Anxiety and cardiovascular reactivity in the Tecumseh population

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Objective **Increased cardiovascular reactivity has been proposed to be a critical mediator in the development of hypertension and cardiovascular disease. The personality factors associated with cardiovascular reactivity are still subject to debate. The studies reported here were undertaken to examine the relationship between trait anxiety and cardiovascular stress reactivity in a community-based sample (Tecumseh).**

Design and methods **All studies were carried out in an outpatient setting. Cardiovascular reactivity to isometric handgrip and mental arithmetic was assessed and recorded by automatic blood pressure monitoring in 832 subjects aged 19–41 years. Spielberger trait and state anxiety measures were collected immediately before the stressors were applied.**

Results **No differences in baseline heart rate, systolic or diastolic blood pressure were observed across anxiety categories. There was a clear negative correlation between trait anxiety and cardiovascular reactivity to mental arithmetic. The pattern was less clear in response to isometric handgrip.**

Conclusions **These results suggest that individuals with high trait anxiety demonstrate reduced cardiovascular reactivity while those with low trait anxiety demonstrate increased reactivity, whereas the opposite might have been expected. *J Hypertens* 1998, 16:1727–1733**

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Introduction

Anxiety disorders affect 24.9% of the population at some time in their lives [1]. While some psychiatric disorders, such as schizophrenia, clearly represent a discontinuity from normal, everyday experiences, others such as anxiety disorders might be viewed as a normal trait or emotion that is excessive for the current situation. Indeed, the definition of anxiety disorders and the boundary between disorder and normality has changed over time. Therefore, examining anxiety as a trait rather than a disorder may help to understand the impact of anxiety on a number of life spheres that are not always captured by the current definitions of anxiety disorders. As originally defined, anxiety was a universal response to a threatening situation. Over time, it was realized that some individuals showed a hyper-response to emotional stimuli, a trait labeled neuroticism by Eysenck and trait anxiety by Spielberger. Trait anxiety, as measured by the Spielberger State Trait Anxiety Index (STAI) taps into a constellation of anxiety proneness and personality variables, including low self esteem, low self-confidence and increased vulnerability to anxiety in situations that involve being evaluated by others [2]. In contrast, state anxiety measures the subjective feelings of tension, worry, apprehensiveness and autonomic arousal [2].

Given the presumed relationship between state anxiety and autonomic arousal, a number of studies have examined the relationship between anxiety and blood pressure in hypertensive individuals, and found positive correlations with state anxiety and systolic blood pressure [3–5]. No relationship between state anxiety and diastolic blood pressure has been found [2]. The relationship between trait anxiety and blood pressure is similarly inconsistent. Trait anxiety was not consistently elevated in hypertensives in a number of studies [6–9]. A recent meta-analysis by Jorgensen *et al.* [10] found that negative affect (similar to trait anxiety) was positively associated with blood pressure in older adults but negatively associated in younger adults. Furthermore, the direction of the relationship was also influenced by whether the subjects were aware of their hypertension or not. Likewise, although personality variables such as anger, hostility and defensive coping may predispose individuals to coronary

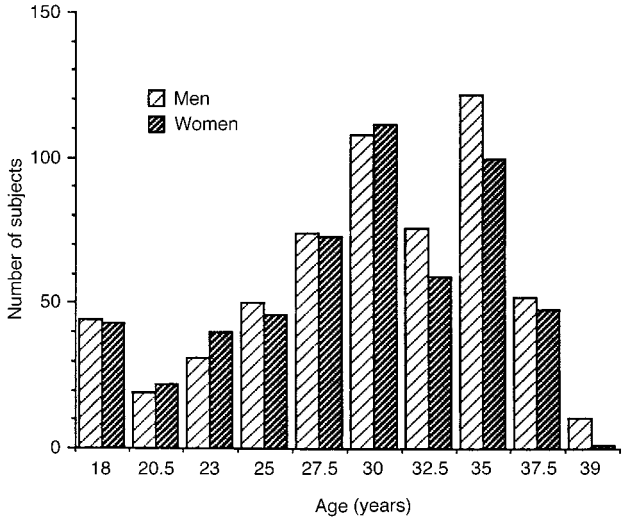
disease, trait anxiety has not been related to the risk of coronary heart disease in any studies [2].

