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Posttraumatic Stress, Heart-Rate Variability, and the Mediating Role of Behavioral Health Risks

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Abstract

Objective—Posttraumatic stress disorder (PTSD) has been linked to reduced heart-rate variability (HRV), which is in turn a risk factor for cardiovascular disease and death. Although hyperarousal and anxiety are thought to underlie this association, behavioral health risks, including smoking, alcohol dependence, obesity, and sleep disturbance, represent potential mechanisms linking PTSD and HRV.

Methods—To test this hypothesis, a combination of short-term laboratory-based and 24-hour ambulatory measures of HRV were collected from 227 young adults (18-39 years old), 107 of whom were diagnosed with PTSD. Latent variable modeling was used to assess the relationship of PTSD symptoms with HRV along with potential behavioral health mediators.

Results—PTSD symptoms were associated with reduced HRV, $\beta = -.21$, $p = .002$. However, this association was reduced in models that adjusted for cigarette consumption and history of alcohol dependence, and was rendered non-significant in a model adjusting for sleep disturbance. Independent mediation effects were deemed significant *via* bootstrapping analysis. Together the three behavioral health factors (cigarette consumption, history of alcohol dependence, and sleep disturbance) accounted for 94% of the shared variance between PTSD symptoms and HRV. Abdominal obesity was not a significant mediator.

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Conclusions—These results indicate that behavioral factors—specifically smoking, alcohol overuse, and sleep disturbance—mediate the association between PTSD and HRV-based indices of autonomic nervous system dysregulation. Benefits from psychiatric and psychological interventions in PTSD may therefore be enhanced by including modification of health behaviors.

Keywords

posttraumatic stress disorder; heart rate variability; cigarette smoking; alcohol dependence; sleep disturbance

Acute stress has long been connected to cardiovascular risk (1). For individuals with posttraumatic stress disorder (PTSD), a disorder characterized by hyperarousal and frequent physiological symptoms related to anxiety and stress, dysregulation of the autonomic nervous system has been identified as an important precursor to cardiovascular disease, diabetes, and other health risks (2, 3). Indeed, a key indicator of autonomic functioning and cardiovascular health, heart-rate variability (HRV), is often depressed amongst individuals with PTSD (4). Although the link between PTSD and HRV is generally discussed as a purely psychosomatic phenomenon (5), a number of behavioral risk factors—namely smoking, alcohol misuse, obesity, and sleep disturbance—may account for this link. In this study, HRV was assessed amongst younger adults (18- to 39-years-old) with and without PTSD to determine whether autonomic dysfunction in individuals with PTSD is in part attributable to the higher rates of smoking, drinking, obesity, and sleep disturbance that often coincide with PTSD (6-9).

Heart-Rate Variability

Under normal circumstances, heart rate varies on a beat-to-beat basis due to the dynamic interplay of the sympathetic (SNS) and parasympathetic nervous system (PNS). The SNS stimulates excitation (e.g., increased heart rate and blood pressure) in response to unexpected changes in the body and/or environment through the release of catecholamines (10). The PNS restores cardiovascular activity to baseline levels *via* vagal innervation. When these two systems are in disequilibrium—either because the SNS is hyperactive or the PNS is hypoactive—HRV attenuates (3).

Low HRV is both an indicator and a precursor of disease. It may signal some underlying irregularity, such as immune dysfunction resulting from diabetes, osteoporosis, arthritis, Alzheimer's disease, and some cancers (11). Reduced HRV may also stimulate deleterious effects on cardiovascular health. Lower HRV is a risk factor for arrhythmia and in turn is predictive of heart disease and cardiac arrest (12-14). Reduced HRV may also accelerate atherosclerosis (2) and result in increased variability in blood pressure, which is itself an independent risk factor for coronary artery disease (15).

Psychophysiology of PTSD

Exposure to psychological trauma increases the risk of developing PTSD, a disorder characterized by persistent re-experiencing of the traumatic event, avoidance of stimuli associated with the event, and increased arousal (16). These symptoms have long been

known to convey autonomic dysregulation, such as elevated heart rate and increased blood pressure both at baseline (17) and in response to stressors (18). Even though the SNS is instrumental in the etiology of anxiety disorders, experimental evidence suggests that the PNS may be responsible for the maintenance of elevated physiology in psychopathology (15). For instance, the administration of catecholamine (SNS) antagonists prior to induced panic attacks does little to reduce heart rate, suggesting that the SNS plays a minor role in such attacks (19). However, lactate, which is a known suppressor of vagal (PNS) activity, accumulates during panic attacks (20), and is even administered in laboratory studies to induce panic attacks (21) and stimulate PTSD symptoms (22). Thus, suppressed PNS activity and consequently reduced HRV are implied in individuals with anxiety or trauma-related disorders such as PTSD. Indeed, individuals with PTSD exhibit reduced HRV in both short-term laboratory-based measurements of HRV (5, 23-26) and 24-hour ambulatory measures (4, 27). In turn, individuals with PTSD are more likely than those without PTSD to develop cardiovascular disease (28) and face an increased risk of cardiac death (29).

Although reduced HRV in PTSD is primarily attributed to the direct impact of psychological hyperarousal and anxiety on the autonomic nervous system (5), behavioral risk factors could partially account for that association. Individuals with PTSD are more likely than individuals without PTSD to smoke and do so heavily (6), abuse alcohol (7), be obese (8), and suffer from sleep disturbance stemming from flashbacks and nightmares (9). Each of these risk factors are independently associated with reduced HRV (30-33), suggesting that the relationship between PTSD and HRV may in part be due to the behavioral health risks that frequently accompany PTSD.

