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Exposure to socioenvironmental stress as a predictor of physical and mental health

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Abstract

The literature demonstrates links between socioenvironmental characteristics, dysregulation of the stress response system, and PTSD, though few studies integrate these factors in one model. In a secondary analysis of cross-sectional data collected by the Survey of the Health of Wisconsin (SHOW), structural equation modeling evaluated the relationships between socioenvironmental stress, cumulative biological risk (CBR), and PTSD symptom severity. The model hypothesized that exposure to socioenvironmental stress was associated with PTSD and that this relationship is mediated by increased CBR. Indices suggest the model provided a good fit to the data and supported socioenvironmental stress and CBR as valid latent constructs. Although the association between CBR and PTSD was not found to be statistically significant in this study, socioenvironmental stress was a significant predictor of PTSD and CBR. Given the role of socioenvironmental factors on CBR and PTSD symptoms, providers need to better assess and incorporate social stressors within evaluation and treatment.

Keywords

model, physical symptoms, risk factors, sociodemographic variables, trauma

The influence of social and physical environments on health has been thoroughly documented in the literature (Barrington et al., 2014; Kawachi and Subramanian, 2007; Pickett and Pearl, 2001). Recognition of the importance of context and systems on the individual began with the onset of the socio-ecological model (Darling, 2007). There are clear relationships between socio-ecological factors, such as social inequalities between neighborhoods and neighborhood disadvantage (Gapen et al., 2011). Disordered neighborhood environments are theorized to influence health via the "wear and tear" of chronic exposure to stress, resulting in heightened activation of the sympathetic nervous system (SNS) and hypothalamic pituitary adrenal (HPA) axis (Brody et al., 2014b). Dysregulated SNS and HPA activity drives physiological responses that in turn are associated with dysregulation of the immune, metabolic, and cardiovascular systems, ultimately

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creating vulnerability to disease and increased mortality (McEwen and Gianaros, 2010; Seeman et al., 2010). This underscores the need to consider health on an individual level and the systems within which individuals operate (Lounsbury and Mitchell, 2009).

The detrimental physical effect of chronic stress can be conceptualized and measured using the construct of allostatic load (McEwen, 1998). Allostasis is described as an organism's active processes of maintaining physiological stability in response to events and stressors by altering its internal milieu to appropriately meet environmental demands (Sterling and Eyer, 1988). Allostatic load is the cumulative consequence of repeated confrontations with stressful environments and/or events and the resulting physiological adaption via allostasis (Crimmins and Seeman, 2004; Juster et al., 2010; McEwen, 1998, 2003). Allostatic load is typically operationalized as an index created by summing multiple biological and anthropometric measures. These biomarkers are selected to represent the different systems expected to be dysregulated by chronic stress: neuroendocrine, immune, metabolic, and cardiovascular. However, there is significant variation across studies as far as the specific markers used in conceptualizing allostatic load (for review, see Juster et al., 2010). Many human studies lack primary neuroendocrine markers (e.g. cortisol, catecholamines) and so create a "cumulative biological risk" (CBR) index using cardiometabolic indicators (King et al., 2011) as a measure of cumulative physiological burden related to allostatic load.

Chronic stress from socio-ecological factors are shown to be related to physiological response with implications for CBR. Factors such as poverty can influence the physical and social environmental conditions of neighborhood residents (Geronimus et al., 2006; Gruenewald et al., 2012; Sabbah et al., 2008; Seeman et al., 2010). Large epidemiological studies using data sources such as the National Health and Nutrition Examination Survey (NHANES) and the Study of Midlife in the US (MIDUS) have shown that various indicators of social status are associated with CBR. Notably, even while controlling for individual characteristics, socio-economic status (SES) and poverty at the *neighborhood* level have been found to predict individual indicators of CBR, and allostatic load (Gruenewald et al., 2012; Sabbah et al., 2008; Schulz et al., 2012). Similarly, experiences of discrimination have a clear link to a variety of the biological markers of stress (Cooper et al., 2009; Cunningham et al., 2012; Lewis et al., 2010; Mays et al., 2007). This suggests that consideration of the larger context including socioenvironmental factors are an important consideration to CBR.