Increased autonomic reactivity to stress has been proposed to be a critical mediator in the development of coronary heart disease and hypertension. It has been hypothesized that increases in blood pressure induced by repeated, exaggerated stress eventually lead to the development of more chronic hypertension (review in [11]). Studies in subjects with borderline hypertension have shown clearly that sympathetic activation is involved in the development of hypertension [12–14]. The longitudinal population-based study of blood pressure and cardiovascular disease in Tecumseh, Michigan, has collected data on stress reactivity, a number of lifestyle variables and trait anxiety on 832 adults aged 19–41 years. The present cross-sectional analyses examine the relationship of state and trait anxiety to demographic variables and the stress reactivity of blood pressure and heart rate.

Subjects and methods

Data were collected in 1986 through 1989. All studies were approved by the University of Michigan Institutional Review Board and informed consent was obtained from the participants. The sample consisted of 432 men and 400 women aged 19–41 years. The age distribution by sex is shown in Figure 1. Subjects were excluded from the study if they were pregnant or within 6 months of delivery, were taking antihypertensive medications or had a medical illness associated with hypertension or coronary heart disease. Blood pressure reactivity studies were conducted in a clinic setting using an automatic blood pressure monitor (Marshall 92 oscillometric sphygmomanometer) with a digital display and readings were manually recorded. All investigators involved in recording blood pressure were blind to the purpose of these studies.

Fig. 1



Distribution of the Tecumseh sample by age and sex. The sexes were closely balanced for age, with a mean age of 32 years.

State and trait anxiety measures were recorded in the same session as the stress reactivity studies. These measures were obtained with the State Trait Personality Index, form X-2, which contains 10 items on trait anxiety and 10 items on state anxiety. The state trait anxiety inventory provides reliable and brief scales for assessing anxiety in research and clinical settings. Separate state and trait anxiety factors have been identified by factor analysis in a number of studies [2]. The scale has been validated in over 6000 high school and college students as well as 600 medical, surgical and psychiatric patients [2]. This scale has been used extensively in experimental investigations [2] over the past 25 years. Data were also collected at the initial interview on smoking habits, alcohol use and exercise.

Upon arrival at the clinic, the subjects rested for 20 min and then three baseline measurements were taken of blood pressure and heart rate. The subjects were next instructed to serially subtract 13 from 1079. If a mistake was made, the subject returned to the last correct answer and began to subtract 13 serially again. This task went on for 5 min. Blood pressure and heart rate were recorded during this mental arithmetic. After a 20 min rest to re-establish baseline, the subjects were directed to squeeze an inflated blood pressure cuff with a manometer in order to determine the maximal grip strength. After several trials of this exercise, the subjects were instructed to generate a one-third maximal grip for 5 min. Blood pressure and heart rate were again recorded. Changes in these parameters during the mental arithmetic and handgrip tests were calculated by subtracting the average of the three baseline measures from the stress measure.

For some data analyses, we constructed categories based upon the normal distribution of trait or state anxiety as measured by the Spielberger instrument. All subjects with anxiety ratings greater than 1 standard deviation (SD) above the mean were classified as 'high anxiety', those with anxiety ratings 1 SD or more below the mean were classified as 'low anxiety' and those with anxiety ratings within 1 SD of the mean were classified as 'medium anxiety'. While many analyses are possible from a large database, we restricted our analyses to specific variables that have been associated with anxiety and to the relationship between anxiety and stress reactivity. All analyses conducted, whether positive or negative, are presented in this paper.

