RESEARCH LETTER

Impact of psychological distress on cardiovagal reactivation after a speech task

Cardiovascular disease (CVD) is more prevalent in individuals with Type-D personality(distressed)[1–3] who tend to avoid confrontation in social situations (social inhibition) and have a greater tendency to experience negative emotions (negative affectivity).[4] Although psychological distress is associated with increased risk of adverse cardiovascular outcomes and impaired heart rate (HR) modulation,[5,6] studies examining cardiac autonomic modulation in distressed individuals through heart rate variability (HRV) and baroreflex functioning during, and more importantly, after stress are scarce. Accordingly, we investigated blood pressure (BP), baroreflex sensitivity (BRS) and HRV responses before, during and after an interpersonal speech task (ST) in individuals with high distress scores (HD) and low distress scores (LD).

Type-D personality, also known as the distressed, is associated with increased likelihood of adverse cardiovascular outcomes including myocardial infarction and tends to be comorbid with major depressive disorder and anxiety.[3,7] In the general population distress also carries, in addition to a cardiovascular risk, a mental health burden as suggested by higher levels of depression and anxiety.[8] The biological mechanism underlying the link between increased CVD and psychological distress is unknown. However, it appears that autonomic deregulation may be implicated. Prior studies have documented blunted HR and impaired cardiovagal modulation in response to social stress in distressed individuals.[9] Martin et al.[10] documented that distressed individuals displayed lower HRV during experimental stress than non-distressed individuals, but they did not examine the immediate recovery period after stress. As cardiovascular recovery from stress is an important window of time when adverse cardiovascular events may occur,[11] and as recovery may be altered in those with increased psychosocial risk factors (anxiety, depression and stress),[12,13] it is reasonable to examine cardiac autonomic modulation in distressed individuals before, during and after stress. We hypothesized that individuals with HD would have impaired cardiovagal modulation and reduced BRS after a stressor.

In a cross-sectional design, 50 individuals were enrolled and grouped into HD or LD using a median split of baseline (BASE) distress scores from the Symptom Checklist-5 scale.[14] Participants were between the ages of 20 and 60 and had no history of hypertension or other chronic conditions. Following established guidelines for cardiovascular autonomic assessments,[15] participants were asked to abstain from caffeine, alcohol and strenuous physical activity for at least 24 h before testing. Testing was conducted during the same period each day (1600–1900 hours) in a quiet, dimly lit, temperature-controlled room (73 ± 2 °F). After instrument calibration, a 10-min resting period was instituted before collecting single lead electrocardiographs (ECG; similar to Lead II) and beat-by-beat finger BP (MP 150, NIBP-100 Biopac Inc., Goleta, CA, USA) measurements for subsequent HRV and BRS analysis. Brachial systolic BP (SBP) was used to calibrate beat-by-beat BP recorded via finger plethysmography.

This method is validated and has been shown to provide accurate measurement of BP when compared with intra-arterial BP.[16]

The ECG tracings were used to calculate the duration of intervals between heartbeats (RRI) and were automatically detected using commercially available software (WinCPRS, Turku, Finland). The RRI were inspected for artifacts, premature beats and ectopic episodes in order to calculate HRV parameters. The time domain total power (TP; global autonomic activity) as well as spectral components of the low frequency HRV (LF; 0.04–0.15 Hz; sympathovagal interplay[15] and baroreflex function[17]) and high frequency HRV (HF; 0.15–0.4 Hz; parasympathetic activity) components of the RRI were calculated by means of Fast Fourier transformation. Normalization, which is the expression of fractal components as a percentage of the TP, was used to exclude the influence of other fractal components and to control for the changes in TP because it is more appropriate to report LF and HF responses to stress in normalized units.[15] The BRS was calculated using the cross correlation method as previously described.[18]

Upon arrival, participants completed a BASE state affect questionnaire to assess levels of distress, State-Trait Anxiety Inventory (STAI), and depression (BDI; Beck Depression Inventory). Participants were seated for a 10-min rest period followed by 4-min BASE measurements. Thereafter, participants engaged in three 1-min interval speaking periods during which they were told to speak about their typical daily activities. Cardiovascular measurements were continuously gathered during the last 4-min of the ST and the following 3-min recovery period (REC). The rationale for using shorter windows of measurement (3–5 min) was based on prior studies using STs with similar measurement times to evaluate the impact of distress and psychosocial stress on cardiovascular reactivity and HRV.[19,20]

