A Meta-Analytic Examination of Basal Cardiovascular Activity in Posttraumatic Stress Disorder

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Objective: The objective of this meta-analytic study was to determine whether individuals with posttraumatic stress disorder (PTSD) have higher levels of basal cardiovascular activity relative to comparable groups of individuals without PTSD. **Methods:** Meta-analytic data methods were applied to 34 studies that gathered indicators of basal cardiovascular activity including: heart rate (HR), systolic blood pressure, and diastolic blood pressure on subjects diagnosed with PTSD and two types of comparison groups. In total, cardiovascular measures were analyzed for 2670 subjects across all studies. **Results:** Results indicate that individuals with a current PTSD diagnosis have higher resting HR relative to both trauma-exposed individuals without a PTSD diagnosis and non–trauma-exposed individuals. The results also suggest that PTSD is associated with elevations in blood pressure; however, the effect sizes were smaller in magnitude than those obtained for heart rate. A subset analysis revealed that the effect sizes for comparisons on basal HR were greatest in studies with the most chronic PTSD samples. **Conclusion:** The meta-analysis supports previous qualitative reviews, finding a positive association between PTSD and basal cardiovascular activity. The discussion addresses possible mechanisms of action and the health-related implications of these findings. **Key words:** posttraumatic stress disorder, cardiovascular, heart rate, blood pressure.

PTSD = posttraumatic stress disorder; HR = heart rate; RR = respiration rate; EEG = electroencephalograph; SBP = systolic blood pressure; DBP = diastolic blood pressure; CI = confidence interval.

Since 1980, the formal diagnostic criteria for PTSD have included symptoms of excessive autonomic nervous system arousal (1), with more recent versions of the DSM nosology adding, "physiological reactivity to trauma related cues" (2). Even before 1980, experts on traumatic stress recognized that individuals exposed to the horrors of combat or other human atrocity (eg, violent sexual assault) often experienced marked autonomic arousal long after the traumatic experience. Conditions known as irritable heart, shellshock, and war neurosis were all characterized by elevated autonomic arousal as part of the symptom profile (3).

Dobbs and Wilson (4) provided the first laboratory-based demonstration of this phenomenon when they examined HR, RR, and EEG responses of World War II and Korean War combat veterans to auditory presentations that simulated combat sounds. Although the majority of the veterans found the protocol too aversive to complete, those who did showed both higher baseline levels of HR and RR and greater magnitude of cardiac response to the auditory stimuli relative to the comparison group of university students. This study showed that humans exposed to strong aversive conditioning events during combat (ie, traumatic stress) produce patterns of autonomic reactivity like those seen in research on fear acquisition with infrahumans—a notion that has been increasingly accepted in recent years (5).

The past 2 decades have witnessed a tremendous increase in research examining both phasic and tonic changes in the autonomic profile of individuals exposed to severe trauma, especially those subsequently diagnosed with PTSD (6). A corresponding increase in the precision of physiological measurement has resulted in a large number of high-quality laboratory-based studies that have examined the autonomic responses of patients with PTSD (7, 8).

These studies have used two primary laboratory paradigms. The first, often referred to as the cue-reactivity paradigm, involves assessment of physiological response to narrative descriptions of trauma (9), pictorial representations (7), or auditory representations of traumatic events (10). The second method, the acoustic startle probe paradigm (11), examines physiological responses evoked by loud and unexpected/unpredictable acoustic stimuli. Both paradigms focus on the direction and magnitude of reactivity of various physiological responses—that is, on *phasic* changes in physiology on exposure to specific stimuli.

Another important physiological feature of PTSD was identified by Blanchard (12), who noted apparent *basal* elevations in cardiovascular activity, particularly heart rate, shown by PTSD groups relative to comparison groups during the resting baseline periods of such studies. Blanchard proposed that PTSD might be a risk factor for hypertension or other cardiovascular problems, in light of evidence that links stress-induced cardiovascular *reactivity* with changes in basal cardiovascular *levels* (13, 14).