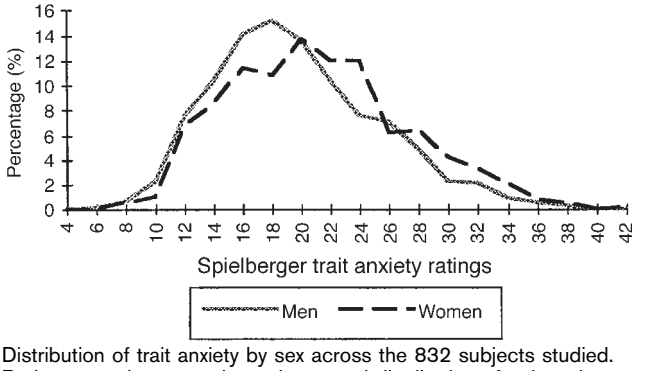
Results

The distribution of state anxiety in the population followed an expected normal distribution with no skewness. Figure 2 shows the distribution of trait anxiety for the entire population by sex. The mean trait anxiety rating

for the entire population was 20.8 with a standard deviation of 5.8. There was a significant sex difference in mean trait anxiety ($F = 13.4$, df = 1, $P = 0.0003$), with the women demonstrating higher trait anxiety than the men, although the shape of the distribution curves was similar in men and women. When we divided the subjects into high, medium and low anxiety categories using ± 1 SD from the overall population mean as the cut-off points, 56 out of 432 males (13%) and 84 out of 400 females (21%) fell into the high anxiety category. Accordingly, there were proportionately more males (47/432, 10%) than females (32/400, 8%) in the low anxiety category.

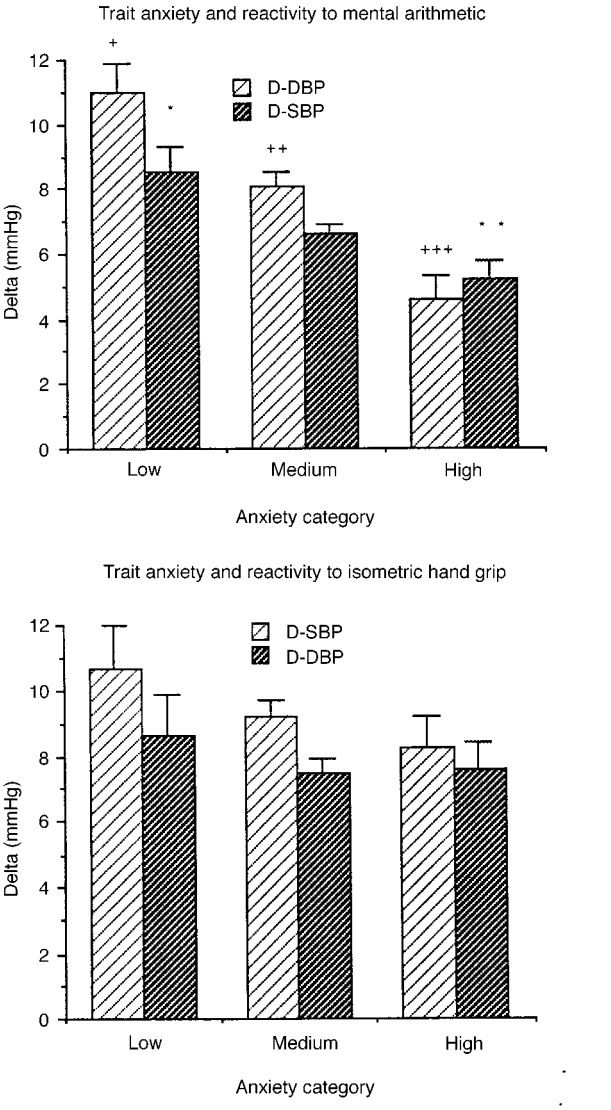
The first set of data analyses explored the links between trait anxiety and cardiovascular response to stress. Two stress tests were administered, isometric handgrip and mental arithmetic. Baseline heart rate and blood pressure and poststress measures were taken, and the changes in the scores were calculated. As shown in Table 1, the three anxiety groups did not differ at baseline in heart rate or systolic or diastolic blood pressure. The three groups also did not differ in body weight. However, following mental arithmetic, there was an inverse relationship between trait anxiety scores and change in heart rate and blood pressure, both systolic and diastolic blood pressure (Table 1, Fig. 3). All three groups were significantly different from each other in the change in the heart rate and in diastolic blood pressure by Bonferroni–Dunn post-hoc testing, but only the high and low anxiety groups were significantly different by Bonferroni–Dunn post-hoc testing for the change in systolic blood pressure (Table 1, Fig. 3). Cardiovascular responses to handgrip were also inversely related to anxiety but these differences were not significant between groups. Within individuals, there were significant correlations in cardiovascular reactivity to the two stressors (change in heart rate: $r = 0.35$, $P < 0.0001$; change in systolic blood pressure: $r = 0.308$, $P < 0.0001$; change in diastolic blood pressure: $r = 0.2$, $P < 0.0001$). Regression analyses of trait anxiety as a continuous variable with changes in the heart rate, and systolic and diastolic blood pressure for the two stressors demonstrated a significant correlation in all cases except the change in diastolic blood pressure with handgrip. A better correlation was shown with mental arithmetic than handgrip (heart rate and mental arithmetic: $r = 0.214$, $P < 0.0001$; diastolic blood pressure and mental arithmetic: $r = 0.126$, $P = 0.0026$; systolic blood pressure and mental arithmetic: $r = 0.116$, $P = 0.0053$; heart rate and handgrip: $r = 0.113$, $P = 0.008$; systolic blood pressure and handgrip: $r = 0.089$, $P = 0.03$).

