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Co-occurrence of posttraumatic stress disorder and cardiovascular disease among ethnic/racial groups in the United States

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Abstract

Objective—Trauma and/or symptoms of posttraumatic stress disorder (PTSD) have been linked to the onset of cardiovascular disease (CVD), but the exact mechanism has not been determined. We examine if the risk of CVD is different among those who have a history of trauma without PTSD symptoms, those who have experienced trauma and developed any symptoms of PTSD, and those with a PTSD diagnosis. Furthermore, we examine whether this association varies across ethnic/racial groups.

Methods—We used two datasets that form part of the Collaborative Psychiatric Epidemiology Surveys (CPES) - the National Latino and Asian American Study (NLAAS) and the National Comorbidity Survey Replication (NCS-R).

Results—We found an increased likelihood of cardiovascular events for those with a diagnosis of PTSD (OR = 2.10, 95% CI, 1.32-3.33) when compared to those who had not experienced trauma. We did not find an increased risk for those who had experienced trauma without symptoms or with sub-clinical symptoms of PTSD. The higher likelihood of having a cardiovascular event in those with PTSD was significant for non-Latino Whites (OR=1.86, 95%CI, 1.08 - 3.11), Latinos (OR = 1.94 95% CI 1.04-3.62) and non-Latino Blacks (OR=3.73, 95%CI, 1.76-7.91, but not for Asian respondents.

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Conflicting Interests

All the authors have completed the journal's copyright transfer agreement that includes a section on potential conflict of interest in which all the authors declare to have no conflicts of interests.

Conclusions—The constellation of symptoms defining PTSD diagnosis reflect adverse reactions to traumatic events and indicate that complex responses to traumatic events may be a risk factor for CVD.

Keywords

posttraumatic stress disorder; cardiovascular disease; ethnicity/racial; national sample

INTRODUCTION

The experience of trauma and Posttraumatic Stress Disorder (PTSD) symptoms are both associated with somatic diseases of the digestive, integumentary, respiratory, and cardiovascular systems (1, 2). However, neither trauma history nor PTSD diagnosis are considered risk factors in cardiovascular disease (CVD; having suffered myocardial infarction, stroke, or hypertension) management guidelines (3, 4). For people with trauma exposure, environmental triggers of previous trauma can elicit chronic stress responses that alter crucial bodily processes and can lead to disease (5). Trauma is defined as an events(s) that threaten someone's life or body, and results in short-term feelings of fear, helplessness, or horror. PTSD refers to the burden of stress experienced and attributed to past traumatic events (5). According to the Diagnostic Statistical Manual of Mental Disorders (DSM-5), PTSD manifests in physiological and psychological pathologies that span four clusters of symptoms: intrusive thoughts or memories or re-experiencing of the traumatic event, avoidance of triggers, negative alterations in mood or cognitions, and increased arousal symptoms (6, 7).

Several studies link trauma exposure and/or symptoms of PTSD with the onset of CVD, but the exact mechanism is unknown. The likelihood of developing cardiovascular risks, such as dyslipidemia, diabetes, or obesity, is higher for those who have been exposed to trauma or who have post-traumatic symptoms (6, 8, 9), and thus possibly explain the link between CVD to PTSD, which has been associated with an increased risk of cardiovascular events, like myocardial infarction, angina, and stroke (10, 11).

Previous research has shown that individuals without trauma exposure have the lowest risk of CVD, followed by those with trauma exposure, among which individuals with PTSD are at greatest risk (12). The more PTSD symptoms experienced (more than 4), the greater the risk of cardiovascular events (11). If trauma occurs early in life, there is a higher risk of several chronic conditions (13) and of cardiovascular events occurring earlier in life (14). Furthermore, a greater number of childhood traumas correlates to a greater risk for chronic disease (15, 16).

Some studies indicate that trauma exposure or PTSD symptomatology constitutes a direct risk factor for CVD (2, 6), as a chronic stressor that generates physiological changes through hyperarousal of the autonomic nervous system and neuroendocrine system(17). Other studies suggest an indirect relationship between trauma or PTSD and CVD, mediated by behavioral factors, like smoking, sleep disturbance, food addiction, and alcohol abuse, that increase the risk of CVD (6, 18). Independent of PTSD, depression can lead to deteriorated health in trauma-exposed individuals (19). Depression can cause changes in the circulatory,

endocrine, and immune systems, which in turn alter the physiology of the coronary artery through the release of norepinephrine and cortisol (15).

McEwen and Stellar first proposed allostasis, the body's adaptation to external stressors, as a psychobiological mechanism to explain the relationship between trauma/PTSD symptoms and CVD (20). With chronic stressors, allostasis is prolonged, causing long-term activation of neuroendocrine responses that can negatively impact health (19, 20). Response to previous trauma can increase allostatic load by chronically activating the hypothalamic-pituitary axis (HPA) and the sympathetic nervous system (19), leading to long-term lowered or elevated levels of norepinephrine and cortisol, respectively (5, 19–21). Dysfunctional regulation of the HPA and the nervous system results in increased blood pressure and lipid levels (6). Neuroendocrine system fluctuations can increase immune function and induce overactive platelet function, resulting in the onset of atherosclerosis, another risk factor for CVD (6, 22).

