

## Type D personality is related to cardiovascular and neuroendocrine reactivity to acute stress

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### Abstract

**Objective:** The relationship between Type D personality (the joint tendency towards negative affectivity [NA] and social inhibition [SI]) and laboratory indices of cardiovascular health was examined. **Method:** 173 undergraduates (86 male) completed a stress protocol involving a mental arithmetic task with harassment. Blood pressure, heart rate (HR), and salivary cortisol were measured both prior to and during the task. **Results:** The relationships between personality and both resting and reactivity levels were examined. Results indicated that socially inhibited men demonstrated height-

ened blood pressure reactivity. NA was related to dampened HR change during the stress task in men. Correlational analyses indicated that both Type D dimensions were associated with greater cortisol reactivity to stress; however, results no longer remained significant in more stringent regression analyses. **Conclusion:** Findings are consistent with the noted relationship between Type D and cardiovascular disease (CVD), and suggest a possible pathway to disease via an association with physiological hyperreactivity. © 2003 Elsevier Inc. All rights reserved.

**Keywords:** Cortisol; Emotional suppression; Negative affect; Reactivity; Stress; Type D personality

### Introduction

Identified biological and lifestyle risk factors for cardiovascular disease (CVD) and hypertension, such as smoking, obesity, dyslipidemia, hypertension, and diabetes [1], account for a small portion of the variance in the development of such diseases. Thus, behaviorally minded researchers have been examining the impact of psychological factors such as personality and perceived social support on the disease process. Recently, a new personality construct has been proposed to be related to CVD outcome, and consistent associations with morbidity and mortality have been noted. The Type D or “distressed” personality construct was developed by Denollet in his investigation of coping styles in men with coronary heart disease. Empirically identified through cluster analysis, Type D individuals score highly on the negative affectivity (NA) and social inhibition (SI) personality dimensions [2]. NA is defined as the “tendency to experience negative emotions,” including anger, hostile feelings, depressed affect, and anxiety

(Ref. [3], p. 209). SI, on the other hand, is defined as “the avoidance of potential dangers involved in social interaction such as disapproval or nonreward by others” (Ref. [3], p. 209). Thus, the “distressed” personality subtype is characterized by the joint tendency to experience negative emotions and to inhibit these emotions while avoiding social contact with others.

Denollet has repeatedly shown that the distressed personality is associated with CVD outcome. For example, Type D carries a six-fold increase in the likelihood of death from cardiac events two to five years postmyocardial infarct (MI) in men [4], a four-fold increase in mortality 6–10 years following a cardiac event [5], and poorer outcome in post-MI patients with a decreased left ventricular ejection fraction, a condition with a particularly poor prognosis [6]. In all of the above cited studies, Type D was shown to be significantly associated with worse disease outcome, even after controlling for traditional risk factors—symptoms of depression, anger, and anxiety did not add to the predictive power of the Type D construct. Furthermore, NA and SI were not predictive of outcome individually; rather, only the joint presence of high scores on both dimensions was linked to disease morbidity and mortality.

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While the studies cited above suggest a potential causal link between Type D and CVD, the specific pathway to disease has yet to be established. Denollet et al. [5] suggest that personality might be linked to disease outcome either directly through psychophysiological mechanisms, such as silent myocardial ischemia or platelet release potentiated by mental stress; or it might be related indirectly through poor health behaviours or psychological factors, such as lack of social support that may arise from the Type D behavior.

Another plausible mediating mechanism linking Type D personality to CVD may be that Type D behavior is linked to physiological hyperresponsivity, i.e., a marker of hypertension/CVD development [7]. In the case of hypertension, reactivity is thought to lead to increased peripheral resistance, which contributes to elevated blood pressure over time [8]. Hyperreactivity could lead to heart disease by causing injury to the endothelial lining of the arteries, thereby promoting the accumulation of plaque, which, over time, can lead to acute events such as thrombosis or ischemia [9]. Personality traits can be conceptualized as contributing to individual differences in cardiovascular reactivity to stress. As such, hyperreactivity can be construed as a mechanism underlying the relationship between personality and disease [10].

