Published in final edited form as:

J Psychosom Res. 2014 July ; 77(1): 45–50. doi:10.1016/j.jpsychores.2014.05.001.

Behavioral Health Mediators of the Link between Posttraumatic Stress Disorder and Dyslipidemia

Paul A. Dennis, Ph.D.¹, Christi S. Ulmer, Ph.D.^{1,3,4}, Patrick S. Calhoun, Ph.D.^{1,2,3,4}, Andrew Sherwood, Ph.D.⁴, Lana L. Watkins, Ph.D.⁴, Michelle F. Dennis, B.A.^{1,4}, and Jean C. Beckham, Ph.D.^{1,2,4}

¹Durham Veterans Affairs Medical Center, Durham, NC, 27705, USA

²Veterans Affairs Mid-Atlantic Region Mental Illness Research, Education, and Clinical Center, Durham, NC 27705, USA

³Veterans Affairs Center for Health Services Research in Primary Care, Durham, NC, 27705, USA

⁴Department of Psychiatry and Behavioral Sciences, Duke University Medical Center, Durham, NC 27705, USA

Abstract

Objectives—Posttraumatic stress disorder (PTSD) has been linked to dyslipidemia, which is a major risk factor for coronary artery disease. Although this link is thought to reflect response to heightened stress, behavioral health risks, including smoking, alcohol dependence, and poor sleep quality, may mediate the relationship between PTSD and dyslipidemia.

Methods—To test this hypothesis, serum lipid levels were collected from 220 young adults (18–39 years old), 103 of whom were diagnosed with PTSD.

Results—PTSD and associated depressive symptoms were negatively related to high-density lipoprotein cholesterol (HDL-C), $p = .04$, and positively related to triglyceride (TG) levels, $p = .04$. Both associations were mediated by cigarette consumption and poor sleep quality, the latter of which accounted for 83% and 93% of the effect of PTSD and depression on HDL-C and TG, respectively.

Corresponding Author: Jean Beckham, Ph.D., Durham Veterans Affairs Medical Center, 508 Fulton St. (116 B), Durham, NC 27705 (USA), Phone: (919) 286-0411 Ext. 7973, Fax: (919) 416-5922, jean.beckham@va.gov or beckham@duke.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Declaration of Interests

The content of this work is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health, the Department of Veterans Affairs, or the United States Government. The authors have no competing interests to report.

Statement of Ethics

All procedures followed were in accordance with the ethical standards of the Duke University Medical Center and Durham Veterans Affairs Institutional Review Boards and with the Helsinki Declaration of 1975, as revised in 2000 (5). Informed consent was obtained from all patients for their inclusion in the study.

Conclusions—These results complement recent findings highlighting the prominence of health behaviors in linking PTSD with cardiovascular risk.

Keywords

posttraumatic stress disorder; dyslipidemia; cholesterol; depression; cigarette smoking; sleep

Introduction

Posttraumatic stress disorder (PTSD) is a disabling mental-health condition that affects individuals exposed to a traumatic experience. As with other anxiety disorders, PTSD conveys an array of cardiovascular risks, including hypertension (1), dyslipidemia (2–7), and coronary artery disease (8). Although the association between PTSD and cardiovascular risk is frequently attributed to symptoms of hyperarousal and elevated anxiety (9), recent evidence (10, 11) suggests that behavioral health risks linked with PTSD—namely smoking, drinking, and sleep quality—could account for much of that association. In this study, serum lipid was collected from 220 younger adults (18- to 39-years-old) with and without PTSD to determine whether the relationship between PTSD and cholesterol and triglyceride (TG) levels may be attributable to the higher rates of smoking, drinking, and poor sleep quality associated with PTSD.

Stress has long been linked to poor cardiovascular outcomes (12). Individuals with PTSD, a disorder characterized by persistent re-experiencing of a traumatic event, avoidance of trauma stimuli, emotional numbing, and hyperarousal (13), face an elevated risk of coronary heart disease (14), myocardial infarction (15), stroke (16), and ultimately cardiac death (14). Much of this risk is thought to be attributable to autonomic dysregulation, whereby individuals with PTSD exhibit repeated and extended episodes of stress response (17). As a result, individuals with PTSD often have higher heart rate and blood pressure than unaffected individuals, both at baseline (18) and in response to startle (19).

To fuel states of elevated arousal, the sympathetic nervous system stimulates the process of lipolysis, whereby free fatty acids are released into the blood stream thereby increasing concentrations of serum-lipid levels (20). Indeed, during periods of high stress, such as medical school examination week (21, 22), or following stressful events, such as public speaking (23) or surgery (24), elevated serum lipid levels have been observed (25). Accumulating evidence suggests that PTSD is also associated with abnormal serum lipid concentrations. For example, Kagan and colleagues (2) found that Vietnam veterans receiving inpatient care for PTSD had significantly higher total cholesterol, low-density lipoprotein cholesterol (LDL-C), and TG levels, and lower levels of high-density lipoprotein cholesterol (HDL-C) than Vietnam veterans receiving inpatient care for substance abuse. Similar PTSD-related disparities in lipid profiles have since been found in veterans of the Bosnian War (3, 4, 6), American military veterans (7), and Brazilian police officers (5).

