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Orthostatic Hypotension in Young Adults with and without Posttraumatic Stress Disorder

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Abstract

OBJECTIVE—The purpose of this research is (1) to evaluate differences in orthostatic hypotension (OH) among young adults with and without posttraumatic stress disorder (PTSD) and (2) to examine whether group differences may be attributable to behavioral risk factors frequently associated with PTSD.

METHOD—Volunteers and U.S. veterans 18–39 years old (n = 222) completed a semi-structured interview assessment of PTSD status. Direct measurements were obtained for supine and standing systolic and diastolic blood pressure at study visits as well as height and weight, from which body-mass index (BMI) was calculated.

RESULTS—After controlling for use of psychotropic medications, a logistic regression model revealed that PTSD status was positively associated with OH, such that participants with PTSD were at 4.51 greater odds of having OH than control participants. Moreover, this effect was partially mediated by lifetime alcohol dependence (bootstrapped 95% CI: −0.83 to −0.20).

CONCLUSIONS—Overall, PTSD may pose a significant risk for OH amongst younger adults. In the present sample, this relationship was primarily driven by the disproportionately high history of

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alcohol dependence amongst individuals with PTSD. These results suggest that traditional therapy for PTSD be coupled with treatment for alcohol dependency where applicable to reap both psychological and physiological benefits.

Keywords

Physical health; alcohol dependence; trauma exposure; posttraumatic stress

Introduction

Posttraumatic stress disorder (PTSD) frequently co-occurs with physical health symptoms and comorbid medical conditions (Buckley, Green, & Schnurr, 1994). For instance, prospective studies have demonstrated that individuals with PTSD are at an increased risk for developing a number of chronic medical conditions, particularly cardiovascular illnesses (Boscarino, 2008). Much of this association may be due to impaired autonomic control, whereby individuals with PTSD exhibit repeated, extended periods of stress response (McFarlane, 2010). Nevertheless, few studies have examined the relationship between PTSD and orthostatic hypotension (OH), a condition defined by weak vagal response to postural change. In the present study, the link between PTSD and OH was investigated along with potential behavioral mediators—namely elevated weight, cigarette smoking, and history of alcohol dependence.

When individuals are exposed to psychological trauma, they run the risk of developing PTSD, a disorder characterized by persistent re-experiencing of the traumatic event, avoidance of stimuli associated with that event, and increased arousal (American Psychiatric Association, 2000). These symptoms have long been known to convey autonomic dysregulation, such as elevated heart rate and blood pressure both at baseline (Blanchard, 1990) and in response to stressors (Pitman, Orr, Forgue, de Jong, & Claiborn, 1987). Furthermore, heart-rate variability, which is an important indicator of autonomic control, is often reduced in individuals with PTSD (e.g., Dennis et al., 2014; Shah et al., 2013).

Not only is low heart-rate variability an indicator of health risk, such as immune dysfunction, osteoporosis, arthritis, Alzheimer’s disease, and some cancers (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002), it can have deleterious effects on cardiovascular health. Low heart-rate variability is a risk factor for arrhythmia, heart disease, and cardiac arrest (Bigger et al., 1992; Dekker et al., 2000). It can also accelerate artherosclerosis (Gorman & Sloan, 2000), resulting in increased variability in blood pressure, which is itself an independent risk factor for coronary artery disease (Sloan, Shapiro, Bagiella, Myers, & Gorman, 1999).

Considering that heart-rate variability is inversely related to blood-pressure variability, it is possible that PTSD is also associated with OH, whereby inadequate sympathetically mediated vasoconstriction renders individuals vulnerable to substantial drops in blood pressure (Rose et al., 2006). Indeed, one study found that Gulf War veterans with PTSD experienced greater immediate and delayed reductions in systolic (SBP)—but not diastolic (DBP)—blood pressure than controls after standing up from a supine position (Peckerman et al., 2003). Furthermore, in a recent study (Hinton et al., 2009) among survivors of the

Cambodian genocide, those participants reporting sudden drops in blood pressure resulting in dizziness, fainting, etc. also had more frequent and intense PTSD symptoms than participants reporting no such drops. Nevertheless, no study to our knowledge has yet examined whether PTSD is associated with a higher incidence of OH as it is conventionally defined (i.e., a decrease in SBP by at least 20mm Hg and/or a decrease in DBP by at least 10mm HG within three minutes of standing up from a prostrate position) (Kaufmann, 1996). Thus, the primary goal of the current study was to test this association.

