Revista Brasileira de Psiquiatria. 2017;39:110–117
Associação Brasileira de Psiquiatria
doi:10.1590/1516-4446-2016-1935

ORIGINAL ARTICLE

Symptoms of anxiety and depression are related to cardiovascular responses to active, but not passive, coping tasks

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Objective: Anxiety and depression have been linked to blunted blood pressure (BP) and heart rate (HR) reactions to mental stress tests; however, most studies have not included indices of underlying hemodynamics nor multiple stress tasks. This study sought to examine the relationships of anxiety and depression with hemodynamic responses to acute active and passive coping tasks.
Methods: A total of 104 participants completed the Hospital Anxiety and Depression Scales and mental arithmetic, speech, and cold pressor tasks while BP, HR, total peripheral resistance, and cardiac output (CO) were assessed.
Results: After adjustment for traditional risk factors and baseline cardiovascular activity, depression scores were negatively associated with systolic BP, HR, and CO responses to the mental arithmetic task, while anxiety scores were inversely related to the systolic BP response to mental arithmetic.
Conclusion: High anxiety or depression scores appear to be associated with blunted cardiac reactions to mental arithmetic (an active coping task), but not to the cold pressor test or speech tasks. Future research should further examine potential mechanisms and longitudinal pathways relating depression and anxiety to cardiovascular reactivity.
Clinical trial registration number: TCTR20160208004

Keywords: Cardiovascular reactivity; active coping; passive coping; anxiety; depression; mental stress test

Introduction

Depression and anxiety have been related to cardiovascular disease (CVD). Further, the association between depression, anxiety, and CVD is complex, and the mechanisms linking them have not been fully elucidated. One pathway linking psychological factors (specifically, depression and anxiety) to CVD and cardiovascular mortality that has received attention is that of cardiovascular reactivity.[1,2] However, the relationship between anxiety and depression and cardiovascular reactivity has been inconsistent. Early studies supported the hypothesis that exaggerated responses to stress were associated with depression, and might therefore account for the raised risk of CVD in people with depression.[3] In particular, a meta-analysis of 11 studies conducted from 1887 to 2001 suggested that there was a small to moderate positive correlation between cardiovascular reactivity and depressive symptoms; however, these effect sizes did not reach conventional criteria for statistical significance.[4] Other, more recent studies have provided limited support for this hypothesis. For example, Pointer et al. reported that state anxiety was positively associated with blood pressure (BP) responses to cold pressor and anger recall in 50 healthy adults.[5] In another study of 60 healthy young women who completed a speech task, depressive symxptoms were positively associated with BP, heart rate (HR), and cardiac output (CO) responses.[3] Thus, these studies suggested that there may be a weak, positive relationship between depressive/anxiety symptoms and cardiovascular reactivity.

Other studies have reported no differences in sympathetic activity between depressed and non-depressed participants.[6,7] For example, Taylor et al. examined cardiovascular reactions to the Trier Social Stress Test. Fifty-nine older depressed patients and 20 non-depressed patients matched for age and cardiovascular risk were tested. The study revealed no significant differences in BP reactivity between depressed and non-depressed individuals.[7] One possible explanation for the lack of association is the small sample size and consequent lack of power to detect possible effects, although studies of a similar size reported significant positive correlations.[3,5]

Finally, other studies have found that anxiety and depressive symptoms are associated with blunted physiological reactivity. In the largest study to date, Carroll et al.[8] assessed depressive symptoms in 1,608 adults. Depression scores were negatively, but modestly, associated with systolic blood pressure (SBP) and HR reactions to the Paced Auditory Serial Arithmetic Test

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Submitted Feb 08 2016, accepted Jun 01 2016, Epub Nov 07 2016.