Current Study

In the present study, the prediction that the link between PTSD and HRV is partially mediated by smoking, lifetime alcohol dependency, abdominal obesity, and sleep disturbance was tested amongst a sample of younger adults (i.e., under 40 years of age) with and without PTSD. Younger adults were targeted to quantify the early health risks posed by PTSD and associated psychopathology. Latent variable modeling was used to model HRV *via* a combination of long- and short-term measures of autonomic functioning to minimize the impact of measurement error on that construct.

Three sets of hypotheses were tested: 1) PTSD symptoms would be associated with lower HRV; 2) PTSD symptoms would be associated with greater smoking, higher rates of lifetime alcohol dependence, higher rates of abdominal obesity, and more sleep disturbance; and 3) each of these behavioral risk factors would partially mediate the association between PTSD symptoms and HRV. That is, accounting for each of these behavioral health risks would attenuate the association of PTSD symptoms with HRV.

Methods

Participants

A sample of 227 participants (18-39 years old; 112 women), consisting of 75 U.S. military veterans, was recruited *via* fliers hung in hospital clinics and waiting rooms as well as online

ads such as Craigslist to complete a study of the metabolic and cardiovascular risk factors associated with PTSD amongst young adults. Criteria for exclusion included presence of a) organic mental disorder, b) schizophrenia, c) bipolar I mixed state or bipolar II, d) lifetime PTSD without current PTSD, e) current substance abuse/dependence, f) current major depressive disorder without PTSD, g) pregnancy, h) AIDS or HIV, and i) uncontrolled medical condition (e.g., liver failure). The study was approved by both the Durham Veterans Affairs and Duke University Medical Center Institutional Review Boards. All patients gave written informed consent prior to participation. Data was collected between August 2009 and September 2013.

Measures

Posttraumatic stress disorder—PTSD status was assessed using the Clinician Administered PTSD Scale (CAPS) (34). The interview was administered by a licensed clinical psychologist or by a trainee under the direct supervision of a licensed clinical psychologist. Interrater reliability among interviewers was high (Fleiss' kappa = .94) across five training tapes. The CAPS interview has excellent reliability (αs from .73 to .85 for the three symptom clusters) and validity within multiple trauma populations, and is widely accepted as the state-of-the-art method for PTSD assessment (35).

The Davidson Trauma Scale (DTS) (36) was used in all analyses to quantify PTSD symptoms along four distinct symptom clusters—re-experiencing (B), avoidance (Av), numbing (Numb), and hyper-arousal (D)—*via* 5-, 2-, 5-, and 5-item scales (37), respectively. Each item measures the frequency (0, “not at all”, to 4, “every day”) and intensity (0, “not at all distressing”, to 4, “extremely distressing”) of corresponding symptoms. Cluster scores were calculated by summing frequency and intensity scores for associated items. The DTS demonstrated strong internal consistency in the present sample (αs from .78 to .89) and good concurrent validity, evident by differences in total DTS scores by CAPS-determined PTSD status, $t(225) = 14.00$, $p < .001$.

Smoking—Smoking was operationalized based on participants' responses to the Fagerström Test for Nicotine Dependence (38): non-smokers were assigned a value of 0; past—but not present—smokers, 1; current smokers who consume 10 or fewer cigarettes/day, 2; current smokers who consume 11 to 20 cigarettes/day, 3; current smokers who consume 21 to 30 cigarettes/day, 4; and current smokers who consume over 30 cigarettes/day, 5.

Lifetime alcohol dependence—The Structured Clinical Interview for the DSM-IV (SCID) (39) was used to assess Axis I disorders, including lifetime alcohol dependence and current MDD. The SCID is a semi-structured diagnostic interview for determining Axis I diagnoses. Study interviewers completed an extensive training program involving the rating of seven video-recorded interviews. Interviewers additionally participated in biweekly reliability meetings and were supervised by licensed clinical psychologists. Interrater reliability among interviewers for Axis I diagnoses was high (Fleiss's kappa = .96).

Sleep disturbance—The Pittsburgh Sleep Quality Index (40) is a self-report questionnaire that assesses seven domains of sleep disturbance *via* 19 items. Global disturbance scores, calculated as the sum of the seven domains scores, range from 0, indicating no sleep disturbance, to 21, indicating severe disturbance. The global disturbance scale was internally consistent (α = .85) in the present study. In previous work, correlations between global disturbance scores and sleep latency (r = .20), number of arousals (r = .47), and percentage of rapid eye movement sleep (r = .34) were demonstrated (40).

Procedure

Participants completed an initial interview that included the above measures along with a demographics questionnaire capturing participants' age, gender, race, and veteran status. Health status and current medications were recorded. Height, weight, waist and hip circumference were directly measured. Abdominal obesity was defined as a waist-hip ratio greater than 0.90 for men and 0.85 for women (41). In addition, participants underwent ECG Holter monitoring using a Del Mar Reynolds Lifecard CF, 3-channel digital recorder. Sessions began at approximately 2:00PM and lasted 24 hours.

Heart-rate variability—ECGs were recorded for 20 to 24 hours and were digitalized at 125 Hz. All QRS intervals were screened, and only beats showing normal sinus rhythm were retained for HRV analyses. Holter recordings were classified as useable if normal sinus rhythm was present 80% of the time or more for each hour in at least 18 hours of the recording.