Aside from irregularities in autonomic functioning, PTSD is also associated with a number of behavioral risk factors. Individuals with PTSD are more likely than unaffected individuals to be obese (Pagoto et al., 2011), smoke frequently and heavily (Fu et al., 2007), and abuse alcohol (McFarlane, 1998). Each of these risk factors have in turn been independently associated with OH (Eigenbrodt et al., 2000; Metzler et al., 2012; Rose et al., 2000), suggesting that the relationship between PTSD and OH may be at least partially due to the behavioral health risks that frequently accompany PTSD. Thus, a second goal of this study was to test the hypothesis that body-mass index (BMI), cigarette consumption, and history of alcohol dependence partially mediate the association between PTSD and OH. In the present study, young adults in particular were sampled to assess the early health risks posed by PTSD and behavioral health risks associated with it.

Methods

Participants & Procedure

Following Institutional Review Board approval of the protocol and obtaining consent, a volunteer sample of 222 participants consisting of young community adults and U.S. military veterans (18–39 years old) responded to fliers in hospital clinics and waiting rooms as well as online ads such as Craigslist. Participants completed study procedures in an ongoing larger study of metabolic and cardiovascular parameters in young adults. Participants completed a semi-structured interview assessment of PTSD status. Smoking status and demographic information, including age, gender, racial minority status, lifetime alcohol dependence, pack year history, the use of blood-pressure medications—namely beta blockers, calcium-channel blockers, ACE inhibitors, or other hypertension medications—and the use of psychotropics that are associated with OH—including prazosin, trazodone, venlafaxine, tricyclic antidepressants (TCAs), and selective serotonin reuptake inhibitors (SSRIs)–were collected (see Table 1). Criteria for study exclusion included (a) organic mental disorder, (b) schizophrenia, (c) bipolar I mixed state or bipolar II, (d) lifetime but not current PTSD, (e) current substance abuse/dependence, (f) current major depressive disorder without PTSD, (g) pregnancy, (h) AIDS or reported positive HIV status, and (i) uncontrolled medical condition (e.g., kidney or liver failure). Direct measurements were obtained for supine and standing systolic and diastolic blood pressure as well as height and weight, from which body-mass index (BMI) was calculated (see Table 1). Data collection for the larger study occurred over three or four study visits between August 2008 and December 2012.

Measures

Clinician Administered PTSD Scale (CAPS)—The CAPS is a 17-item structured interview that corresponds to the DSM-IV criteria for PTSD. This instrument yields a total score and subscale scores for re-experiencing, avoidance/numbing and hyperarousal symptoms (Blake et al., 1995). The presence of each symptom was determined using the frequency ≥ 1/intensity ≥ 2 rule (Blake et al., 1995; Weathers et al., 2001) requiring each symptom to be endorsed at a frequency of at least once per month and intensity of at least moderate distress or impairment in functioning.

Body mass index (BMI)—Height in inches was self-reported and weight in pounds was determined by a scale. BMI was calculated by dividing weight (pounds) by height (inches) squared and multiplying the quotient by 703.

Orthostatic hypotension (OH)—Consistent with convention, we defined OH as a decrease in systolic blood pressure by at least 20mm Hg and/or a decrease in diastolic blood pressure by at least 10mm Hg from lying down to standing up (Kaufmann, 1996). Participants rested for five minutes in supine position on a bed, and their blood pressure was taken on the right arm with an Accutorr V blood pressure machine. They then stood upright for 60 seconds after which their blood pressure was measured again on the right arm. Heart rate was not measured at this time.

