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RESEARCH ARTICLE

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The role of pain and socioenvironmental factors on posttraumatic stress disorder symptoms in traumatically injured adults: A 1-year prospective study

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Abstract

Approximately 20% of individuals who experience a traumatic injury will subsequently develop posttraumatic stress disorder (PTSD). Physical pain following traumatic injury has received increasing attention as both a distinct, functionally debilitating disorder and a comorbid symptom related to PTSD. Studies have demonstrated that both clinician-assessed injury severity and patient pain ratings can be important predictors of nonremitting PTSD; however, few have examined pain and PTSD alongside socioenvironmental factors. We postulated that both area- and individual-level socioeconomic circumstances and lifetime trauma history would be uniquely associated with PTSD symptoms and interact with the pain–PTSD association. To test these effects, pain and PTSD symptoms were assessed at four visits across a 1-year period in a sample of 219 traumatically injured participants recruited from a Level 1 trauma center. We used a hierarchal linear modeling approach to evaluate whether (a) patient-reported pain ratings were a better predictor of PTSD than clinician-assessed injury severity scores and (b) socioenvironmental factors, specifically neighborhood socioeconomic disadvantage, individual income, and lifetime trauma history, influenced the pain–PTSD association. Results demonstrated associations between patient-reported pain ratings, but not clinician-assessed injury severity scores, and PTSD symptoms, $R^{2(fvm)} = .65$. There was a significant interaction between neighborhood socioeconomic disadvantage and pain such that higher disadvantage decreased the strength of the pain–PTSD association but only among White participants, $R^{2(fvm)} = .69$. Future directions include testing this question in a larger, more diverse sample of trauma survivors (e.g., geographically diverse) and examining factors that may alleviate both pain and PTSD symptoms.

Posttraumatic stress disorder (PTSD) is characterized by a constellation of symptoms including hyperarousal, avoidance of trauma-related stimuli, negative alterations in mood and cognition, and reexperiencing (e.g., flashbacks; American Psychiatric Association, 2013). In the United States, national studies have shown that of the 90% of residents exposed to a traumatic event, approximately 8%–10% will go on to develop PTSD (Kilpatrick et al., 2013). However, it remains unclear as to what sets apart this minority of individuals given the heterogeneity of PTSD symptoms and its various comorbidities (Galatzer-Levy & Bryant, 2013). One area of work that has received increasing attention in the literature is the co-occurrence of PTSD and pain (Otis et al., 2003).

Patient-reported pain is highly comorbid with and predictive of PTSD symptoms (Brasel et al., 2010), and individuals diagnosed with PTSD report higher levels of pain sensitivity and higher pain-related impairment ratings (Phifer et al., 2011). Compared to individuals with chronic pain symptoms alone, those with comorbid PTSD and chronic pain report more severe pain and poorer quality of life (Scioli-Salter et al., 2015). These individuals have also been shown to experience more symptoms of depression and anxiety (Irwin et al., 2014). Previous work has demonstrated that self-reported pain in the acute aftermath of a traumatic injury is more predictive of future PTSD symptoms than clinician-reported injury severity scores (ISSs), with individuals who report chronic pain 4 months postinjury displaying significantly more symptoms of depression, anxiety, and PTSD (Trevino et al., 2014). This suggests "objective" measures of injury severity (e.g., an ISS determined in the emergency room by a clinician) do not help predict mental health–related injury outcomes over individuals' own perceptions of their pain.

Two prominent theories have sought to explain why PTSD and chronic pain are related (Asmundson et al., 2002; Sharp & Harvey, 2001). Sharp and Harvey (2001) reported seven "mutual maintenance factors" between pain and PTSD, including attentional biases to pain or trauma stimuli, higher anxiety sensitivity, persistent reminders of the traumatic event, avoidant coping styles, depression, higher pain perception, and depleted cognitive resources. They proposed that painful sensations can remind the patient of the trauma, the avoidance of pain may lead to similar avoidance and maintenance or exacerbation of PTSD symptoms, and intensified anxiety can increase a patient's perceived pain levels (Sharp & Harvey, 2001). Asmundson et al. (2002) described "shared vulnerability factors" between PTSD and pain, which are contingent upon the role of anxiety in the individual's life. The authors propose that increased anxiety sensitivity during a stressful situation may predispose an individual to PTSD and chronic pain due to heightened alerting and awareness (Asmundson et al., 2002).

Researchers have explored the possible mechanisms underlying this link between diagnosed PTSD and pain, the role of mediators like depression or anxiety in this association, and the etiological underpinnings surrounding trauma and pathological factors (Brennstuhl et al., 2015). The neurobiological mechanisms, including preexisting neural vulnerabilities that may predispose individuals to PTSD and/or pain, have also received considerable attention. Importantly, there is substantial overlap in the neural circuitry impacted by pain and PTSD, supporting a biological mutual maintenance mechanism by which pain and PTSD may synergistically relate (Scioli-Salter et al., 2015). However, the role of sociodemographic risk and resiliency factors as they relate to associations between pain and PTSD remains unclear (Lillis et al., 2018).

