

UCSF

UC San Francisco Previously Published Works

Title

Understanding Onset, Dynamic Transitions, and Associated Inequality Risk Factors for Adverse Posttraumatic Neuropsychiatric Sequelae After Trauma Exposure.

Permalink

<https://escholarship.org/uc/item/33m2c9c7>

Journal

Psychiatric Research and Clinical Practice, 7(1)

Authors

Lee, Chiyoungh

House, Stacey

Beaudoin, Francesca

et al.

Publication Date

2025

DOI

10.1176/appi.prcp.20240017

Peer reviewed

Understanding Onset, Dynamic Transitions, and Associated Inequality Risk Factors for Adverse Posttraumatic Neuropsychiatric Sequelae After Trauma Exposure

Chiyong Lee, Ph.D., R.N. , Stacey L. House, M.D., Ph.D., Francesca L. Beaudoin, M.D., Ph.D., Thomas C. Neylan, M.D., Gari D. Clifford, D.Phil., Sarah D. Linnstaedt, Ph.D., Laura T. Germine, Ph.D., Scott L. Rauch, M.D., John P. Haran, M.D., Ph.D., Alan B. Storrow, M.D., Christopher Lewandowski, M.D., Paul I. Musey, Jr., M.D., Phyllis L. Hendry, M.D., Sophia Sheikh, M.D., Brittany E. Punches, Ph.D., R.N., Robert A. Swor, D.O., Lauren A. Hudak, M.D., M.P.H., Jose L. Pascual, M.D., Ph.D., Mark J. Seamon, M.D., Erica Harris, M.D., Claire Pearson, M.D., David A. Peak, M.D., Robert M. Domeier, M.D., Niels K. Rathlev, M.D., Brian J. O'Neil, M.D., Paulina Sergot, M.D., Leon D. Sanchez, M.D., M.P.H., Steven E. Bruce, Ph.D., John F. Sheridan, Ph.D., Steven E. Harte, Ph.D., Karestan C. Koenen, Ph.D., Ronald C. Kessler, Ph.D., Samuel A. McLean, M.D., M.P.H., Qing Yang, Ph.D., Xinming An, Ph.D.

Objective: Several gaps remain in the understanding of the onset, dynamic transitions, and associated risk factors of adverse posttraumatic neuropsychiatric sequelae (APNS) in the acute post-trauma window. Based on serial assessments of symptoms from a large cohort study, we identified homogeneous statuses across multiple APNS symptom domains and investigated the dynamic transitions among these statuses during the first 2 months after trauma exposure. Furthermore, we studied how symptom onset and transitions are affected by equity-relevant characteristics.

Methods: The analysis was based on 2557 participants enrolled in the Advancing Understanding of Recovery after Trauma (AURORA). APNS symptoms comprised pain, depression, sleep discontinuity, nightmares, avoidance, re-experience, anxiety, hyperarousal, somatic symptoms, and mental fatigue. We identified the homogeneous status of APNS symptoms at baseline, 1 month, and 2 months, and explored transition probabilities among these statuses using latent transition analysis. Equity-relevant characteristics

included gender, race, education, family income, childhood trauma, and area deprivation.

Results: Three homogeneous statuses—low-, moderate-, and severe-symptom—were identified. While the majority of trauma survivors with severe- or moderate-symptom status maintained the same status over time, some transitioned to a less severe symptom status, particularly within the first month. Specifically, females, non-whites, and those with higher childhood trauma were associated with a decreased likelihood of transitioning to a less severe symptom status. From one to 2 months, lower income was associated with a decreased likelihood of transitioning from moderate- to low-symptom status.

Conclusions: The findings can inform early intervention strategies for APNS, potentially reducing health disparities among trauma survivors.

Psych Res Clin Pract. 2025; 7:53–62; doi: 10.1176/appi.prcp.20240017

Adverse posttraumatic neuropsychiatric sequelae (APNS) are common among civilians and military veterans who have experienced traumatic events, such as motor vehicle collisions and sexual assault (1–3). These APNS greatly increase the risk of developing chronic diseases, such as cancer and heart disease, and are also among the leading causes of drug use, suicide, and disability (4, 5). Furthermore, APNS can have harmful and long-lasting impacts,

including psychosocial and financial burdens not only for individuals with the disorder but also for their families, communities, and society (4).