(PASAT).[8] In a second, large recent study, de Rooij et al. reported that 725 healthy Dutch participants with high depressive or anxiety scores on the Hospital Anxiety and Depression Scale (HADS) exhibited more marked SBP and HR reactions to psychological stressors (i.e., Stroop, mirror-tracing, and speech tasks) than those with low depression and anxiety scores.[9] Young et al. assessed trait anxiety in 832 healthy participants, and found that those with higher levels of trait anxiety had lower cardiovascular reactivity than participants with low levels of anxiety.[10] Thus, a number of studies do not support a hyper-reactivity hypothesis; rather, they suggest that blunted physiological reactivity is related to anxiety and depression. Indeed, a meta-analysis by Chida & Hamer of studies from 1950 to 2008 found that anxiety and depressive mood were associated with decreased cardiovascular reactivity.[11] In short, larger recent studies have reported negative relationships of symptoms of anxiety and depression with cardiovascular reactivity.

However, to date, few published studies have examined hemodynamic responses (including CO and total peripheral resistance [TPR]) to mental stress tests in relation to anxiety and depressive symptoms in healthy volunteers, and the pattern of such findings has not been consistent. For example, Matthews' findings suggest a vascular mechanism underpinning relationships between depression and blood pressure reactivity,[12] whereas Light's findings suggest a myocardial mechanism.[3] In addition, these studies used a variety of different measures of depression (including clinical diagnosis and questionnaires) and had a limited range of active coping tasks.

Moreover, most published studies have examined cardiovascular responses to a single task. Only two published studies investigated responses to both active and passive coping tasks in healthy participants.[5,13] Pointer et al. reported that state anxiety was positively associated with BP responses to both active and passive coping tasks (anger recall and cold pressor) in 50 adults.[5] In contrast, Schwerdtfeger & Rosenkaimer found that BP responses to an active coping task (public speaking) were negatively associated with depressive symptoms, whereas BP responses to a passive coping task (cold pressor test) were not related to depressive symptoms.[13] However, only 55 people participated in the study, nine of whom were excluded from analysis of the cold pressor task, i.e., the sample was smaller than for the active coping task analysis and thus relatively underpowered to detect effects.

Recently, it has been suggested that cardiovascular reactivity may be related to negative health outcomes and behaviors.[8,14] One proposed mechanism is motivation; blunted cardiovascular reactivity may be a physiological marker of motivational dysregulation shared by people with depression and anxiety.[15] Carroll et al. found lower cardiovascular reactivity to be related to PASAT performance scores and increased symptoms of anxiety and depression.[8] Similarly, Salomon et al. found that participants with major depression rated speech and mirror-tracing tasks as more demanding, threatening, and stressful than participants without major depression.[16] However, subjective ratings did not mediate the relationship between depression and blunted reactivity. Both studies measured cardiovascular responses to active coping tasks alone. Of note, the present study is the first to explore the relationships between hemodynamic responses (cardiac and vascular patterns) to both active and passive coping tasks and HADS depression and anxiety scores.

In brief, there are inconsistent findings regarding the relationship between cardiovascular reactions to active and passive coping tasks and symptoms of anxiety and depression. Thus, the objectives of the present study were: to examine the associations of anxiety and depression with hemodynamic reactions to both active and passive coping tasks; and to examine whether these associations remained after statistical adjustments for performance and self-reported stress.

Materials and methods

Participants

Participants were recruited from staff and students at the University of Derby and from the local community. One hundred and four people aged 18-65 years agreed to participate. Participants had no known history of CVD or surgery (including prior myocardial infarction and percutaneous coronary intervention or coronary artery bypass graft), no history of neurological disorder, no peripheral vascular disease, no history of stroke or symptomatic cerebral ischemia, no chronic renal failure or liver disease, and no history of diagnosed psychiatric problems. Participants who had a fever (high temperature) prior to or during experimental tests were also excluded from the sample. Participants who reported having a history of any circulatory disorder, including Raynaud's disease were also excluded. The study was approved by the Ethics Committee of the University of Derby; all participants gave informed consent before the study began.