Sociodemographic factors have been shown to be related to both PTSD and pain, with lower socioeconomic position (SEP), quantified using income, employment status, educational attainment, and neighborhood poverty level, associated with more severe PTSD symptoms and worse pain outcomes (Davies et al., 2009; Phifer et al., 2011). Inequities in SEP are highly correlated with race and ethnicity due to systems of oppression maintaining higher neighborhood poverty rates, fewer educational opportunities, and lower income for individuals from minoritized backgrounds (Lillis et al., 2018; Williams et al., 2010). As such, in the United States, there is a higher prevalence of PTSD among Black individuals compared to their non-Hispanic White counterparts. This is not necessarily due to a higher frequency of trauma exposure among the Black community but rather disparities in exposure to trauma types and/or racialized life experiences (Harnett & Ressler, 2021). Lillis and colleagues (2020) found that SEP factors mediated the associations among PTSD, pain intensity, and pain interference in Black women. More explicitly, their findings suggest that the elevated pain observed in Black participants may be due to racialized SEP inequities (Lillis et al., 2020).

Although neighborhood socioeconomic disadvantage (NSD) and individual SEP are related, they represent independent factors that influence mental health. NSD, operationalized using a composite score of various neighborhood-level factors (e.g., the number of individuals in a neighborhood who are employed), serves as a proxy for a neighborhood's SEP. A neighborhood's SEP is critical, as it shapes access to myriad financial, material, and social resources (Diez Roux & Mair, 2010; Kind & Buckingham, 2018). Even after adjusting for individual-level SEP factors, NSD has been shown to explain alterations in biological markers of the stress response (Barrington et al., 2014; Finegood et al., 2017). Overall, the literature supports investigating associations

between pain and PTSD in populations that have been systemically stripped of resources, both at the individual and neighborhood levels.

The current study had two aims, which broadly sought to disentangle associations among clinician-assessed injury severity, participant-reported pain, socioenvironmental factors, and PTSD. We recruited adults who had recently experienced a traumatic injury and conducted follow-up visits for 1 year (i.e., a total of four visits). First, we tested whether participant-reported pain ratings were a better predictor of PTSD than clinician-assessed ISS and examined how pain and PTSD changed across the 1-year study. Given previous work (Brasel et al., 2010), we anticipated that participant-reported pain ratings would more accurately track symptoms, with higher levels of pain associated with more severe PTSD symptoms. Second, we tested whether socioeconomic variables, specifically NSD and individual income, were significantly related to PTSD symptoms after adjusting for individual life events and gender. We anticipated that socioeconomic variables may interact with pain and time such that participants in lower socioeconomic positions would report higher pain levels and experience nonremitting PTSD symptoms. Finally, we explored whether these associations varied by ethnoracial group. We expected to replicate Lillis et al. (2020) and demonstrate stronger associations between pain and PTSD in Black participants, which would be explained by socioenvironmental variables.

METHOD

Participants

Participants who experienced a physical traumatic injury were recruited from the emergency department (ED) of a Level 1 trauma center in southeastern Wisconsin, United States, as part of the Imaging Study on Trauma and Resilience (iSTAR). Participants were included in iSTAR if they were English-speaking, between 18and 60 years of age, and scored a minimum of 3 on the Predicting PTSD Questionnaire (PPQ; Rothbaum et al., 2014) or endorsed that the trauma was a near-death experience. The PPQ cutoff score was implemented to recruit participants who had a heightened risk of developing PTSD (Rothbuam et al., 2014). Participants were excluded from the larger project if they scored 13 or lower on the Glasgow Coma Scale (Sternbach, 2000), which was administered to exclude moderate-to-severe head injuries; had a spinal cord injury accompanied by neurological deficits or were diagnosed with any neurological conditions; had a self-inflicted injury; had severe visual or hearing impairments; disclosed a history of psychotic or manic symptoms or current antipsychotic medication use; were unable to verbally communicate; had experienced a sexual assault traumatic event that necessitated the ED visit; were on a police hold; displayed moderate-to-severe cognitive impairment secondary to trauma-related head injury; tested positive for alcohol, defined as a blood alcohol level than 0.08%, and/or illegal drugs and/or prescription narcotics at the time of the traumatic injury; or had eye conditions that prevented the use of an eye-tracking assessment.

Procedure

The project protocol was approved by the Medical College of Wisconsin Institutional Review Board. Participants provided written informed consent prior to participation and completed study visits at 2 weeks (i.e., baseline; Time 0 [T0]), 3 months (Time 1 [T1]), 6 months (Time 2 [T2]), and 12 months postinjury (Time 3 [T3]). All participants were financially compensated for their time.