Investigations of the link between PTSD and CVD have been conducted with specific populations of trauma survivors, including veterans, victims of terrorism, or certain professionals, like police or medical personnel (22–24). Race and ethnicity have been identified as possible confounders in previous studies because their samples have been composed primarily of non-Latino Whites (22), and thus racial/ethnic variation in the association between PTSD and CVD risk has not been adequately characterized (25).

Our aim is to explore whether the risk of CVD is different among those who have not experienced trauma, those who have a history of trauma without PTSD symptoms, those who have experienced trauma and developed any symptoms of PTSD, and those who have a diagnosis of PTSD. We also investigate whether these associations differ across ethnic/racial groups.

METHODS

Data

We utilized data from two samples of the Collaborative Psychiatric Epidemiology Surveys (CPES) pooled dataset - the National Latino and Asian American Study (NLAAS) and the National Comorbidity Survey Replication (NCS-R) (26–29). We excluded the National Survey of American Life (NSAL) sample, as we did not have data on cardiovascular disease. The studies, conducted between 2001 and 2003, share a common sampling strategy, allowing data to be treated as a single, nationally representative study (27). The sampling weights are inversely proportional to the selection probabilities, and are used in survey analysis for population level inferences (27). The data consist of a sample of those over the age of 18 in the non-institutionalized population of the contiguous United States. The sample (n=10,165) is comprised of 4,180 non-Latino Whites, 3,081 Latinos, 2,178 Asians, and 717 non-Latino Blacks. We only included participants of the NCS-R who completed the long survey and all participants from the NLAAS study.

The sample was drawn as household clusters and weighted to represent the U.S. population. Weighted response rates from the NLAAS (with interviews conducted in English, Spanish,

Mandarin, Tagalog, and Vietnamese) were 75.5% for Latinos and 65.6% for Asians (29). The response rate in the NCS-R (conducted in English) was 70.9%. The standard errors (SEs) of the estimates for all analyses consider the complex sample design. Study methods were approved by the Institutional Review Boards of all participating institutions and written informed consent was obtained from all participants.

Measures

PTSD—*PTSD* DSM-4 diagnoses were based on Version 3.0 of the Composite International Diagnostic Interview (CIDI) (30), a fully structured lay interview that generates diagnoses per the criteria of both the World Health Organization's International Classification of Diseases, 10th Revision (31) and the American Psychiatric Association's DSM, Fourth Edition Revised (DSM-4) (32). Disorders assessed included anxiety disorders (panic disorder, generalized anxiety disorder, agoraphobia without panic disorder, social phobia, posttraumatic stress disorder), mood disorders (major depressive disorder, dysthymic disorder) and substance use disorders (alcohol and drug abuse and dependence). As described elsewhere (33), generally good concordance was found between DSM-4 diagnoses based on the CIDI and those based on blinded clinical reappraisal interviews with the Structured Clinical Interview for DSM-4 (SCID) (34). However, previous studies suggest that the CIDI presents problems for assessing PTSD among Latinos (35). The CIDI quality was thus enhanced by including several methodologic improvements based on findings from the debriefing interviews (29, 30). Because CIDI assessed PTSD age-of onset retrospectively, special question sequence was added to improve accuracy of reporting. This specific sequence started with questions designed to highlight the importance of correct response: *"can you remember your exact age the very first time when you had (the symptom)."* Participants who answered "no" were then probed for a bound of uncertainty by asking the earliest age they could remember having the disorder. Onset was set at the upper end of the bound of uncertainty. Previous research has shown that this approach yields more plausible onset distributions than standard onset questions (36, 37). In our analyses, we assigned respondents to four categories that represented the range of trauma exposure and PTSD symptoms: no trauma exposure; trauma exposure without PTSD symptoms; trauma exposure and reported PTSD symptoms (but no diagnosis); and trauma exposure with PTSD diagnosis. To standardize PTSD diagnoses across the two surveys, reported reactions were based only on the worst traumatic event (as defined by the respondent). A list of the assessed traumatic events is provided (see Text, Supplemental Digital Content 1).

Cardiovascular disease—CVD was defined as having reported suffering myocardial infarction (AMI), stroke (CVA), or hypertension (HTA). The information was obtained through self-report, but respondents were asked whether they had ever received the medical diagnosis from a doctor.

We also conducted sensitivity analyses where CVD excluded hypertension to determine if results varied. Because around 60% of people who reported AMI and/or CVA also reported having hypertension, there is not enough sample to test the correlation of trauma and PTSD separately for each of the cardiovascular dysfunctions, especially for AMI and stroke.

