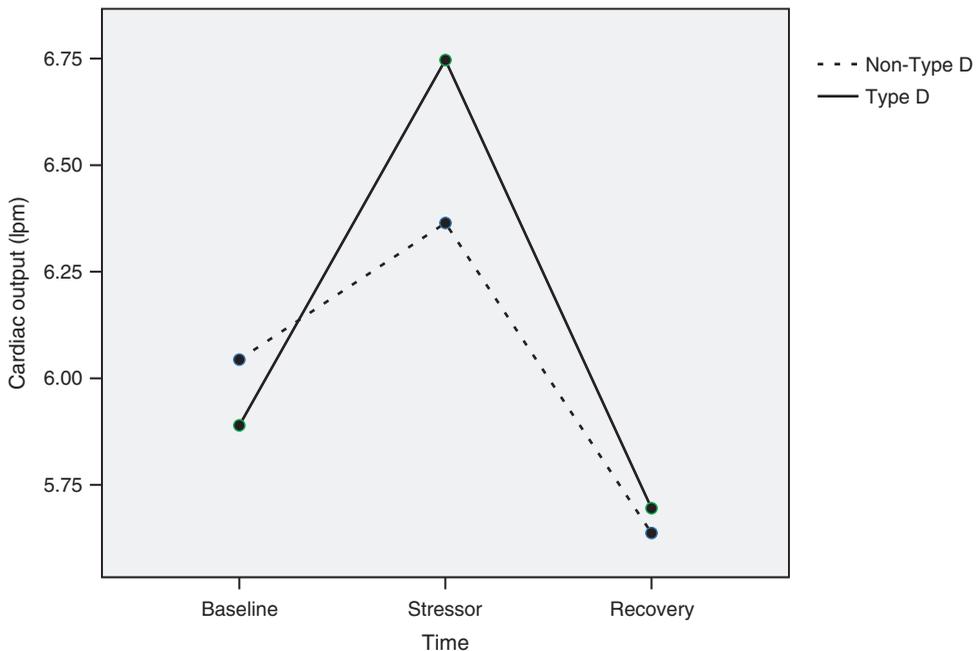


Figure 1. The effect of Type D on cardiac output in the male participants across the baseline, stress and recovery phases.

Notes: lpm = Litres per minute.

The graph shows a significant group effect of Type D on cardiac output [ $F(2, 79)=6.7, p<0.03$ ]. In addition, a significant time  $\times$  Type D effect on CO in males [ $F(3, 37)=3.4, p<0.05$ ]. ANCOVA (controlling for baseline cardiac output) revealed that the difference in cardiac output between Type D and non-Type D's occurs during the stressor phase [ $F(1, 82)=4.91, p<0.05$ ].

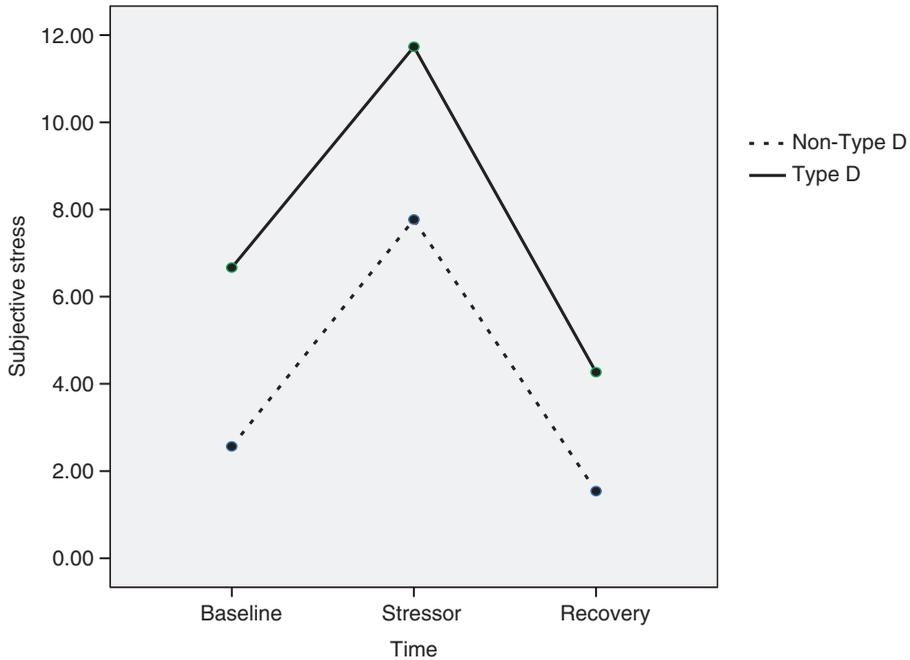


Figure 2. The effect of Type D on subjective stress arousal across the baseline, stress and recovery phases.

