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Posttraumatic stress disorder, alone or additively with early life adversity, is associated with obesity and cardiometabolic risk

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Abstract *Background and aims:* There is some evidence that posttraumatic stress disorder (PTSD) and early life adversity may influence metabolic outcomes such as obesity, diabetes, and cardiovascular disease. However, whether and how these interact is not clear.
Methods: We analyzed data from a cross-sectional and longitudinal study to determine how PTSD severity influences obesity, insulin sensitivity, and key measures and biomarkers of cardiovascular risk. We then looked at how PTSD and early life adversity may interact to impact these same outcomes.
Results: PTSD severity is associated with increasing risk of obesity, diabetes, and cardiovascular disease, with higher symptoms correlating with higher values of BMI, leptin, fibrinogen, and blood pressure, and lower values of insulin sensitivity. PTSD and early life adversity have an additive effect on these metabolic outcomes. The longitudinal study confirmed findings from the cross sectional study and showed that fat mass, leptin, CRP, sICAM-1, and sTNFRII were significantly increased with higher PTSD severity during a 2.5 year follow-up period.
Conclusions: Individuals with early life adversity and PTSD are at high risk and should be monitored carefully for obesity, insulin resistance, and cardiometabolic risk.
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Abbreviations: PTSD, posttraumatic stress disorder; BMI, body mass index; MetS, metabolic syndrome; ELA, early life adversity; sICAM-1, soluble intercellular adhesion molecule-1; sTNFRII, soluble tumor necrosis factor receptor II; BP, blood pressure; DBP, diastolic BP; SBP, systolic BP; HPA, hypothalamic-pituitary-adrenal; OGTT, oral glucose tolerance test; FBG, fasting blood glucose; BDI, Beck Depression Inventory; BMI, body mass index; WC, waist circumference; SiM, insulin sensitivity; TC, total cholesterol; CRP, c-reactive protein; PAI-1, plasminogen activator inhibitor-1; RIA, radioimmunoassay; ELISA, enzyme-linked immunosorbent assay; SD, standard deviation; SE, standard error of the mean; OGTT, oral glucose tolerance test; FFQ, food frequency questionnaire.

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Introduction

Posttraumatic stress disorder (PTSD), a psychological trauma-related disorder, is a relatively common occurrence in veterans, estimated at current rates between 15 and 19% with lifetime incidence rates of up to 30% [1], and in the general population at about 7.8% [2]. More recently, evidence has accumulated to suggest that PTSD increases the risk of developing metabolic disorders such as type 2 diabetes, dyslipidemia, obesity [3,4] and cardiovascular diseases [5]. Early life adversity (ELA), which includes emotional, physical, and sexual abuse or neglect before the age of 18, is also known to increase metabolic and cardiovascular disorders [6,7]. How these two factors, ELA and PTSD, may interact to impact metabolic outcomes is less clear.

Indeed, PTSD has been associated with higher body mass index (BMI), blood pressure (BP), and metabolic syndrome (MetS), even when compared to other psychiatric disorders [8]. When PTSD is comorbid with depression, the risk for MetS is drastically increased [9]. However, in a low income population, PTSD still predicted MetS even when depression, demographic factors, and antipsychotic use were controlled [4]. Further, PTSD symptom severity proved to be a predictor of MetS in veterans, controlling for other significant predictors, such as antipsychotic use [3]. Similarly, ELA has been found to increase central obesity and BMI controlling for established adult psychosocial and heath behavior risk factors [7,10]. ELA has also been seen to increase the risk of diabetes, cardiovascular disease, and premature death when controlling for potential confounders [6,7].

Disrupted activation of the hypothalamic-pituitary-adrenal (HPA) axis and increased activity of the sympathetic nervous system may lead to the metabolic and cardiovascular problems frequently seen in PTSD [11], similar to the proposed mechanisms of dysfunction resulting from ELA [12]. However, whether PTSD and ELA may combine or interact to alter BMI, insulin sensitivity, and hormonal outcomes remains unknown. Here, we performed a cross-sectional and a longitudinal study of a diverse community population to determine how the severity of PTSD symptoms, based on self-report, or probable PTSD (PTSD), may interact with ELA to alter metabolic outcomes and to better define how PTSD may impact biomarkers for adiposity, inflammation and insulin sensitivity to alter risk of diabetes, obesity, and cardiovascular disease.

Methods

Cross-sectional study: study population

We examined 158 adults cross-sectionally. Participants between the ages of 35 and 55 were recruited from the greater Boston area via advertisements to be representative of the general population in terms of socioeconomic status. Participants were White European Americans and Black/African Americans. We excluded individuals with a history of myocardial infarction or stroke, an active diagnosis of diabetes mellitus, active intravenous drug use, hepatitis, cirrhosis, dialysis, long-term steroid use, and/or current treatment for cancer or active infection.

The study was approved by the Institutional Review Board at the Judge Baker Children's Center (JBCC) and Beth Israel Deaconess Medical Center (BIDMC). Written informed consent was obtained from all participants.

Among 158 participants, 55 returned for a follow-up visit 2.5 years after the cross-sectional visit. Follow up sample size was limited by funding constraints. Procedures at the follow-up visit were identical to the cross-sectional visit. Biomarkers and anthropometric data were collected as described previously [7] and in Appendix 1.

Psychosocial data

Information on ELA, probable PTSD (PTSD) and psychosocial measurements was obtained via validated interviews and questionnaires at JBCC. An overall ELA score was created by multiplying the number of ELA$\times$ the overall severity of each ELA$\times$ the overall chronicity of ELA (chronic/acute) as described previously [7,10,13]. PTSD severity scores and subscale scores were measured with the UCLA PTSD scale [14,15]. For additional information on psychosocial data, see Appendix 2.

Statistical analysis

General characteristics of the study participants according to three categories ($Q_1 + Q_2$ vs. Q_3 vs. Q_4) of PTSD severity scores, with the first two quartiles ($Q_1 + Q_2$) collapsed and then the third (Q_3) and fourth quartiles (Q_4) presented separately, per standard epidemiology practices to correct for the skewedness of the data while still representing the distribution of the scores, were compared using ANOVA or chi-square test and presented as means (or geometric means) $\pm$SD or frequency (%). Normality of the distributions was assessed with frequency histograms and the Shapiro–Wilk test. The linear trend was calculated by simple linear regression analysis (continuous variables) or by linear-by-linear association (categorical variables). If there was a significant difference in continuous variables amongst the three groups, a *post-hoc* test using the Bonferroni method was performed for the comparison between two groups amongst three categories. Spearman's correlation analyses were used to compare PTSD severity scores and individual PTSD subscale severity scores with other variables. Cardiometabolic and biomarker values in the three PTSD severity score categories were compared using ANOVA or ANCOVA and presented as means (or geometric means) $\pm$ SE. Subsequent models were used to test comparisons while controlling for different potential confounders. For instance, Model 1 was uncorrected, while Model 2 was adjusted for age and gender.

Subsequent analyses were then performed to examine possible interactions between ELA and PTSD. Thus, we divided overall ELA scores as low ($T_1 + T_2$; 0–15) or high (T_3; 16–156) using the highest tertile point of 16 as a cut-off point. We divided the PTSD severity scores as lower

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