Clinical Factors—Since both the risk of PTSD and CVD increase in the presence of other psychiatric disorders, we adjusted for the presence of major depressive disorder, generalized anxiety disorder, and substance use disorder based on DSM-4 diagnoses from the WMH-CIDI. We also adjusted for other variables that the literature has associated with increased cardiovascular risk, including diabetes, tobacco use, and body mass index (BMI).

Sociodemographic Factors—Four categories for race/ethnicity were obtained from self-reported data consistent with U.S. Census definitions (non-Latino White, non-Latino Black, Latino and Asian). As the largest group, non-Latino White was used as the reference category. Nativity was coded as a dichotomous variable (born in the U.S./immigrant), with born in the U.S. as the reference category. Sex was coded using a dichotomous variable (male/female) with male as the reference category. Age was a continuous variable. Marital status was categorized as married, never married, and widowed/separated/divorced, with married as the reference category. Education level was categorized based on the number of years of education (≤11, 12, 13-15 and ≥16) with less than 11 years of education as the reference category.

Social Support—We measured social support using selected survey items that were equivalent across the NLAAS and NCS-R. Participants were asked to report how often they spoke by phone or spent time with family or friends, as well as the degree to which they sought help or discussed concerns with family or friends. Both scales were transformed into a continuous variable with a range from 0 to 1, with higher scores indicating greater support.

Statistical Analyses—We described sociodemographic, clinical, and social support factors as well as the pattern of heart disease and trauma exposure for each disaggregated ethnic/racial subsample. To investigate the relation between traumatic experience and subsequent development of CVD, we restricted the sample by excluding 314 participants who reported having CVD before their exposure to trauma (n=9,842). Rao-Scott statistics from the Pearson χ^2 test were used for testing racial/ethnic group differences. We then reported the prevalence rate of trauma and/or CVD across the four ethnic/racial groups in the full sample (n=10,165) and tested for mean differences in the prevalence rates across groups, using Wald test statistics. We assessed the relation of trauma/PTSD to the likelihood of having CVD in a benchmark logistic model that adjusted for sociodemographic characteristics, clinical, and social factors, using the restricted sample. The likelihood of having CVD was estimated for three groups – a group who had experienced trauma and reported no PTSD symptoms, a group who had experienced trauma and reported some PTSD symptoms, and a group who had experienced trauma and were classified as having a PTSD diagnosis (Model 1). This relation was further evaluated using different model specifications where known cardiovascular risk factors (BMI, current smoker, and diabetes; Model 2) and psychiatric conditions commonly associated with PTSD (major depressive disorder, generalized anxiety disorder, and substance abuse disorder; Model 3) were added into the benchmark model. Odds ratios of Trauma/PTSD severity categories were pair-wise compared. Significance of difference in the odds ratios were assessed using P-values from Wald test. Next, we present the results of our most complete model specification (the full model), which included an interaction term between PTSD diagnosis and each racial/ethnic

group. Our sensitivity analyses further tested two separate three-way interactions using the full model specification, i.e., interact PTSD diagnosis and racial/ethnic group interaction with sex and with age, respectively. All statistical analyses were conducted in Stata 14. Sampling weights designed for the combined NCS-R and NLAAS sample were used (27). The survey design was considered in calculating the SEs of the estimates, using the SVY package in Stata.

RESULTS

Sociodemographic, clinical, and social support factors and the pattern of heart disease and trauma exposure for each ethnic/racial group are presented in Table 1. Compared with non-Latino Whites, there were more participants from minority groups (Latinos, Asians and non-Latino Blacks) who were immigrants, between ages 18-65, and who lived below the poverty line in metropolitan areas. Latinos and non-Latino Blacks were more likely to have less education and be unemployed than their white counterparts. Although these variables are associated with an increased risk of PTSD (38), we found that a lower percentage of Asian and non-Latino Blacks reported experiencing some traumatic event in their life (64.8%, 68.9% respectively). A higher percentage of non-Latino Whites reported experiencing some traumatic event without having symptoms of PTSD (71%). Consistent with previous studies (38), non-Latino Blacks had the highest rate of having experienced a traumatic event and being diagnosed with PTSD (77%).

We found differences in the prevalence rate of experiences of trauma, cardiovascular events, and both categories combined (has experienced trauma and had a cardiovascular event) across racial and ethnic groups (Table 2). Non-Latino Whites (84%) and non-Latino Blacks (82%) reported the highest rates of trauma and CVD. Non-Latino Blacks (35%) and non-Latino Whites (29%) also reported the highest rates of CVD. The co-occurrence of trauma and CVD was highest for non-Latino Blacks (30%) and lowest for Asians (15%).