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Three hypotheses have been advanced to explain why PTSD may be associated with elevated basal cardiovascular activity. First, elevations at baseline may reflect systemic changes in cardiovascular function that result from repeated cardiovascular responses to stress (13, 14). Indeed, numerous studies show that, relative to traumatized subjects without PTSD, those with PTSD show exaggerated cardiovascular responses to trauma reminiscent cues in laboratory challenge studies (6). It is likely that these cardiovascular responses to trauma cues are mediated by the sympathetic branch of the autonomic nervous system, because individuals diagnosed with PTSD show elevated catecholamine levels, relative to comparison subjects without PTSD, after exposure to stressors (15, 16). Such repeated autonomic reactivity may produce structural and/or functional changes in the cardiovascular system. For example, chronic, stress-related sympathetic activation has been linked to down-regulation of beta-adrenergic receptors in the heart and peripheral vasculature, which increases peripheral vascular resistance and can ultimately lead to an increase in blood pressure (17, 18).

The second hypothesis attributes baseline differences in cardiovascular activity to either emotional priming or apprehension. Priming can occur when exposure to trauma-relevant cues takes place just before physiological assessment and lowers the threshold for subsequent responding. Anticipatory anxiety is a state of arousal shown by participants awaiting exposure to aversive stimuli in cue-reactivity studies (19). Some investigations have used ambulatory monitoring or other procedural maneuvers to reduce the likelihood of either trauma-specific emotional priming or anticipatory anxiety. Findings from these studies are mixed, with some demonstrating elevated basal levels of heart rate and blood pressure in subjects with PTSD, relative to control groups (20–22) and others not doing so (23–25).

A third hypothesis proposes that the association between PTSD and basal cardiovascular activity is mediated through variables known to have direct effects on cardiovascular health. For example, it is relatively well documented that alcohol consumption of >3 drinks per day is associated with increased blood pressure and heart rate, as well as increased mortality from coronary artery disease and stroke (26–28). PTSD is associated with higher rates of alcohol abuse/dependence (29), with especially high rates occurring in some high-risk trauma populations such as Vietnam veterans (30). PTSD also is associated with elevated rates of smoking (31), which is known to adversely affect cardiovascular health. These influences and others like them (eg, lower aerobic fitness) point to ways in which PTSD can have an indirect impact on basal cardiovascular levels.

To date, no meta-analytic examination of baseline cardiovascular indices in persons with PTSD has been conducted to quantify the magnitude of baseline differences. Therefore, we sought to address three questions with this set of meta-analyses. (1) Are observed differences in basal cardiovascular activity found between PTSD and non-PTSD comparison samples statistically reliable? (2) If such differences are reliable, do they vary in relation to chronicity of the disorder? This question aims to addresses the hypothesis that repeated cardiovascular reactions to stress over long periods of time can have an adverse impact on cardiovascular health. (3) Are differences in baseline cardiovascular levels dependent on emotional priming or anticipatory anxiety? This final question allows us to examine whether certain study methodologies result in artificially high basal cardiovascular measurement in PTSD groups because of emotional priming or anticipatory anxiety. Our meta-analysis was not able to address the potential contribution of other specific health habits on effect size outcome, because the individual studies did not provide data on which such information could be coded.

METHODS

Candidate studies were identified by means of PsycLit, Medline, and PILOTS database searches with the use of the following terms: PTSD, along with one of the following: heart rate, blood pressure, physiology, or psychophysiology. In addition, an issue-by-issue search was conducted on 10 journals that often publish empirical articles with content relevant to the current analyses. The literature search was conducted from 1980 forward, that being the first year PTSD was recognized in the psychiatric nomenclature (1). The reference sections of relevant review articles and identified empirical articles also were examined for potential candidate studies. Studies were included in the meta-analyses if they met all of the following criteria. (1) There was at least one dependent variable in the study that was a measure of resting heart rate, SBP, or DBP. (2) The physiological measures were collected by use of reliable devices and methods that corresponded to published recording guidelines (32). (3) Either the study report contained enough information such that group means and standard deviations on the dependent variables could be directly determined, calculated from test statistics (33), or relevant means and standard deviations could be obtained from study authors. (4) There was at least one analysis comparing a PTSD-diagnosed group with either a trauma-exposed non-PTSD group or a nontrauma control group (which were comparable in age and gender). (5) Diagnosis for the groups was established via a structured or clinical interview that corresponded to formal diagnostic criteria from DSM-III, DSM-IIIR, or DSM-IV (1, 2, 34). The most common interview formats were the Structured Clinical Interview for DSM-IV (35), The Clinician Administered PTSD Scale (36), and the Anxiety Disorder Interview Schedule (37).