Beyond the cardiovascular risks posed by autonomic dysregulation, PTSD has been associated with a number of behavioral risk factors that may indirectly link PTSD with poorer cardiovascular outcomes. Individuals with PTSD are more likely to smoke and do so more heavily than individuals without PTSD (26), abuse alcohol (27), and report nightmare-

related sleep disturbance (28). Evidence from recent studies suggests that these behaviors to some degree account for the relation of PTSD with orthostatic hypotension (11) and reduced heart-rate variability (10). Considering that smoking (29, 30), alcohol abuse (31), and fragmented sleep (32) are independently associated with dyslipidemia, it is possible that these three behavioral variables mediate the relationship between PTSD and lipid levels.

This study tested the hypothesis that the link between PTSD and abnormal lipid levels is partially mediated by cigarette consumption, lifetime alcohol dependency, and sleep quality. Younger adults (i.e., under 40 years of age) were targeted in order to quantify the early health risks posed by PTSD and associated psychopathology. Because depression is often comorbid with PTSD (33) and has been independently associated with cholesterol and TG levels (34), latent variable modeling was used to also consider the contribution of depression to the association between PTSD and lipid levels. This allowed us to evaluate the overlapping effects of PTSD and depression on lipid levels rather than their relatively inscrutable unique effects.

Three sets of hypotheses were tested: 1) PTSD and depressive symptoms would be associated with lower HDL cholesterol, higher LDL cholesterol, and higher TG levels; 2) PTSD and depressive symptoms would be associated with greater cigarette consumption, higher rates of lifetime alcohol dependence, and poorer sleep quality; and 3) each of these behavioral risk factors would partially mediate the association between PTSD and depressive symptoms and lipid levels. That is, accounting for each of these behavioral health risks would render the association of PTSD and depressive symptoms with each of the three lipid levels non-significant.

Methods

Participants

A sample of 220 participants (18–39 years old; 108 females), consisting of 74 U.S. military veterans, was recruited as part of an ongoing study of risk factors associated with metabolic and cardiovascular disorders amongst young adults (the YoungHost study). 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).

Measures

Posttraumatic stress disorder—PTSD status was assessed using the Clinician Administered PTSD Scale (CAPS) (35). Presence of each symptom was determined using the frequency > 1/intensity > 2 rule, which requires a symptom to be endorsed at a frequency of at least once per month with at least moderate impairment or distress to be counted as present. The CAPS interview has excellent reliability and validity within multiple trauma populations and is widely accepted as the state-of-the-art method for PTSD assessment (36). The Davidson Trauma Scale (DTS) (37) was used 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 4-item scales (38), respectively. Given the hypothesis that sleep quality would mediate the association between PTSD symptoms and dyslipidemia, item 12 (“Have you had trouble falling asleep or staying asleep?”) was not included in the calculation of the hyper-arousal cluster score as is otherwise typically the case. 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 along a 5-point Likert scale. Cluster scores were calculated by summing frequency and intensity scores for associated items.

Depressive symptoms—The Beck Depression Inventory-II (BDI) (39) is a 21-item questionnaire that assesses the severity of depressive symptoms along two dimensions: cognitive/affective and somatic. The cognitive/affective subscale was calculated as the sum of 8 items and ranged from 0 to 24. As with the DTS, an item addressing changes in respondents’ sleeping pattern (item 16), which usually contributes to the somatic subscale, was not included in the present analyses. As such, the somatic subscale was calculated as the sum of the remaining 12 items, with a range of 0 to 36.

Cigarette consumption—Cigarette consumption was operationalized based on participants’ responses to the Fagerström Test for Nicotine Dependence (40): non-smokers were assigned a value of 0; past—but not present—smokers a value of 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) (41) was used to assess Axis I disorders, including lifetime alcohol dependence and current major depressive disorder (MDD).

Sleep quality—The Pittsburgh Sleep Quality Index (42) is a self-report questionnaire that assesses seven domains of sleep quality via 19 items. Global sleep quality scores, calculated as the sum of the seven domain scores, were used to quantify sleep quality. Scores may range from 0 to 21, with those exceeding 5 indicating poor sleep quality.

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 also recorded. Anthropometric measures were taken, including height and weight, from which body-mass index (BMI) was calculated. Serum samples were taken on a subsequent session visit to assess overnight fasting lipid profile.

Analytic Plan

Given high co-occurrence of PTSD and depression (33), latent variable modeling was used to test the hypothesis that PTSD and related depressive symptoms would be associated with lipid levels, with subsequent models conducted to test the mediation hypotheses.

Specifically, a latent variable was specified to capture PTSD symptoms along with associated depressive symptoms using the four DTS scales and two BDI scales. The adequacy of this variable was determined prior to further modeling using standard fit criteria: root mean square error of approximation (RMSEA) $< .10$, comparative fit index (CFI) $> .90$, and standardized root mean square residual (SRMR) $< .08$. Chi-squares were also consulted to facilitate model comparison. Factor scores were generated from the measurement model and were used in subsequent analyses.

To analyze lipid (i.e., HDL-C, LDL-C, and TG) levels as a function of PTSD and depressive symptoms as well as to test the mediation hypotheses, multivariate gamma regression models with an inverse power link were conducted. Gamma regression is appropriate for modeling positively skewed continuous data, and its use in analyzing lipid levels is consistent with previous research (43, 44). Follow-up univariate models were analyzed where indicated by the multivariate models. All models included age, gender, minority status, and BMI as control variables. Age and BMI were centered and the PTSD/depression variable was z-scored to facilitate interpretation. Multivariate gamma regression was conducted using PROC GLIMMIX, available with SAS v9.2.

Results

PTSD and Depressive Symptoms

One-hundred three participants (47% of the sample) met CAPS criteria for PTSD. Nearly half of these were veterans (see Table 1). A quarter of participants with PTSD also met criteria for current MDD. As expected, PTSD and depressive symptoms were highly intercorrelated (see Table 2), justifying the use of latent variable modeling to approximate the psychological disturbance underlying both sets of symptoms.