To understand the association between trauma exposure and the development of CVD, our sample in further analyses included all participants who had experienced a traumatic event and excluded those who reported having cardiovascular pathology before the age of the reported traumatic event. Table 3a shows the different logistic regression models that were tested to estimate the likelihood of having CVD based on the reported trauma/PTSD history. We adjusted for age and sex in Model 1. In Model 2, we additionally adjusted for variables that traditionally were related with an increased risk of cardiovascular events: BMI, tobacco dependence, and report of having diabetes in Model 1 to determine if these explained the relationship between PTSD and CVD. In Model 3, we additionally adjusted for psychiatric conditions that are frequently comorbid with PTSD: major depressive disorder, generalized anxiety disorder, and substance use disorder. When adjusted for age, sex and other sociodemographics (Model 1), all the conditions where trauma was present (trauma without PTSD symptoms, trauma and some PTSD symptoms and trauma and PTSD diagnosis) increase the likelihood of having CVD. When we included traditional cardiovascular risk factors in the model (Model 2), having trauma with some PTSD symptoms no longer significantly increases the likelihood of having CVD. In this model, having trauma with a PTSD diagnosis still significantly increases the likelihood of CVD (OR=2.62, 95% CI,

1.74-3.95). The link between trauma with no PTSD and CVD diagnosis appears to be accounted for by comorbid psychiatric diagnoses, so does the link between trauma with some PTSD symptoms and CVD diagnosis. Once we adjusted for the diagnoses of MDD, GAD, and substance use disorder, only PTSD diagnosis significantly increases the risk of CVD (Model 3 OR = 2.10, 95% CI, 1.32-3.33). We also identified positive age effects on development of CVD after controlling for a large set of cardiovascular risk factors (Model 3). Our sensitivity analysis examined the observed relationship between PTSD diagnosis and CVD, with hypertension excluded from CVD, without finding significant changes to the relationship. However, because comorbidity of different CVD conditions was relatively high we also present distributions of CVD comorbidity in our sample to better understand the studied population (Table 3b).

The model in Table 4 includes interaction terms for race/ethnicity and PTSD diagnosis, with those who had not experienced trauma as the reference group. Having a concurrent PTSD diagnosis significantly increased the likelihood of having CVD for non-Latino Whites (OR=1.86, 95%CI, 1.10-3.17), Latinos (OR = 1.94, 95% CI 1.04-3.62), and non-Latino Blacks (OR=3.73, 95%CI, 1.76-7.91) except for Asians. We also tested whether the relation of PTSD diagnosis to CVD was different for minorities versus Whites using an Omnibus test for equality of the regression coefficients of concurrent PTSD diagnosis. Results of the Omnibus test suggested that, for minority groups, the relationship was not significantly different when compared to Whites ($F(3,109) = 0.85$, $p=0.468$). Given PTSD diagnosis was highly correlated to CVD, we concluded that a concurrent PTSD diagnosis is positively related to CVD, but there was not enough evidence to suggest a differential relationship for minorities as compared to Whites (Table 3a). The three-way interaction results from the sensitivity analyses suggested that there are no significant differences of the relationship between race/ethnicity and PTSD by age or by sex. We also tested whether the relationship between CVD diagnosis and age (stratified as 35-49 years; 50-65 years; 65+ years) differed from our main results, and found no significant differences by age or interaction with race/ethnicity (see Tables S1-S2, Supplemental Digital Content 2 for results).

DISCUSSION

Using a nationally representative sample, we studied the relationship between CVD and trauma/PTSD. After controlling for variables that increase cardiovascular risk and restricting the sample to those who reported developing CVD after trauma exposure, we conclude that trauma alone or the presence of some PTSD symptoms appears to not be associated with an increased risk for CVD. However, presenting with clinical symptoms that meet diagnostic criteria for PTSD does significantly seem to increase the risk of CVD. Although many psychiatric illnesses are associated with PTSD (21), the risk of CVD associated with PTSD diagnosis remains significant, even after adjusting for depression, anxiety, and substance use and other risk factors of CVD. Results from our study open a new door for research that considers PTSD as a contributing factor for CVD. However, the underlying mechanisms of how PTSD leads to CVD development remain unclear, but may suggest that detrimental reactions to traumatic events confer risk for CVD, not the traumatic event itself.

Trauma exposure or having PTSD symptoms may act as a limited stressor, whereas clinical levels of PTSD may represent both a cognitive and physiological response to processing traumatic events. Research has examined indirect physiological mechanisms that explain the link between cardiovascular risk and PTSD, based on alterations of the HPA axis and the autonomic nervous system. These alterations would lead to a neurohormonal imbalance of the pituitary gland, and an increased risk for metabolic syndrome (19, 39). Psychologically, the impact of trauma on health could operate through two processes: reactivation of stress pathways, or demands on the body related to symptom management through dissociation and suppression (5). Multiple traumas could have a cumulative effect on physical health independent of the presence of PTSD symptoms (12), and this relationship is due to excessive, chronic activation of physiological stress pathways. While our study is one of the first to show a strong association between PTSD diagnosis and CVD development after adjusting for many confounders, further research must identify the characteristics of traumatic experiences, including type, duration and severity, as well as the coping mechanisms which may influence the relationship between PTSD diagnosis and medical conditions. Relationship between specific CVD condition and PTSD diagnosis should be better studied in future research as our sample did not have enough power to study each condition separately.