Apparatus and procedure

Participants were asked to complete a series of medical and family history questionnaires, which included traditional risk factors, namely family history of CVD, current cigarette smoking, and hypertension status. History of hypertension status was defined as high BP (SBP $>$ 140 mmHg and/or diastolic blood pressure [DBP] $>$ 90 mmHg) or taking any antihypertensive medication. In addition, participants were asked to complete the HADS to assess symptoms of anxiety and depression.[17]

Prior to arrival at the laboratory, the participants were asked to refrain from drinking coffee, tea, and other caffeinated beverages, as well as from smoking, for at least 2 hours prior to the study. Upon arriving for the laboratory session, each participant was seated upright in a comfortable armchair with feet on the floor. The experimental session was initiated by a 15-minute rest period while wearing a Portapres continuous BP monitor (Finapres Medical Systems, Amsterdam). The arterial pressure signal was measured through appropriate-sizes

finger cuffs wrapped around the middle phalanx of the middle and fourth fingers of the participant's left hand and alternated every 30 minutes. BP, HR, CO, and TPR were recorded at baseline and continuously during the tasks. Following the baseline period, participants were engaged in three mental stress tests, a mental arithmetic task, a speech task, and a cold pressor test; task order was randomized and counterbalanced. A rest period of 8 minutes between each task was enforced.

The present study focused on active and passive coping tasks. According to Obrist, active coping tasks are psychological stressors that demand attention and vigilance or mental effort, and primarily elicit cardiac responses. In contrast, passive coping tasks are defined as those during which an individual has no control over the outcomes of the situation or no ability to control the situation; these primarily elicit vascular responses.[18] Therefore, mental arithmetic tasks and speech tasks were represented as active coping tasks, and the cold pressor test was classified as a passive coping task.

Mental arithmetic

For a 5-minute serial subtraction task, participants were requested to subtract the number 13 repetitively starting from 1,079 as quickly and accurately as possible while mistakes were corrected by the experimenter. During this task, a metronome was set at a frequency of 2 Hz to elicit time pressure. Performance scores (the number of correct responses) were recorded.

Speech task

Participants were instructed to read and prepare a speech on an assigned topic (i.e., a salesman who refused to honor an advertised sale price) for 2 minutes. They were then asked to speak for 3 minutes on this topic. A video camera was set up to record during this task; participants looked at the camera and were asked to talk continuously. Participants who faltered were encouraged to continue.

Cold pressor test

A tank of circulating cold water at 7°C was used for the cold pressor test. The participants were instructed to try to hold their hand in the cold water for as long as possible up to 2 minutes. Pain tolerance (defined as the total time of hand immersion in water) was recorded.

After all three tasks were completed, participants were asked to rate the perceived stress of each task on a 10-point Likert scale, with zero corresponding to not stressful and 10 corresponding to very stressful.

Data reduction and analyses

Baseline values were computed by averaging the values from 5 to 13 minutes of the rest period. Cardiovascular reactions were calculated by subtracting baseline cardiovascular values from task levels for each cardiovascular index: SBP, DBP, HR, CO, and TPR.

Correlations of HADS depression and anxiety scores with traditional risk factors and cardiovascular responses were calculated. To evaluate the relationships between depression and anxiety scores and cardiovascular reactivity, multiple linear regression was applied. In the first models, traditional risk factors (sex, age at entry, body mass index [BMI] at entry, family history of CVD, current cigarette smoking) and baseline cardiovascular activity measurements were entered at step one. Depression or anxiety scores were then entered at step two. In the second set of models, the traditional risk factors and cardiovascular activity measurements were entered in step one, performance scores and self-reported perceived stress were entered in step two, and HADS depression or anxiety scores were then entered in step three.

Analyses then focused on the partial correlations between HADS anxiety and depression scores after controlling for self-reported perceived stress, performance (number of correct answers for the mental arithmetic task). and pain tolerance (total amount of time each participant's hand remained immersed in cold water, measured with a stopwatch).

Results

Demographic and HADS depression and anxiety scores

The demographic and HADS anxiety and depression scores of the sample are presented in Table 1. Using cutoff scores of 8 for anxiety and depression as indicators of elevated risk,[17] only six participants had elevated depression scores, whereas nearly half the sample (n=47) had elevated anxiety scores.