An initial measurement model of the PTSD/depression latent variable indicated a poor fit on two out of three indices: RMSEA = .28, CFI = .88, SRMR = .07, $\chi^2(9) = 161.89$, $p < .01$. Examination of the residual covariance matrix, however, suggested that the residual errors for the two BDI scales were correlated as were the re-experiencing and avoidance-symptom scales. Indeed, specifying these correlations significantly improved the model, $\chi^2(2) = 135.66$, $p < .01$, rendering an adequate fit: RMSEA = .12, CFI = .98, SRMR = .02, $\chi^2(7) = 26.23$, $p < .01$. The resulting latent variable is depicted in Figure 1. Factor scores for the latent variable demonstrated satisfactory construct validity: The resulting variable was strongly correlated with both current PTSD, $r(218) = .71$, $p < .01$, and current MDD, $r(218) = .49$, $p < .01$. As predicted, the PTSD/depression variable was also correlated with cigarette consumption, $r(218) = .45$, $p < .01$, lifetime alcohol dependence, $r(218) = .38$, $p < .01$, and sleep quality, $r(218) = .67$, $p < .01$.

Lipid Levels

The multivariate model of HDL-C, LDL-C, and TG revealed significant main effects for age, $F(3, 642) = 9.31$, $p < .01$, gender, $F(3, 642) = 12.88$, $p < .01$, minority status, $F(3, 642) = 17.60$, $p < .01$, and BMI, $F(3, 642) = 14.71$, $p < .01$. The multivariate effect of PTSD and depressive symptoms was also significant, $F(3, 642) = 2.92$, $p = .03$. According to the

univariate models, PTSD and depressive symptoms were negatively associated with HDL-C (see Table 3, Model 1) and positively associated with TG (see Table 4, Model 1), partially supporting the first hypothesis. However the relationship between PTSD and depressive symptoms and LDL-C was non-significant (see Table 5, Model 1).

To test mediation of the relationship between PTSD and depressive symptoms and lipid levels by cigarette consumption, a second multivariate model was specified with cigarette consumption added as a predictor. Age, $F(3, 639) = 8.16, p < .01$, gender, $F(3, 639) = 11.78, p < .01$, minority status, $F(3, 639) = 17.73, p < .01$, and BMI, $F(3, 639) = 14.76, p < .01$, remained significant predictors of lipid levels. However, the effect of PTSD and depressive symptoms was no longer significant, $F(3, 639) = 0.62, p = .60$, in the presence of cigarette consumption, $F(3, 639) = 4.61, p < .01$, suggesting mediation. Inspection of the univariate models revealed that PTSD and depressive symptoms were not significantly associated with either HDL-C (see Table 3, Model 2) or TG levels (see Table 4, Model 2) when cigarette consumption was included in those models. In fact, the addition of cigarette consumption resulted in a reduction of the PTSD/depression effect on HDL-C and TG by 54% and 45%, respectively.

To test mediation of the relationship between PTSD and depressive symptoms and lipid levels by history of alcohol dependence, another multivariate model was specified with lifetime alcohol dependence added as a predictor in place of cigarette consumption. Age, $F(3, 639) = 8.54, p < .01$, gender, $F(3, 639) = 12.20, p < .01$, minority status, $F(3, 639) = 17.13, p < .01$, and BMI, $F(3, 639) = 14.53, p < .01$, remained significant predictors of lipid levels. Although the effect of PTSD and depressive symptoms was somewhat attenuated in this model, $F(3, 639) = 2.37, p = .07$, the effect of lifetime alcohol dependence failed to reach significance, $F(3, 639) = 0.13, p = .94$. Thus, there was no evidence that history of alcohol dependence was a mediator.

Lastly, to test mediation of the relationship between PTSD and depressive symptoms and lipid levels by sleep quality, a multivariate model was specified in which sleep quality was entered as the potential mediator. Age, $F(3, 639) = 8.81, p < .01$, gender, $F(3, 639) = 10.97, p < .01$, minority status, $F(3, 639) = 15.20, p < .01$, and BMI, $F(3, 639) = 12.79, p < .01$, were significant predictors of lipid levels. Moreover, the effect of PTSD and depressive symptoms was not significant, $F(3, 639) = 0.03, p = .99$, in the presence of sleep quality, $F(3, 639) = 2.82, p = .04$. According to the univariate models, the effect of PTSD and depressive symptoms on HDL-C (see Table 3, Model 3) and TG levels (see Table 4, Model 3) was attenuated when sleep quality was controlled. In fact, the addition of sleep quality to the models resulted in a reduction of the PTSD/depression effect on HDL-C and TG by 83% and 93%, respectively.¹

¹When total scores on the DTS were substituted for PTSD/depression factor scores, nearly identical results were achieved. That is, total DTS score was negatively associated with HDL-C, $p = .04$, positively associated with TG, $p = .04$, and unassociated with LDL-C, $p = .38$. Moreover, the former two associations appeared to be mediated by cigarette consumption and sleep quality, such that cigarette consumption explained 54% of the effect PTSD on HDL-C and 45% of its effect on TG, whereas sleep quality explained 78% of the effect of PTSD on HDL-C and 81% of its effect on TG. PTSD/depression factor scores were in fact highly correlated with total DTS scores, $r(218) = .98, p < .01$.