The impact of socioenvironmental stress relates not only to physical health but extends to mental health and have reciprocal relationships. Aspects of neighborhood disadvantage, such as poverty, have measurable negative effects on the mental health of residents (Antoni et al., 2006; Leventhal and Brooks-Gunn, 2003). Both acute and chronic exposure to socioenvironmental stressors, such as unemployment and low SES, can negatively influence mental health outcomes, including elevated anxiety, depression, and symptoms of posttraumatic stress disorder (PTSD) (Jain et al., 2018; Williams, 2003). PTSD can significantly impact functioning and quality of life, and it is associated with HPA axis dysregulation and medical comorbidities (De Kloet et al., 2006; Ryder et al., 2016; Yehuda, 2001). Socioenvironmental factors related to disadvantaged neighborhoods (e.g. perceived mistrust) are proposed to be related to PTSD (Gapen et al., 2011). As such, PTSD, a disorder marked by avoidance and hypervigilance, may further promote negative socioenvironmental factors. Beyond factors such as SES, one's perception of neighborhood disorder (e.g. breakdown of social order through signs such as vandalism, criminal activity) and experiences of discrimination have also been tied to psychopathology, specifically, PTSD (Carter et al., 2013; Ross, 2000). Although possibly not directly linked to onset of PTSD, the broader impact of one's environment, such as those discussed previously, are unknown. It is important to consider how these factors are linked within the construct of socio-environmental stress and how that may influence psychological health independent of an index trauma event.

There is a well-established association between PTSD and physical health comorbidities, such as cardiovascular disease. Further, studies have found that individuals with PTSD are at a higher risk for physical conditions, including metabolic syndrome, autoimmune disorders, and gynecologic conditions like polycystic ovarian syndrome and endometriosis (Cohen et al., 2015; Heppner et al., 2009; Levine et al., 2014; O'Donovan, 2016). These findings suggest that the link between PTSD and health-related comorbidities may be at least partially explained by increased CBR. Indeed, initial evidence demonstrates a relationship between PTSD and allostatic load (Thayer et al., 2017). For instance, one study demonstrated that PTSD development following early life trauma exposure had higher allostatic load. This may be concerning given PTSD symptoms of avoidance and hypervigilance may make management of physical health conditions challenging. Therefore, it is critical to understand how individual factors and neighborhood characteristics play a role in managing PTSD and physical health conditions.

The literature demonstrates links between socioenvironmental characteristics, dysregulation of the stress response system and physiology, and PTSD, suggesting further investigation is warranted. While links between different the components of the proposed model have been investigated (Barber et al., 2016; Brody et al., 2014a; Glover et al., 2006; Guidi et al., 2021; Schulz et al., 2012; Thayer et al., 2017), this study presents the first examination of a model linking PTSD, cumulative biologic risk, and neighborhood environmental factors together. Few studies integrate these overlapping factors in one model and, furthermore, ways of assessing socioenvironmental stress can vary significantly; addressing this gap will contribute to the literature on the complex and inter-related effect of social environments on individuals' mental and physical health (Barber et al., 2016; Brody et al., 2014a; Glover et al., 2006; Guidi et al., 2021; Schulz et al., 2012; Thayer et al., 2017). Socio-ecological models highlight that individual health is impacted by larger systems, and likewise, intervention should be multifaceted (McLeroy et al., 1988). By accounting for the relationship of physical and mental health in the context of our social environment, we can better understand how socio-ecological factors may intersect with treatment planning (e.g. maintaining adherence, reducing recidivism). In this study, we hypothesized that the stress of living in disadvantaged conditions contributes to a dysregulated stress response, resulting in increased PTSD symptom severity as well as physiological wear-and-tear over time. We further hypothesized that CBR, as a down-stream result of exposure to chronic stress, would positively predict PTSD symptom severity.

Method

Participants

Secondary cross-sectional analyses of a subset of Survey of the Health of Wisconsin (SHOW) participants was conducted. The SHOW is an ongoing population based health examination study of the Wisconsin population. The current secondary analysis utilized a sample of adults aged 21–74 who participated in the 2010, 2011, and 2012 cohorts of SHOW. These three study cohorts included a total of 2321 individuals. If participants did not have complete data for the measures of interest they were removed from the study sample. This resulted in 362 participants being excluded for incomplete data, leaving a final study sample of 1959.

The average age of the study population was 46 years, with approximately half (50.2%) of the population identifying as female. The majority of the sample identified as White American (87.0%), and 73.9% had some college education or an associate degree. When examining income, 13.0% had a household income lower than $20,000 annually, while

Table 1. Demographics of study sample ($N = 1959$).