The 34 studies selected for the meta-analyses had an average sample size of 82 and included 20 laboratory-based cue-reactivity studies, 6 acoustic startle studies, and 8 nonreactivity studies (eg,

ambulatory blood pressure studies or medical clinic basal cardiovascular examinations). Because the majority of phasic reactivity studies to date have been conducted with combat veterans, 59% of the studies in our analysis had exclusively male samples. The length of time since trauma for the PTSD groups ranged from a low of 1 month to a high of >20 years. Additional procedural and sample characteristics, as well as effect sizes for individual studies, are summarized in Table 1. All studies had age- and gender-comparable (relative to the PTSD samples) control groups for comparison purposes.

Meta-analytic tests of group mean differences for HR, SBP, and DBP were first applied to pooled data from all studies and then to data from subsets of studies, in order to address specific hypotheses. Cohen's *d* was the input variable for all between group comparisons (38). This was calculated by subtracting the respective control group mean from the PTSD group mean and then dividing by the pooled standard deviation for the dependent variable of interest or calculated from test statistics (eg, F ratios and degrees of freedom). Larger effect sizes indicate greater values on the dependent variables for the PTSD samples. Meta-analyses were conducted by use of the Meta-Analysis Programs Version 5.3 software package. The unweighted effect sizes were adjusted according to criteria outlined in Hedges and Olkin (39) to correct for the upward bias in estimation of effect size determined from simply averaging Cohen's *d* values. In addition, the weighted effect sizes were based on the weighted integration method outlined in Hedges and Olkin (39). Homogeneity of study effect sizes was evaluated by use of the criteria outlined in Hunter et al. (40).

RESULTS

The first step in the analysis was an examination of overall mean differences between the PTSD group and both comparison groups. Table 2 presents both unweighted and weighted effect sizes (with associated 95% CI), which show that PTSD is associated with elevated basal levels of heart rate and diastolic blood pressure relative to the two comparison groups. The results for SBP were slightly different, with the PTSD groups showing elevated basal levels relative to the nontrauma comparison group but not the traumatized non-PTSD comparison group. As suggested by meta-analytic methodologists (40), our distributions of effect sizes were examined for outliers. For 5 of the 6 comparisons that appear in Table 2 (excepting the comparison between PTSD and trauma-exposed non-PTSD on DBP), the amount of variance in the data set explained by sampling error was >75%, and the corresponding tests of homogeneity were nonsignificant, which suggested that the data set was sufficiently homogenous to conduct a meta-analysis (ie, no outlying values had undue influence on the effect size analyses; 40). The average amount of variance explained by sampling error across the six comparisons was 86%.

Our second meta-analysis sought to determine whether the between group effect sizes for HR varied as a function of the chronicity of PTSD. Analysis was limited to the HR data because there were too few studies with blood pressure data to support this subanalysis. Chronicity information for each study can be found in Table 1. The chronicity of the PTSD samples clustered into two groupings: samples tested <8 years posttrauma and more chronic samples tested >13 years posttrauma (no studies included samples between 8 and 12 years posttrauma). In order to have an adequate number of studies for comparison purposes on this chronicity analysis, we conducted a meta-analysis on these clusters of studies (<8 and >12 years). Studies that did not report chronicity information were excluded from this subanalysis.

The comparison between the PTSD samples <8 years posttrauma and age-comparable trauma-exposed non-PTSD samples produced statistically significant unweighted ($d = 0.24$) and weighted effects ($d = 0.23$, 95% CI = 0.04–0.43) across 9 studies (409 subjects). The same effect size comparisons for the PTSD samples >12 years posttrauma were larger in magnitude (unweighted $d = 0.53$, weighted $d = 0.39$, 95% CI = 0.29–0.49) and were statistically significant across 17 studies (1802 subjects). The amount of variance in the underlying data for these subset analyses explained by sampling error was 90% and 82% respectively, which suggests sufficient homogeneity of effect size across studies (40).