A total of 246 participants were initially enrolled in the study; however, we experienced participant attrition across all study time points. Specifically, approximately 89% (n = 219) of the sample completed T0, with 163 total participants (n = 163) returning for T1. Although 79.3% of the full sample (n = 195) returned for T2, only 50.0% (n = 123) returned for T3. Despite this drop-off, we included participants who completed baseline assessments even if they had missing data from other time points. This resulted in a final sample size of 219 (n = 117 women; M_{age} = 33.30 years, SE = 0.73) for the present analyses. Descriptive statistics for self-report measures administered at each time point can be found in Table 1.

Measures

Demographic information

Participants self-reported their gender (male, female, or other), race, ethnicity, and annual household income (see Table 2 for sociodemographic information). Income was reported using a semicontinuous scale in $10,000 increments (e.g., an annual household income ranging from $0 to 10,000 was coded as 1; all incomes over $100,000 were coded as 11).

PTSD symptom severity

The PTSD Checklist for *DSM-5* (PCL-5; Weathers, Litz, et al., 2013) was used to assess PTSD symptoms at all study visits. Participants were asked to indicate how much each

TABLE 1 Descriptive statistics of the sample, by follow-up time

Variable	Assessment point[a]							
	T0		T1		T2		T3	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Pain	4.37	0.26	3.18	0.27	2.77	0.28	2.80	0.29
PCL-5	26.4	1.68	24.2	1.62	19.6	1.56	19.1	1.76
ADI	70.9	1.43	-	-	-			
ISS	0.84	0.15	-	-	-			
LEC	31.5	1.14	-	-	-			

Note: T0 = Time 0, T1 = Time 1; T2 = Time 2; T3 = Time 3; PCL-5 = Posttraumatic Stress Disorder Checklist for *DSM*-5; ADI = Area Deprivation Index; ISS = Injury Severity Score; LEC = Lifetime Events Checklist.

[a]Assessments took place 2 weeks postinjury (i.e., T0, baseline), 3 months postinjury (T1), 6 months postinjury (T2), and 12 months postinjury (T3).

of the 20 items bothered them, scoring responses on a 5-point Likert scale ranging from 1 (*not at all*) to 5 (*extremely*). A total symptom severity score was calculated by summing all responses, with higher scores indicating a higher level of PTSD severity (Blevins et al., 2015). In the present sample, Cronbach's alpha was .94 at baseline.

Physical pain ratings

At all assessment points, participants completed the widely used Visual Analogue Scale for Pain (VAS; Holdgate et al., 2003) to assess physical pain severity. Participants were asked to rate their pain using a numbered line with labels ranging from 0 (*no pain*) to 10 (*worst possible pain*).

Neighborhood socioeconomic disadvantage

A National Area Deprivation Index (ADI) score was derived from the participant's home address as provided at T0 (Kind & Buckingham, 2018). ADI is a factor-based percentile ranking of a neighborhood's disadvantage comprising of 17 socioeconomic variables (e.g., number of households with complete plumbing). Each census-block group has a ranking between 1 and 100, where 100 is indicative of the most disadvantaged neighborhoods in the United States relative to all the other neighborhoods in the country, and 1 corresponds with the most advantaged neighborhoods.

Injury severity

The ISS was clinician-assessed in the ED at baseline (i.e., T0; Osler et al., 1997). Briefly, the ISS is used to evaluate trauma severity by first assigning an abbreviated injury scale (AIS) score for six major body regions (e.g., head, face, extremities). The AIS is then squared, and each region's score is summed to create a total ISS. The ISS can range from 1 to 7, with a value of 15 indicating severe trauma.

Lifetime trauma history

The Life Events Checklist (LEC; Weathers, Blake, et al., 2013) was used at T0 to assess lifetime exposure to traumatic and/or stressful experiences. Participants were asked by a trained research staff member whether they experienced, witnessed, or learned about any of the 16 events listed (e.g., natural disaster). A recently developed weighted-total scoring method (Weis et al., 2021) was used. Items the participant endorsed as having experienced were

TABLE 2 Baseline sociodemographic characteristics

Variable	*n*	%	M_{age} (years)	*SE*
Gender				
Male	102	46.6	32.8	1.05
Female	117	53.4	33.8	1.02
Ethnicity				
Hispanic or Latino	20	9.1	29.6	2.16
Not Hispanic or Latino	197	90.0	33.7	0.77
Race				
Black or African American	138	63.0	34.3	0.94
White	51	23.3	33.4	1.53
More than one race	14	6.4	26.5	1.71
Unknown or not reported	13	5.5	30.1	2.64
Annual income (USD)				
< $10,000	49	22.4	31.4	1.50
$10,001–$20,000	34	15.5	35.1	1.84
$20,001–$30,000	37	16.9	31.9	1.65
$30,001–$40,000	16	7.3	33.9	2.73
$40,001–$50,000	20	9.1	37.9	2.77
$50,001–$60,000	14	6.4	33.7	3.47
$60,001–$70,000	13	5.9	31.1	2.92
$70,001–$80,000	12	5.5	37.9	3.39
≥ $80,001	23	10.5	31.9	1.82
Educational attainment				
Did not finish high school	24	11.0	31.3	2.50
High school graduate	60	27.4	30.8	1.40
GED	14	6.4	37.9	2.76
Some college, no degree	58	26.5	32.2	1.44
Associate's degree	30	13.7	38.2	1.73
Bachelor's Degree	25	11.4	35.7	1.92
Master's degree and above	8	3.7	31.6	2.31
Trauma type				
Assault/altercation	27	12.3	34.4	2.03
Motor vehicle crash	150	68.5	33.1	0.88
Other	42	19.2	33.5	1.75