Fig. 2



Distribution of trait anxiety by sex across the 832 subjects studied. Both men and women showed a normal distribution of trait anxiety but the women showed significantly higher mean anxiety levels.

Fig. 3



Relationship between trait anxiety and blood pressure reactivity to mental arithmetic (upper panel) and handgrip (lower panel). With the mental arithmetic stressor, the changes in both systolic (D-SBP) and diastolic (D-DBP) blood pressure in the low anxiety group were significantly different from those in the high anxiety group by Bonferroni–Dunn post-hoc testing. Isometric handgrip showed no significant effect by analysis of variance.

Table 1 Cardiovascular variables on stress tests in subject groups stratified according to trait anxiety

	Heart rate (beats/min)	SBP (mmHg)	DBP (mmHg)
Baseline			
Low anxiety	66 ± 1.3	118.9 ± 1.75	73.1 ± 1.3
Medium anxiety	66.6 ± 0.5	119.9 ± 0.5	74 ± 0.4
High anxiety	66.8 ± 1.0	117.5 ± 1.2	73 ± 0.9
Δ with mental arithmetic			
Low anxiety	11 ± 0.9*	10.2 ± 0.7**	8.5 ± 0.8***
Medium anxiety	8.1 ± 0.4*	8.5 ± 0.3**	6.6 ± 0.3***
High anxiety	4.5 ± 0.74*	6.8 ± 0.8**	5.2 ± 0.6***
Δ with isometric handgrip			
Low anxiety	4.0 ± 0.8	10.7 ± 1.3	8.65 ± 1.2
Medium anxiety	3.0 ± 0.4	9.2 ± 0.48	7.48 ± 0.47
High anxiety	1.4 ± 0.9	8.3 ± 0.9	7.59 ± 0.86

Values are means ± SEM. SBP, systolic blood pressure; DBP, diastolic blood pressure. *$F = 12.05$, df = 2, $P < 0.0001$; Bonferoni–Dunn post-hoc: low versus medium $P = 0.0086$; low versus high $P < 0.0001$; medium versus high $P = 0.0002$; **$F = 4.3$, df = 2, $P = 0.014$; Bonferoni–Dunn post-hoc: $P = 0.0041$ for low versus high; ***$F = 5.1$, df = 2, $P < 0.0062$; Bonferoni–Dunn post-hoc: $P = 0.0015$ for low versus high.