Notes: The graph shows a significant group effect of Type D on subjective stress [ $F(1, 83) = 6.43$ ,  $p < 0.03$ ]. In addition, a significant time  $\times$  Type D interaction was established [ $F(2, 79) = 3.38$ ,  $p < 0.05$ ]. ANCOVA (controlling for baseline subjective stress) showed that the difference in subjective stress between the Type D and non-Type D individuals occurred during the stressor phase [ $F(1, 82) = 5.32$ ,  $p < 0.05$ ].

participants who were high on Type D personality reported significantly higher feelings of subjective stress compared to those low on Type D. In addition, a significant time  $\times$  Type D interaction  $F(2, 79) = 3.38$ ,  $p < 0.05$  was established. ANCOVA (controlling for baseline subjective stress) showed that the difference in subjective stress between the Type D and non-Type D individuals occurred during the stressor phase,  $F(1, 82) = 5.32$ ,  $p < 0.05$ .

For the ratings obtained from the visual analogue scale, there was a significant effect of time  $F(2, 82) = 47.5$ ,  $p < 0.001$ , but there was no group effect of Type D  $F(1, 83) = 2.16$ , ns, and no time  $\times$  Type D interaction  $F(2, 79) = 0.87$ . Again, in the interest of completeness it should be noted that no gender differences were found on either of the subjective measures of stress.

## Discussion

The study had four main aims: (1) to investigate the relationship between Type D personality and cardiovascular reactivity to stress; (2) to investigate the relationship between the components of Type D (NA and SI) and CVR to stress, (3) to investigate if the cardiovascular stress response is more evident in Type D males, as reported by

Habra et al. (2003) and (4) to investigate the relationship between Type D and subjective feelings of stress.

The first aim of the study was to establish if Type D individuals exhibit a differential pattern of cardiovascular reactivity to stress compared to non-Type D's. Type D's were compared to non-Type D's on several physiological measures; HR, blood pressure, peripheral resistance and cardiac output. Although no differences were found between Type D's and non-Type D's on measures of HR, blood pressure and peripheral resistance, there was a significant effect of Type D on cardiac output. Furthermore, Type D males exhibited significantly higher cardiac output during the stressor phase of the experiment compared to the non-Type D male participants. This is in line with previous research (Habra et al., 2003) which also identified differences in the reactivity of males who were high in the components of Type D-NA and SI. However, the latter study failed to demonstrate any relationship between the global Type D construct and cardiovascular reactivity. The current study is therefore an extension of previous research demonstrating a relationship between the global Type D construct and CVR for the first time. Indeed, the current study failed to find an association between the components of Type D-NA and SI and physiology, signalling that it is the interaction of the components, as described by Type D, which is crucial. Similarly, Denollet et al. (2006) demonstrated that it is the combination of the two traits that is toxic, with SI moderating the effects of NA on cardiac prognosis.

The relationship observed between Type D and increased cardiac output during stress is important for a number of reasons. By establishing that Type D is related to heightened physiological reactions to acute stress, a further mechanism by which Type D may potentially influence health has been found. Specifically, increased cardiac output means that the heart has to work at an increased level in order to pump blood to the rest of the body during stress. Importantly, increased cardiac output over time has been implicated early in the disease course of hypertension (Julius, 1998).

The third aim of the current study was to examine the relationship between Type D and subjective stress. It was found that Type D individuals report higher feelings of subjective stress during a stressor compared to non-Type D's. Type D is related to a number of negative emotional states, such as anxiety and depression (Denollet et al., 1995; Pedersen et al., 2004). Therefore, the association between Type D and heightened feelings of stress provides further evidence for the link between Type D and psychological distress. Establishing a link between Type D and increased feelings of stress is particularly important given the role of stress in the etiology of CHD.

It is important to consider the sex differences observed in the current study. A significant relationship was found between Type D and cardiac output in males, but not in females. Similarly, Habra et al. (2003) also found a significant relationship between Type D and CVR in males only. Further research is required in order to determine if this is a robust and replicable sex difference effect. If it is, then it may be that Type D operates differentially in males and females. For example, in females, Type D may have a more toxic effect on outcome through psychosocial mechanisms rather than via CVR.