Note: $N = 219$. Exact percentages of groups with less than 5% of participants are not reported to ensure anonymity.

weighted with a 3, items witnessed were weighted with a 2, and events learned about were weighted with a 1. The scores for each item were summed to create a weighted total score (range: 0–102; Weis et al., 2021). In the present sample, Cronbach's alpha was .91 at T1.

Data analysis

Bivariate associations between measures are presented in Table 3. We conducted independent *t* tests to examine whether there were significant differences between individuals who completed the study and those who were lost to follow-up. We also conducted two one-way analyses of variance (ANOVAs) to better understand changes in pain and PTSD symptom levels across study visits. For the primary analyses, data were analyzed using a hierarchal linear modeling approach. Time was coded such that the initial 2-week posttraumatic event time point was 0 (i.e., T0 = 0). Income was coded such that the lowest income group (i.e., $0–$10,000) was 0. As no participants selected "other" for gender and wrote in a gender identity, gender was coded as 0 for male and 1 for female. Physical pain ratings, ADI, ISS, and LEC, were grand-mean centered. All models employed the maximum likelihood (ML) method. Gender and income were covariates in the models and treated as

TABLE 3 Bivariate correlations between study variables

	T1 Pain	T2 Pain	T3 Pain	T0 PCL	T1 PCL	T2 PCL	T3 PCL	LEC[a]	ADI	ISS	Income	Gender
T0 Pain	.537*	.480*	.496*	.280*	.263*	.221*	.301*	.100	.186*	−.033	−.177*	.024
T1 Pain	-	.659*	.665*	.153	.289*	.186*	.230*	.072	.219*	−.138	−.158*	.039
T2 Pain		-	.615*	.197*	.252*	.376*	.279*	.136	.184*	−.033	−.228*	.014
T3 Pain			-	.311*	.322*	.230*	.381*	−.052	.203*	− .027	−.136	−.002
T0 PCL				-	.746*	.544*	.654*	.263*	.019	.068	−.091	.113
T1 PCL					-	.616*	.709*	.177*	.160*	.006	−.247*	.107
T2 PCL						-	.556*	.173*	.068	.085	−.229*	.042
T3 PCL							-	.148	.093	.121	−.225*	.064
LEC[a]								-	−.041	−.026	−.018	−.056
ADI									-	−.104	−.472*	−.008
ISS										-	−.025	−.126
Income											-	−.121

Note: T0 = Time 0 (baseline, 2 weeks postinjury), T1 = Time 1(3 months postinjury); T2 = Time 2 (6 months postinjury); T3 = Time 3 (12 months postinjury); PCL = Posttraumatic Stress Disorder Checklist for *DSM-5*; ADI = Area Deprivation Index (national ranking); LEC = Life Events Checklist; ISS = Injury Severity Score.

[a]Weighted score.

*$p < .05$; **$p < .01$; ***$p < .001$.

Level 2 predictor variables with fixed effects. All Level 1 variables were nested within each participant.

Effects of pain and time of study visit on PTSD

First, we examined whether VAS ratings (Level 1 predictor) and time of study visit (Level 1 predictor) predicted PCL-5 scores and evaluated whether time and VAS ratings should be treated as fixed or random effects in predicting PCL-5 score, using separate models for each. The results of the full multilevel model revealed that time as a random slope did not significantly contribute to predicting PCL-5 score over and above a model where time was treated as a fixed effect, $\chi^2(1, N = 219) < .001$, $p > .999$. Similarly, we found that the full model containing VAS ratings as a random effect did not significantly contribute to predicting PCL-5 score compared to the model with VAS as a fixed effect, $\chi^2(1, N = 219) < .001$, $p > .999$. All subsequent models included time and VAS ratings as fixed effects.

Next, we assessed how the addition of ISS (Level 2 predictor) contributed to predicting PCL-5 score over and above the model containing time and VAS ratings by conducting two reduced versus full model comparisons. We then examined whether the VAS x Time interaction (Level 1 predictor) significantly contributed to predicting PCL-5 score by conducting two reduced versus full model comparisons, similar to the ISS analyses.

Effects of socioenvironmental factors and pain on PTSD across time

We were also interested in examining whether the slopes between VAS and PCL-5 scores, and time and PCL-5 scores, changed based on ADI, income, and LEC. First, we compared a reduced model (Level 1 predictors: time, VAS, VAS x Time; Level 2 predictors: income, LEC, ADI, and gender) against a full model including these variables along with the interaction VAS x ADI interaction term. Our second model comparison included the same reduced model variables against a full model with these same variables and the Time x ADI interaction term. Finally, we conducted a model comparison between our previously described reduced model against a full model that included these variables and the VAS x Income interaction term. Any interaction terms that significantly contributed to our ability to predict PCL-5 score were included in the final model.