Relation between cardiovascular reactivity and HADS anxiety and depression scores

Negative associations were found between HADS depression scores and SBP, HR, and CO reactions to the mental

Table 1 Demographic data, self-perceived stress, and HADS scores

Characteristic	Result
Sex, n (%)	
Male	45 (43.27)
Female	59 (56.73)
Current cigarette smoking, n (%)	33 (31.73)
Family history of CVD, n (%)	32 (30.77)
Age, years	31.52 (11.55)
BMI, kg/m^2	25.53 (5.09)
Performance score on mental arithmetic	23.89 (15.78)
Pain tolerance to cold pressor (seconds)	86.19 (41.69)
Self-perceived stress	
Mental arithmetic task	6.28 (2.38)
Speech task	5.20 (2.61)
Cold pressor test	6.68 (3.03)
HADS anxiety score	7.62 (3.85)
HADS depression score	3.38 (2.86)

Data presented as mean (standard deviation), unless otherwise specified.
BMI = body mass index; CVD = cardiovascular disease; HADS = Hospital Anxiety and Depression Scale.

Table 2 Bivariate correlations between HADS scores and cardiovascular reactivity

	Anxiety	Depression
Mental arithmetic task		
SBP response	-0.245*	-0.206*
DBP response	-0.082	-0.056
HR response	-0.153	-0.263†
CO response	-0.140	-0.349‡
TPR response	-0.043	0.088
Speech task		
SBP response	-0.096	-0.086
DBP response	-0.053	-0.153
HR response	0.138	-0.103
CO response	-0.090	-0.162
TPR response	-0.126	0.030
Cold pressor test		
SBP response	-0.139	0.016
DBP response	-0.043	0.005
HR response	0.108	-0.075
CO response	-0.146	-0.159
TPR response	-0.113	-0.064

CO = cardiac output; DBP = diastolic blood pressure; HADS = Hospital Anxiety and Depression Scale; HR = heart rate; SBP = systolic blood pressure; TPR = total peripheral resistance.
* $p < 0.05$; † $p < 0.01$; ‡ $p < 0.001$.

arithmetic task. HADS anxiety scores were negatively associated with SBP responses to mental arithmetic. These correlations are displayed in Table 2.

Next, hierarchical regression was performed to determine the relationships between depression and anxiety scores and cardiovascular reactivity; these are shown in Table 3. HADS anxiety scores were negatively associated with SBP reactions to the mental arithmetic task. Further, HADS depression scores were inversely associated with SBP, HR, and CO responses to this task.

On analysis of the pattern of hemodynamic responses to speech tasks (preparation and delivery) and HADS depression and anxiety scores, bivariate correlations suggested that neither the speech task nor the cold pressor test were associated with anxiety or depression scores ($p > 0.05$). Therefore, regression analyses of cardiovascular reactivity were not performed.

Since motivation has been posited as one possible pathway linking depression to blunted cardiovascular reactivity,[8] our analyses then focused on the correlations between HADS anxiety and depression scores and cardiovascular reactions to mental arithmetic after controlling for self-reported perceived stress and performance (number of correct responses on the mental arithmetic task). Hierarchical regression showed that, after controlling for traditional risk factors, baseline cardiovascular activity, self-reported perceived stress, and performance, lower SBP, HR, and CO responses to the mental arithmetic task were still associated with higher depression scores (β = -0.232, β = -0.260, and β = -0.338). In addition, the hierarchical regression between anxiety scores and SBP reactions to the mental arithmetic task remained significant after adjustment for self-reported perceived stress, performance, traditional risk factors, and baseline SBP activity (β = -0.221).

Finally, associations of pain tolerance with anxiety and depression have been reported.[19] However, the present study found no relationship between pain tolerance and depression or anxiety scores (r = -0.085, p = 0.389, and r = 0.064, p = 0.521, respectively). In addition, previous studies of the cold pressor test have found that BP generally peaks within 2 minutes of immersion.[20,21] A subgroup analysis that included only those participants who were able to withstand more than 90 seconds of immersion (n=60) revealed that CO responses to the cold pressor test were negatively associated with depression and anxiety scores (r = -0.307, and r = -0.407, respectively). Further, hierarchal regression revealed that CO responses to the cold pressor task were associated with anxiety scores (β = -0.404, p = 0.002) and depression scores (β = -0.274, p = 0.053) after adjustment for traditional risk factors and cardiovascular activity, although the relationship of depression scores did not reach conventional criteria for significance ($p < 0.05$).