It is widely recognized that APNS disorders are highly heterogeneous and share strong comorbidity based on traditional classification and diagnoses. For the past several decades, little progress has been made in advancing research for APNS as the majority of studies on APNS have

been focused on individual disorders (e.g., post-traumatic stress disorder [PTSD], depression, and pain) with heterogeneous symptom presentations (5–7).

Investigating the dynamic prognosis of APNS in the early aftermath of trauma exposure has the potential to advance the understanding of APNS and help identify effective intervention options and timing on the individual level. Unfortunately, no large-scale studies have yet been conducted to examine the onset, dynamic transitions (e.g., recovery and relapse), and associated risk factors of APNS across multiple disorders. The early aftermath of trauma is characterized by fluid symptoms, which if left unaddressed, potentially lead to the transition from acute to chronic conditions. Hence, exploring these dynamics during this phase is critical.

STUDY AIMS

In this study, using a unified analysis approach and serial assessments of symptoms from a large cohort study ($n = 2557$), we aimed to identify homogeneous statuses across multiple APNS symptom domains and characterize the transitions among these statuses during the first 2 months after trauma exposure. Furthermore, considering that the trauma impact is well known to be different across groups defined by equity-relevant characteristics, such as gender (8–10), race (11–13), childhood trauma (14, 15), and socioeconomic position (SEP) (16–18), we examined how symptom onset and transitions might also vary based on these characteristics.

METHODS

Study Participants

This study used a series of self-report flash survey data collected from the Advancing Understanding of Recovery after trauma (AURORA) study (4). AURORA is a large-scale emergency department (ED)-based longitudinal study of trauma survivors across the United States (4). Survivors aged 18–75 years who presented to an ED for care within 72 h of a trauma event were screened to determine enrollment eligibility. Trauma cases related to motor vehicle collisions, falls greater than 10 ft, physical or sexual assaults, or mass casualty incidents were automatically qualified for enrollment, with most survivors being involved in motor vehicle collisions. This study also included patients who had experienced trauma from non-motorized collisions (e.g., bicycle accidents), large-scale traumatic incidents (e.g., natural disasters), poisoning, burns, or animal-related injuries (e.g., bites). Other inclusion criteria included fluency in English and having an Android or iOS smartphone with internet access. Additionally, participants had to complete at least one flash survey within the first 2 months after enrollment to be included in the current study.

HIGHLIGHTS

- We investigated the onset, dynamic transitions, and associated risk factors of adverse posttraumatic neuropsychiatric sequelae (APNS) using latent transition analysis.
- Three homogeneous APNS symptom statuses (low, moderate, and severe) were identified.
- While most survivors maintained the same status, some followed the recovery trajectory by transitioning to a lesser symptom status.
- Females, non-whites, those with childhood trauma, and those with a lower income were less likely to follow a recovery trajectory.
- Our findings can guide preventive interventions for APNS among trauma survivors.

The AURORA study is specifically designed to monitor stress-induced psychological symptoms in ED patients who do not have severe physical injuries, such as significant hemorrhage or intracranial injuries, thereby steering clear of the complexities introduced by severe physical injuries. It meticulously excludes individuals who have undergone or are experiencing conditions that might confound the assessment of psychological symptoms, including general anesthesia, long bone fractures, significant lacerations with hemorrhage, and visual or auditory deficits that could impede participation in web-based neurocognitive evaluations and/or telephone follow-ups. The study also does not include pregnant or breastfeeding individuals. A total of 2557 participants were analyzed. Supplementary Figure S1 represents the flowchart for participants in the final analytical sample. Further details on AURORA can be found elsewhere (4). Patients were informed about the general nature of the study, expectations for participation, the voluntary nature of participation, and the risks and benefits before seeking written informed consent (4).

Variables

APNS symptom domains. Ten most common/burdensome APNS symptoms across traditional domain were selected based on review of prior studies: pain (19), depressive symptoms (20), sleep discontinuity (21), nightmares (22), somatic symptoms (23), concentration/thinking/fatigue (24), avoidance, re-experiencing, anxiety, and hyperarousal (25, 26). These symptoms were defined by survey items selected by domain experts from a rotating battery of smartphone-based “flash” questionnaires, administered using the Mindstrong Discovery™ application. AURORA integrates the assessment of broader bodily pain as an APNS domain, predicated on the assumption that such pain is predominantly *stress-induced* rather than stemming directly from physical injuries. The corresponding survey items,

their response options, and the study day on which they were administered are shown in Supplementary Table S1.