Table 2 Items completed by mental arithmetic by subjects stratified according to trait anxiety

	Total no. of items	No. correct	No. incorrect	% Correct
Low anxiety	43 ± 2.5*	38 ± 2.7**	4.6 ± 0.36	89.7
Medium anxiety	38 ± 0.9*	33 ± 0.9**	4.9 ± 0.15	87
High anxiety	32 ± 1.7*	26 ± 1.8**	5.49 ± 0.40	81

Values are means ± SEM. *$F = 7.7$, df = 2, $P = 0.0005$; Bonferroni–Dunn post-hoc comparisons: low versus high, $P = 0.0002$; medium versus high, $P = 0.003$; **$F = 8.2$, df = 2, $P = 0.0003$; Bonferroni–Dunn post-hoc comparisons: low versus high, $P = 0.0001$; medium versus high, $P = 0.0017$.

Since mental arithmetic is a psychological stressor, and differences in perceived stress or cooperation may have affected the outcome, we examined performance measures to determine whether there were differences between the groups in total numbers of item completed or numbers of items correct versus incorrect. There were significant differences between groups in both total number of items and total number correct, with the high anxiety subjects completing fewer items and having fewer correct items overall (Table 2). Since the high trait anxiety subjects were predominantly female, we also examined the data by sex (Table 3). There was a significant sex difference in baseline cardiovascular measures. There was a significant sex effect on the change in diastolic, but not systolic, blood pressure during mental arithmetic ($F = 38$, df = 1, $P = 0.0367$) and a significant sex effect on the change in the heart rate in response to mental arithmetic ($F = 4.98$, $P = 0.026$), although the directions of the changes were the same in men and women (no sign for sex by anxiety group interaction). There were no significant sex differences in the cardiovascular reactivity to handgrip.

In addition to trait anxiety, state anxiety may be expected to influence the cardiovascular response to stress. Additionally, state anxiety and trait anxiety may be expected to correlate. In fact, state anxiety showed a weak but significant correlation ($r = 0.26$, $P < 0.0001$) with trait anxiety. Table 4 shows the number of persons who fell into each of the nine possible cells, demonstrating that although the measures overlap, 45% of the subjects low on trait anxiety were also low on state anxiety but only 22% of the subjects high on trait anxiety were also high on state anxiety. The data examining the link between cardiovascular reactivity and state anxiety are shown in Table 5. At baseline, there was a suggestion of differences in systolic and diastolic blood pressure, although this was not significant by analysis of variance ($P = 0.054$). There was no significant differences between groups in systolic blood pressure reactivity to mental arithmetic, although the direction of the effect was similar to that seen with mental arithmetic and trait anxiety. Following isometric handgrip, systolic blood pressure in the different anxiety groups increased to essentially the same level, but there was now a significant effect of state anxiety on the systolic blood pressure change with isometric handgrip ($F = 6.09$, $P = 0.002$). Figure 4 presents the data graphically and indicates that the decreased systolic blood

Table 3 Trait anxiety and cardiovascular variables by sex

	Males			Females		
	SBP (mmHg)	DBP (mmHg)	Heart rate (beats/min)	SBP (mmHg)	DBP (mmHg)	Heart rate (beats/min)
Baseline						
Low anxiety	122 ± 1.4	74 ± 1.4	65.6 ± 1.4	113 ± 3.9	71 ± 2.7	66.8 ± 2.4
Medium anxiety	124.6 ± 0.7	76.5 ± 0.6	65.1 ± 0.65	114 ± 0.6	71 ± 0.6	68 ± 0.6
High anxiety	121 ± 1.9	74.8 ± 1.4	64.7 ± 1.3	115 ± 1.3	71.8 ±1.2	68.4 ± 1.4
Δ with mental arithmetic						
Low anxiety	10.3 ± 0.96	9.3 ± 0.9	12.2 ± 1.1	10.2 ± 1.1	6.9 ± 1.4	9.1 ± 1.3
Medium anxiety	9.2 ± 0.5	6.9 ± 0.4	8.3 ± 0.56	7.5 ± 0.5	6.3 ± 0.5	7.8 ± 0.56
High anxiety	7.7 ± 1.3	6.1 ± 0.9	6.3 ± 1.1	6.1 ± 0.9	5.8 ± 0.8	3.4 ± 0.95
Δ with isometric handgrip						
Low anxiety	11.3 ± 1.7	10.4 ± 1.3	4.4 ± 1.2	9.8 ± 2	7.13 ± 1.9	3.4 ± 0.96
Medium anxiety	10.3 ± 0.7	9.3 ± 0.6	3.5 ± 0.59	7.9 ± 0.6	6.4 ± 0.7	2.45 ± 0.57
High anxiety	10.2 ± 1.2	5.6 ± 1	3.6 ± 1.1	6.7 ± 1.3	6.8 ± 1.1	0.13 ± 1.3