Interactions between ethnoracial group, socioenvironmental factors, and pain

We performed exploratory analyses to examine whether associations between study measures varied by ethnoracial group. Due to small sample sizes across certain ethnoracial groups, we restricted our sample to examine only participants who identified as Black and/or African American (coded as 0; $n = 138$) or White (coded as 1; $n = 51$). We computed three separate interaction terms by multiplying ADI, income, and LEC grand-mean centered scores with the coded ethnoracial grouping variable. To aid interpretation and enable us to examine whether certain effects were driven by ethnoracial categories, we ran two separate models, one for each ethnoracial group.

RESULTS

There were no significant differences in ADI, T0 ISS, T0 VAS, T0 PCL-5, and LEC between the individuals who were enrolled and did not complete the study and those who completed assessments at all follow-up points. However, there was a significant difference in income, $t(242) = -2.62$, $p = .009$ such that participants who completed the study reported significantly higher income ($M = 4.61$, $SD = 3.19$), equal to approximately $10,000 more per year, than those who did not complete the study ($M = 3.64$, $SD = 2.63$).

Improvement in pain and PTSD symptoms across time

The results of a one-way ANOVA indicated that PCL-5 scores significantly decreased over time, $F(2.66, 303.04) = 12.50$, $p < .001$, $\eta_p^2 = .099$, with Bonferroni post hoc comparisons indicating significant decreases in PCL-5 score between T0 and both T2, $t(114) = 4.20$, $p < .001$, and T3, $t(114) = 5.23$, $p < .001$, but not between T0 and T1, $t(114) = 2.05$, $p = .257$. High PCL-5 scores were reported for T1 compared to T2, $t(114) = 3.07$, $p = .016$, and T3, $t(114) = 3.91$, $p < .001$. However, PCL-5 scores did not significantly decrease from T2 to T3, $t(114) = 0.30$, $p > .999$.

A one-way ANOVA revealed that VAS ratings significantly decreased over time, $F(2.81, 319.83) = 17.30$, $p < .001$, $\eta_p^2 = .131$. Follow-up Bonferroni post hoc comparisons indicated significant decreases in VAS between T0 and T1, $t(114) = 4.54$, $p < .001$; T2, $t(114) = 5.69$, $p < .001$; and T3, $t(114) = 5.67$, $p < .001$. VAS ratings did not significantly decrease between T1 and T2, $t(114) = 1.92$, $p = .341$, or T3, $t(114) = 1.65$, $p = .616$, or between T2 and T3, $t(114) = -0.10$, $p > .999$.

Contributions of pain, ISS, and time to PTSD symptom severity

The findings indicated that including ISS as a fixed effect or random effect did not significantly contribute to predicting PCL-5 scores over the reduced model, $\chi^2(1, N = 219) = 2.52$, $p = .113$ and $\chi^2(2, N = 219) = 0.10$, $p = .951$, respectively. Therefore, ISS was excluded in subsequent models. The addition of the VAS x Time interaction term did not contribute to the prediction of PCL-5 score when treated as a fixed effect, $\chi^2(1, N = 219) = 1.75$, $p = .186$, but significantly contributed over and above the reduced model when treated as a random effect, $\chi^2(2, N = 219) = 41.55$, $p < .001$. Despite this, we chose not to include this variable as a random effect due to a violation of the assumption that the fixed effects should not covary with the random effects specified in the model (McNeish & Kelley, 2019). Specifically, the VAS x Time interaction term showed a covariance of 13.05 with VAS and a covariance of −0.61 with time.

In our final model, which included time, VAS ratings, income, and gender, only the fixed effects of income, $t(216.05) = -2.29$, $p = .023$; time, $t(517.47) = -5.53$, $p < .001$; and VAS, $t(693.05) = 7.04$, $p < .001$, were significant predictors of PCL-5 score. Specifically, PCL-5 scores were predicted to decrease by 2.22 per subsequent assessment point and 0.78 per every $10,000 increase in annual income and increase by 1.49 for every 1-unit increase in VAS rating. In contrast, gender, $t(215.04) = 1.00$, $p = .319$, did not significantly contribute to predicting PCL-5 score. The final model yielded variability values of 191.3 for the intercepts and a residual variability of 117.6.