Discussion

This study examined the link between PTSD and associated depressive symptoms and dyslipidemia in a sample of younger adults. To our knowledge, this is the first study to further evaluate whether smoking, alcohol use, or poor sleep quality mediates that association. As with previous research (2–7), PTSD was negatively related to HDL-C levels and positively related to TG levels, although no relationship was found between PTSD and LDL-C levels. A novel finding was that both of these relationships were almost completely explained by increased cigarette consumption and sleep quality.

These findings are consistent with recent evidence that behavioral health risks may account for a substantial portion of the cardiovascular risk linked to PTSD, particularly amongst younger adults. In two other subsets of the present study sample, the link between PTSD and orthostatic blood pressure (11) as well as that between PTSD and reduced heart-rate variability (10) was substantially attenuated when smoking, history of alcohol dependence, and sleep quality were controlled. Similarly, in a recent study of 2,981 Dutch adults, the association between anxiety severity and dyslipidemia was partially mediated by tobacco use, albeit not alcohol use (34).

Together these findings highlight the substantial behavioral health risks associated with PTSD along with the potential health benefits of minimizing said risks. Individuals with PTSD are at least 2 times as likely as non-affected individuals to smoke (45, 46) and 1.5 times as likely to report sleep disturbance (47). Nevertheless, a number of studies have demonstrated the benefits of treating these behaviors. For example, according to Maeda and colleagues' (48) meta-analysis, smoking cessation resulted in the normalization of HDL-C levels, although not LDL-C and TG, with change evident in as little of 17 days (49). Similarly, therapies aimed at alleviating apnea-related sleep disturbance have been shown to be effective at increasing HDL-C levels (50, 51) and decreasing LDL-C and TG levels (51) within eight months. Thus, an emphasis on targeting these behavioral health risks amongst individuals with PTSD is warranted.

Counter to our expectations, history of alcohol dependence was unrelated to dyslipidemia. Therefore, no mediation effect was detected for this risk factor. Given that the link between alcohol abuse and dyslipidemia is well-established (31), it is likely that the failure to observe an association between alcohol use and dyslipidemia is due to the exclusion of individuals with current alcohol abuse or dependence. It is also possible that our measurement of alcohol consumption was not sufficiently high-resolution to capture that link. In future research, a more precise measure of current alcohol consumption may be more likely to demonstrate mediation of the association between PTSD and dyslipidemia.

Three other limitations to the present study are also worth noting. First, the differences in HDL-C, LDL-C, and TG by PTSD status (see Table 1) were rather modest in comparison to other similar studies (2–5). These small group differences may be attributable to the relatively high level of trauma experienced by the control group. In the present sample, controls had a mean DTS score of 17.15, well above the 9.33 observed in a normative sample of individuals with either no or sub-threshold PTSD (52). Indeed, in one study that

compared the lipid levels of combat veterans with PTSD to those with MDD rather than unaffected controls (6), similarly small group differences were observed. Nevertheless, just 7% of participants with PTSD ($n = 7$) in the current study presented with a lipid profile considered normal or optimal by National Cholesterol Education Program guidelines (53) (HDL-C >60 mg/dl, LDL-C < 100 mg/dl, and TG < 150 mg/dl), whereas 17% of participants without PTSD ($n = 20$) did so, $\chi^2(2) = 5.40, p = .02$. In light of recent evidence that non-optimal lipid levels in early adulthood is a significant predictor of coronary artery calcification in middle age, independent of later lipid levels (54), the present results highlight the need to intervene early, before even small discrepancies are exacerbated over time.

A second limitation is that the sampling of younger adults conservatively limits the generalizability of the present results. For instance, dyslipidemia amongst older adults with PTSD may be less attributable to behavioral health risks and more tied to the cumulative effect of years of elevated stress on the hypothalamic-pituitary-adrenal axis. Conversely, years of smoking, drinking, and poor sleep could render an even greater mediation effect amongst older adults with PTSD than evident in this study. This remains an empirical question, one that we hope to answer with further research. Nevertheless, that behavioral factors play such a major role in the development of dyslipidemia in our young sample sheds light on the multi-faceted and relatively immediate risks posed by PTSD.

A third limitation was the absence of an objective measure of sleep. It is possible that participants with more severe psychiatric symptoms over-reported their impaired sleep quality, in which case the substantial mediation effect of sleep quality may be inflated. In addition, the possible presence of sleep apnea, known to increase risk of cardiovascular disease, was not evaluated in our study sample. Regardless, the effect of subjectively impaired sleep quality on HDL-C and TG levels independent of PTSD and depressive symptoms was quite robust, suggesting at least partial mediation. In any case, future research should incorporate objective measures of sleep, for instance *via* sleep actigraphy, to validate these results.

A notable innovation in the present study was the use of latent variable modeling to capture PTSD and depressive symptoms. Because depression is often comorbid with PTSD in the most severe cases of the latter (33) and is independently associated with dyslipidemia (34), it neither made sense to ignore the effect of depression on serum lipid levels nor to examine the relatively inscrutable unique effect of PTSD on lipid levels after controlling for depression. With latent variable modeling, we were able to highlight the underlying features of PTSD and depressive symptoms that are associated with dyslipidemia, thereby efficiently analyzing the influence of both sets of symptoms in the same model. In previous work (10), we made similar use of a PTSD/depression latent variable to demonstrate its relationship to reduced heart-rate variability. This technique may represent a productive approach for analyzing the overlapping influence of PTSD and depressive symptoms on various outcomes.