Variable	Frequency	%
Gender		
Male	848	49.5
Female	1111	50.5
Race		
White	1715	87
Black/African American	109	5.2
Other	135	7.8
Family income		
<$20,000	272	13
$20,000–34,999	317	14.5
$35,000–49,999	275	14
$50,000–74,999	390	20.2
$75,000 +	643	35.5
Missing	62	2.9
Education		
Less than HS	116	5.5
HS/GED	425	20.6
Some college/Assoc.	784	40.3
Bachelors	419	22.5
Above Bach	215	11.1
Insurance		
None	176	8.6
Private	1305	70.7
Government	464	20.1
Other	14	0.6

35.5% fell in the highest bracket of $75,000 or over. Demographic characteristics describing the study sample are displayed in Table 1.

Measures

PTSD checklist—civilian version (PCL-C; Weathers et al., 1993). Posttraumatic Stress Disorder (PTSD) symptom severity was obtained using the PCL-C (Weathers et al., 1993) for the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV; American Psychiatric Association, 2000). This self-report measure is comprised of 17 items corresponding to key PTSD symptoms as described in the DSM-IV (e.g. repeated, disturbing dreams of the stressful experience). Items are on a 5-point Likert scale from 1 (not at all) to 5 (extremely). Items were summed with higher total scores suggesting greater PTSD symptom severity. The PCL-C has been shown to have a high internal reliability ($\alpha = 0.94$; Blanchard et al., 1996). The Cronbach's alpha for the current study was $\alpha = 0.93$.

Cumulative biological risk. Data from six biomarkers and anthropometric measures were objectively assessed by SHOW staff and used to create a summary index of cumulative biological risk (CBR) using previously defined and clinically relevant parameters (McEwen and Stellar, 1993; Seeman et al, 2010). These measures included the following: systolic blood pressure $\geq$140 mmHg, diastolic blood pressure $\geq$90 mmHg, waist circumference of 102 cm or above in men and 80 cm or above in women, glycosylated hemoglobin of 6.5 or higher or self-reported use of diabetes medication, total cholesterol of ⩾240 mg/dcl or self-reported use of hyperlipidemia medication, high-density lipoprotein (HDL) cholesterol under 40 mg/dcl in men and under 50 mg/dcl in women, and white blood cell count outside of the normal range of 4000–10,000 white blood cells per microliter. A dichotomous indicator was created for each of these observed variables (0 = no risk, 1 = high risk) based on clinically indicated high-risk criteria (McPherson, 2011; Ross et al., 2020), with CBR analyzed in the model as a latent variable defined by the six observed CBR variables. Only participants with data for all six observed variables were included in the analysis.

Socioenvironmental stress. Socioenvironmental stress was included in the model as a latent variable defined by three observed variables: Economic Hardship Index, perceived neighborhood environment stress, and experienced discrimination.

Economic hardship index (EHI; Montiel et al., 2004). The EHI, developed by the Rockefeller Institute of Government, was used to assess neighborhood-level economic hardship. The measure is created from block group-level socioeconomic indicators contained within the SHOW dataset including: poverty (the percent of households living below the federal poverty level), crowded housing (the percent of housing units with over one person per room), education (the percent of people over 25 years old without a high school education), unemployment (the percent of people over 16 years old that are unemployed), dependency (the percent of population under 18 years or over 64 years), and income level (median per capita). Following the method described by Montiel and colleagues, the individual indicators were standardized and then averaged to create an overall EHI score. EHI scores can range from 1 to 100, with higher indices indicating higher neighborhood-level economic hardship.

Neighborhood environment walkability survey (NEWS; Saelens et al., 2003). Perceived neighborhood environmental stress was assessed in SHOW using six items from the NEWS. The items asked respondents if their community was well maintained, free from garbage, safe from crime, safe from traffic, a good place to be physically active, and if there was easy access to fresh fruits and vegetables. Items are on a 4-point Likert scale from 1 (strongly disagree) to 4 (strongly agree). The responses were summed to create a measure of neighborhood environmental stress, with greater scores suggesting greater stress. The NEWS has been shown to have moderate to high reliability ($r = 0.58–0.80$; Saelens et al., 2003). For the current study, Cronbach's alpha was $\alpha = 0.75$.