Values are means ± SEM. SBP, systolic blood pressure; DBP, diastolic blood pressure. Analysis of variance by sex: baseline SBP in males, $F = 47.0$, $P < 0.0001$; baseline DBP in males, $F = 11.4$, $P = 0.0008$; baseline heart rate in males and females, $F = 5.4$, $P = 0.02$; mental arithmetic change in DBP for males, $P = 0.0367$.

Table 4 Relationship between state and trait anxiety

	State anxiety		
	Low	Medium	High
Trait anxiety			
Low	35	38	6
Medium	97	406	109
High	12	98	31

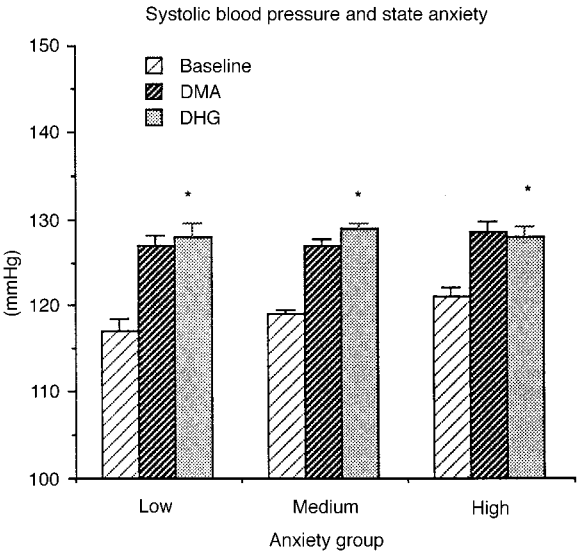
Values are numbers of patients in the study who fell into each category.

Table 5 Effects of state anxiety on stress reactivity

	Heart rate (beats/min)	SBP (mmHg)	DBP (mmHg)
Baseline			
Low anxiety	67.1 ± 1.51	117 ± 1	73.9 ± 0.9
Medium anxiety	67.9 ± 0.61	119 ± 0.56	74 ± 0.46
High anxiety	70.9 ± 1.56	121 ± 1.1	74.6 ± 0.88
Δ with mental arithmetic			
Low anxiety	8.7 ± 0.7*	9.8 ± 0.7	7.1 ± 0.6
Medium anxiety	8.2 ± 0.4*	8.2 ± 0.4	6.6 ± 0.3
High anxiety	6.3 ± 0.8*	7.7 ± 0.7	5.8 ± 0.5
Δ with isometric handgrip			
Low anxiety	4.1 ± 0.7	12.1 ± 1.0**	8.65 ± 1.2
Medium anxiety	3.5 ± 0.5	9.2 ± 0.5**	7.48 ± 0.47
High anxiety	1.6 ± 0.7	7.3 ± 0.88**	7.59 ± 0.86

Values are means ± SEM. SBP, systolic blood pressure; DBP, diastolic blood pressure. *$P = 0.045$, $F = 3.1$, df = 2; Bonferroni–Dunn post-hoc: no significant differences between groups; **$P = 0.0024$, $F = 6.09$, df = 2; Bonferroni–Dunn post-hoc: low versus medium, $P = 0.01$; low versus high, $P = 0.0006$.

Fig. 4



Relationship between baseline and poststress systolic blood pressure and state anxiety. As might be expected, subjects with high state anxiety showed an increase in baseline systolic blood pressure and subsequently showed a smaller response to isometric handgrip ($F = 6.09$, $P = 0.002$) and no difference in the response to mental arithmetic. DMA, during mental arithmetic; DHG, during isometric handgrip.

pressure reactivity seen in subjects with high state anxiety is related to the baseline differences. There were weak but significant state anxiety effects on the heart rate during mental arithmetic (Table 5). There was no significant state anxiety effect on diastolic blood pressure reactivity to isometric handgrip. Overall, the blood pressure reactivity pattern observed with state anxiety appeared to be less robust than the pattern observed with trait anxiety, with evidence suggesting an elevation in baseline values in those subjects with high state anxiety.