Data Analytic Plan

Hierarchical logit-linked logistic regression was used to assess the association between PTSD and OH while controlling for demographic variables. To test mediation of the relationship between PTSD and OH by each behavioral health risk variable, a bootstrapped 95% confidence interval (CI) of the indirect effect of PTSD status, controlling for all other independent variables, was generated from probit-linked logistic models employing 5,000 re-samples. Probit-linked, as opposed to logit-linked, models were used because of the presence of at least one dichotomous mediator (see MacKinnon, 2008). The advantage of bootstrapped CIs over conventional tests, such as Sobel's *z*, is that they take into account the positive skew inherent to indirect effects (for further discussion, see Preacher & Hayes, 2008). As such, bootstrapped mediation analysis is more powerful than conventional tests, with mediation deemed significant when the resulting CI does not span 0.

Results

At baseline and supine, participants with PTSD had higher DBP (M = 74.34)—but not SBP (M = 125.40)—than participants without PTSD (DBP M = 70.02, $t(220) = 3.93$, $p < .01$, Cohen's $d = 0.53$; SBP M = 122.0, $t(220) = 1.30$, $p = .19$, Cohen's $d = 0.18$). Ten participants with PTSD and two without PTSD had OH. Given the low prevalence of OH, Firth's (1993) penalized likelihood method was used rather than the maximum likelihood method, and profile penalized 95% confidence intervals are reported.

To limit over-specification of the models, demographic and baseline variables were initially entered into unadjusted logistic regression models of OH to determine the selection of

control variables for the adjusted models. Only the use of psychotropics approached significance (see Table 2) and was retained as a covariate in subsequent models. Next, PTSD status and use of psychotropics were entered into a single model of OH. As hypothesized, participants with PTSD were at 4.51 greater odds of having OH than control participants. In a second step, three behavioral health risk variables—BMI, pack years, and lifetime alcohol dependence—representing potential mediators of the association between PTSD status and OH were entered. PTSD status was no longer significant with the addition of these behavioral health risk variables, yet only lifetime alcohol dependence remained significant, suggesting possible mediation.

The indirect effect of PTSD on OH *via* BMI, pack years, and lifetime alcohol dependence was tested using bootstrapped 95% confidence intervals. Only the indirect effect for lifetime alcohol dependence was significant (bootstrapped 95% CI: −0.83 to −0.20, $p < .01$), accounting for 63% of the total effect of PTSD on OH. No significant mediation was detected for BMI (bootstrapped 95% CI: −0.14 to 0.11, $p = .88$) or pack year history (bootstrapped 95% CI: −0.20 to 0.17, $p = .70$).

Discussion

Chronic PTSD is associated with poor health outcomes, including increased cardiovascular morbidity and mortality (Boscarino, 2008). Prior research has suggested that rates of orthostatic hypotension may be elevated amongst individuals with PTSD (Hinton et al., 2009; Peckerman et al., 2003). In this study, we identified a link between PTSD and OH, as it is clinically defined. To our knowledge this is the first study to do so. We further demonstrated that a strong history of alcohol dependence amongst individuals with PTSD accounted for much of that link.

That the association between PTSD and OH was found in a sample of young adults is both compelling and alarming. OH is a known risk factor for cardiovascular disease (Verwoert, 2008) and death (Rose et al., 2006; Verwoert, 2008) in middle-aged and older populations. Although less research has focused on OH amongst young adults, one study employing age-adjusted analyses found that even young adults with OH faced a higher risk of 5-year mortality than those without OH (Davis et al., 1987). Thus, the present findings highlight the urgency of identifying and treating PTSD relatively early.

The finding that history of alcohol dependence accounted for such a substantial portion of the association between PTSD and OH—63% by our reckoning—is consistent with previous work. Indeed, the link between alcohol abuse and OH is well-established (e.g., Asensio et al., 2011; Mathias, 1995). The present results thus serve to emphasize the importance of efforts to treat alcohol dependence amongst young adults affected by PTSD. Considering recent evidence that cognitive behavioral therapy may be sufficient to improve SBP response to orthostatic challenge amongst individuals with PTSD (Hinton et al., 2009), combined treatment of PTSD symptoms and alcohol abuse and dependence could reap considerable psychological and physiological benefits. Certainly more research in this area is warranted.