We also investigated differences between ethnic/racial groups in patterns of trauma exposure/PTSD and CVD. Although non-Latino Whites reported the highest percentage of experiencing trauma, a greater percentage of non-Latino Blacks met diagnostic criteria for PTSD. This finding is consistent with previous research (38, 40), and may indicate that non-Latino Blacks are at greater risk of exposure to hostile environments and racial discrimination that could exacerbate the risk of PTSD (38). Similarly, non-Latino Blacks have higher rates of cardiovascular events than other racial/ethnic groups (41). In our analysis, the risk of CVD increased in all racial/ethnic groups in the presence of a PTSD diagnosis, but the prevalence of PTSD was found to be higher in Blacks, indicating the importance of addressing the vulnerability of co-occurring PTSD and CVD. The “weathering hypothesis” links accumulated experiences of exclusion and discrimination to higher rates of impairing and disabling chronic health conditions among Blacks (42). Furthermore, biological evidence has demonstrated the long-term physical impact of discrimination and race-related stressors on Blacks as compared to whites (42, 43), including an increased risk of adiposity (44) and higher rates of hypertension among Black people (45). While we found an association between PTSD and CVD, the mechanisms linking PTSD and CVD remained mostly unexplained. Future studies should examine longitudinal pathways between the diagnosis of PTSD and subsequent health status to characterize the connection between PTSD and cardiovascular health.

Limitations

There are several limitations to consider. Some CVD risk factors were not available in the dataset, like prior diagnosis of dyslipidemia, nutritional habits, lifestyle, or taking drugs (e.g. antipsychotics), and thus could not be considered in the analyses. Studies have shown an association between PTSD and metabolic syndrome, independent of antipsychotic medication use (46, 47). These risk factors should be investigated in future studies to

determine if they attenuate the relationship of interest. The identification of CVD and related health factors was based on self-report and would exclude patients who are unaware of their health status. Use of cross-sectional data is also a limitation, although we limited the study sample to those who developed CVD after experiencing trauma. Finally, the reliability of WMH-CIDI for assessing PTSD among Latinos has been questioned and should be considered in interpreting results with caution (35).

Clinical implications

Patients who meet diagnostic criteria for PTSD should receive optimal mental health services to lessen CVD risk that may be related to adverse reactions from PTSD. There should be an emphasis on preventive interventions tailored for the non-Latino Black population, who were found to be at greater risk of PTSD and cardiovascular events. While not all patients who experience trauma will develop PTSD, the course of developing PTSD may in turn increase risk of CVD, highlighting the importance of prevention strategies at all points in the risk continuum. Population level interventions to cope with adverse reactions of PTSD and increase resilience and perceived control should be further tested and implemented as a preventive strategy for CVD (9, 48–50). At the individual level, interventions for PTSD should include strategies to promote physical health, reduce CVD risk factors, and ensure medical follow-up (9, 51, 52).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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TABLE 1

Sociodemographic, Clinical, and Social Support Variables among the NCSR and NLAAS Sample (N= 9842)

Variables	Non-Latino White		Latino			Asian American			Black/African American			Total	
	n = 4032		*n* = 2991			*n* = 2136			*n* = 9842			*n* = 9842	
	%	SE	%	SE	Sig	%	SE	Sig	%	SE	Sig	%	SE
Born in US					*a*			*a,b*			*b,c*		
US-born	96.8	0.5	48.9	2.1		22.6	2.9		96.1	1.2		85.9	1.1
Immigrant	3.2	0.5	51.1	2.1		77.4	2.9		3.9	1.2		14.1	1.1
Age Category					*a*			*a,b*			*a,b,c*		
18-34 years	28.7	1.5	49.4	1.6		40.2	1.6		40.7	3.1		32.5	1.2
35-49 years	30.9	1.2	30.7	1.1		32.8	1.7		30.7	2.1		31.0	0.9
50-65 years	22.7	1.3	13.0	0.8		17.6	1.1		18.0	1.9		20.9	1.0
65+ years	17.7	1.2	6.9	0.8		9.4	1.6		10.6	2.3		15.6	1.0
Gender					*a*			*b*			*a,b,c*		
Male	47.9	1.2	52.0	1.5		47.3	1.2		42.0	2.1		48.3	0.9
Female	52.1	1.2	48.0	1.5		52.7	1.2		58.0	2.1		51.7	0.9
Marital					*a*			*a,b*			*a,b,c*		
Married	55.1	1.5	51.0	1.6		64.8	1.9		26.2	2.6		54.4	1.2
Never Married	22.6	1.5	31.1	1.3		25.6	1.4		46.3	3.4		24.5	1.2
Widowed/Divorced/Separated	22.3	0.8	17.9	1.1		9.5	1.0		27.5	2.3		21.1	0.7
Education					*a*			*a,b*			*a,b,c*		
11 years or less	12.8	1.0	42.3	1.6		14.8	1.4		21.8	2.9		17.3	0.9
12 years	31.4	1.7	27.5	1.0		17.1	1.2		35.8	2.4		30.2	1.3
13 – 15 years	28.9	1.2	20.0	1.2		25.3	1.4		28.8	1.6		27.4	0.9
16 years or more	26.9	1.4	10.2	0.9		42.8	2.0		13.5	2.0		25.1	1.1
Employment					*a*						*b,c*		
Employed	66.3	0.9	65.3	1.6		65.3	1.4		62.5	3.4		66.0	0.8
Unemployed	4.9	0.5	7.4	0.9		6.0	0.6		3.0	0.7		5.2	0.4
Out of labor force/other	28.8	1.0	27.4	1.6		28.7	1.5		34.5	3.4		28.7	0.8
Region					*a*			*a,b*			*a,b,c*		
Northeast	20.7	4.0	14.4	1.2		15.2	3.3		17.3	2.8		19.5	3.2