Two lifestyle variables that can influence blood pressure and have a potential effect on stress response are smoking and exercise. The second set of data analyses explored the association between trait anxiety, state anxiety and the lifestyle variables of smoking, exercise and alcohol use. There was a significant difference ($\chi^2 = 148.8$, df = 14, $P < 0.0001$) in smoking behavior by anxiety category, with the high anxiety group smoking more. As trait anxiety increased, the percentage of subjects who never smoked fell and the percentage of subjects smoking more than half a pack per day increased. The exercise data showed a negative relationship between exercise and anxiety category ($\chi^2 = 27.5$, df = 8, $P = 0.0006$). As trait anxiety levels increased, the proportion of subjects never exercising increased and the proportion of subjects exercising at least once a week decreased. There was no significant relationship between trait anxiety and alcohol abuse. In contrast to the data on trait anxiety, there were no significant associations between state anxiety and tobacco use, exercise or alcohol use.

Discussion

The purpose of these analyses was to examine the relationship between trait anxiety and cardiovascular reactivity to two simple stressors, mental arithmetic and isometric handgrip, which represent different types of stimuli to cardiovascular reactivity. While mental arithmetic is a pure sympathetic challenge, isometric handgrip involves muscle sensory feedback and local chemical signals as well as sympathetic stimuli. The data on trait anxiety and stress reactivity do not support the hyper-reactivity hypothesis, which postulates that subjects with high trait anxiety are more reactive to stress. Instead, there was an inverse relationship between anxiety and stress reactivity. A number of previous studies examining the relationship between trait anxiety and stress responsiveness have found no relationship between trait anxiety and cardiovascular reactivity [15–21]. Differences between the current study and those negative studies include our very large sample size and our use of the extremes, those subjects with at least ± 1 SD from the mean, to examine the relationship. Even with our large sample size, the correlation between trait anxiety and cardiovascular reactivity was small ($r = 0.214$–0.116) and of questionable biological significance. Thus, the effect is stronger in subjects with very high trait anxiety outside the 'normal' range.

One of the possible explanations for the decreased reactivity in high anxiety subjects is that the subjects with the highest anxiety were less engaged in the task and less 'stressed' or that chronic anxiety leads to changes in cardiovascular reactivity. The data on the number of items completed and the number of items correct during mental arithmetic showed that those subjects with high trait anxiety completed fewer items and made more mistakes than those with low trait anxiety. While fewer items completed might suggest less cooperation, the larger number of errors suggest that these individuals were 'stressed'. Thus, the association with lesser reactivity cannot be explained by a lesser engagement in the experimental procedure.

The reactivity hypothesis of cardiovascular disorders suggests that higher reactivity ought to be associated with higher baseline blood pressure readings, and it has been repeatedly suggested that hyper-reactivity to mental stress is a precursor of human hypertension [22,23]. In the present study we found no differences in baseline blood pressure between the three trait anxiety groups. Since the high trait anxiety group in this study showed a decreased response to mental stress, it is important to note that there is no uniform agreement on the effect of baseline blood pressure on reactivity. In fact, the evidence suggests that a higher baseline blood pressure is associated with lesser reactivity in two states with high baseline sympathetic activity, borderline hypertension [12,13,24] and panic disorder [25], as well as in the subjects who rated high on state anxiety in the present study. Julius *et al.* [26] found a normal reactivity to mental stress in subjects with baseline hypertension. Furthermore, these authors [26] reported a consistent negative relationship between baseline blood pressure and the responsiveness to mental arithmetic in 283 participants in the Tecumseh study [26]. However, the relationship was weak, and significant only for systolic blood pressure in women and diastolic blood pressure in men.