Similar analyses based on the distinction between PTSD samples along the chronicity continuum were also conducted with age-comparable nontrauma control groups. For the PTSD sample <8 years posttrauma vs. nontrauma control group comparison, the unweighted ($d = 0.46$) and weighted effects ($d = 0.32$, 95% CI = 0.07–0.58) were statistically significant (5 studies with 247 subjects). Repeating this comparison with the PTSD samples >12 years posttrauma revealed a larger weighted effect size (unweighted $d = 0.44$, weighted $d = 0.52$, 95% CI = 0.22–0.82; 6 studies with 178 subjects). The amount of variance in the underlying data for these subset analyses explained by sampling error was 91% and 100% respectively, which suggests sufficient homogeneity of effect size across studies (40).

The third set of analyses controlled for the potential influences of both emotional priming and anticipatory anxiety by using only studies that *did not* include either PTSD diagnostic assessment or other study-related trauma reminders within the 48 hours preceding the physiological assessment or study procedures involving exposure to trauma-related aversive stimuli after basal measurement. Studies selected for this secondary meta-analysis have been indicated with double asterisks in the Reference section.[1] Again, analysis was

[1]References marked with a single or double asterisk indicate studies included in the first meta-analysis. Those with double asterisks were included in the second meta-analysis.

TABLE 1. Study Characteristics

Study	Method	Trauma Type	Sex	*N*	Mean Age	Time Posttrauma	ES1 (d)	ES2 (d)
Beckham et al. (51)	BCA	COM	M	117	50	>20 years	HR = .27 SBP = .05 DBP = .05	
Blanchard et al. (52)	CR	COM	M	22	34	≈14 years	—	HR = .83 SBP = 1.03 DBP = .81
Blanchard et al. (53)	CR	COM	M	91	37	≈17 years	HR = .98	—
Blanchard et al. (54)	CR	COM	M	71	39	≈19 years	HR = .76	—
Blanchard et al. (55)	CR	MVA	M/F	90	34	2.5 months	HR = −.05	HR = .18
Blanchard et al. (49)	CR	MVA	M/F	159	38	2.5 months	HR = .28 SBP = .25	HR = .25 SBP = .02
Carson et al. (56)	CR	COM	F	38	54	≈25 years	HR = .85	—
Cohen et al. (20)	BCA	CIV	M/F	18	38	7 years	—	HR = 1.08
Davis et al. (57)	CR	COM	NR	29	30	≈2 years	HR = .69	—
Davis et al. (58)	CR	COM	NR	28	35	≈3 years	HR = .37	—
Gerardi et al. (59)	CR	COM	M	36	37	≈17 years	HR = .86 DBP = 1.25	—
Gerardi et al. (21)	ME	COM	M	58	40	≈20 years	—	HR = .79 SBP = .62 DBP = .78
Keane et al. (7)	CR	COM	M	1,148	44	≈24 years	HR = .30 SBP = .01 DBP = .08	HR = .31 SBP = .01 DBP = .07
Kinzie et al. (60)	CR	CON	M/F	34	39	≈20 years	HR = .44	
		COM	M/F	48	49	≈23 years	HR = .73	HR = .52
Litz et al. (61)	CR	COM	M	61	51	≈25 years	HR = .10	—
*Malloy et al. (62)	CR	COM	M	20	35	≈13 years	—	HR = .58
McFall et al. (63)	CR	COM	M	21	39	≈18 years	HR = −.03 SBP = .22 DBP = .36	—
McFall et al. (23)	BCA	COM	M	22	40	≈20 years	—	HR = .02 SBP = .42 DBP = .38
Metzger et al. (64)	SP	SA	F	57	41	NR	HR = .15	HR = .83
Muraoka et al. (22)	BCA	COM	M	18	46	≈26 years	HR = .98 SBP = .44 DBP = 1.21	—
Murburg et al. (65)	BCA	COM	M	18	41	≈21 years	—	HR = 0 SBP = .48 DBP = .96
Orr et al. (8)	CR	COM	M	20	67	NR	HR = −.02	—
Orr et al. (66)	SP	COM	M	56	44	≈24 years	HR = .48	—
Orr et al. (67)	SP	COM	M	39	49	≈29 years	HR = .62	—
Orr et al. (68)	SP	COM	M	93	42	≈22 years	HR = .35	—
*Orr et al. (69)	CR	SA	F	47	40	>20 years	HR = .64	—
Orr et al. (24)	BCA	COM	M	35	44	≈24 years	HR = −.27 SBP = −.20 DBP = −.07	—
*Pallmeyer et al. (70)	CR	COM	M	27	35	≈15 years	HR = 1.08	—
Pitman et al. (9)	CR	COM	M	33	41	≈20 years	HR = .93	—
Shalev et al. (25)	ME	COM	M/F	98	33	≈6 years	HR = .12 SBP = .17 DBP = .00	—
*Shalev et al. (11)	SP	CIV	M/F	48	33	5.75 years	HR = −.12	HR = .67
Shalev et al. (71)	CR	CIV	M/F	26	32	5 years	HR = −.35	—
Shalev et al. (72)	SP	CIV	M/F	58	36	3.8 years	HR = .69	—
Shalev et al. (73)	CR	CIV	M/F	40	30	≈3 years	HR = .57	HR = .19