Every day discrimination scale (Williams et al., 1997). Experiences of discrimination were measured with items from the every day discrimination subscale of the Detroit Area Study Discrimination Questionnaire (DAS-DQ; Williams et al., 1997), with a focus on every day discrimination. Seven items assessed chronic and/or repetitive experiences of unfair treatment that could be based on age, gender, race, culture or ethnicity, physical appearance, religion, sexual orientation, or another reason. Items are on a 8-point Likert scale from 0 (never) to 7 (several times a day). Example items include: "People act as if they think you are dishonest" and "You are called names, insulted, threatened, or harassed." The mean score from the seven items was used as a continuous score of experienced everyday forms of discrimination, with higher scores indicating greater experiences of discrimination. The every day discrimination subscale has good internal consistency ($\alpha = 0.80$; Taylor et al., 2004). For the current study, Cronbach's alpha was $\alpha = 0.88$.

Procedure

This study is a secondary analysis of data collected by SHOW, an annual survey that gathers health-related data from a representative sample of Wisconsin residents; the study rationale and detailed methods have been described previously (please see Nieto et al., 2010 for more detail). In brief, each year a representative sample of non-institutionalized Wisconsin residents aged 21–74 are selected from random households based on a two-stage cluster sampling approach. First, using Census 2000 data, 4388 census groups were created and stratified based on congressional district and percentage of population living below poverty level. Second, using commercially available United States Postal Service delivery sequence data, the study used simple random sampling to recruit from 12 to 28 addresses in a given census group. Participants are then approached in person and sent information via mail about the SHOW study. Inclusion criteria include: (1) The selected household is the individual's primary residence, (2) Ages 21–74, (3) Individuals who have multiple residencies and live outside of selected household more than 50% of the time, (4) Individuals who voluntarily disclose mental incapacity, without someone who can serve as proxy. Ability to give full written consent, 4. Ability to respond to survey questions. Exclusion criteria include: (1) Individuals living in institutions (e.g. in jail), college dormitories, or nursing homes. (2) Full time members of armed forces or those away from home. (3) Participants have bio-specimen samples collected, complete anthropometric assessments, and provide responses to a number of health-related survey measures including the measures listed below that are the variables of interest for the current study.

Data sharing statement

Data can be accessed by contacting the Survey of the Health of Wisconsin. Data taken for the current study include DAS-DQ, NEWS, EHI, PCL-C, systolic blood pressure ⩾140 mmHg, diastolic blood pressure ⩾90 mmHg, waist circumference of 102 cm or above in men and 80 cm or above in women, glycosylated hemoglobin of 6.5 or higher or self-reported use of diabetes medication, total cholesterol of ⩾240 mg/dcl or self-reported use of hyperlipidemia medication, high-density lipoprotein (HDL) cholesterol under 40 mg/dcl in men and under 50 mg/dcl in women, and white blood cell count outside of the normal range of 4000–10,000 white blood cells per microliter. For greater detail on protocol and other variables available see Nieto et al. (2010).

Statistical analysis

The goal of this study was to evaluate the relationship between increased socioenvironmental stress, CBR, and PTSD. First, descriptive statistics were utilized for the study variables of interest, including PTSD symptom severity, CBR scores, EHI, perceived neighborhood environment stress, and experienced discrimination. The data was evaluated for multivariate normality by creating and examining probability-probability (P-P) plots, with no concerns for non-normality noted. SAS 9.4 was used to analyze demographic characteristics and descriptive statistics.

To test these hypotheses, we used structural equation modeling (SEM) to examine whether our hypothesized model is an appropriate portrayal of the relationships between socioenvironmental stress, CBR, and PTSD symptom severity in a civilian sample. As no other models have evaluated these variables all together, no rival models exist to compare the proposed model to. Because of this, a strictly confirmatory approach was taken to simply evaluate the proposed model using data from SHOW. This approach allows for evaluation of the latent variables as constructs (i.e. CBR, socioenvironmental stress) as well as how these latent variables are related to PTSD. Latent variables examined in the model included CBR (defined by the six observed CBR indicator variables) and socioenvironmental stress (defined by three observed variables: EHI, perceived neighborhood environment stress, and experienced day-to-day discrimination), and PTSD symptom severity (defined by the 17 items of the PCL-C).

MPlus Version 7.4 was used to calculate correlations between variables and to examine relationships between variables with SEM. For the SEM analysis, a weighted least square mean and variance (WLSMV) adjusted

Table 2. Descriptive statistics for study variables.