Counter to our expectations, no mediation effect was observed for either BMI or cigarette consumption (pack years). Perhaps this is because the process by which elevated BMI and smoking influence OH is less accelerated than that observed for alcohol abuse. It is worth noting that the observed impact of BMI (e.g., Masaki et al., 1998; Rose et al., 2000; Shin, Abbott, Lee, Kim, & Kimm, 2004) and smoking (e.g., Applegate et al., 1991; Eigenbrodt et al., 2000; Rose et al., 2000; Rutan et al., 1992) on risk of OH has not been particularly consistent. Thus, the association of BMI and smoking with OH may in fact be tenuous or subject to other as-yet-unknown factors. In any case, further clarification of these associations is needed.

As compelling as these findings are, the present study has several limitations that should be highlighted. For one, the sample size was relatively small and the incidence of OH, low. Consequently, the number of statistical controls we could implement was limited. For instance, a larger study of PTSD and OH would benefit by controlling for psychotropic medications, many of which can influence OH (Montastruc, Laborie, Bagheri, & Senard, 1997), by drug class and dosage.

Future research in this area would also benefit from a more granular assessment of alcohol consumption. The exclusion of individuals with current alcohol abuse or dependence and the dichotomous nature of the lifetime-alcohol-dependence variable likely diminished the predictive power of that construct in the present study. Nevertheless, history of alcohol dependence proved a strong mediator in the present analyses, demonstrating the importance of replicating these findings with higher-resolution measures of alcohol consumption.

In sum, the current study demonstrates that PTSD may be associated with OH, even amongst young adults, highlighting the potential need to screen for this condition. That history of alcohol dependence accounted for much of this association emphasizes the importance of coupling traditional PTSD therapies with alcohol treatment. That said, future work should be directed toward replicating these findings and testing whether the proposed treatments can effectively ameliorate OH.

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Table I

Participant Characteristics by PTSD Status

	PTSD (n = 102)	Control (n = 120)	Significance Test
Age	31.60 (5.31)	28.15 (5.52)	$t(220) = 4.72$, $p < .01$
Females	44 (43%)	64 (53%)	$X^2(1) = 2.29$, $p = .13$
Minorities	61 (60%)	63 (53%)	$X^2(1) = 1.19$, $p = .27$
Using BP meds	9 (9%)	6 (5%)	$X^2(1) = 1.28$, $p = .26$
Using Psychotropics	47 (46%)	11 (9%)	$X^2(1) = 38.92$, $p < .01$
BMI	30.13 (6.29)	28.07 (6.53)	$t(207) = 2.29$, $p = .02$
Pack years	6.30 (8.37)	2.54 (5.85)	$t(207) = 3.88$, $p < .01$
LT alcohol dep.	48 (47%)	18 (15%)	$X^2(1) = 27.12$, $p < .01$

Note. Counts and proportions/means and standard deviations (in parentheses).

Table II

Logistic Models of Orthostatic Hypotension

	Unadjusted Model		Step 1 (Adjusted Model)		Step 2 (Adjusted Model)	
	Est. (SE)	**OR (95% CI)**	**Est. (SE)**	**OR (95% CI)**	**Est. (SE)**	**OR (95% CI)**
Intercept	–		−2.90** (0.37)		−2.90* (1.42)	
Age	0.05 (0.05)	1.05 (0.95 – 1.17)	–	–	–	–
Male	0.31 (0.30)	1.85 (0.59 – 6.60)	–	–	–	–
Minority	0.40 (0.32)	2.24 (0.69 – 9.19)	–	–	–	–
Use of BP meds	0.62 (0.39)	3.48 (0.63 – 13.74)	–	–	–	–
Use of Psychotropics	0.55† (0.29)	3.02 (0.97 – 9.42)	0.24 (0.31)	1.62 (0.48 – 5.84)	0.10 (0.31)	1.23 (0.35 – 4.24)
PTSD	–	–	0.75* (0.38)	4.51 (1.12 – 25.35)	0.47 (0.38)	2.55 (0.61 – 14.92)
BMI	–	–	–	–	0.00 (0.05)	0.99 (0.91 – 1.05)
Pack years	–	–	–	–	−0.01 (0.04)	1.00 (0.91 – 1.10)
Alcohol Dep.	–	–	–	–	1.06** (0.37)	8.29 (2.04 – 47.73)

† $p < .10$,

* $p < .05$,

** $p < .01$