Socioenvironmental factors as predictors of PTSD symptom severity

The cross-level interaction variable of VAS x ADI significantly predicted PCL-5 score over and above the reduced model that included the Level 1 predictors of VAS and time as well as the Level 2 variables of income, gender, LEC, and ADI, $\chi^2(1, N = 219) = 5.43$, $p = .020$. However, the second model comparison demonstrated that the full model, which included the ADI x Time cross-level interaction variable, did not significantly contribute to our ability to predict PCL-5 scores over the reduced model, $\chi^2(1, N = 219) = 0.70$, $p = .402$. The final model comparison included the cross-level interaction of VAS x Income, which also did not significantly predict PCL-5 score beyond the reduced model, $\chi^2(1, N = 219) = 0.05$, $p = .821$. Therefore, a final model was composed of the Level 1 variables time and VAS, Level 2 variables LEC, ADI, income, and gender, and the cross-level interaction variable of VAS x ADI. All predictor

variables were treated as fixed effects. The adjusted intraclass correlation coefficient was .596, indicating that this model accounted for approximately 59.6% of the variability in PCL-5 scores.

At T0, male participants with an annual income of less than $10,000, with an average VAS rating, average LEC score, and average ADI were associated with a PCL-5 score of 27.38, 95% CI [23.36, 31.40]. Time significantly predicted PCL-5, $t(518.51) = -5.46$, $p < .001$. Specifically, PCL-5 scores were predicted to decrease by 2.18, 95% CI [−2.96, −1.40], across each assessment point when all other variables were held constant. VAS ratings also significantly predicted PCL-5 score, $t(691.96) = 7.07$, $p < .001$, such that for every 1-unit increase in VAS ratings, PCL-5 scores were predicted to increase by approximately 1.49, 95% CI [1.08, 1.91], when all other variables were held constant. PTSD symptoms were also significantly predicted by LEC scores, $t(220.50) = 3.32$, $p = .001$. Specifically, PCL-5 scores were predicted to increase by 0.20, 95% CI [0.08, 0.31], for every 1-unit increase in LEC score. The VAS x ADI interaction also significantly predicted PCL-5 score, $t(694.14) = -2.34$, $p = .020$, such that the association between VAS and PCL-5 was predicted to decrease by 0.02, 95% CI [−0.04, −0.003], for every 1-unit increase in ADI. Income also significantly predicted PTSD symptoms, $t(694.14) = -2.34$, $p = .020$, such that PCL-5 scores decreased by 0.75, 95% CI [−1.50, −0.008], for every 1-unit increase in income. However, ADI, $t(214.41) = -0.23$, $p = .818$, and gender, $t(217.937) = 1.41$, $p = .160$, did not significantly predict changes in PCL-5 scores in our final model. The final model yielded a variability value of 170.8, 95% CI [135.16, 216.52], for the intercepts and a residual error variability of 116.0, 95% CI [102.46, 131.99].

Interactions between ethnoracial group, socioenvironmental factors, and pain

Exploratory independent *t* tests with only participants who identified as White or Black ($n = 189$) indicated that White participants lived in more advantaged neighborhoods ($M = 52.02$, $SE = 3.22$ vs. $M = 78.73$, $SE = 1.30$), $t(187) = 9.23$, $p < .001$, and reported higher income than Black participants ($M = 6.39$, $SE = 0.49$ vs. $M = 3.50$, $SE = 0.21$), $t(187) = -6.29$, $p < .001$. There were no differences between Black and White participants with regard to LEC score, $t(187) = -0.73$, $p = .465$; baseline PTSD symptoms, $t(187) = -0.57$, $p = .570$); or ISS, $t(187) = -1.60$, $p = .111$. However, Black participants reported significantly higher VAS ratings ($M = 4.75$, $SE = 0.26$) than White participants ($M = 3.40$, $SE = 0.34$), $t(187) = 2.86$, $p = .005$.

Additional exploratory analyses revealed similar findings compared to the final model described previously: income, time, pain, LEC, and the VAS x ADI interaction predicted PTSD symptoms; gender was no longer significant. Adding ethnoracial group to this final model did not significantly improve the model fit, $\chi^2(1, N = 189) = 2.55$, $p = .255$. Additional model comparisons found that the interaction terms of Ethnoracial Group x Income, $\chi^2(1, N = 189) = 1.20$, $p = .273$, and Ethnoracial Group x ADI, $\chi^2(1, N = 189) = 0.05$, $p = .831$, did not significantly predict PCL-5 over and above the reduced model. However, a VAS x ADI x Ethnoracial Group three-way interaction significantly improved the ability to predict PCL-5 scores compared with the reduced model, $\chi^2(1, N = 189) = 18.85$, $p < .001$. The addition of the Ethnoracial Group x LEC interaction term did not significantly improve our ability to predict PCL-5 score compared to the previous three-way interaction model, $\chi^2(3, N = 189) = 6.56$, $p = .087$. We disaggregated the data to examine White and Black participants separately and examined the results from a model predicting PCL-5 with income, time, VAS, LEC, and VAS x ADI.