Variable	Mean (SE)	95% CL for mean	Range
Age (years)	45.9	44.8–47.0	21–74
PCL-IV	24.3	23.7–24.9	17–78
CBR score	1.7	1.6–1.8	0–6
EHI	24.5	23.9–25.1	16.0–57.8
NES	4.6	4.3–4.8	0–17
DISC	0.03	0.7–0.8	0–6

PCL-IV: PTSD checklist – civilian version, for DSM-IV; CBR: cumulative biological risk; EHI: economic hardship index; NES: neighborhood environment score; DISC: experienced discrimination.

Table 3. Correlations[a] between study variables of interest (N = 1959).

	CBR score	PCL-IV	DISC	NES
PCL-IV	0.081***			
DISC	0.011	0.399***		
NES	0.068**	0.197***	0.253***	
EHI	0.069**	0.105***	0.143***	0.313***

CBR: cumulative biological risk; PCL-IV: PTSD checklist – civilian version, for DSM-IV; DISC: experienced discrimination; NES: neighborhood environment score; EHI: economic hardship index.
[a]Two-tailed Spearman's rank correlation coefficients.
**$p \leqslant 0.05$ (two-tailed). ***$p \leqslant 0.001$ (two-tailed).

estimator was used. Default criteria including iterations and tolerance were not adjusted to achieve convergence and the model does not include any significant interaction effects. Model goodness-of-fit was evaluated utilizing indices and cutoffs previously described in the literature (Hu and Bentler, 1999; Kenny, 2014), including Root Mean Square Error of Approximation (RMSEA) ⩽0.6, Comparative Fit Index (CFI) and Tucker Lewis Index (TLI) ⩾0.95, and Standardized Root Mean Square Residual (SRMR) below 0.08 indicating satisfactory model fit.

Results

The descriptive statistics of the variables are displayed in Table 2. The mean PTSD symptom severity score was 24.3 (95% CL for M: 23.7–24.9, Range: 21–74), and the mean CBR score was 1.7 (95% CL for M: 1.6–1.8, Range: 0–6). The mean EHI was 24.5 (95% CL for M: 23.9–25.1, Range: 16.0–57.8), mean perceived neighborhood environment score was 4.6 (95% CL for M: 4.3–4.8, Range: 0–17), and mean experienced discrimination score was 0.03 (95% CL for M: 0.7–0.8, Range: 0–6). As shown in Table 3, examining correlations between the study variables of interest found CBR is significantly correlated with PCL-C score (r=0.081, p<0.001), neighborhood environment score (r=0.068, p=0.003), and EHI (r=0.070, p=0.002), but not experienced discrimination (r=0.011, p=0.6312).

The resulting model and fit statistics are shown in Figure 1 and Table 4. Indices suggest the model provides a good fit to the data, with a CFI of 0.965, TLI of 0.962, and RMSEA estimate of 0.041, 90% CI [0.039, 0.043]. Additionally, the parameter estimates reported in Table 5 demonstrate the validity of the latent structures included in the model. In addition to the well-established PTSD construct, the model supports the validity of socioenvironmental stress as latent variable defined by exposure to discrimination, neighborhood-level socio-economic disadvantage, and increased perception of disordered neighborhood environment. The results also support the validity of the CBR measure utilized in the analysis.

In order to assess the individual relationships between socioenvironmental stress, CBR, and PTSD within the context of the model, the parameter estimates were evaluated (Table 5). Socioenvironmental stress is a significant predictor of PTSD symptom severity (β: 0.364,