Variables	Non-Latino White		Latino			Asian American			Black/African American			Total	
	n = 4032		*n* = 2991			*n* = 2136			*n* = 9842			*n* = 9842	
	%	SE	%	SE	Sig	%	SE	Sig	%	SE	Sig	%	SE
Midwest	28.7	2.6	9.2	1.6		9.0	1.9		17.9	2.6		24.6	2.0
South	30.6	3.1	32.0	3.8		8.5	1.8		53.5	3.8		30.1	2.4
West	20.0	2.8	44.3	3.7		67.3	4.0		11.3	1.8		25.8	2.2
Poverty								*a,b*			*a,c*		
Above poverty level	91.0	0.7	74.3	1.8		82.6	1.2		74.2	3.6		87.8	0.7
Below poverty level	9.0	0.7	25.7	1.8		17.4	1.2		25.8	3.6		12.2	0.7
Urbanicity								*a*			c		
Non-metro counties	26.8	7.1	8.2	3.7		3.5	1.6		19.3	9.0		22.7	5.9
Metro counties	73.2	7.1	91.8	3.7		96.5	1.6		80.7	9.0		77.3	5.9
Family Support								*a,b*			*a,b*		
Scales [0-1] Mean (SE)	0.7	0.0	0.7	0.0		0.6	0.0		0.7	0.0		0.68	0.004
Friend Support								*a,b*			*a,b,c*		
Scales [0-1] Mean (SE)	0.7	0.0	0.5	0.0		0.6	0.0		0.6	0.0		0.66	0.004
PTSD								*a,b*			*b,c*		
No Trauma, No PTSD	16.4	1.0	25.6	1.4		29.7	1.4		18.6	2.1		18.5	0.9
Trauma, No PTSD	71.0	0.8	65.0	1.5		64.8	1.3		68.9	2.2		69.7	0.7
Trauma, Some PTSD symptoms	5.5	0.4	4.8	0.5		3.5	0.5		4.8	0.7		5.3	0.3
Trauma, PTSD Diagnosis	7.1	0.6	4.6	0.4		1.9	0.5		7.7	0.8		6.5	0.5
Any Formal Mental Health Service Use During Past 12 Months					*a*			*a,b*					
No	52.8	1.1	72.3	1.3		82.0	1.3		72.6	2.4		57.6	1.0
Yes	47.2	1.1	27.7	1.3		18.0	1.3		27.4	2.4		42.4	1.0
Any Formal Mental Health Service Use in the Past					*a*			*a,b*					
No	84.8	0.9	90.4	0.7		93.6	0.7		91.8	1.3		86.2	0.7
Yes	15.2	0.9	9.6	0.7		6.4	0.7		8.2	1.3		13.8	0.7

Note: This analysis includes 4649 participants from NLAAS and 5507 participants from NCSR-who completed the long form survey. The sample further excludes 314 participants who had cardiovascular disease before exposure to trauma. The total number of participants used in the analytical sample is 9842. Summary statistics are calculated using NLAAS_NCS long form weights from the CPES study.

a: Significant difference ($p < .05$) compared to Whites;

b: Significant difference ($p < .05$) compared to Latinos;

[c]: Significant difference ($p < .05$) compared to Asians. Further detail available upon request.

TABLE 2

Prevalence of Trauma, Cardiovascular Disease Among the NCSR and NLAAS Sample (N=10156)

	Prevalence	Std. Errs.	P-values
Ever had any trauma experience			
Non-Latino White	84%	0.01	<0.001
Latino	75%	0.01	
Asian	71%	0.01	
Black/African American	82%	0.02	
Ever had cardiovascular disease: stroke, heart attack, and high blood pressure			
Non-Latino White	29%	0.01	<0.001
Latino	18%	0.01	
Asian	19%	0.02	
Black/African American	35%	0.03	
Ever had both trauma and cardiovascular disease			
Non-Latino White	26%	0.01	<0.001
Latino	16%	0.01	
Asian	15%	0.01	
Black/African American	30%	0.03	

Note: This analysis includes 4649 participants from NLAAS and 5507 participants from NCSR-who completed the long form of survey. Prevalence rates and their standard errors are calculated using NLAAS_NCS long form weights from the CPES study. We tested whether prevalence rates are equal across racial/ethnic groups using Wald Chi-square test. *p* values from the test are reported.