* Denotes studies that included additional comparison groups beyond those reported in the meta-analyses (thus, tabled *N* will not equal overall *N* from original article), CR = cue-reactivity, SP = startle probe, BCA = baseline cardiovascular assessment, COM = combat, CON = concentration camp, CIV = civilian, SA = sexual abuse/rape, ME = medical exam, MVA = motor vehicle accident, M = male, F = female, NR = not reported, ES1 = effect size comparing PTSD vs. trauma-exposed non-PTSD sample, ES2 = effect size comparing PTSD vs. non-trauma-exposed sample, d = Cohen's *d*.

TABLE 2. Omnibus Meta-Analysis on Pooled Data Across All Studies

	PTSD vs. Trauma Non-PTSD	PTSD vs. Nontrauma Control
Heart rate (BPM)		
Number of studies (no. of subjects)	28 (2313)	12 (459)
Unweighted *d* (SE)	.42* (.07)	.48* (.10)
Weighted *d* (SE)	.36* (.04)	.43* (.10)
Weighted *d* 95% CI	.27–.45	.24–.62
Systolic blood pressure (mm Hg)		
Number of studies (no. of subjects)	7 (1353)	5 (212)
Unweighted *d* (SE)	.13 (.07)	.50* (.16)
Weighted *d* (SE)	.04 (.06)	.35* (.14)
Weighted *d* 95% CI	−.07–.16	.07–.63
Diastolic blood pressure (mm Hg)		
Number of studies (no. of subjects)	7 (1319)	4 (120)
Unweighted *d* (SE)	.39* (.21)	.71* (.12)
Weighted *d* (SE)	.10* (.06)	.71* (.19)
Weighted *d* 95% CI	.00–.21	.34–1.08

* = $p < .05$.

limited to heart rate data because too few studies with blood pressure measures fit these criteria.

Relative to trauma-exposed non-PTSD samples, those with PTSD continued to show elevated HR in this subset of 7 studies containing 415 subjects (unweighted $d = 0.33$, weighted $d = 0.26$, 95% CI for weighted $d = 0.06-0.46$). The comparison to the non–trauma-exposed control samples involved 5 studies with 150 subjects and also revealed higher resting HR for the PTSD samples (unweighted $d = 0.53$, weighted $d = 0.60$, 95% CI for weighted $d = 0.26-0.93$). The amount of variance in these two analyses attributed to sampling error was 70% and 79%, respectively. Again, the corresponding tests of homogeneity were nonsignificant, indicating sufficient homogeneity for a meta-analytic examination (40).

In order to convey the distribution of cardiovascular measures across groups, we have provided a graphical representation of the group mean values for each individual study on the heart rate and blood pressure variables in Figures 1 and 2, respectively. It is important to note that some studies only reported test statistics (eg, F ratios and degrees of freedom), without group mean data for the diagnostic groups in question. Thus, the number of data points that appear in the graphs is slightly lower than the number of data points that comprised the studies which went into the data analysis. The graphs do however, represent the vast majority of the studies and nicely display the shapes of the distributions of data.