Shapiro–Wilk normality tests were used to examine absolute values for tested variables. Results indicated TP and BRS were not normally distributed and therefore a logarithmic transformation (Ln) was performed for these variables. T-tests revealed that HD ($n = 25$; male = 15) had higher STAI ($P < 0.001$) and BDI ($P < 0.001$) scores at the BASE and a higher level of reported state stress ($P < 0.05$) after the ST compared with LD ($n = 25$; male = 12) group (Table 1). To investigate cardiovascular and autonomic variable distress group changes during the experiment, 3 (time: BASE, ST and REC) by 2 (group: HD and LD) repeated measures analysis of covariance using least significant difference as alpha adjustment and that controlled for anxiety and depression scores were conducted as followed. Significant group-by-time interactions occurred for diastolic BP (DBP), mean arterial pressure (MAP), normalized low frequency (nLF), normalized high frequency (nHF) and LnBRS (see Table 1 and Figure 1). Follow up analyses to significant group-by-time interactions ($P < 0.05$) indicate that, during BASE, the only cardiovascular and autonomic variable difference between the distressed groups existed for DBP, which was significantly higher in HD ($P < 0.05$).

During ST, in comparison with LD, the HR responses were significantly lower (blunted) and DBP significantly higher ($P < 0.05$) for HD. Significant increases from BASE to ST occurred for SBP, DBP, MAP, nLF as well as a significant decrease in nHF, although these changes did not differ between the distress groups.

Table 1. Summarizes demographics, psychological status, and blood pressure responses to speech task between the individuals with LD and HD scores

Variables	LD (n = 25; males = 12)			HD (n = 25; males = 15)		
	BASE	ST	REC	BASE	ST	REC
Age (years)	37 ± 2	—	—	37 ± 2	—	—
Weight (kg)	88.7 ± 3.1	—	—	87.1 ± 5.0	—	—
Height (m)	1.78 ± 0.17	—	—	1.76 ± 0.18	—	—
BMI (kg m^{-2})	28.1 ± 1.3	—	—	28.8 ± 1.8	—	—
Distress	4.9 ± 0.1	—	—	12.8 ± 0.3[a]	—	—
Anxiety trait	14.1 ± 0.7	—	—	19.8 ± 1.1[a]	—	—
BDI	4.1 ± 1.0	—	—	13.7 ± 1.1[a]	—	—
HR (b.p.m.)	70 ± 2	74 ± 2[b]	69 ± 2	72 ± 2	73 ± 2	71 ± 2
SBP (mm Hg)	124 ± 3	126 ± 3[b]	125 ± 3	127 ± 3	131 ± 3[b]	131 ± 3
DBP (mm Hg)	78 ± 2	81 ± 2[b]	78 ± 2	82 ± 2[a]	87 ± 2[a,b]	85 ± 2[a,b]
MAP (mm Hg)	93 ± 2	96 ± 2[b]	93 ± 2	97 ± 2	102 ± 2[b]	100 ± 2[a]

Abbreviations: BASE, baseline; BMI, body mass index; BDI, Beck Depression Inventory; DBP, diastolic blood pressure; HD, high distress; HR, heart rate; LD, low distress; MAP, mean arterial pressure; REC, recovery; SBP, systolic blood pressure; ST speech task. Data are mean ± s.e. [a]$P < 0.05$ different from LD. [b]$P < 0.05$ different from BASE.
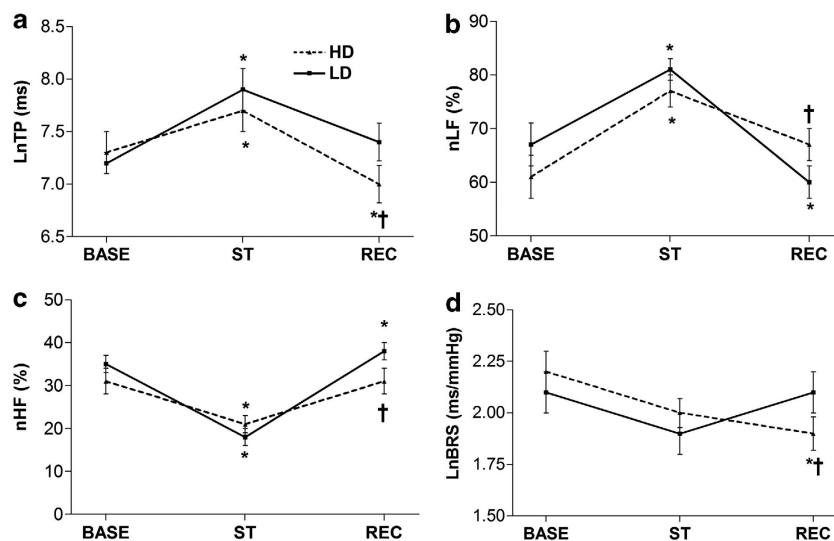