TABLE 3a

Association of Trauma and/or PTSD Symptoms and the Likelihood of Having Cardiovascular Disease (N=9842)

	Model 1		Model 2		Model 3	
Variables	**Sociodemographics**		**Model 1 + Current smoker, BMI, Diabetes**		**Model 2 + MDD, GAD, Substance Abuse**	
No Trauma, No PTSD (Reference)						
Trauma, No PTSD	1.54*	[1.08,2.20]	1.32	[0.98,1.79]	1.27	[0.93,1.73]
Trauma, Some PTSD Symptoms	1.73*	[1.10,2.70]	1.28	[0.77,2.14]	1.14	[0.68,1.91]
Trauma, PTSD Diagnosis	3.08***	[2.02,4.69]	2.62***	[1.74,3.95]	2.10**	[1.32,3.33]
Race/ethnicity (ref=White)						
Latino	1.02	[0.80,1.30]	0.87	[0.67,1.12]	0.89	[0.69,1.14]
Asian	1.21	[0.89,1.64]	1.38	[0.99,1.91]	1.44*	[1.04,2.00]
Black	2.15***	[1.65,2.80]	1.89***	[1.42,2.52]	2.00***	[1.50,2.66]
Nativity (ref=US-born)						
Immigrant	0.80	[0.64,1.01]	0.97	[0.76,1.24]	1.01	[0.79,1.29]
Age	1.06***	[1.05,1.07]	1.06***	[1.05,1.08]	1.07***	[1.06,1.08]
Gender (ref=Male)						
Female	0.63***	[0.53,0.75]	0.61***	[0.50,0.75]	0.61***	[0.50,0.74]
Marital Status (ref=Married)						
Never Married	0.83	[0.64,1.08]	0.99	[0.74,1.32]	0.99	[0.75,1.31]
Widowed/Divorced/Separated	1.04	[0.83,1.30]	1.06	[0.84,1.32]	1.02	[0.81,1.27]
Education (ref=11 years or less)						
12 years	1.01	[0.71,1.43]	1.03	[0.76,1.39]	1.04	[0.77,1.40]
13-15 years	0.95	[0.70,1.30]	0.98	[0.74,1.29]	0.98	[0.73,1.31]
16 years or more	0.81	[0.58,1.14]	0.92	[0.67,1.25]	0.92	[0.67,1.25]
Employment (ref=Employed)						
Unemployed	1.42	[0.91,2.20]	1.59*	[1.04,2.41]	1.60*	[1.06,2.42]
Out of labor force/other	1.07	[0.86,1.33]	1.12	[0.88,1.43]	1.11	[0.87,1.42]
Region (ref=Northeast)						
Midwest	1.02	[0.89,1.17]	0.94	[0.78,1.14]	0.95	[0.79,1.14]
South	0.94	[0.74,1.21]	0.99	[0.76,1.29]	1.00	[0.77,1.30]

	Model 1		Model 2		Model 3	
Variables	**Sociodemographics**		**Model 1 + Current smoker, BMI, Diabetes**		**Model 2 + MDD, GAD, Substance Abuse**	
West	0.92	[0.77,1.09]	0.99	[0.80,1.23]	0.98	[0.79,1.21]
Poverty (ref= Above poverty level)						
Below poverty level	1.06	[0.80,1.40]	1.00	[0.73,1.36]	1.00	[0.73,1.36]
Urbanicity (ref=Non-metro counties)						
Metro counties	0.81	[0.64,1.01]	0.73**	[0.58,0.92]	0.73**	[0.58,0.92]
Family support scale	1.00	[0.70,1.43]	0.88	[0.62,1.24]	0.94	[0.66,1.32]
Friend support scale	1.05	[0.77,1.44]	1.62**	[1.15,2.30]	1.62**	[1.14,2.29]
BMI			1.09***	[1.07,1.11]	1.09***	[1.07,1.11]
Current Smoker			1.24	[0.93,1.65]	1.19	[0.89,1.59]
Diabetes			2.68***	[1.90,3.80]	2.67***	[1.90,3.76]
Major Depressive Disorder					1.36**	[1.08,1.71]
General Anxiety Disorder					1.20	[0.95,1.52]
Substance Abuse (Alcohol or Drug)					1.21*	[1.01,1.44]
P values from Wald Test using Odds Ratios						
Trauma/No PTSD vs. Trauma/Some PTSD Symptoms	0.447		0.884		0.574	
Trauma/No PTSD vs. Trauma/PTSD Diagnosis	0.000		0.000		0.001	
Trauma/Some PTSD Symptoms vs. Trauma/PTSD Diagnosis	0.003		0.008		0.025	
Observations	9411		8852		8852	

Note: This analysis includes 4649 participants from NLAAS and 5507 participants from NCSR who completed the long form survey. The sample further excludes 314 participants who had cardiovascular disease before exposure to trauma. The total number of participants used in the analytical sample is 9842. For each model, observations with missing values in the covariates are list wise deleted. Predictions are based on NLAAS_NCSR long form weights. Results are presented as Odds Ratios from logistic regressions.