The negative correlations between trait anxiety and reactivity in the present study may reflect the interaction between sympathetic tone and cardiovascular responsiveness. A downregulation of receptors in the presence of high autonomic tone and an upregulation in response to a decreased tone is a well documented general physiologic phenomenon. Both reactivity tests used in this study involve a β-receptor-mediated increase in cardiac output and heart rate, and a downregulation of β-adrenergic responses in conditions of sympathetic overactivity is well known. The evidence of increased sympathetic tone in young patients with borderline hypertension is overwhelming: catecholamine levels are elevated [12], the response to receptor blockade is increased [13,14], norepinephrine spillover is excessive [27] and an increased sympathetic tone has been reported in peroneal nerve microneurography [28]. Such patients with borderline hypertension show a characteristic decrease in β-adrenergic responses [29,30].

While anxiety symptoms such as panic attacks are presumably mediated by activation of the sympathetic nervous system, this is difficult to establish conclusively. White and Baker [31] found a mean increase of 27 mmHg in systolic blood pressure and 5 mmHg in diastolic blood pressure and an increase of 14 beats/min in the heart rate during the hour of a panic attack, providing some validation that panic attacks/anxiety are accompanied by sympathetic activation. Baseline catecholamine and 3-methoxy-4-hydroxyphenylglycol levels appear to be either normal or mildly elevated in patients diagnosed with panic [32–34]. Decreases in the number of lymphocyte β-adrenergic receptors [35–37] and in β-adrenergic mediated cyclic AMP generation [38,39] have been reported in patients with panic disorder, providing additional evidence that panic attacks are accompanied by activation of β-adrenergic systems. Aronson *et al.* [36] have also demonstrated a negative correlation between trait anxiety and the number of lymphocyte β-adrenergic receptors. Studies of patients with panic disorder given isoproterenol infusions have shown a smaller change in the heart rate [40], again suggesting that chronic anxiety, by increasing sympathetic outflow, can lead to β-adrenergic receptor downregulation. While the present data on isometric handgrip only partly support the hypothesis of β-adrenergic downregulation, the systolic blood pressure changes were in the hypothesized direction although they were not significant. Subjects with high trait anxiety demonstrated less cardiovascular reactivity, suggesting that autonomic hyper-responsiveness is not a feature of chronic illness but may represent only an early feature of high anxiety levels, which lead to compensatory β-adrenergic downregulation.

In additional analyses, we examined lifestyle variables which have been linked to both cardiovascular disease and anxiety disorders. In epidemiological samples, Breslau *et al.* [41] noted a strong association between nicotine dependence and anxiety disorders and a relationship between nicotine dependence and neuroticism, as measured by the Eysenck index, even when a past history of depression and anxiety disorders and educational levels were taken into account in the analysis [42].

It is widely believed that exercise reduces anxiety and the failure to engage in exercise may have maintained the high levels of anxiety in the subjects in the present study. Alternatively, low levels of exercise may have been associated with the low-energy/fatigability component of trait anxiety. This further suggests that any possible cardiovascular morbidity associated with anxiety may act through these other well known cardiovascular risk factors.

In conclusion, these analyses of a population-based study demonstrate that high trait anxiety is associated with

increased smoking behavior and decreased routine exercise, lifestyle variables that are associated with high cardiovascular risk. However, high trait anxiety does not appear to be associated with increased autonomic reactivity. Several limitations in the study affect the ultimate conclusions and these include the use of discontinuous measurements of blood pressure and heart rate reactivity, the use of only one stress session and the use of an artificial stressor. Future studies using continuous blood pressure monitoring in an ambulatory setting in subjects exposed to naturalistic stressors are needed to confirm the findings of decreased reactivity in subjects with high trait anxiety. Our data support the hypothesis that increased sympathetic activation produced by anxiety leads to a compensatory decreased sensitivity to sympathetic arousal. More direct challenges with isoproterenol may provide further support for these findings. Finally, these studies addressed the effect of only one personality variable, trait anxiety, on blood pressure reactivity. Other factors such as defensive coping, which was not measured in these studies, may show a stronger correlation with cardiovascular reactivity than trait anxiety.

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