In White participants, PCL-5 score were predicted to decrease by 2.55, $t(139.42) = -3.46$, 95% CI [−3.99, −1.10], $p < .001$, CI], across each assessment point. VAS x ADI significantly predicted PCL-5 score, $t(171.07) = -2.94$, $p = .003$, such that the association between VAS ratings and PCL-5 scores was predicted to decrease by 0.06, 95% CI [−0.09, −0.02], for every 1-unit increase in ADI score. Income also significantly predicted PTSD symptoms, $t(53.53) = -3.33$, $p = .002$, such that PCL-5 scores decreased by 1.94 points, 95% CI [−3.10, −0.08], for every 1-unit increase in income. However, ADI, $t(54.74) = -1.16$, $p = .253$; VAS, $t(174.63) = 1.51$, $p = .131$; LEC score, $t(54.18) = -1.97$, $p = .054$; and gender, $t(50.82) = 1.50$, $p = .140$, did not significantly predict changes in PCL-5 in the final model.

For Black participants, PCL-5 score were predicted to decrease by 1.64, $t(309.23) = -3.22$, $p = .001$, 95% CI [−2.64, −0.64], across each assessment point. VAS ratings significantly predicted PTSD symptoms, $t(425.94) = 1.20$, $p < .001$, such that for every 1-unit increase in VAS rating, PCL-5 scores were expected to increase by 1.20, 95% CI [0.65, 1.76]. Lifetime trauma exposure also predicted PTSD symptoms, $t(137.85) = -4.57$, $p < .001$, such that PCL-5 scores increased by 0.34, 95% CI [0.19, 0.47], for every 1-unit LEC increase. Income significantly predicted PCL-5, $t(53.53) = -3.33$, $p = .002$, in that PCL-5 scores decreased by 1.94, 95% CI [−3.10, −0.08], for every 1-unit increase in income. ADI, $t(130.99) = 0.05$, $p = .590$; VAS x ADI, $t(409.40) = 0.007$, $p = .676$; and gender, $t(136.74) = 0.70$, $p = .487$, did not significantly predict changes in PCL-5 score.

DISCUSSION

In this 1-year prospective study in a sample of traumatically injured adults, we assessed the effects of pain, clinician-assessed ISS, and socioenvironmental factors

on PTSD symptoms and examined whether interactions among these variables played a role in PCL-5 scores across assessment points. Overall, we found a significant decrease in PTSD symptoms across time, with the highest symptoms reported at baseline. Further, we replicated previous work indicating that participant-reported pain, and not ISS, was associated with PTSD symptoms across time (Brasel et al., 2010). Surprisingly, individual income, but not NSD, was related to PTSD symptoms such that every additional $10,000 in income was associated with a 1-unit decrease in PTSD symptoms.

Clinician-assessed ISS has been proposed as a more objective measure of assessing injury severity than self-report measures; however, the predictive ability of the ISS for posttraumatic outcomes has been mixed (Delahanty et al., 2003). Although Delahanty and colleagues (2003) demonstrated that cortisol levels mediated the association between ISS and PTSD in participants who experienced a motor vehicle crash, the present results align with recent work that patient-reported pain has more utility in predicting quality of life after a traumatic injury (Brasel et al., 2010; Geiger et al., 2011; Trevino et al., 2014). Other individual factors may be relevant in identifying how pain and PTSD bidirectionally contribute to each other. For example, insomnia has also been reported in veterans experiencing PTSD and co-occurring pain, which may affect cognition, irritability, and rehabilitation efforts (Lang et al., 2014).

Socioenvironmental factors, including both area- (e.g., NSD) and individual-level (e.g., income) variables, are well-documented predictors of posttrauma outcomes (Gapen et al., 2011; Schuck & Widom, 2019). However, to our knowledge, no previous work has tested the role of socioenvironmental variables in the association between pain and PTSD after adjusting for lifetime trauma history. We found that income, but not ADI, was a significant predictor of PTSD symptoms. Importantly, this effect was significant even after controlling for LEC score. Individual income has been shown to be directly related to financial and physical access to physical and mental health care (Lurie & Dubowitz, 2007). Lower income is broadly related to exposure to more stressors, which may exacerbate acute PTSD symptoms and maintain persistent nonremitting PTSD (Green & Hart-Johnson, 2012; Lillis et al., 2018). In addition, individual income and NSD have also been significantly associated with chronic pain (Green & Hart-Johnson, 2012). Interestingly, although neighborhood characteristics are hypothesized to contribute to PTSD symptoms through various mechanisms (Gapen et al., 2011; Lowe et al., 2016), we did not find a direct association between ADI and PCL-5 scores. One potential explanation for this outcome is that we were underpowered to observe neighborhood-level effects, which are well-documented to have more modest effect sizes compared to individual-level socioeconomic variables (Diez Roux & Mair, 2010).

Surprisingly, we observed a significant ADI x Pain interaction, suggesting that living in a disadvantaged neighborhood was related to a decrease in the strength of the association between pain and PTSD. In our exploratory analyses, we tested whether the associations we observed were racialized. Due to small sample sizes across ethnoracial groups, we were compelled to restrict our investigation to Black and White participants. There were significant differences between Black and White individuals on income and ADI rankings, which reflected structural forms of racism in the U.S. population (Williams et al., 2010, 2019). The racialized patterns of ADI and income necessitates cautious interpretation. Upon further investigation, we found that among White participants, pain and LEC scores did not significantly predict PCL-5 scores, although lower income was related to more severe PTSD symptoms. However, for Black participants, income, pain, and lifetime trauma exposure were significant predictors of PTSD.