Overall HRV was estimated from the 24-hour ECG recordings from the standard deviation of all normal R-R intervals (SDNN) and from the triangular (TR) index using the HRV Tools 1.72 software (Del Mar Reynolds). SDNN provides a measure of overall HRV and has been found to consistently predict mortality in several populations (42, 43). The TR index, estimated by the integral division of the total number of R-R intervals by the height of the density distribution (modal number of R-R intervals), is considered to be more resistant than other HRV measures to variations in the quality of ECG data (44).

HRV was further examined for the two-hour period immediately following waking, a period of increased risk of cardiac events, when sympathovagal balance shifts towards lower parasympathetic control relative to sleep (45). For the waking HRV measures, frequency domain analysis was used to estimate low-frequency (LF) power in the range of 0.04 Hz to 0.15 Hz and high-frequency (HF) power in the range of 0.15 to 0.40 Hz. Raw power was log-transformed before analysis to normalize their distributions.

HRV was also examined during a 5-minute period of quiet supine rest. These short-term recordings were reviewed for artifacts and edited in a similar fashion as the long-term recordings. Short-term HRV was estimated from the standard deviation of the normal-to-normal R-R intervals (RRSD).

Analytic Plan

Latent variable modeling was used to test the hypothesis that PTSD symptoms would be associated with HRV, with subsequent models conducted to test the mediation hypotheses.

Specifically, a latent variable representing HRV was specified using SDNN, TR index, log HF power, log LF power, and RRSD. The purpose of this was to minimize the measurement error in HRV. A second latent variable was specified to capture PTSD symptoms using the four DTS scales. The adequacy of the HRV and PTSD latent variables was determined prior to further modeling using standard fit criteria: root mean square error of approximation (RMSEA) < .08, comparative fit index (CFI) > .90, and standardized root mean square residual (SRMR) < .05. The chi-squared test of model fit was also consulted, with non-significance indicative of good model fit.

Initially a direct-effects model was conducted to test the association between PTSD symptoms and HRV. In subsequent models, the indirect effect of PTSD symptoms on HRV *via* associated behavioral risk factors was tested. To test the significance of mediation, bootstrapped confidence intervals around the indirect effects of PTSD symptoms on HRV were generated using resampling. This method offers an advantage over conventional tests, such as Sobel's *z*, because it takes into account the positive skew inherent to indirect effects (46). As such, bootstrapping methods are more powerful than conventional tests, with mediation deemed significant when the resulting confidence interval does not span 0.

Missing HRV data were imputed *via* multiple imputations (10 imputation datasets, using the Monte Carlo Markov chain method), which were subsequently used for latent variable modeling. Multiple imputation was not available for bootstrapped mediation analyses; thus, data with case-wise deletions were used for those analyses. In each of the latent variable models and bootstrapping analyses, models were adjusted for age, sex, and minority statusAnalyses were conducted using Mplus 7. Estimated effects were deemed significant at $p < .05$.

Results

PTSD Symptoms

One-hundred seven participants (47% of the sample) met CAPS criteria for PTSD. Nearly half ($n = 49$) of these were veterans (see Table 1). Indeed, 37% ($n = 42$) cited combat as the trauma precipitating PTSD; 17% ($n = 20$), childhood physical or sexual abuse; 11% ($n = 13$), adulthood violence; 10% ($n = 11$), adulthood physical or sexual assault; 9% ($n = 10$), death of a close friend or family member; 4% ($n = 5$), childhood violence; 3% ($n = 4$), domestic violence; 3% ($n = 3$), a serious accident; and 6% ($n = 7$), some other event. Mean time since trauma was 10.05 years ($SD = 8.45$). A quarter of participants with PTSD ($n = 27$) also met criteria for current MDD. As expected, PTSD symptoms—particularly re-experiencing and avoidance symptoms—were negatively correlated with the HRV indices (see Table 2).

An initial measurement model of the PTSD latent variable indicated an inadequate fit: RMSEA = .28, CFI = .96, SRMR = .03, and $X^2(2) = 38.64$, $p < .001$. Examination of the residual covariance matrix, however, suggested that the residual errors for the re-experiencing and avoidance symptoms were correlated. Indeed, specifying this correlation significantly improved the model, $\Delta X^2(1) = 38.64$, $p < .001$, rendering a good fit: RMSEA

= .00, CFI = 1.00, SRMR = .00, and $X^2(1) = 0.00$, $p = .97$. The resulting latent variable was strongly correlated with current PTSD status, $r(225) = .69$, $p < .001$.

Heart-Rate Variability

Of the 227 participants, 15 were missing data for one or more HRV indicators. The majority of these ($n = 12$) were missing waking HRV data (log HF power and log LF power) due to sleeping through the end of the 24-hour recording period. Two of the 12 were also missing SDNN and TR index data due to ectopy or early monitor removal. Three participants were missing RRSD data. Participants with missing HRV data were younger ($M = 26.00$ years) than those with no missing data ($M = 29.56$), $t(225) = 2.40$, $p = .017$. Otherwise, participants with missing HRV data were comparable to those without missing data with regard to sex, $X^2(1) = 3.30$, $p = .069$, minority status, $X^2(1) = 0.02$, $p = .89$, PTSD status, $X^2(1) = 0.00$, $p = .97$, current MDD status, $X^2(1) = 0.42$, $p = .52$, smoking, $t(225) = 0.82$, $p = .41$, lifetime alcohol dependence, $X^2(1) = 0.64$, $p = .42$, abdominal obesity, $X^2(1) = 0.00$, $p = .95$, and sleep disturbance, $t(225) = 0.49$, $p = .62$. Means for the five HRV indicators by PTSD status are listed in Table 1. All but one of the indicators, RRSD, were reduced amongst participants with PTSD. None of the HRV indicators varied by current MDD status (ps > . 16).