In sum, the results of this study further emphasize the detrimental effects of PTSD on health, particularly cardiovascular disease risk, even among younger adults. PTSD and related

depressive symptoms were significantly associated with reduced HDL-C and elevated TG levels, both of which are major risk factors for coronary artery disease (55). The finding that these associations were almost entirely explained by elevated smoking levels and poor subjective sleep quality complements recent research (10, 11) on the cardiovascular risks posed by PTSD by highlighting the behavioral risks associated with PTSD. These findings also suggest that interventions aimed at reducing smoking and improving sleep, as well as alleviating psychiatric symptoms, should feature prominently in the treatment of PTSD.

Acknowledgments

Preparation of this work was supported by the National Institute of Mental Health (2R01MH062482), the Durham, NC Veterans Affairs Medical Center; and the Department of Veterans Affairs office of Research and Development Clinical Science.

REFERENCES

1. Player MS, Peterson LE. Anxiety disorders, hypertension, and cardiovascular risk: A review. *International Journal of Psychiatry in Medicine*. 2011; 41:365–377. [PubMed: 22238841]
2. Kagan BL, Leskin G, Haas B, Wilkins J, Foy D. Elevated lipid levels in Vietnam veterans with chronic posttraumatic stress disorder. *Biological Psychiatry*. 1999; 45:374–377. [PubMed: 10023518]
3. Karlović D, Buljan D, Martinac M, Marcinko D. Serum lipid concentrations in Croatian veterans with post-traumatic stress disorder, post-traumatic stress disorder comorbid with major depressive disorder, or major depressive disorder. *Journal of Korean Medical Science*. 2004; 19:431–436. [PubMed: 15201512]
4. Karlović D, Martinac M, Buljan D, Zorić Z. Relationship between serum lipid concentrations and posttraumatic stress disorder symptoms in soldiers with combat experiences. *Acta Medica Okayama*. 2004; 58:3–28.
5. Maia DB, Marmar CR, Mendlowicz MV, Metzler T, Nóbrega A, Peres MC, Figueira I. Abnormal serum lipid profile in Brazilian police officers with post-traumatic stress disorder. *Journal of Affective Disorders*. 2008; 107:259–263. [PubMed: 17888517]
6. Solter V, Thaller V, Karlović D, Crnković D. Elevated serum lipids in veterans with combat-related chronic posttraumatic stress disorder. *Clinical Sciences*. 2002; 43:685–689.
7. Trief PM, Ouimette P, Wade M, Shanahan P, Weinstock RS. Post-traumatic stress disorder and diabetes: Co-morbidity and outcomes in a male veterans sample. *Journal of Behavioral Medicine*. 2006; 29:411–418. [PubMed: 16865552]
8. Vaccarino V, Goldberg J, Rooks C, Shah AJ, Veledar E, Faber TL, Bremner JD. Post-traumatic stress disorder and incidence of coronary heart disease: A twin study. *Journal of the American College of Cardiology*. 2013; 62:970–978. [PubMed: 23810885]
9. Bedi US, Arora R. Cardiovascular manifestations of posttraumatic stress disorder. *Journal of the National Medical Association*. 2007; 99:642–649. [PubMed: 17595933]
10. Dennis PA, Watkins L, Calhoun PS, Oddone A, Sherwood A, Dennis MF, Beckham JC. Posttraumatic stress and heart-rate variability: An examination of the mediating role of behavioral health risks. Manuscript submitted for publication. 2014
11. Oddone A, Dennis PA, Calhoun PS, Watkins L, Sherwood A, Dedert EA, Beckham JC. Orthostatic hypotension in young adults with and without posttraumatic stress disorder. *Nicotine and Tobacco Research*. (in press).
12. Krantz DS, Manuck SB. Acute psychophysiological reactivity and risk of cardiovascular disease: A review and methodological critique. *Psychological Bulletin*. 1984; 96:435–464. [PubMed: 6393178]
13. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. rev. Washington, DC: American Psychiatric Press; 2000.