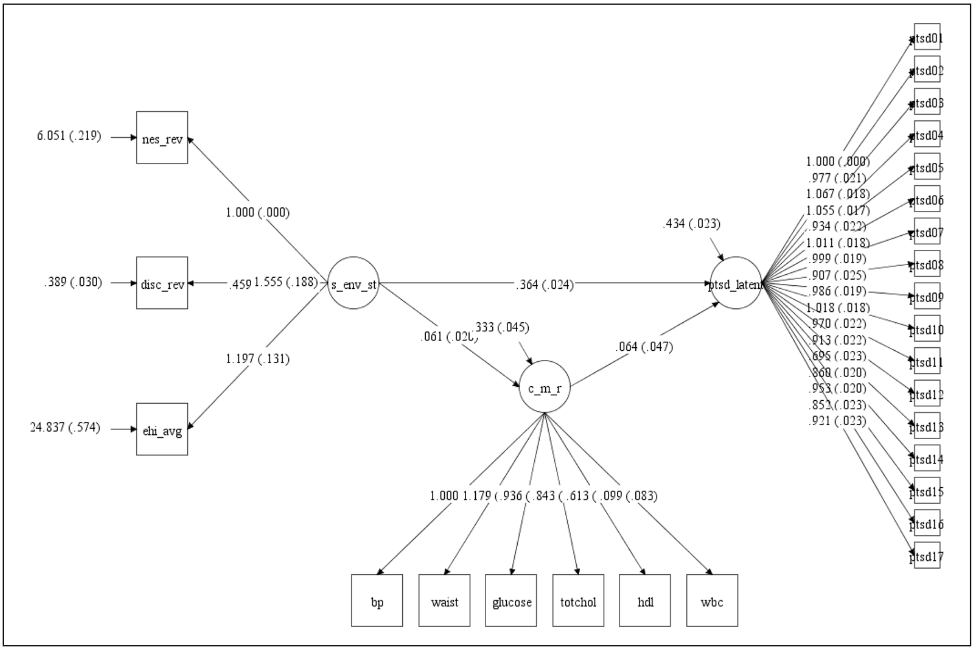

Figure 1. Path diagram of factor loadings and path coefficients for proposed structural equation model.

Table 4. Fit statistics of proposed model ($N = 1959$).

	df	RMSEA (90% CI)	CFI	TLI
Model 1	296	0.041 (0.039, 0.043)	0.965	0.962

RMSEA: root-mean-square error of approximation; CFI: comparative fit index; TLI: Tucker Lewis index.

SE: 0.024, $p < 0.001$) as well as CBR (β: 0.061, SE: 0.020, $p = 0.003$). The association between CBR and PTSD was positively associated but not statistically significant (β: 0.064, SE: 0.047, $p = 0.179$).

Discussion

The purpose of this study was to examine the relationship between socioenvironmental stress, CBR, and PTSD. Although many studies have demonstrated a significant relationship between PTSD and medical comorbidities (for a review, see Ryder et al., 2016), specific mechanisms and pathways are poorly understood. We found that increased socioenvironmental stress was significantly associated with both increased PTSD severity and increased CBR. The inclusion and evaluation of socioenvironmental stress as a latent variable, comprised of an index of neighborhood-level hardship, perceived neighborhood disorder, and experienced discrimination, has not been previously described in the literature. Results of the current study suggest this is a valid latent construct to be used when evaluating stress induced by social environments. Furthermore, it underscores the continued need to consider physical and mental health from a broader socio-ecological perspective.

Another latent construct validated by the model described in this study is CBR, a measure shown to be predictive of health outcomes (King et al., 2011). Results indicate that the index of CBR created by using the biomarkers available in the SHOW dataset (i.e. blood pressure, waist circumference, glycosylated hemoglobin, total cholesterol, high density lipoprotein, and white blood cell count) function as a robust latent construct.

Table 5. Model parameter estimates ($N = 1959$).

Variable	Estimate	SE Est.	*p*-value
SE stress by			
NES	1	0.0	–
DISC	0.46	0.04	0.000
EHI	1.20	0.13	0.000
CBR by			
Blood pressure	1	0.0	–
Waist circumference	1.18	0.12	0.000
Glucose	0.94	0.11	0.000
Total cholesterol	0.84	0.09	0.000
High-density lipoprotein	0.61	0.08	0.000
White blood cell count	0.10	0.08	0.233
PTSD on			
CBR	0.06	0.05	0.179
SE stress	0.36	0.024	0.000
CBR on			
SE stress	0.06	0.02	0.003
Intercepts			
NES	4.56	0.06	0.000
DISC	0.73	0.03	0.000
EHI	25.04	0.15	0.000

CBR: cumulative biological risk; PCL-IV: PTSD checklist – civilian version, for DSM-IV; SE stress: socio-environmental stress; DISC: experienced discrimination; NES: neighborhood environment score; EHI: economic hardship index.