Several limitations of the present study should be noted. First, the generalisability of the current findings to a more 'at-risk' population is limited by the fact that the sample consisted of healthy young adults. Second, as the experiment was conducted in the laboratory the findings may be limited to how Type D and physiology are related to stress experienced in the lab. Indeed, reactivity in the lab may not be representative of stress experienced during daily life. However, a recent study by Johnston, Tuomisto, and Patching (2007) has demonstrated that reactivity in the lab predicts reactivity to stressors

in real life. Third, it would have been useful to include a prospective component to the study in order to test if the association between Type D and cardiac output is consistent across time. Fourth, out of 166 potential participants who were invited to take part in the experimental phase, only 84 participants took part in the study, representing a 50% recruitment rate. Finally, in order to maximise the likelihood of detecting significant effects we only tested participants at the low and high range of Type D. Therefore, the generalisability of these findings to other studies which classify Type D in the standard way may be limited.

Future research is needed to test if these are robust and replicable findings, and to investigate if the relationship between Type D and cardiac output is also present in a cardiac population. Furthermore, in order to make the findings generalisable beyond laboratory stress, ambulatory monitoring should be used to see if the relationships observed still hold as the individual faces stressors in everyday life.

The current study has extended the existing research on Type D in several important ways. First, it has identified for the first time that Type D is related to heightened cardiac output to acute stress in males. This may represent a direct physiological pathway by which Type D is associated with ill-health. Second, it has established that Type D is associated with increased subjective feelings of stress following acute stress. These findings suggest that Type D individuals may be at a particular risk for stress-related cardiac events.

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### References

- Al-Ruzzeh, S., Athanasiou, T., Mangoush, O., Wray, J., Modine, T., George, S., et al. (2005). Predictors of poor mid-term health related quality of life after primary isolated coronary artery bypass grafting surgery. *Heart*, *91*, 1557–1562.
- Aquarius, A.E., Denollet, J., Hamming, J.F., & De Vries, J. (2005). Role of disease status and Type D personality in outcomes in patients with peripheral arterial disease. *American Journal of Cardiology*, *96*, 996–1001.
- Chafin, S., Roy, M., Gerin, W., & Christenfeld, N. (2004). Music can facilitate blood pressure recovery from stress. *British Journal of Health Psychology*, *9*, 393–403.
- Conraads, V.M., Denollet, J., De Clerck, L.S., Stevens, W.J., Bridts, C., & Vrints, C.J. (2006). Type D personality is associated with increased levels of tumour necrosis factor (TNF) –  $\alpha$  and TNF-  $\alpha$  receptors in chronic heart failure. *International Journal of Cardiology*, *113*, 34–38.
- Denollet, J. (2005). DS14: Standard assessment of negative affectivity, social inhibition, and Type D personality. *Psychosomatic Medicine*, *67*, 89–97.
- Denollet, J., & Brutsaert, D.L. (1998). Personality, disease severity, and the risk of long-term cardiac events in patients with decreased ejection fraction after myocardial infarction. *Circulation*, *97*, 167–173.
- Denollet, J., Conraads, V.M., Brutsaert, D.L., De Clerck, L.S., Stevens, W.J., & Vrints, C.J. (2003). Cytokines and immune activation in systolic heart failure: The role of Type D personality. *Brain, Behavior and Immunity*, *17*, 304–309.