14. Kubzansky LD, Koenen KC, Spiro A, Vokonas PS, Sparrow D. Prospective study of the posttraumatic stress disorder symptoms and coronary heart disease in the Normative Aging Study. *Archives of General Psychiatry*. 2007; 64:109–116. [PubMed: 17199060]
15. Boscarino JA, Chang J. Electrocardiogram abnormalities among men with stress-related psychiatric disorders: Implications for coronary heart disease and clinical research. *Annals of Behavioral Medicine*. 1999; 21:227–234. [PubMed: 10626030]
16. Lauterbach D, Vora R, Rakow M. The relationship between posttraumatic stress disorder and self-reported health problems. *Psychosomatic Medicine*. 2005; 67:939–947. [PubMed: 16314599]
17. McFarlane AC. The long-term costs of traumatic stress: Intertwined physical and psychological consequences. *World Psychiatry*. 2010; 9:3–10. [PubMed: 20148146]
18. Blanchard EB. Elevated basal levels of cardiovascular responses in Vietnam veterans with PTSD: A health problem in the making? *Journal of Anxiety Disorders*. 1990; 4:233–237.
19. Pitman RK, Orr SP, Forgue DF, de Jong JB, Claiborn JM. Psychophysiological assessment of posttraumatic stress disorder imagery in Vietnam combat veterans. *Archives of General Psychiatry*. 1987; 44:970–975. [PubMed: 3675137]
20. Arner P. Human fat cell lipolysis: Biochemistry, regulation and clinical role. *Best Practice & Research Clinical Endocrinology & Metabolism*. 2005; 19:471–482. [PubMed: 16311212]
21. Grundy S, Griffin A. Relationship of periodic mental stress to serum lipoprotein and cholesterol levels. *Journal of the American Medical Association*. 1959; 171:1794–1796. [PubMed: 13829859]
22. Thomas C, Murphy E. Further studies on cholesterol levels in the John Hopkins medical students: The effect of stress at examination. *Journal of Chronic Diseases*. 1958; 8:661–668. [PubMed: 13598777]
23. Taggart P, Carruthers M, Somerville W. Electrocardiogram plasma catecholamines and lipids and their modification by oxprenolol when speaking before an audience. *Lancet*. 1973; 2:341–346. [PubMed: 4124526]
24. Zarafonitis CJ, Seifter J, Baeder D, Kalas JP. Lipid mobilization as a consequence of surgical stress. *American Journal of the Medical Sciences*. 1959; 237:418–433. [PubMed: 13637070]
25. Dimsdale JE, Herd JA. Variability of plasma lipids in response to emotional arousal. *Psychosomatic Medicine*. 1982; 44:413–430. [PubMed: 6758015]
26. Beckham JC, Roodman AA, Shipley RH, Hertzberg MA, Cunha GH, Kudler HS, Fairbank JA. Smoking in Vietnam combat veterans with post-traumatic stress disorder. *Journal of Traumatic Stress*. 1995; 8:461–472. [PubMed: 7582610]
27. McFarlane AC. Epidemiological evidence about the relationship between PTSD and alcohol abuse: the nature of the association. *Addictive Behaviors*. 1998; 23:813–825. [PubMed: 9801718]
28. Morrison AR. Sleep disturbance as the hallmark of posttraumatic stress disorder. *American Journal of Psychiatry*. 1989; 146:697–707.
29. Craig WY, Palomaki GE, Haddow JE. Cigarette smoking and serum lipid and lipoprotein concentrations: An analysis of published data. *British Medical Journal*. 1989; 298:784–788. [PubMed: 2496857]
30. Rose JC, Vijayarani H, Rani HN. A comparative study on of lipid profile of smokers and non-smokers. *Indian Journal of Biotechnology and Biochemistry*. 2013; 1:63–72.
31. Baraona, E.; Lieber, CS. Alcohol and lipids. In: Galanter, M., editor. *Recent Developments in Alcoholism*. New York: Kluwer Academic Publishers; 1998. p. 97-134.
32. Ekstedt M, Åkerstedt T, Söderström M. Microarousals during sleep are associated with increased levels of lipids, cortisol, and blood pressure. *European Respiratory Journal*. 2004; 27:121–127.
33. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*. 1995; 52:1048–1060. [PubMed: 7492257]
34. van Reedt Dortland AK, Vreeburg SA, Giltay EJ, Licht CM, Vogelzangs N, van Veen T, Zitman FG. The impact of stress systems and lifestyle on dyslipidemia and obesity in anxiety and depression. *Psychoneuroendocrinology*. 2013; 38:209–218. [PubMed: 22717171]
35. Blake DD, Weathers FW, Nagy LM, Kaloupek DG, Gusman FD, Charney DS, Keane TM. The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress*. 1995; 8:75–90. [PubMed: 7712061]

36. Weathers FW, Keane TM, Davidson JR. Clinician-Administered PTSD Scale: A review of the first ten years of research. *Depression and Anxiety*. 2001; 13:132–156. [PubMed: 11387733]
37. Davidson JR, Book SW, Colket JT, Tupler LA, Roth S, David D, Feldman ME. Assessment of a new self-rating scale for post-traumatic stress disorder. *Psychological Medicine*. 1997; 27:153–160. [PubMed: 9122295]
38. McDonald SD, Beckham JC, Morey R, Marx C, Tupler LA, Calhoun PS. Factorial invariance of posttraumatic stress disorder symptoms across three veteran samples. *Journal of Traumatic Stress*. 2008; 21:309–317. [PubMed: 18553409]
39. Beck, AT.; Steer, RA.; Brown, GK. *Manual for Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation; 1996.
40. Heatherston TF, Kozlowski LT, Frecker RC, Fagerström K. The Fagerström Test for Nicotine Dependence: A revision of the Fagerström Tolerance Questionnaire. *British Journal of Addiction*. 1991; 86:1119–1127. [PubMed: 1932883]
41. First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JB. *Structured clinical interview for DSM-IV axis I disorders, clinician version (SCID-CV)*. Washington, DC: American Psychiatric Association; 1997.
42. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research*. 1989; 28:193–213. [PubMed: 2748771]
43. Hetz SP, Latimer AE, Buchholz AC, Martin Ginis KA. Increased participation in activities of daily living is associated with lower cholesterol levels in people with spinal cord injury. *Archives of Physical Medicine and Rehabilitation*. 2009; 90:1755–1759. [PubMed: 19801067]
44. Iwane MK, Wittes J, Collins DE, Rubins HB. Simultaneous screening for three correlated lipids in the VA HDL Intervention Trial. *Journal of Clinical Epidemiology*. 1999; 52:685–693. [PubMed: 10391662]
45. Hapke U, Schumann A, Rumpf HJ, John U, Konerding U, Meyer C. Association of smoking and nicotine dependence with trauma and posttraumatic stress disorder in a general population sample. *Journal of Nervous and Mental Disease*. 2005; 193:843–846. [PubMed: 16319709]
46. Kalman D, Morissette SB, George TP. Co-morbidity of smoking in patients with psychiatric and substance use disorders. *American Journal of Addiction*. 2005; 14:106–123.
47. Maher MJ, Rego SA, Asnis GM. Sleep disturbances in patients with post-traumatic stress disorder: Epidemiology, impact and approaches to management. *CNS Drugs*. 2006; 20:567–590. [PubMed: 16800716]
48. Maeda K, Noguchi Y, Fukui T. The effects of cessation from cigarette smoking on the lipid and lipoprotein profiles: A meta-analysis. *Preventative Medicine*. 2003; 37:283–290.
49. Moffatt RJ, Stamford BA, Owens SG, Chitwood LF. Cessation from cigarette smoking: Lipoprotein changes in men and women. *Journal of Smoking-Related Disorders*. 1992; 3:11–19.
50. Börgel J, Sanner BM, Bittlinsky A, Keskin F, Bartels NK, Buechner N, Müggue A. Obstructive sleep apnoea and its therapy influence high-density lipoprotein cholesterol serum levels. *European Respiratory Journal*. 2006; 27:121–127. [PubMed: 16387944]
51. Chin K, Shimizu K, Nakamura T, Narai N, Masuzaki H, Ogawa Y, Ohi M. Changes in intra-abdominal visceral fat and serum leptin levels in patients with obstructive sleep apnea syndrome following nasal continuous positive airway pressure therapy. *Circulation*. 1999; 100:706–712. [PubMed: 10449691]
52. Davidson JRT, Tharwani HM, Connor KM. Davidson Trauma Scale (DTS): Normative scores in the general population and effect sizes in placebo-controlled SSRI trials. *Depression and Anxiety*. 2002; 15:75–78. [PubMed: 11891997]
53. Expert Panel on Detection, E. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on Detection, Evaluation, and Treatment of high blood cholesterol in adults (Adult Treatment Panel III). *JAMA: The Journal of the American Medical Association*. 2001; 285:2486–2497. [PubMed: 11368702]
54. Pletcher MJ, Bibbins-Domingo K, Liu K, Sidney S, Lin F, Vittinghoff E, Hulley SB. Non-optimal lipids commonly present in young adults and coronary calcium later in life: The Coronary Artery