DISCUSSION

The results of the main meta-analysis support previous qualitative reviews (12) that found an association between PTSD and elevations in basal heart rate and diastolic blood pressure relative to comparison groups. The most pronounced effect was for HR, with the PTSD samples having average resting values approximately 5 beats per minute faster than the comparison samples (see Figure 1). The effect sizes for SBP and DBP were statistically significant, but pressure differences were relatively small, ranging from 1 to 5 mm Hg between PTSD samples and the comparison samples (see Figure 2).

The second meta-analysis indicates that the effect size is larger for comparisons of basal HR involving the most chronic PTSD samples. Although our analysis does not allow for causal inferences, it is worthy to note that this pattern is consistent with the cardiovascular reactivity hypothesis whereby the association between PTSD and elevated basal cardiovascular activity is a result of cardiovascular adaptation to repeated stress responses over many years.

The results of the third meta-analysis are not entirely consistent with the hypothesis that that PTSD-related elevation in basal cardiovascular activity is attributable to anticipatory anxiety and/or emotional priming that is often present in laboratory studies of cue-reactivity. The HR difference for this subanalysis were of smaller magnitude than for the omnibus analysis but were still consistent with the general finding of higher resting HR for PTSD groups relative to non-PTSD groups. Although we excluded studies that involved exposure to explicit trauma-related threat cues, it is possible that PTSD samples are more reactive to a variety of stressors, which could also influence the baseline measures. Indeed, there is preliminary evidence that PTSD samples may be more reactive to seemingly "neutral" demands (ie, demands without

