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Type D personality is associated with a sensitized cardiovascular response to recurrent stress in men

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ABSTRACT

The present study sought to examine the role of gender and Type D personality on cardiovascular reactivity to stress, by examining patterns of cardiovascular adaptation to recurrent laboratory-based stress. Cardiovascular data were collected from 76 students who, following an initial 10-min baseline period, underwent two cognitive stress tasks. Type D personality was assessed using the 16-item Type D scale. Adaptation of cardiovascular response to recurrent stress was examined by scrutinizing the changes in systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) across the procedure. Female participants and non-Type D males showed cardiovascular habituation to recurrent stress. For Type D males, however, cardiovascular sensitization was evident. The results implicate Type D personality in maladaptive cardiovascular responses, particularly in men, highlighting a possible direct mechanism of psychosomatic cardiovascular pathogenesis.

1. Introduction

Identified by a tendency to experience negative emotions (negative affectivity; NA) along with the propensity to inhibit self-expression in social situations (social inhibition; SI), the "distressed" personality type, better known as Type D personality, has been shown to predict poor health-related outcomes following coronary events (for reviews see Denollet, Schiffer, & Spek, 2010; O'Dell, Masters, Spielmans, & Maisto, 2011), although contrary findings have also been reported (see Coyne et al., 2011; Grande, Romppel, & Barth, 2012; Pelle et al., 2010). Type D personality appears to contribute negatively to cardiovascular health both via indirect (increased social alienation and failure to adhere to medical treatment; Molloy et al., 2012; Williams et al., 2008) and direct (immune activation; Conraads et al., 2006) mechanisms. One psychosomatic mechanism garnering support posits that that Type D personality influences stress-appraisal in ways that moderate cardiovascular responses to stressors (Habra, Linden, Anderson, & Weinberg, 2003; Howard, Hughes, & James, 2011; Kupper, Denollet, Widdershoven, & Kop, in press; Williams, O'Carroll, & O'Connor, 2009).

Cardiovascular reactivity to psychological stress (CVR) is believed to be a marker of risk of future illness among healthy persons, such that high CVR is associated with increased risk of cardiovascular disease (Blascovich & Katkin, 1993). This is interesting given that previous research has suggested that the individual components of Type D personality (namely, NA and SI or proxy measures thereof) are indeed each linked to physiological reactivity to stress (Gross & Levenson, 1997; Habra et al., 2003; Horsten et al., 1999; Marshall & Stevenson-Hinde, 1998; von Känel et al., 2005) as well as the overall Type D personality profile (Howard et al., 2011; Kupper et al., in press; Williams et al., 2009). One note-worthy aspect about previous studies using healthy mixed-gender samples (Habra et al., 2003; Williams et al., 2009) is that effects have been restricted to males. Gender differences in CVR have been well-documented, with males consistently showing higher resting blood pressure and greater blood pressure reactivity to standardized laboratory-based stressors than females (e.g., Light, Turner, Hinderliter, & Sherwood, 1993; Newton, Watters, Philhower, & Weigel, 2005). Furthermore, the majority of epidemiological research on Type D personality has focused on male cardiac patients. Thus, it remains to be clearly established whether gender moderates the effect of Type D personality on either health-related outcomes or physiological reactivity.

An interesting extension of the CVR hypothesis includes examination of patterns of cardiovascular adaptation to recurrent stress, which may be particularly elucidating when detailing the influence of individual differences on physiological reactivity. Rather than relying on the assessment of cardiovascular responses arising from a single exposure to a novel laboratory-based stressor, the introduction of the same stressor, either within the same laboratory session (Howard & Hughes, 2012a; Hughes, 2007a, 2007b; Hughes, Howard, James, & Higgins, 2011), over time (Carroll, Turner, Lee, & Stephenson, 1984; Johnson, Lavoie, Bacon, Carlson, & Campbell,

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2012), or within the period of a single exposure to stress (Hughes & Black, 2006), might reveal important differences in the individual's capacity to habituate to recurrent stress which may well be a crucial element of psychosomatic pathogenesis (Kelsey, 1993).

Type D personality may be expected to impact on initial responses to a stressor, given that a Type D individual might be particularly sensitive to stress (due to the tendency toward NA) while feeling inclined to conceal this sensitivity (due to SI). Moreover, the combination of SI and NA might serve to impede a Type D individual's capacity to habituate to the stressor in comparison to non-Type D participants. If so, then scrutiny of cardiovascular adaptation patterns may reveal a greater adverse impact of Type D personality than would be suggested by examining the initial stress response in isolation. Furthermore, such an assessment would likely have greater external validity than one of a single stress response in that it intrinsically incorporates an evaluation of whether the observed associations are short-term or longer lasting.

As such, the present study sought to investigate if Type D personality and/or gender influences adaptation of cardiovascular responses to recurrently presented stress in male and female Type D and non-Type D individuals. If Type D impacts adversely on CVR patterns over time, then it can be implicated in the psychosomatic etiology of cardiovascular disease, thereby corroborating the claim that Type D personality represents a particularly cardio-toxic combination of psychological traits. Such mechanisms may help account for the already well-established association between Type D and cardiovascular disease outcomes in clinical populations, in ways that might not be satisfactorily explained by invoking indirect behavioral pathways. Secondly, the present study sought to offer an investigation of whether the Type D personality may be associated with physiological reactivity differently in healthy male, and female, participants.