The initial measurement model of the HRV latent variable yielded a poor fit: RMSEA = .26, CFI = .87, SRMR = .07, and $X^2(5) = 83.13$, $p < .001$. However, after specifying the correlation between the residuals for SDNN and TR index, the model improved significantly, $\Delta X^2(1) = 74.83$, $p < .001$, rendering an adequate fit: RMSEA = .07, CFI = .99, SRMR = .02, and $X^2(4) = 8.30$, $p = .081$.

According to an initial latent-variable model, HRV was diminished amongst older participants, women, and minorities (see Figure 1, Model A), which is consistent with previous findings (44, 47, 48). HRV was also negatively associated with PTSD symptoms, lending support to the first hypothesis.

In support of the second hypothesis, PTSD symptoms were positively associated with smoking, lifetime alcohol dependence, abdominal obesity, and sleep disturbance (see Figures 2-5, Models B-E). Moreover, the direct of effect of PTSD symptoms on HRV was attenuated in the presence of smoking, lifetime alcohol dependence, and sleep disturbance, suggesting mediation. No such attenuation was evident with abdominal obesity in the model.

To test the significance of smoking, alcohol-dependence, and sleep-disturbance mediation effects, bootstrapped confidence intervals around the indirect effects of PTSD and depressive symptoms on HRV were generated from 5,000 re-samples. In support of the third hypothesis, the indirect effects for smoking (bootstrapped 95% CI of standardized effect: -. 15 to -.00), lifetime alcohol dependence (bootstrapped 95% CI of standardized effect: -.11 to -.01), and sleep disturbance (bootstrapped 95% CI of standardized effect: -0.23 to -0.02) were each significant in separate models, independently accounting for 36%, 27%, and 57% of the effect of PTSD symptoms on HRV, respectively. In combination, the three mediators accounted for 94% of the effect of PTSD symptoms on HRV (bootstrapped 95% CI of combined standardized effects: -.33 to -.08). However, only the individual indirect effect of

sleep disturbance (bootstrapped 95% CI of standardized effect: -0.21 to -0.00) remained statistically significant in the combined model (bootstrapped 95% CI for standardized smoking effect: -.14 to .02; bootstrapped 95% CI for standardized lifetime alcohol dependence effect: -.10 to .02), likely due to the strong correlation between smoking and lifetime alcohol dependence, $r(225) = .40$, $p < .001$.

Discussion

The present study examined the association of PTSD symptoms with HRV, along with potential health-behavior mediators. Consistent with previous work (4, 5, 23-27), PTSD symptoms were negatively related to a combination of short- and long-term measures of HRV. A novel finding was that nearly all of this relationship was explained by increased smoking, lifetime alcohol dependence, and sleep disturbance.

These findings represent an important contribution to the growing literature linking PTSD with lowered HRV. Furthermore, the demonstration that 24-hour ambulatory measurements of HRV are negatively associated with PTSD symptoms is relatively novel. The majority of prior studies linking PTSD with HRV used short-term laboratory measures of HRV, which carry less prognostic power than long-term ECG data (48). Those few studies that have employed 24-hour ECG monitoring (4, 27, 50) have produced somewhat inconsistent results, likely due to small sample size (27) and restricted sampling (50). One recent investigation, a large-scale twin study of middle-aged combat veterans (4), did, however, demonstrate significantly lower 24-hour HRV in high- and low-frequency spectra amongst individuals with PTSD. Moreover, the authors noted substantial reductions in the effect of PTSD by 18.4% up to 39.3% after adjusting for demographic characteristics, health history, and behavioral factors, such as smoking and alcohol consumption. The present findings extend those results, demonstrating that the link between PTSD and reduced HRV is present amongst young adults and may be almost entirely due to behavioral health risks.

That smoking, drinking, and sleep disturbance account for so much of the association between PTSD symptoms and HRV emphasizes the importance of efforts to reduce nicotine and alcohol consumption as well as treat insomnia and other sleep disturbances among younger adults with PTSD. Individuals with PTSD are at least twice as likely as non-affected individuals to smoke (51, 52), 1.5 times as likely to suffer from alcohol abuse or dependence (53), and 1.5 times as likely to report sleep disturbances (54). All three health risks are strongly associated with reduced HRV (30, 32, 33). Indeed, just one week of smoking cessation can result in significant increases in HRV (55), as can six months of temperance by recovering alcoholics (56), and prolonged cognitive-behavioral therapy for insomnia (57). Thus, an emphasis on targeting these behavioral health risks amongst individuals with PTSD is warranted.

Counter to our expectations, no mediation effect for abdominal obesity was observed. Compared to a recent nationally representative sample (8), for which the relative odds of obesity for PTSD (based on body-mass index) was 1.51, the association between PTSD and (abdominal) obesity was weak (odds ratio = 1.16). This discrepancy may in large part be due to the relative youth of the present sample. That is, the relative odds of obesity for

individuals with PTSD may increase with age as the consequences of poor dieting and exercise habits accumulate. If so, the mediating role played by obesity between PTSD and HRV may increase with age. This is a question we hope to explore in future efforts.