- Risk Development in Young Adults (CARDIA) study. *Annals of Internal Medicine*. 2010; 153:137–146. [PubMed: 20679558]
55. Dahlen GH, Guyton JR, Attar M, Farmer JA, Kautz JA, Gotto AM. Association of levels of lipoprotein Lp(a), plasma lipids, and other lipoproteins with coronary artery disease documented by angiography. *Circulation*. 1986; 74:758–765. [PubMed: 2944670]

Highlights

- Fasting serum lipid levels of young adults with and without PTSD were assessed.
- PTSD was significantly associated with lower HDL-C and higher TG levels.
- Smoking accounted for around half of the association between PTSD and lipid levels.
- Poor sleep accounted for nearly the entire link between PTSD and lipid levels.

Figure 1.

PTSD/depression measurement model (standardized coefficients). BDI Cog/Aff = Beck Depression Inventory cognitive/affective subscale; BDI Som = somatic subscale; DTS B = Davidson Trauma Scale re-experiencing subscale; DTS Av = avoidance subscale; DTS Numb = numbing subscale; DTS D = hyper-arousal subscale.

Table 1

Participant Characteristics by PTSD Status

	PTSD (<i>n</i> = 103)	Non-PTSD (<i>n</i> = 117)	Test of Difference
Age	30.49 (5.48)	27.90 (5.52)	$t(218) = 3.48, p < .01$
Sex (Female)	47 (46%)	61 (52%)	$X^2(1) = 0.93, p = .34$
Minority Status	62 (60%)	58 (50%)	$X^2(1) = 2.49, p = .11$
Veterans	48 (47%)	26 (22%)	$X^2(1) = 14.59, p < .01$
Total DTS score	70.18 (64.07)	17.15 (12.74)	$t(218) = 14.18, p < .01$
Current MDD	26 (25%)	0 (0%)	$X^2(1) = 33.49, p < .01$
BMI	30.28 (7.89)	28.57 (6.81)	$t(218) = 1.72, p = .09$
Cholesterol Med ^a	3 (3%)	2 (2%)	Fisher's Exact $p = .67$
Diabetes	2 (2%)	2 (2%)	Fisher's Exact $p > .99$
Cig Consum			Fisher's Exact $p < .01$
0. non-smoker	38 (37%)	74 (63%)	
1. past smoker	17 (17%)	20 (17%)	
2. 10 cigarettes/day	24 (23%)	8 (7%)	
3. 11–20 cigarettes/day	21 (21%)	13 (11%)	
4. 21–30 cigarettes/day	2 (2%)	2 (2%)	
5. >30 cigarettes/day	1 (1%)	0 (0%)	
LT Alc Dep	45 (44%)	18 (15%)	$X^2(1) = 21.47, p < .01$
Sleep Quality	9.56 (3.53)	5.15 (3.02)	$t(218) = 10.00, p < .01$
HDL-C ^b	48.44 (11.53)	53.85 (16.51)	Wald $X^2(1) = 8.12, p < .01$
LDL-C ^b	112.40 (34.53)	106.80 (34.09)	Wald $X^2(1) = 1.54, p = .21$
TG ^b	101.80 (62.24)	99.60 (73.10)	Wald $X^2(1) = 0.09, p = .77$

Note. Means/frequencies and standard deviations/ percentages (in parentheses). For cigarette consumption, 0 = non-smoker; 1 = past—but not present—smoker; 2 = currently smokes 10 or fewer cigarettes/day; 3 = currently smokes 11 to 20 cigarettes/day; 4 = currently smokes 21 to 30 cigarettes/day; 5 = currently smokes over 30 cigarettes/day.