The negative interaction between ADI x Pain held in White participants but was not present in Black participants. NSD is not protective; rather, we interpret this result to reflect the racialized social systems in the United States, which are known to benefit White people (i.e., white privilege; Malat et al., 2018). Whiteness grant access to social and material resources that are intentionally withheld from people of color (Malat et al., 2018). We speculate that even when living in a disadvantaged neighborhood, White individuals may have reliable and abundant resources that can buffer against the associations between pain and PTSD symptoms. We did not find an effect of ADI in Black participants. One potential explanation is that Black people disproportionately live in more disadvantaged neighborhoods, limiting our ability to observe a statistically significant effect. Although these were exploratory analyses, the results suggest disparities in how NSD influences health outcomes. Future work should employ more nuanced and intersectional approaches to understand how interactions between socioeconomic position, NSD, and physical and psychological symptoms drive ethnoracial health disparities.

These findings reflect several limitations regarding how individual socioeconomic position and NSD were evaluated. Participants were asked to report their annual household income; however, the size of the household, which influences income and expenses, was not assessed. Therefore, income could not be normalized to household size. In addition, both income and NSD were measured only at the first visit. Changes in income and NSD may have occurred for participants across the year-long study. In

addition, no information was collected on income or housing stability. The absence of this information may help explain why we did not observe a significant association between NSD and PTSD symptoms. There may be a dose-dependent effect wherein the length of time an individual resides in a given neighborhood influences the association between NSD and health outcomes. We encourage future researchers to approach the collection of sociodemographic information deliberately to ensure appropriate capture of such variables at each visit, particularly in longitudinal studies.

Our results align with the large body of work indicating a clinically meaningful and robust interplay between pain and PTSD (Stratton et al., 2014). As larger samples are recruited and longitudinal designs are employed, the directionality and specificity of pain and PTSD symptom development and influence can better be tested. For example, more recent work suggests that among civilians who experience trauma, PTSD intrusion and hyperarousal symptoms may significantly influence pain (de Vries et al., 2021; Liedl et al., 2010). Interestingly, de Vries and colleagues (2021) found that across time, the connection between pain and PTSD switched between theoretical models. Specifically, the mutual maintenance of pain and intrusion-related PTSD symptoms was apparent only in the earlier study phases, and later phases suggested that PTSD symptoms drove pain symptoms. Given the expanse of trauma types, individual characteristics, and symptom presentations, it seems unlikely that a single theory or statistical model will entirely explain the directionality or specificity of the association between pain and PTSD across time. However, the present work adds to the current literature by considering socioenvironmental characteristics.

Most of our participants experienced a motor vehicle crash, which represents only a subset of physical traumatic events that can occur. In addition, participants were not admitted to the hospital, were excluded for moderate to severe head injuries, and scored relatively low on the clinician-assessed ISS, suggesting our recruitment strategy targeted individuals who were less severely injured. Although this study and others (e.g., Brasel et al., 2014) suggest that patient-reported pain is a better predictor of future PTSD symptoms, this finding should be tested in a sample with a larger range of injury severity, particularly as more severe injuries typically require hospitalization and the need for in-hospital pain management strategies. Finally, although we assessed pain and PTSD symptoms up to 12 months postinjury, the VAS was not designed to evaluate chronic pain. A necessary assumption was made that the pain assessed at baseline and follow-up visits was entirely associated with the index traumatic injury for which the participant was recruited; however, a longer pain questionnaire or measure that explicitly assesses chronic pain, as well as the quality and quantity of pain symptoms, may yield different results (Bahreini et al., 2014).

The present results have implications for the field's clinical understanding of the treatment of pain and PTSD symptoms. Specifically, this study provides evidence that over a 1-year period, individuals who experienced a traumatic injury and reported higher levels of pain also reported more severe PTSD symptoms. Assessing patient-reported pain after a traumatic injury is an efficient way to help identify individuals with a high risk of nonremitting PTSD. This is particularly important, as nonremitting pain and PTSD are two of the most significant contributors to poor quality of life following an injury (Kiely et al., 2006). If traumatic injury is to be treated comprehensively, the connection between psychological health and physical recovery should be assessed and managed concurrently.

The present findings further demonstrate the robust connection between pain and PTSD (Brasel et al., 2010; de Vries et al., 2021). Although socioenvironmental factors did not moderate the association between pain and PTSD, individual income and trauma history contributed to PTSD symptoms across time. Future work should probe factors that may interrupt the pain–PTSD association, with the intention of identifying a mechanism to improve posttraumatic symptoms.

OPEN PRACTICES STATEMENT

The analysis reported in this article was not formally preregistered. Deidentified data along with a code book are shared via the National Institutes of Mental Health Data Archive at https://nda.nih.gov/edit_collection.html?id=2297; access to the data is limited to qualified researchers. The materials used in these studies are widely publicly available.

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