As compelling as these findings are, there are limitations to the present study. For one, the cross-sectional nature of the data limits our interpretation of the directionality of the associations. For instance, it is possible that smoking, alcohol misuse, and sleep disturbance exacerbate PTSD symptoms. Nevertheless, that the effect of PTSD symptoms on HRV was subsumed by the behavioral risk factors suggests that those risk factors represent an intermediary set of mechanisms linking PTSD and HRV. Considering that this interpretation is consistent with the conventional view that PTSD engenders greater substance misuse and sleep disturbance (51-54), the conclusion forwarded here appears to be the most logical one.

Another limitation was the exclusion of individuals with current alcohol abuse or dependence and the dichotomous nature of the lifetime-alcohol-dependence variable. These likely diminished the predictive power of that construct. Nevertheless, its influence on HRV was sufficient in the present study to demonstrate a significant mediation effect; in the absence of the exclusion of individuals with current alcohol abuse/dependence it is likely that HRV would have been further reduced in the PTSD group. In any case, future research in this area should make use of higher-resolution measures of alcohol consumption.

Although by design, the sampling of younger adults limits the generalizability of the present results. For instance, Shah and colleagues (4) found that smoking and alcohol use played a much more modest role in linking PTSD with HRV amongst middle-aged adults than did the present study. If these age differences are reproducible, they would suggest that younger adults with PTSD in particular should be targeted for smoking and drinking interventions in light of the relatively prominent role that they play in decreased HRV compared to older populations. In any case, this remains an empirical question and should be addressed with future research.

A notable innovation in the present study is the use of latent variable modeling to capture both PTSD symptoms and HRV. In particular, analyzing HRV as a latent variable provides an efficient multivariate method for modeling the shared features of multiple HRV indicators absent the random variance. That said, use of latent variable modeling for HRV may be a productive approach in future studies.

In sum, these results further emphasize the deleterious effects of PTSD on health, even among younger adults. In the present study, PTSD symptoms were significantly associated with dysregulation of the PNS, which both indicates and prognosticates cardiovascular risk. Moreover, major contributors to this linkage were smoking, alcohol dependence, and sleep disturbance. These findings complement evidence from two sub-samples of the current study indicating that PTSD is also associated with increased risk of orthostatic hypotension (58), which is symptomatic of SNS dysfunction (59), and dyslipidemia (60). In those analyses, smoking, alcohol dependence, and sleep disturbance were also identified as potential mechanisms for cardiovascular risk. Together, these findings underscore the extent to which interventions for individuals with PTSD aimed at smoking and alcohol cessation as

well as sleep improvement could reap meaningful, long-term benefits, both psychiatric and cardiovascular.

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Acronyms

PTSD	posttraumatic stress disorder
HRV	heart-rate variability
SNS	sympathetic nervous system
PNS	parasympathetic nervous system
CAPS	Clinician Administered PTSD Scale
DTS	Davidson Trauma Scale
SCID	Structure Clinical Interview for the DSM-IV
MDD	major depressive disorder
SDNN	standard deviation of all normal R-R intervals
TR index	triangular index
LF power	low-frequency power
HF power	high-frequency power
RRSD	standard deviation of normal-to-normal R-R intervals
RMSEA	root mean square error of approximation
CFI	comparative fit index
SRMR	standardized root mean square residual
CI	confidence interval

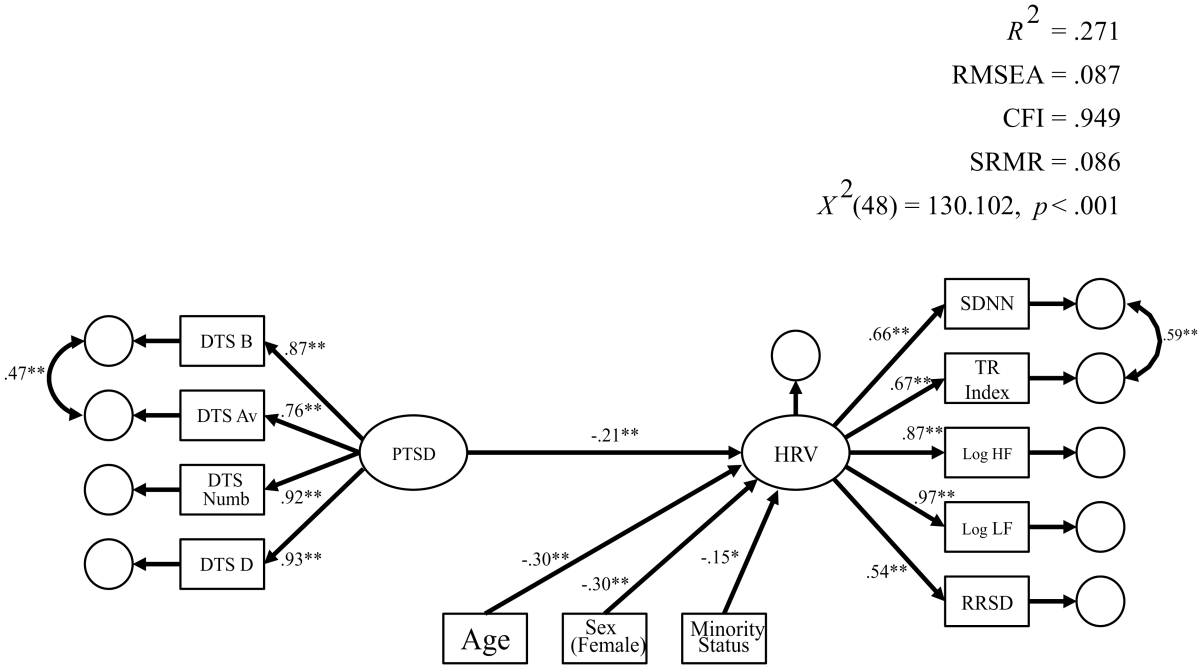

Figure 1. Latent variable Model A of HRV depicts the standardized direct effect of PTSD symptoms on HRV