^aCholesterol medication includes statins and fibrates.

^bDifference test reported from gamma regression.

Table 2

Means and Inter correlations of PTSD and Depressive Symptoms

	Mean (SD)	BDI Som	DTS B	DTS Av	DTS Numb	DTS D
BDI Cog/Aff	4.01 (4.55)	.80	.61	.50	.71	.61
BDI Som	7.38 (7.18)	-	.61	.53	.76	.69
DTS B	11.56 (11.39)		-	.82	.80	.81
DTS Av	5.03 (5.49)			-	.70	.70
DTS Numb	11.13 (11.78)				-	.86
DTS D	10.90 (10.27)					-

Note. BDI Cog/Aff = Beck Depression Inventory cognitive/affective subscale; BDI Som = somatic subscale; DTS B = Davidson Trauma Scale re-experiencing subscale; DTS Av = avoidance subscale; DTS Numb = numbing subscale; DTS D = hyper-arousal subscale. All correlations were significant at $p < .01$.

Table 3
Univariate Gamma Models of Effect of PTSD and Depressive Symptoms on HDL Cholesterol

	Model 1			Model 2			Model 3		
	Coeff (SE)	Est Effect		Coeff (SE)	Est Effect		Coeff (SE)	Est Effect	
Intercept	0.019** (0.001)	26.36		0.019** (0.001)	26.13		0.019** (0.001)	26.35	
Age	0.000* (0.000)	0.99		0.000 [†] (0.000)	0.99		0.000 [†] (0.000)	0.99	
Gender (Male)	0.003** (0.001)	0.86		0.003** (0.001)	0.87		0.003** (0.001)	0.86	
Minority	-0.001* (0.001)	1.08		-0.001* (0.001)	1.08		-0.001 [†] (0.001)	1.07	
BMI	0.000** (0.000)	0.99		0.000** (0.000)	0.99		0.000** (0.000)	0.99	
PTSD/Dep Sx	0.008* (0.004)	0.70		0.004 (0.004)	0.84		0.001 (0.005)	0.93	
Cig Consum	-	-		0.001* (0.000)	0.96		-	-	
Sleep Quality	-	-		-	-		0.000* (0.000)	0.99	
Scale	16.521** (1.560)	-		17.016** (1.607)	-		16.828** (1.589)	-	

Note. The estimated effects represent the multiplicative effect on the dependent variable of a 1-unit increase in the independent variables. For instance, because the variable representing PTSD and associated depressive symptoms was standardized, a 1-unit increase is equivalent to a 1-standard deviation increase, which is in turn associated with a decrease in HDL cholesterol by 30% in Model 1.

[†] $p < .10$,

* $p < .05$,

** $p < .01$

Table 4

Univariate Gamma Models of Effect of PTSD and Depressive Symptoms on TG

	Model 1			Model 2			Model 3		
	Coeff (SE)	Est Effect		Coeff (SE)	Est Effect		Coeff (SE)	Est Effect	
Intercept	0.010** (0.001)	52.13		0.010** (0.001)	52.36		0.010** (0.001)	51.89	
Age	-0.000** (0.000)	1.02		-0.000** (0.000)	1.02		-0.000** (0.000)	1.02	
Gender (Male)	-0.003** (0.001)	1.34		-0.002** (0.001)	1.31		-0.002** (0.001)	1.30	
Minority	0.005** (0.001)	0.67		0.005** (0.001)	0.67		0.005** (0.001)	0.68	
BMI	-0.000** (0.000)	1.02		-0.000** (0.000)	1.02		-0.000** (0.000)	1.02	
PTSD/Dep Sx	-0.001* (0.000)	3.56		-0.000 (0.000)	1.66		-0.001 (0.005)	1.05	
Cig Consum	-	-		-0.001* (0.000)	1.06		-	-	
Sleep Quality	-	-		-	-		-0.000* (0.000)	1.02	
Scale	4.512** (0.415)	-		4.598** (0.424)	-		4.597** (0.423)	-	

Note. The estimated effects represent the multiplicative effect on the dependent variable of a 1-unit increase in the independent variables.

* $p < .05$,

** $p < .01$

Table 5
Univariate Gamma Models of Effect of PTSD and Depressive Symptoms on LDL Cholesterol

	Model 1			Model 2			Model 3		
	Coeff (SE)	Est Effect		Coeff (SE)	Est Effect		Coeff (SE)	Est Effect	
Intercept	0.009** (0.000)	54.75		0.009** (0.000)	54.90		0.009** (0.000)	54.72	
Age	-0.000** (0.000)	1.01		-0.000** (0.000)	1.01		-0.000** (0.000)	1.01	
Gender (Male)	-0.000 (0.000)	1.05		-0.000 (0.000)	1.04		-0.000 (0.000)	1.04	
Minority	0.001 (0.000)	0.95		0.001 (0.000)	0.95		0.001 (0.000)	0.95	
BMI	0.000 (0.000)	1.00		0.000 (0.000)	1.00		0.000 (0.000)	1.00	
PTSD/Dep Sx	-0.001 (0.002)	1.16		0.000 (0.000)	0.95		-0.000 (0.003)	1.00	
Cig Consum	-	-		-0.000 [†] (0.000)	1.03		-	-	
Sleep Quality	-	-		-	-		-0.000 (0.000)	1.00	
Scale	11.045** (1.038)	-		11.190** (1.051)	-		11.058** (1.039)	-	

Note. The estimated effects represent the multiplicative effect on the dependent variable of a 1-unit increase in the independent variables.

[†]
p < .10,
**
p < .01