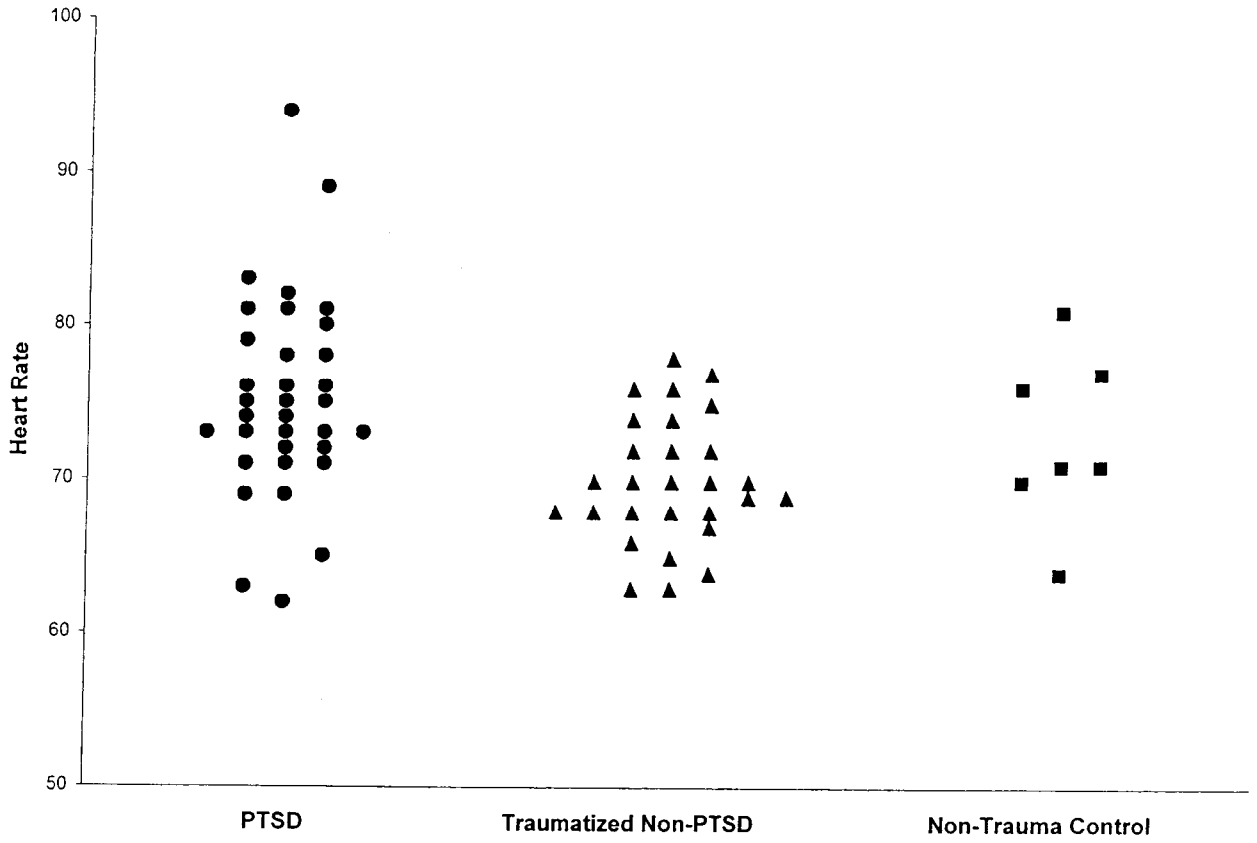

Fig. 1. Individual study group mean values for heart rate as a function of diagnostic group.

explicit threat cues related to traumatic experiences; Ref. 41).

Most subjects were in the normotensive range, even though the effect size analyses show that the PTSD samples have greater basal blood pressure than the comparison groups. The preponderance of nonpathologic levels is at least partially attributable to selection criteria applied by the individual studies, which typically excluded individuals taking hypertensive medications (eg, beta-adrenergic blocking agents). The likely impact of this medication exclusion is selective enrollment of subjects with the lowest basal levels of both heart rate and blood pressure. This sampling bias complicates interpretation of PTSD-related elevations in basal cardiovascular activity (eg, ≈5 mm Hg difference in DBP) as a substantive health risk. It is apparent however, that an elevation of 5 mm Hg in DBP in *hypertensive* populations does constitute a health risk, given literature reviews that conclude that long-term DBP elevations of this magnitude resulted in increased probability of stroke and coronary heart disease in prospectively studied hypertensive samples (42). Furthermore, a recent large-scale study found that PTSD was associated with an increased risk (relative to a comparable group of veterans without PTSD) of myocardial infarction and atrioventricular conduction problems in a cohort of prospectively followed combat veterans (43). For these reasons, the effect of PTSD on basal cardiovascular activity in the hypertensive spectrum is especially worthy of further exploration.

The elevated HR found in PTSD samples is also worthy of further study, given both infrahuman and human research on cardiac reactivity to stress, which shows that repeated phasic increases in cardiac activity and sustained elevated HR as a function of chronic stress can facilitate atherosclerotic buildup and contribute to coronary artery disease (13, 44). Also, several large-scale cohort studies have found higher resting heart rate to be positively associated with early mortality from cardiovascular diseases. For example, the Chicago Heart Association Detection Project prospectively followed thousands of study participants and found such an association in both men and women (45). Although not all studies support such an association (46), the majority of studies that have examined this issue have (45).

If repeated cardiovascular reactivity to stress accounts for the relationship between PTSD and elevated basal cardiovascular activity, one would expect the probability of HR and blood pressure elevations to