* $p < .05$,

** $p < .01$,

*** $p < .001$.

TABLE 3b

Distribution of Different Types of Cardiovascular Comorbidity(N=9842)

Pattern of illnesses	%	S.E.	95% CI	
No CVD Illness	75.2%	0.006	74.1%	76.3%
Stroke Only	0.9%	0.002	0.7%	1.3%
Heart Attack Only	0.7%	0.001	0.4%	1.0%
Heart Disease Only	1.5%	0.001	1.2%	1.8%
High Blood Pressure Only	17.0%	0.006	15.9%	18.1%
Stroke and Heart Attack Comorbidity	0.0%	0.000	0.0%	0.1%
Stroke and Heart Disease Comorbidity	0.0%	0.000	0.0%	0.1%
Stroke and High Blood Pressure Comorbidity	0.8%	0.002	0.5%	1.4%
Heart Attack and Heart Disease Comorbidity	0.5%	0.001	0.3%	0.8%
Heart Attack and High Blood Pressure Comorbidity	0.5%	0.001	0.3%	0.8%
Heart Disease and High Blood Pressure Comorbidity	1.5%	0.002	1.2%	1.9%
Three Comorbidities	1.1%	0.002	0.8%	1.5%
All Four Comorbidities	0.3%	0.001	0.1%	0.5%

TABLE 4

Ethnic/Racial Variations in Association of Trauma and/or PTSD Symptoms and the Likelihood of Having Cardiovascular Disease (N=9842)

	OR	95% CI
No Trauma, No PTSD (Reference)	1.00	—
Trauma, No PTSD	1.26	[0.92,1.73]
Trauma, Some PTSD Symptoms	1.13	[0.67,1.90]
Trauma, PTSD Diagnosis × White	1.86*	[1.10,3.17]
Trauma, PTSD Diagnosis × Latino	1.94*	[1.04,3.62]
Trauma, PTSD Diagnosis × Asian	1.73	[0.58,5.19]
Trauma, PTSD Diagnosis × Black	3.73***	[1.76,7.91]
Race/ethnicity (ref=White)		
Latino	0.88	[0.68,1.15]
Asian	1.44*	[1.04,2.01]
Black	1.87***	[1.36,2.57]
Nativity (ref=US-born)		
Immigrant	1.01	[0.78,1.29]
Age	1.07***	[1.06,1.08]
Gender (ref=Male)		
Female	0.61***	[0.50,0.74]
Marital Status (ref=Married)		
Never Married	0.98	[0.74,1.30]
Widowed/Divorced/Separated	1.02	[0.82,1.28]
Education (ref=11 years or less)		
12 years	1.04	[0.77,1.40]
13-15 years	0.98	[0.73,1.30]
16 years or more	0.92	[0.68,1.25]
Employment (ref=Employed)		
Unemployed	1.59*	[1.05,2.41]
Out of labor force/other	1.11	[0.87,1.42]
Region (ref=Northeast)		
Midwest	0.94	[0.78,1.13]
South	1.00	[0.77,1.29]
West	0.97	[0.79,1.20]
Poverty (ref= Above poverty level)	1.00	[1.00,1.00]
Below poverty level	1.00	[0.73,1.36]
Urbanicity (ref=Non-metro counties)		
Metro counties	0.73**	[0.57,0.92]
Family support scale	0.94	[0.66,1.33]
Friend support scale	1.61**	[1.14,2.28]

	OR	95% CI
BMI	1.09***	[1.07,1.11]
Current Smoker	1.19	[0.89,1.60]
Diabetes	2.66***	[1.89,3.75]
Major Depressive Disorder	1.36**	[1.08,1.72]
General Anxiety Disorder	1.22	[0.97,1.53]
Substance Abuse (Alcohol or Drug)	1.21*	[1.02,1.45]
P values from omnibus test for joint significance of the interactions	0.004	
P values from omnibus test for racial differences in PTSD diagnosis coefficient	0.468	
Observations	8852	

Note. This analysis includes 4649 participants from NLAAS and 5507 participants from NCSR who completed the long form survey. The sample further excludes 314 participants who had cardiovascular disease before exposure to trauma. The total number of participants used in the analytical sample is 9842. Observations with missing values in the covariates are list wise deleted. Predictions are based on NLAAS_NCSR long form weights. Results are presented as Odds Ratios from logistic regressions.

* $p < .05$,

** $p < .01$,

*** $p < .001$.