DTS B = Davidson Trauma Scale (DTS) re-experiencing (B cluster) subscale; DTS Av = DTS avoidance subscale; DTS Numb = DTS numbing subscale; DTS D = DTS hyperarousal (D cluster) subscale; SDNN = standard deviation of all normal R-R intervals; TR index = triangular index; Log LF = log-transformed low-frequency power, Log HF = log-transformed high-frequency power, RRSD = standard deviation of normal-to-normal R-R intervals.

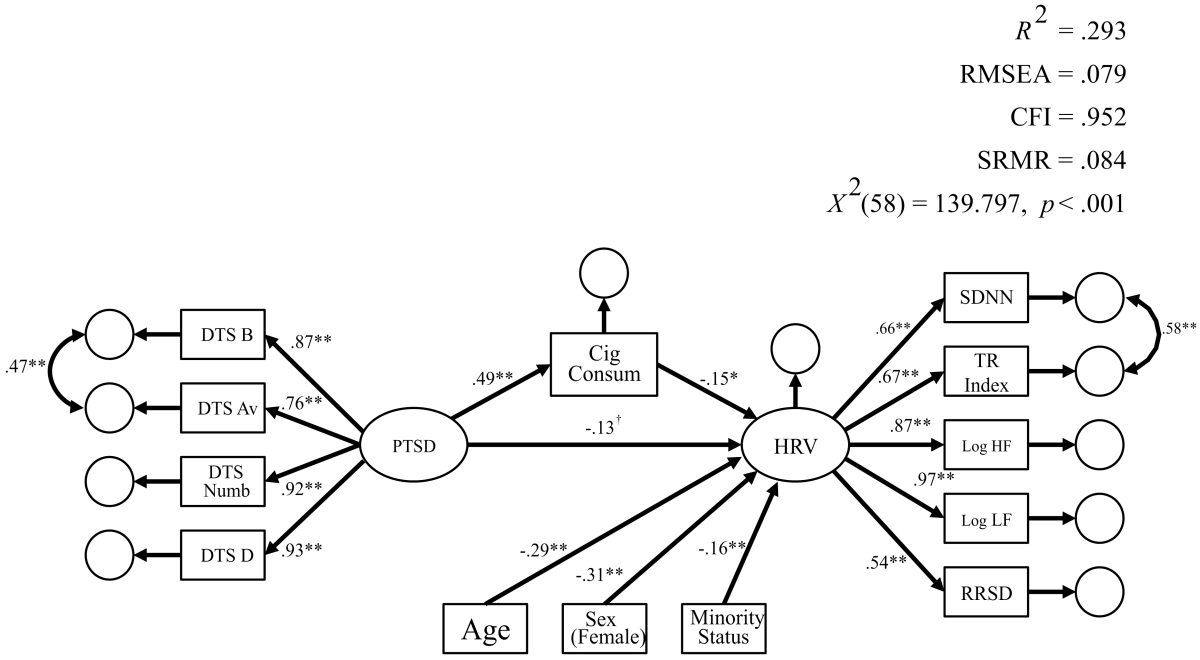

Figure 2. Latent variable Model B of HRV depicts the standardized indirect effect of PTSD symptoms on HRV *via* smoking