Discussion

The primary objective of the present study was to examine the relationships between HADS-measured symptoms of depression and anxiety and cardiovascular responses to mental arithmetic, speech, and cold pressor tasks. These tasks were chosen as they represent different types of stimuli that elicit different cardiovascular response patterns associated with active and passive coping.[22] The main findings were that depression scores were negatively associated with SBP, HR, and CO responses to the mental arithmetic task. Anxiety scores were only related to SBP responses to mental arithmetic; again, there was an inverse relationship. Further, the relationships between depression scores and SBP, HR, and CO responses to the mental arithmetic task, as well as SBP responses to the mental arithmetic task and anxiety scores, remained significant after adjustment for traditional risk factors and baseline cardiovascular activity. Finally, these negative relationships remained significant after adjusting for traditional risk factors and baseline cardiovascular activity, in addition to self-reported perceived stress and performance scores.

With respect to anxiety and depressive symptoms, participants had high trait anxiety, whereas few had depression. Age might explain the large number of participants with anxiety symptoms. The mean age of participants with anxiety symptoms was 28.92 years, vs. 33.75 years for participants with no anxiety symptoms. It has been reported that older adults may have lower levels of stress and anxiety than younger adults, as older individuals have had more experiences and learned to cope with them.[23]

In the present study, participants with higher depression scores exhibited blunted SBP, HR, and CO responses to the mental arithmetic task; the relationship with these responses remained after statistical adjustment for traditional risk factors and baseline cardiovascular activity. In contrast, depression scores were not associated with cardiovascular reactions to the speech task or cold pressor test. These findings are consistent with a growing body of

Table 3 Summary of hierarchical regression outcomes between SBP responses, HR reactions, and CO reactions to mental arithmetic and HADS scores

	β	T	R^2	F	ΔR^2	ΔF
SBP responses						
Step 1			0.111*	2.022	0.111	2.022*
Age	0.040	0.408				
Sex†	-0.165	-1.556				
BMI	-0.005	-0.050				
FH‡	0.238	2.426§				
Smoking‖	0.112	1.102				
Baseline SBP	-0.045	-0.449				
Step 2						
Step 2.1			0.158	2.583§	0.047	5.400§
Anxiety	-0.229	-2.324§				
Step 2.2			0.159	2.594§	0.048	5.468§
Depression	-0.227	-2.338§				
HR reactions						
Step 1			0.105	1.904	0.105	1.904*
Age	-0.130	-1.255				
Sex†	0.184	1.730*				
BMI	-0.088	-0.833				
FH‡	0.126	1.277				
Smoking‖	-0.056	-0.546				
Baseline HR	-0.235	-2.257§				
Step 2			0.167	2.758§	0.062	7.161¶
Depression	-0.258	-2.676¶				
CO reactions						
Step 1			0.091	1.628	0.091	1.628
Age	-0.119	-1.184				
Sex†	-0.020	-0.179				
BMI	0.139	1.246				
FH‡	-0.033	-0.332				
Smoking‖	0.007	0.069				
Baseline CO	-0.320	-2.819¶				
Step 2			0.194	3.302¶	0.103	12.214¶
Depression	-0.332	-3.495¶				

BMI = body mass index; CO = cardiac output; FH = family history of cardiovascular disease; HR = heart rate; SBP = systolic blood pressure.
* $p < 0.1$; § $p < 0.05$; ¶ $p < 0.01$.
† Male = 1; female = 2.
‡ Positive = 1; negative = 0.
‖ Current cigarette smoking status: smoking = 1; non-smoking = 0.