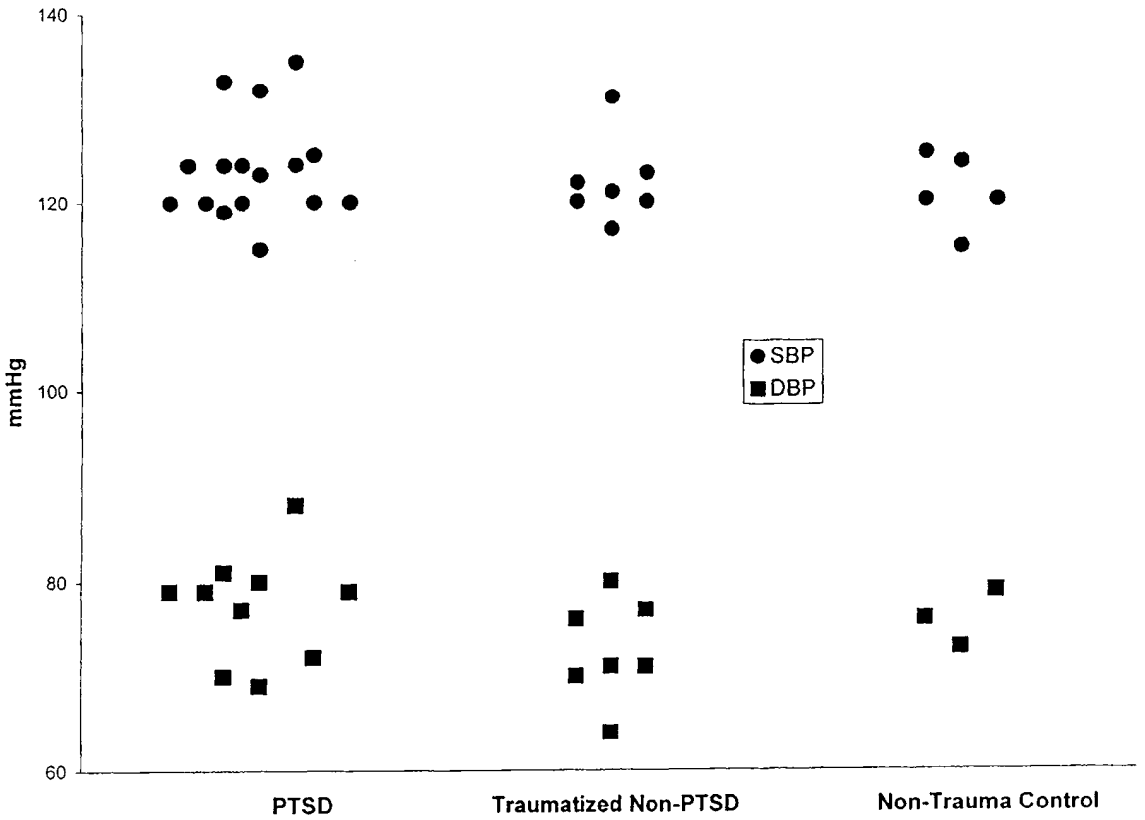

Fig. 2. Individual study group mean values for systolic and diastolic blood pressure as a function of diagnostic group

increase along with the chronicity of PTSD because of functional and/or structural changes in the cardiovascular system due to repeated stress that occurs over long periods of time (47). Our results are partially consistent with this hypothesis, in that studies with the most chronic PTSD samples tended to show the largest differences in basal heart rate relative to trauma-exposed comparison samples. Unfortunately, we could not address this question with analyses on blood pressure measures. Examination of Table 1 reveals that the most chronic samples were often exclusively men, relative to the less chronic samples, which more often included both genders or all women. Thus, it may be tempting to attribute our chronicity findings to a gender confound. However, women tend to have higher resting heart rates than men (48) and, therefore, the fact that more male samples were in the most chronic samples would run counter to our noted effects.

Future investigations of the relationship between PTSD and basal cardiovascular functioning would benefit from less restrictive sampling so that more variables known to affect cardiovascular functioning can be measured. For example, it is well documented that PTSD is associated with high rates of substance use involving agents known to influence cardiovascular functioning (29, 31). Unfortunately, as was noted earlier, the range of potential mediating variables was restricted because most of the studies contributing to the meta-analysis excluded individuals on the basis of substance use. In addition, smoking, body mass, and aerobic fitness level have not been routinely assessed, so important indicators of health behavior were not available for our analyses. It is also the case that the extant research has examined fairly "gender-specific" traumas. Specifically, the combat studies have included exclusively male samples, whereas the rape survivor studies have examined exclusively female samples. Thus, examination of the effects of gender and trauma types on such outcomes is not possible, given the gender by trauma confound. More research on traumas proportionally common to both genders (eg, motor vehicle accidents; Ref. 49) is needed to address these important questions.

By including the aforementioned measures in future research, multivariate procedures such as structural equation modeling (50) might then be used to evaluate the contribution of PTSD as a risk factor for conditions such as hypertension in the context of a multivariate model that accounts for other well-known risk factors for cardiovascular problems. An advantage of that type of analysis is that it allows for concurrent examination of direct, mediating, and moderating effects of PTSD

on cardiovascular functioning such as those outlined in the present article. Longitudinal designs that follow patients diagnosed with acute PTSD and assess the covariation between PTSD status and basal cardiovascular functioning would also be an area worthy of investigation. To date, no such comprehensive studies have been conducted. Future investigations might also use ambulatory blood pressure monitoring, which is a fairly common methodology used in cardiovascular research but has not been used frequently with PTSD samples (22). The use of such methodology will allow for more accurate assessment of basal heart rate and blood pressure because it rules out the potential confounds of anticipatory anxiety and emotional priming.

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