Figure 1. Summarizes cardiovascular autonomic modulation responses and baroreflex functioning in response to ST between individuals with LD and HD scores. Data are mean ± s.e.; BASE, baseline; ST, speech task; REC, recovery; (**a**) TP, total power: (**b**) nLF, normalized low frequency component of HR variability; (**c**) nHF, normalized high frequency component of HR variability; (**d**) BRS, baroreflex sensitivity. *$P < 0.05$ different from BASE. †$P < 0.05$ different from LD.

During REC, HD had significantly ($P < 0.05$) higher DBP, MAP, nLF and LnBRS than LD. Compared with BASE values, the LD group displayed significant increases in nHF ($P < 0.05$) and decreases in nLF ($P < 0.05$), but the HD group reported significant increases in DBP ($P < 0.05$) and nLF ($P < 0.05$) and decreases in LnBRS ($P < 0.05$).

The present study found an increase in nHF after ST in the LD group that was not observed in the HD group. In addition, we did not find differences between the groups in cardiac autonomic modulation at rest or during ST, but we did find differences in BRS during the REC period. Findings suggest that psychological distress leads to an attenuated cardiovagal reactivation and sympathetic hyperactivity during REC from an acute stressor. Hence, distress may carry increased cardiovascular risk by attenuating vagal reactivation and evoking sympathetic stimulation during the REC period after stress. These responses seem to be mediated psychologically by negative affectivity and physiologically by increased MAP (distention pressure) and attenuated BRS.

Individuals with HD did not demonstrate vagal rebound, an increase in parasympathetic activity above baseline levels after stress,[21] but did maintain residual sympathetic activity as illustrated by nHF fully recovering but nLF remaining elevated after the ST. Our results are somewhat in agreement with those of Von Kanel et al.[22] who reported lower HR recovery, a marker of cardiovagal reactivation, in patients with congestive heart disease with Type-D personality. These responses could be attributed to negative affectivity and stress regarding social interactions in distressed individuals[4] as demonstrated here by increased stress reported post-ST in HD. Physiologically, distress may induce impaired cardiovagal reactivation after the ST due to decreased BRS.[23] As impaired cardiovagal reactivation and decreased BRS are associated with increased risk of arrhythmias,[24] distress induced autonomic deregulation and vascular dysfunction may be potential biological factors linking increased cardiovascular risk to psychological distress. It is also worth noting that in HD individuals there was increased DBP across all time frames along

with a blunted HR response during the ST. The finding of blunted HR responses in HD in the present study is in agreement with previous reports suggesting impaired cardiovagal modulation in response to stress in individuals with HD scores.[9,25] Similarly, individuals with high depressive and anxiety symptoms have blunted responses in BP and HR during psychological stress in addition to increased perceptions of stress.[26] In fact, it has previously been shown that increased acute anxiety to a stressor, which was found in the HD group, is likely mediated via increased social inhibition.[26,27] Together, prior studies and our results suggest that distress, which carries high negative affectivity and social inhibition, may lead to higher sympathetic activity and decreased parasympathetic tone especially during acute stress and the early recovery period after the stress when cardiac events are more prevalent.

The results of the present study suggest that HD individuals were unable to appropriately recover after the ST due to sustained sympathetic activity and reduced cardiovagal modulation, likely owing to a reduced BRS. Distress seems to affect cardiovagal activity by increasing levels of perceived stress after the ST. In conclusion, individuals with HD demonstrated hyperactivity and attenuated recovery to a stressor suggesting dysautonomia as a potential biomarker linking HD and the development of CVD. Prospective studies evaluating cardiovascular autonomic modulation and arterial stiffness using physiological and psychological stressors in individuals with Type-D personality are warranted.

What is known about this topic?
- Psychological distress is associated with increased cardiovascular risk.
- Distressed individuals display negative affectivity and decreased HRV to stress.
- Type-D personality increases HR recovery in patients with congestive heart failure.

What this study adds?
- Psychological distress leads to an attenuated cardiovagal reactivation and sympathetic hyperactivity during recovery after acute stress.
- Psychological distress decreases BRS after stress.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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