evidence that attenuated or blunted cardiovascular reactions to active coping tasks are associated with depressive symptoms.[8,16,24] Although the relationship between CO responses and symptoms of depression has been inconsistent,[3,12] studies comparing clinically depressed patients with non-depressed individuals have consistently found that the magnitude of CO reactions is smaller in the former. For example, Ehrenthal et al. compared cardiovascular reactivity to anger recall and mental arithmetic tasks in 25 individuals with severe depression without heart disease and 25 non-depressed participants.[25] The depressed participants exhibited overall reductions in HR, BP, and cardiac index. Increases in TPR index were also found in the depression groups, but with no significant difference between groups. In another study, clinically depressed individuals experienced significantly reduced SBP, HR, and CO reactivity during exposure to two stressors (speech and mirror tracing) than non-depressed individuals.[16] There were no differences in DBP or TPR responses to the stressors. Therefore, the present findings are consistent with previous studies of depressed patients and corroborate the hypothesis of a beta-adrenergic mechanism.[26]

Regarding anxiety, the present study found only one small negative correlation after controlling for traditional risk factors and baseline cardiovascular activity: anxiety scores were associated with blunted SBP responses to the mental arithmetic task. However, anxiety scores were unrelated to cardiovascular reactions to the speech and cold pressor tasks. Other investigators have also found negative correlations between anxiety scores and SBP responses to mental arithmetic tasks.[10] Symptoms of anxiety may influence cardiac function via multiple pathways. For example, Nesse et al. found decreased beta-adrenergic receptor sensitivity and decreased stimulation of the beta-adrenergic system in patients with panic disorders.[27] In addition, decreases in the number of lymphocyte beta-adrenergic receptors have been reported in participants with high trait anxiety scores.[28] Therefore, the present findings support the hypothesis of beta-adrenergic downregulation in people with high levels of anxiety.

One possible explanation for the blunted responses seen in individuals with high anxiety and depression scores is that such subjects may be less engaged in the task or less motivated to perform well.[29,30] The present study found that, after controlling for performance (where possible) or self-reported perceived stress, negative relationships of depression and anxiety scores with cardiovascular reactions were still observed. For example, the association between depression scores and CO reactions to the mental arithmetic task remained significant after controlling for traditional risk factors, baseline CO, self-reported perceived stress, and performance scores; there was little evidence that performance or self-reported stress mediated depression-cardiovascular reactivity relationships, possibly because of the small effect sizes observed. Other studies have also found that relationships between depression scores and reactivity remained after adjusting for performance[8] and perceived stress.[9,16] In the present study, self-reported perceived stress was higher for the mental arithmetic task than the speech task; neither differed from the ratings assigned to the cold pressor test. The relatively low perceived stress ratings for the speech task might partially explain why cardiovascular responses to this task were not related to depression or anxiety scores. However, it is possible that the measures used in our study (performance and stress) are poor proxies of effort and motivation and, so, do not mediate relationships between cardiovascular reactivity and psychological factors. Alternatively, other mechanisms might better account for these relationships. Phillips et al. reviewed the potential mechanisms underlying blunted cardiovascular reactivity and negative health outcomes and suggested a number of possible mechanisms, including lower effort on the part of the participant, reduced awareness or perception of stress, and motivational dysregulation within the brain.[14] Ginty et al. compared neural activation differences in participants with exaggerated and blunted cardiac reactors,[31] and found that, during a functional magnetic resonance imaging (fMRI) testing session, exaggerated cardiac reactors demonstrated significant increases in HR during the PASAT, whereas blunted reactors had no change in HR. Further, blunted reactors had reduced activation in the anterior midcingulate cortex and insula compared to exaggerated reactors during a stress test, as well as greater deactivation in the amygdala and posterior cingulate.