- Denollet, J., Pedersen, S.S., Ong, A.T., Erdman, R.A., Serruys, P.W., & van Domburg, R.T. (2006). Social inhibition modulates the effect of negative emotions on cardiac prognosis following percutaneous coronary intervention in the drug eluting stent era. *European Heart Journal*, *27*, 171–177.
- Denollet, J., Sys, S.U., & Brutsaert, D.L. (1995). Personality and mortality after myocardial infarction. *Psychosomatic Medicine*, *57*, 582–591.
- Denollet, J., Sys, S.U., Stroobant, N., Rombouts, H., Gillebert, T.C., & Brutsaert, D.L. (1996). Personality as independent predictor of long-term mortality in patients with coronary heart disease. *Lancet*, *34*, 417–21.
- Denollet, J., Vaes, J., & Brutsaert, D.L. (2000). Inadequate response to treatment in coronary heart disease: Adverse effects of Type D personality and younger age on 5-year prognosis and quality of life. *Circulation*, *102*, 630–635.
- Frasure-Smith, N., Lesperance, F., & Talajic, M. (1995). Depression and 18 month prognosis after myocardial infarction. *Circulation*, *91*, 999–1005.
- Gronwall, D.M.A. (1977). Paced Auditory Serial Addition Task: A measure of recovery from concussion. *Perceptual and Motor Skills*, *44*, 367–373.
- Habra, M.E., Linden, W., Anderson, J.C., & Weinberg, J. (2003). Type D personality is related to cardiovascular and neuroendocrine reactivity to acute stress. *Journal of Psychosomatic Research*, *55*, 235–245.
- Johnston, D.W., Tuomisto, M., & Patching, G.R. (2007). The relationship between cardiac reactivity in the laboratory and in real life. *Health Psychology* in press.
- Julius, S. (1998). Transition from high cardiac output to elevated vascular resistance in hypertension. *American Heart Journal*, *116*, 600–606.
- Lesperance, F., & Frasure-Smith, N. (1996). Negative emotions and coronary heart disease: Getting to the heart of the matter. *Lancet*, *347*, 414–415.
- Lett, H.S., Blumenthal, J.A., Babyak, M.A., Strauman, T.J., Robins, C., & Sherwood, A. (2005). Social support and coronary heart disease: Epidemiologic evidence and implications for treatment. *Psychosomatic Medicine*, *67*, 869–878.
- Mackay, C.J., Cox, T., Burrows, G.C., & Lazzarini, A.J. (1978). An inventory for the measurement of self-reported stress and arousal. *British Journal of Social and Clinical Psychology*, *17*, 283–284.
- Pedersen, S.S., & Denollet, J. (2006). Is Type D personality here to stay? Emerging evidence across cardiovascular patient groups. *Current Cardiology Reviews*, *2*, 205–213.
- Pedersen, S.S., Lemos, P.A., van Vooren, P.R., Liu, T.K., Daemen, J., Erdman, R.A.M., et al. (2004). Type D personality predicts death or myocardial infarction after bare metal stent or sirolimus-eluting stent implantation: A Rapamycin-Eluting Stent Evaluated at Rotterdam Cardiology Hospital (RESEARCH) Registry Sub-study. *Journal of the American College of Cardiology*, *44*, 997–1001.
- Pedersen, S.S., & Middel, B. (2001). Increased vital exhaustion among Type-D patients with ischemic heart disease. *Journal of Psychosomatic Research*, *51*, 443–449.
- Pedersen, S.S., Van Domburg, R.T., Theuns, D.A.M.J., Jordaens, L., & Erdman, R.A.M. (2004). Type D personality is associated with increased anxiety and depressive symptoms in patients with an implantable cardioverter defibrillator and their partners. *Psychosomatic Medicine*, *66*, 714–719.
- Ring, C., Burns, V.E., & Carroll, D. (2002). Shifting hemodynamics of blood pressure control during prolonged mental stress. *Psychophysiology*, *39*, 585–590.
- Schiffer, A.A., Pedersen, S.S., Widdershoven, J.S., Hendriks, E.H., Winter, J.B., & Denollet, J. (2005). The distressed (type D) personality is independently associated with impaired health status and increased depressive symptoms in chronic heart failure. *European Journal of Cardiovascular Prevention and Rehabilitation*, *12*, 341–346.
- Shekelle, R.B., Gale, M., Ostfeld, A.M., & Paul, O. (1983). Hostility, risk of coronary heart disease, and mortality. *Psychosomatic Medicine*, *45*, 109–114.

- Whitehead, D.L., Perkins-Porras, L., Strike, P.C., Magid, K., & Steptoe, A. (2007). Cortisol awakening response is elevated in acute coronary syndrome patients with Type-D personality. *Journal of Psychosomatic Research, 62*, 419–425.
- Willemson, G., Ring, C., Carroll, D., Evans, P., Clow, A., & Hucklebridge, F. (1998). Secretory immunoglobulin A and cardiovascular reactions to mental arithmetic and cold pressor. *Psychophysiology, 35*, 252–259.
- Winzer, A., Ring, C., Carroll, D., Willemsen, G., Drayson, M., & Kendall, M. (1999). Secretory immunoglobulin A and cardiovascular reactions to mental arithmetic, cold pressor, and exercise: Effects of beta-adrenergic blockade. *Psychophysiology, 36*, 591–601.
- Wright, C.E., O'Donnell, K., Brydon, L., Wardle, J., & Steptoe, A. (2007). Family history of cardiovascular disease is associated with cardiovascular responses to stress in healthy young men and women. *International Journal of Psychophysiology, 63*, 275–282.