While most of the past research linking reactivity to hypertension/CVD has focused on the sympathetic nervous system (sympathetic–adrenal–medullary or SAM system), there has been a recent move towards also examining the role of adrenocortical activity in the disease process. The seminal work of Selye [11], building on the work of Cannon [12], initially identified the influential role of the hypothalamic–pituitary–adrenal (HPA) axis in mobilizing resources in the general adaptation syndrome (GAS). The HPA axis, via release of glucocorticoids such as cortisol, is involved in increasing glucose levels in the blood, enhancing cardiovascular tone, and suppressing immune function [13]. While these functions are essential for readying the body to cope with immediate stressors, it has been suggested that prolonged or repeated activation of this system may lead to increased risk for disease [14,15]. The term *allostatic load* has been used to describe the “cumulative strain on the body produced by repeated ups and downs of physiologic response, as well as by the elevated activity of physiological systems under challenge” [16]. Thus, individual differences in allostatic loads are thought to be related to the incidence of disease.

There are several possible pathways linking cortisol to hypertension/CVD. First, we know that excess cortisol, as seen in Cushing’s syndrome, is associated with an increased risk for hypertension. Indeed, 85% of patients with the disorder are hypertensive. While the mechanisms by which excess cortisol leads to hypertension are still unclear, several possibilities have been proposed: (a) cortisol might lead to increases in blood pressure by increasing vascular resistance, possibly by enhancing catecholamine pressor response, by increasing angiotensin II levels, or by potentiating sodium and calcium uptake in smooth muscles [17];

(b) cortisol could cause hypertension by increasing cardiac output, via an increase in circulating extracellular fluid [18]. Excess cortisol has also been linked to dyslipidemia and increased insulin levels, the combination of which can contribute to and hasten the atherosclerotic process [17]. Finally, cortisol may have an impact on the disease process through its interaction with SAM hormones such as epinephrine and norepinephrine [18].

Review of the above literature suggests that both the SAM and the HPA systems are involved in the disease process. Indeed, several researchers have indicated that it is the concerted, excessive activation of *both* the SAM and the HPA systems that results in pathological effects on the organism [14,19]. If we propose that a personality trait is involved in the development of CVD via its impact of physiological hyperresponsivity, it follows from the above that one should look at its relationship to both SAM and HPA reactivity to acute stress.

Thus, as noted above, although a consistent relationship between Type D personality and CVD has been noted, the pathways to disease have yet to be established. To date, there has been no published study relating Type D to cardiovascular or neuroendocrine reactivity as a potential explanatory construct for the observed relationship between disease and personality. As such, the purpose of the current study was to examine the association between Type D personality and reactivity to acute stress, using a stress protocol shown in our laboratory to affect both the SAM and the HPA systems [20]. A secondary aim of the current study was to relate the Type D construct to other personality and psychological factors known to be associated with markers of cardiovascular health: hostility, perceived social support, and defensiveness [21–23]. In addition, because hostility has been extensively studied as a personality risk factor for CVD, we wanted to examine whether Type D personality would emerge as a stronger predictor of reactivity. In other words, for a new personality risk factor to be useful and parsimonious, it should possess incremental validity, i.e., it should add to our ability to understand what contributes to hyperresponsivity, rather than overlapping with already existing constructs.

## Method

### *Participants*

Participants were 182 University of British Columbia undergraduates who took part in the study for course credit. The data from nine participants were excluded from the analyses due to experimenter error or equipment failure. The final sample consisted of 173 participants (86 male, 87 female, average age: 20.4). The ethnic composition of the sample was diverse as is typical for our student population (41% North American/European, 35% Chinese, 14% Other East Asian, 4% South-Asian, 6% Other). Almost half of the

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