2. Methods

2.1. Participants

The participants were 40 male and 36 female (mean age = 21.65, $SD = 4.48$) undergraduate university students. Participation was voluntary and all participants gave informed consent prior to participation in the study. All participants had a negative family history of cardiovascular disease and were free of major illnesses and medications that could affect cardiovascular function. The mean body mass index (BMI) of the sample was 23.23 kg/m^2 ($SD = 3.24$). There was no gender difference in age, $t(74) = 1.68$, $p = .091$. ANOVA also confirmed that there were no gender, personality, or personality × gender differences in BMI or age (all $ps > .07$).

2.2. Design

The present study was of a 2 × 2 × 4 mixed factorial design, within which the between-subjects component comprised a 2 × 2 design. The between-subjects factors were gender (two levels: male; female) and personality (two levels: Type D; non-Type D). The within-subjects factor was phase (four levels: baseline, task exposure 1, intertask interval, task exposure 2). The dependent variables were systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR).

2.3. Measurement apparatus and materials

2.3.1. Psychometric assessment

Type D personality was assessed using the 16-item Type D scale (DS16; Denollet, 1998). The DS16 comprises of two 8-item subscales that measure NA and SI. Conventionally a median split on both subscales is used to identify Type D status (Denollet, 1998; Denollet, Vaes, & Brutsaert, 2000; Pedersen & Middel, 2001). Good internal consistency has been demonstrated in previous research (Denollet, 1998) and in the present sample (Cronbach's $\alpha = 88$ for the NA subscale; Cronbach's $\alpha = 77$ for the SI subscale). Because there was no gender difference in the medians of the NA scores (Mann–Whitney) $U = 703$, $p = .859$, or the SI scores, $U = 644$, $p = .428$, a median split based on the whole sample ($N = 76$) was used. Participants scoring ≥11.5 on NA and ≥14 on SI were identified as Type D. This resulted in 25 individuals identified as Type D (12 males and 13 females) and 51 as non-Type D (28 males and 23 females). This distribution of gender × Type D did not deviate from chance levels, $\chi^2(1) = .06$, $p = .57$.

2.3.2. Physiological assessment

A Finometer (Finapres Medical Systems BV, BT Arnhem, The Netherlands) was used to assess cardiovascular function. The Finometer is a continuous blood pressure monitor that measures beat-to-beat blood pressure and HR (Schutte, Huisman, Van Rooyen, Oosthuizen, & Jerling, 2003). It has been shown to provide highly accurate assessments of cardiovascular parameters in both healthy (Schutte et al., 2003) and clinical populations (Guelen et al., 2003), meeting the validation criteria of the Association for the Advancement of Medical Instrumentation. The Finometer is based on the volume-clamp method first developed by Peñaz (1973). A finger cuff is attached to the participant's middle phalanx of their middle finger. An in-built photo-plethysmograph detects changes in the diameter of the arterial wall and inflates the cuff, ensuring the arterial walls are kept at a set diameter. When the volume clamp is active at the proper unloaded diameter, intra-arterial pressure equals that of the finger cuff pressure.

3. Procedure

To minimize the impact of variations in environmental cues on reactivity (Christenfeld, Glynn, Kulik, & Gerin, 1998), all testing took place in the same laboratory. Further, to limit circadian cardiovascular rhythms (Giles, 2006), testing took place within the same time-period each day (between 09:00 and 13:00). Participants who were smokers were instructed to abstain from smoking for 1 h before arriving at the laboratory. Upon arrival at the laboratory, participants were greeted by the researcher and seated at a desk in a comfortable chair with arm support and completed the DS16. The Finometer was attached to the participant's middle finger of their non-dominant hand. Participants were asked to rest for a 30-min period prior to the start of the procedure. Reading material was supplied in order to facilitate genuine relaxation and the establishment of cardiovascular baselines (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992). After the initial acclimatization period, the participant was asked to face a computer screen and sit quietly for a 10-min period. The Finometer was operated by the researcher from behind an opaque partition and out of view of the participant, and baseline cardiovascular measures were assessed. Following this, the participant was asked to complete a 5-min mental arithmetic task, followed by a 10-min rest period, and then perform the mental arithmetic task again for 5 min. The stressor task consisted of a number of subtraction problems that the participant was required to solve without the aid of a calculator or other materials. Subtraction items became harder for every three consecutive correct responses therefore controlling for mathematical ability, employing the principle of standardized flexibility previously recommended for CVR assessment (Hughes, 2001; Turner, 1994; Turner et al., 1986). Participants entered their answers using a computer keypad. Beat-to-beat measures of SBP, DBP, and HR were obtained continuously throughout the procedure. A task performance score was computed as the number of mental arithmetic problems attempted, as well as the number of correct answers returned. Due to equipment malfunction performance scores are not available for $n = 11$ participants.

4. Results

4.1. Data reduction and analysis

Mean levels of SBP, DBP, and HR were computed for each phase of the experiment, namely, baseline, task exposure 1, inter-task interval, and task exposure 2 and are shown in Table 1. Internal reliability consistency for each measure was excellent ($\alpha > .90$ for each SBP, DBP, and HR mean). First, a series of 2 × 2 × 2 mixed ANOVAs were conducted to establish if there were any differences in performance from task exposure 1 to task exposure 2. For cardiovascular measures, a series of 2 × 2 between-subjects ANOVAs were conducted to examine any gender or personality differences on baseline measures. Whether cardiovascular measures returned to baseline levels during the intertask interval was examined using