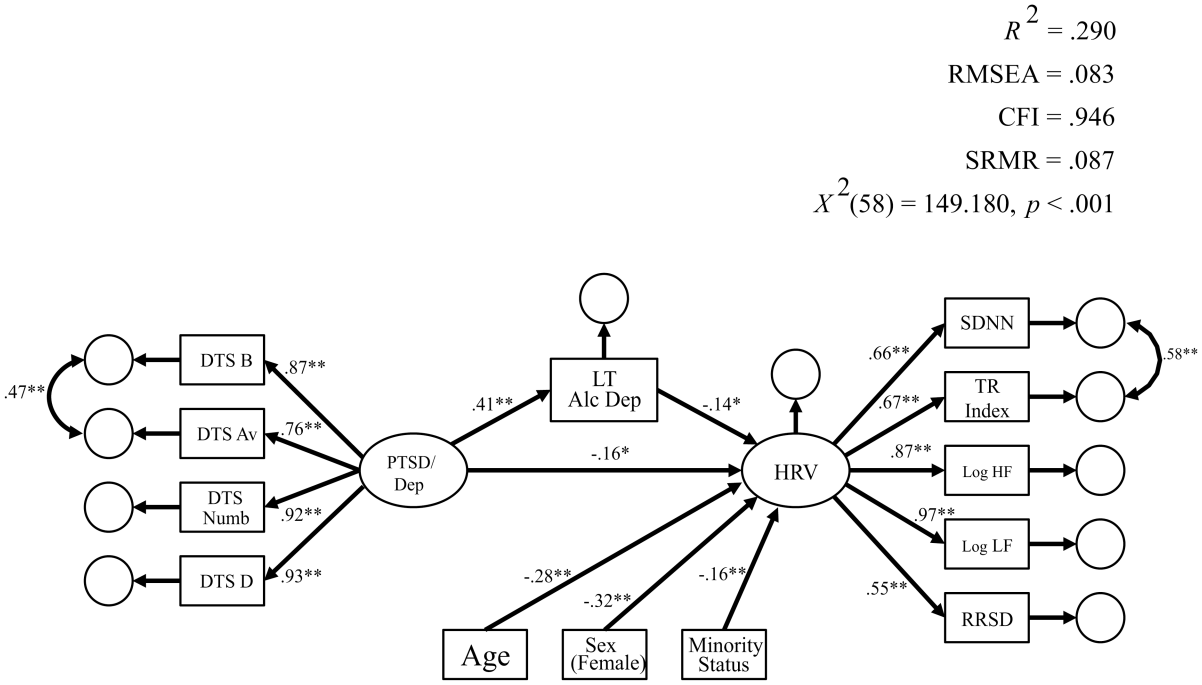

Figure 3. Latent variable Model C depicts the standardized indirect effect of PTSD/ symptoms on HRV *via* lifetime alcohol dependence

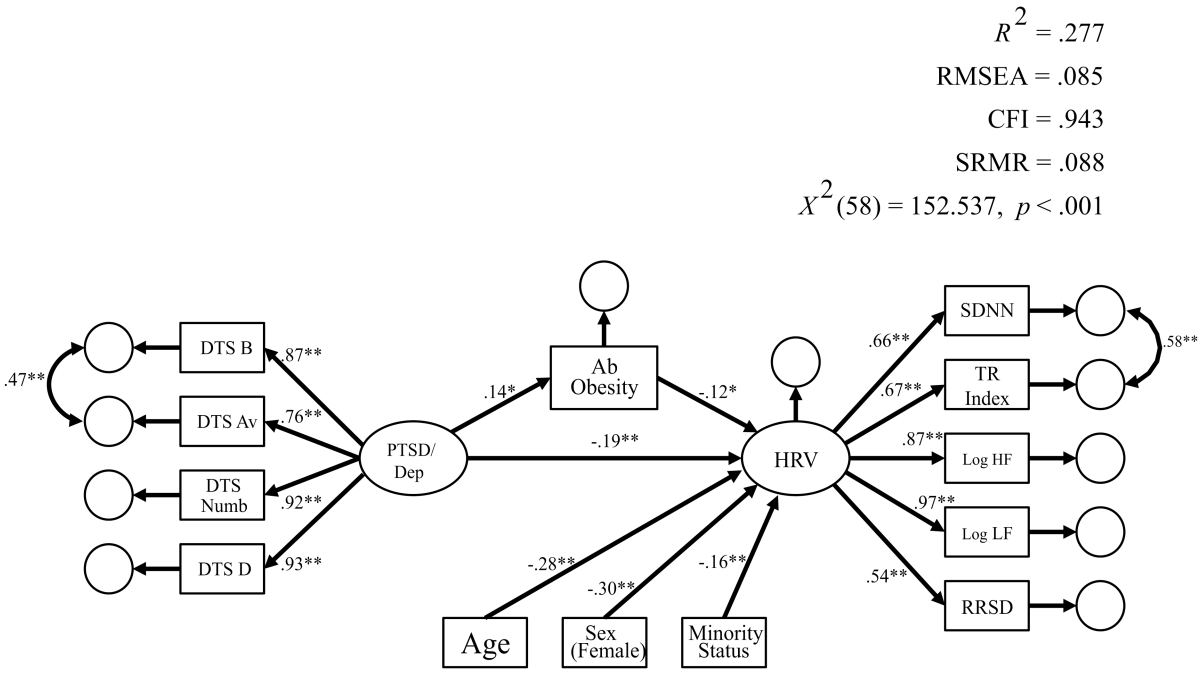

Figure 4. Latent variable Model D depicts the standardized indirect effect of PTSD/ symptoms on HRV *via* abdominal obesity