Cardiovascular responses to the mental arithmetic task, an active coping task, were associated with anxiety and depression scores after adjusting for baseline cardiovascular activity and traditional risk factors, but there were no relationships with any responses to the passive coping task (cold pressor test). Pain tolerance may explain this. The present study found that CO reactions to the cold pressor test were associated with anxiety scores after controlling for baseline cardiovascular reactivity and traditional risk factors only in those 60 participants who withstood hand immersion in cold water for more than 90 seconds. This suggests that pain tolerance may offer a simple explanation for cardiovascular responses to the cold pressor test in participants who are able to withstand the test for more than 90 seconds. However, the small size of this subgroup of participants should be considered. A plausible explanation for the lack of associations with the speech task is that this task might elicit a mixed alpha- and beta-adrenergic response, as the hemodynamic responses observed revealed a more mixed pattern. Since this task involved both speech preparation and speech delivery, participants may have been coping passively during preparation.[32,33] However, there were no significant associations of cardiovascular responses to each element of the speech task (preparation and delivery) with depression and anxiety scores. Thus, it is unclear why cardiovascular responses to the speech task were unrelated with depression and anxiety when other studies have indicated a negative correlation.[13,16]

Active coping tasks have been shown to involve a beta-adrenergic mediated pattern of responses and increase BP via central mechanisms. Further, increases in catecholamines, HR, SBP, stroke volume, and CO or minor increases in DBP and TPR are elicited via sympathetic nervous system responses. Passive coping tasks, conversely, involve vasoconstriction through alpha-adrenergic receptor stimulation, stimulating noradrenaline release and resulting in changes in DBP and TPR.[34,35] Of note, the active task was associated most markedly with myocardial reactivity patterns, consistent with a beta-adrenergic mechanism, whereas the passive coping task was associated with vascular reactivity patterns, consistent with an alpha-adrenergic mechanism.[36] Therefore, the present study demonstrated that specific coping strategies may be related to responses to active tasks and involve an active (myocardial) coping mechanism.

Cardiac output responses to mental arithmetic were negatively associated with depression scores; in contrast, TPR responses were not associated with anxiety and depression scores. Interestingly, this finding is consistent with two studies that compared hemodynamic reactivity in depressed and non-depressed individual groups.[13,16] Salomon et al. found that participants with major depressive disorders had larger increases in SBP, HR, and CO during speech tasks than non-depressed participants.[16] Previous studies have also demonstrated relationships between depression or anxiety and reactivity, and suggested a role for the sympathetic nervous system.[37,38] For example, several studies have shown that participants with depression and anxiety have fewer beta-adrenergic receptors.[22,23,39] Yu et al. found that mood states (tension-anxiety, depression-dejection, and anger-hostility) were associated with downregulation of beta-adrenergic receptors in individuals without a clinical diagnosis of depression or anxiety.[39] Responses to active coping tasks have been extensively characterized by relatively large increases in CO, indicative of a beta-adrenergic mediated pattern of responses via central mechanisms.[40] The present study provides further evidence for the involvement of blunted beta-adrenergic receptor responsiveness in participants with high anxiety and depression scores, as these participants showed a smaller magnitude of CO response (a marker of beta-adrenergic responsiveness) to the active coping task.

The present study has a number of limitations. First, causality cannot be inferred, as the design was cross-sectional. To date, only one prospective study has examined relationships between anxiety/depression and cardiovascular reactivity over time. Phillips et al. examined cardiovascular reactivity and symptoms of depression and anxiety and reassessed HADS depression and anxiety scores after 5 years of follow-up.[41] They found HR reactions to the PASAT were negatively associated with subsequent depressive symptoms. The small effect sizes observed in this and other studies suggests that large-scale investigations are needed to examine the contribution of possible mediating variables.

This was the first study to investigate the relationships of cardiovascular reactivity with depression and anxiety in different types of tasks (namely, a mental arithmetic task and a speech task, which involve the beta-adrenergic system, and a cold pressor test, which disturbs the alpha-adrenergic system) with measures of both myocardial and vascular responses. We conclude that high levels of anxiety or depression symptoms appear to be associated with blunted myocardial reactions to some active coping tasks (mental arithmetic), but not to passive coping tasks, implicating beta-adrenergic mechanisms.

Acknowledgements

The authors would like to thank Neale Samways for technical support. We also extend our thanks to the students and staff from the University of Derby for participating in this study.

Disclosure

The authors report no conflicts of interest.

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