Socio-ecological models emphasize that need to consider the impact of systems and socio-ecological factors on health (Darling, 2007; Lounsbury and Mitchell, 2009; McLeroy, et al., 1988). The novel SEM model tested by this study includes socioenvironmental stress as a latent factor contributing to both physical and mental health outcomes, which is consistent with past studies reporting the health effects of neighborhood environments (Bird et al., 2010; Schulz et al., 2012). Living in disadvantaged neighborhoods is associated with significantly greater CBR in analyses of data from the Jackson Heart Study (Barber et al., 2016) and from the National Health and Nutrition Examination Survey (NHANES) (Merkin et al., 2009). The finding that our latent socioenvironmental stress variable is significantly associated with CBR is also in agreement with the literature on discrimination and health. Exposure to discrimination is not only linked to poor health outcomes (Krieger and Sidney, 1996; Landrine and Klonoff, 2000; Williams et al., 1997), but it is also associated with a variety of the biological markers of stress, including glucocorticoids and markers of inflammation, such as C-reactive protein and proinflammatory cytokines, a set of proteins produced by the immune system important in cell signaling and orchestrating the healing process (Mays et al., 2007; Cooper et al., 2009; Lewis et al., 2010; Cunningham et al., 2012). Additionally, experiencing racial discrimination is associated with an increased risk of hypertension, carotid artery disease, and atherosclerosis, in addition to an increase in the anthropometric measurements of waist circumference and body mass index (Din-Dzietham et al., 2004; Everson-Rose et al., 2015; Troxel et al., 2003). Taken together with the results of the current study, these findings suggest the cumulative physiologic burden on the body from socioenvironmental stressors can have concrete and measurable implications for physical health.

Contrary to our initial hypothesis, the results did not indicate that CBR significantly predicted PTSD in this sample, with no statistically significant relationship between CBR and PTSD found. This was inconsistent with previous literature demonstrating a clear relationship between PTSD and general physical health (Cohen et al., 2015; Heppner et al., 2009; Levine et al., 2014; O'Donovan, 2016). As such, it is notable that no relationship was found with CBR. This lack of significant relationship could be due to several factors. Data on trauma type is not available in the SHOW dataset and is likely highly variable, and the time since trauma among participants is unknown. Because our summary score of cumulative risk is comprised of secondary indicators which presumably take time to develop and manifest physiologically, the length of time since traumatic event is particularly relevant information, and its absence represents a significant limitation. Finally, the ability to detect an association may be limited due to small sample size. Future studies that prospectively investigate history of exposure to socioenvironmental stress and include a more thorough trauma history assessment may be able to create a clearer understanding of the relationships between these factors.

In addition to these constraints, other limitations of this analysis must be taken into consideration. Because this study used cross-sectional data, true cause and effect cannot be determined and future longitudinal studies are needed to establish temporal relationships between exposure to socioenvironmental stress and outcomes such as PTSD and CBR. The lack of data on trauma exposure or information on participant's experience with specific events (for instance, through the Life Events Checklist) represents a gap, as it is possible that increased exposure to traumatic events plays a role in the relationship between socioenvironmental stress and PTSD. Finally, as the study population used in this analysis is predominantly White American, future work is needed to examine socioenvironmental stress and health in underrepresented ethnic and racial groups. This is particularly important in light of studies showing that African Americans who report higher levels of discrimination have higher levels of allostatic load and literature showing associations between discrimination and a variety of biological markers of stress and inflammation including glucocorticoids, C-reactive protein, and proinflammatory cytokines (Cooper et al., 2009; Cunningham et al., 2012; Lewis et al., 2010; Mays et al., 2007; Tomfohr et al., 2016). Likewise, while the current study incorporated experiences of discrimination into socioenvironmental stress, specific types of discrimination were not differentiated. Future studies should consider using validated measures of experiences of discrimination to assess its role on CBR and PTSD symptoms.

Clarifying the relationships between socioenvironmental stressors and physiological reactions to stress is a critical step in designing interventions, and reducing the health disparities that exist in the PTSD patient population. Given the clear influence of socioenvironmental factors on CBR and PTSD symptoms in the current study, this underscores a need to better assess for social stressors and barriers when considering treatment. Furthermore, considering the role of potential exposure to discrimination is a necessary aspect of the assessment of socioenvironmental stress. Providers might assess for these as potential barriers to treatment, providing better access to needed health care. Additionally, socio-ecological perspectives underscore the need to address systemic issues within intervention, highlighting need to craft policy and create community-based intervention to address socioenvironmental stress such as discorded neighborhood environments (Lounsbury and Mitchell, 2009; McLeroy et al., 1988). Lastly, it is critical to gain better understanding of the interaction of socioenvironmental stress and biological mechanisms that link to mental health. The role of socioenvironmental factors as a contributor to stress exposure and subsequently increased biologic vulnerability and decreased mental health provides a potential explanatory pathway between social environment and poor health.

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