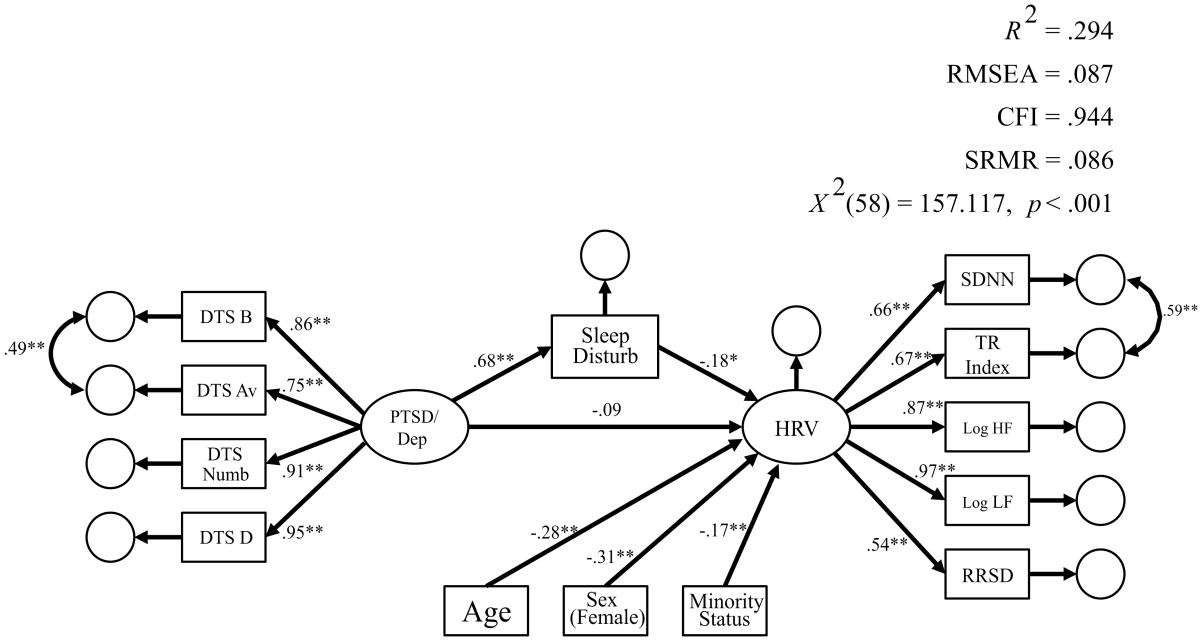

Figure 5. Latent variable Model E depicts the standardized indirect effect of PTSD/ symptoms on HRV *via* sleep disturbance

Table 1
Participant Characteristics and HRV by PTSD Status

	PTSD (n = 107)	Non-PTSD (n = 120)	Test of Difference	Effect Size
Age	30.79 (5.31)	28.01 (5.53)	$t(225) = 3.86$, $p < .001$	Cohen's $d = 0.53$
Sex (Female)	49 (46%)	63 (53%)	$X^2(1) = 1.02$, $p = .313$	$OR_{PTSD} = 0.76$
Minority Status	65 (61%)	60 (50%)	$X^2(1) = 2.64$, $p = .104$	$OR_{PTSD} = 1.55$
Veterans	49 (46%)	26 (22%)	$X^2(1) = 14.88$, $p < .001$	$OR_{PTSD} = 3.05$
Current MDD	27 (25%)	0 (0%)	$X^2(1) = 34.37$, $p < .001$	-
Anti-hypertensives[a]	7 (7%)	6 (5%)	$X^2(1) = 0.25$, $p = .618$	$OR_{PTSD} = 1.33$
Cholesterol meds[b]	3 (3%)	2 (2%)	Fisher's Exact $p = .669$	$OR_{PTSD} = 1.70$
Diabetes	2 (2%)	2 (2%)	Fisher's Exact $p > .999$	$OR_{PTSD} = 1.12$
Smoking	1.85 (1.72)	0.88 (1.47)	$t(225) = 4.57$, $p < .001$	Cohen's $d = 0.61$
LT Alc Dep	48 (45%)	18 (15%)	$X^2(1) = 24.46$, $p < .001$	$OR_{PTSD} = 4.61$
Ab Obesity	60 (56%)	63 (53%)	$X^2(1) = 0.29$, $p = .590$	$OR_{PTSD} = 1.16$
Sleep Disturbance	9.58 (3.39)	5.18 (3.05)	$t(225) = 10.29$, $p < .001$	Cohen's $d = 1.37$
HRV Indices				
SDNN	135.40 (41.97)	150.10 (49.51)	$t(223) = 2.37$, $p = .019$	Cohen's $d = 0.32$
TR Index	37.10 (12.95)	41.26 (13.17)	$t(223) = 2.38$, $p = .018$	Cohen's $d = 0.32$
Log HF	5.72 (1.12)	6.22 (1.31)	$t(213) = 2.96$, $p = .003$	Cohen's $d = 0.40$
Log LF	6.76 (0.85)	7.11 (0.91)	$t(213) = 2.92$, $p = .004$	Cohen's $d = 0.40$
RRSD	53.42 (30.23)	54.90 (29.72)	$t(222) = 0.37$, $p = .713$	Cohen's $d = 0.05$

Note. Means/frequencies and standard deviations/ percentages (in parentheses). All values based on non-imputed data. MDD = major depressive disorder; Ab Obesity = abdominal obesity; SDNN = standard deviation of all normal R-R intervals; TR index = triangular index; Log LF = log-transformed low-frequency power, Log HF = log-transformed high-frequency power, RRSD = standard deviation of normal-to-normal R-R intervals.

[a] Anti-hypertensives includes beta blockers, prazosin, ACE inhibitors, and calcium-channel blockers.

[b] Cholestorol medication includes statins and fibrates.

Table 2

Means and Intercorrelations of PTSD Symptoms for Sample (N = *227*)

	Mean (SD)	DTS Av	DTS Numb	DTS D	SDNN	TR Index	Log HF	Log LF	RRSD
DTS B	11.52 (11.34)	.81**	.80**	.81**	-.23**	-.24**	-.25**	-.26**	-.16*
DTS Av	4.99 (5.45)		.71**	.71**	-.23**	-.27**	-.19**	-.21**	-.14*
DTS Numb	11.13 (11.80)		-	.85**	-.19**	-.18**	-.25**	-.23**	-.12†
DTS D	14.48 (12.72)			-	-.23**	-.28**	-.26**	-.25**	-.16*

Note. DTS B = Davidson Trauma Scale (DTS) re-experiencing (B cluster) subscale; DTS Av = DTS avoidance subscale; DTS Numb = DTS numbing subscale; DTS D = DTS hyperarousal (D cluster) subscale; SDNN = standard deviation of all normal R-R intervals; TR index = triangular index; Log LF = log-transformed low-frequency power, Log HF = log-transformed high-frequency power, RRSD = standard deviation of normal-to-normal R-R intervals.

† $p < .10$,

* $p < .05$,

** $p < .01$

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