Equity-relevant characteristics (i.e., covariates). Equity-relevant characteristics included gender (males or females), race (whites or non-whites), education (less than a high school degree or a high school degree or higher), total family income ($\leq \$75,000$ or $> \$75,000$), childhood trauma (continuous), and area deprivation (continuous). According to the U.S. Census Bureau (27), a household income of \$75,000 represents the approximate median household income; therefore, it was selected as the cut point for income stratification. Childhood trauma was measured after 2 weeks from ED admission using the Childhood Trauma Questionnaire–Short Form (CTQ–SF) (28). The CTQ–SF is comprised of 28 items and assesses the extent to which the respondent experienced five types of childhood maltreatment: physical neglect, emotional neglect, emotional abuse, physical abuse, and sexual abuse. The possible responses on each item represent the frequency of maltreatment experiences and range from 0 to 4, with 4 being “very often true.” The scores can be generated for each type of maltreatment individually, or for a total maltreatment score. The AURORA study only surveys 11 of the 28 items in the CTQ–SF, two items each from the physical neglect, emotional neglect, emotional abuse, and physical abuse subtypes, and three items from the sexual abuse subtype, yet follows the same scoring methodology as the CTQ–SF. In the current study, a total score was used. Area Deprivation Index (ADI) national percentiles were used to measure area deprivation and were computed from a participant’s geographic location within the US according to their census block group. The 2019 ADI national percentile rankings were obtained for all participants from their 9-digit ZIP code through The Neighborhood Atlas® at the University of Wisconsin-Madison (<https://www.neighborhoodatlas.medicine.wisc.edu/>). The ADI quantifies multiple context-level socioeconomic status factors (neighborhood income, employment, education, and housing quality) in a single metric. The index ranges from 0 to 100, with higher scores indicating greater deprivation.

Statistical Analyses

Using survey items (see Supplementary Table S1 for details) as indicator variables, joint measurement models were developed to create latent variables for each of the 10 symptoms. Factor scores were then computed for each participant for each of the 10 symptoms (see Supplementary S1.1 for details in Supporting Information S1). In this study, to facilitate analysis and interpretation, highly correlated symptoms were combined to form five symptom domains: pain, sleep discontinuity and nightmares; avoidance and re-experience; depression, anxiety, and hyperarousal; and somatic symptoms and mental fatigue.

Latent transition analysis (LTA) (29) was used to identify latent statuses of APNS and transitions among these statuses over time as well as to study characteristics

that can affect symptom onset and transitions. To ensure that latent statuses across different time points are comparable, they were determined jointly using data across all time points in LTA. First, we fit successive unconditional LTA models with increasing numbers of latent statuses to determine the most parsimonious model that provides an adequate fit to the data (29). Model fit indices, interpretability and clinical meaningfulness were used to guide decisions of the best model (29). Following the determination of the best LTA model, we investigated the prevalence of the latent statuses and studied the transition probabilities among latent statuses over time (29). Finally, covariates were incorporated into the model both individually and collectively to study the unadjusted and adjusted effect of each predictor on the onset and transitions of APNS symptom status. All binary covariates were coded as 0 or 1 and continuous covariates were standardized according to Lanza et al. (30). More details about this analytical approach are discussed extensively in Supplementary S1.2 in Supporting Information S1.

Missing data for the survey items were treated as missing at random and were imputed based on the joint measurement model using Full Information Maximum Likelihood (FIML) (31) estimation since missingness was not associated with outcome measures (see Supplementary S1.3 for details in Supporting Information S1). All analyses were completed using *Mplus*, version 8.8 (Muthén and Muthén). Example statistical codes used in this study are shown in Supplementary S2 in Supporting Information S1.

RESULTS

Table 1 presents the baseline characteristics of the participants. The LTA model with three latent statuses was identified as the best model based on model fit indices (see Supplementary Table S2 for details) and interpretability, and was used for the subsequent analysis. Results from the unconditional LTA model that estimated the general onset and transition probabilities on the population level are summarized in Table 2. Based on the symptom profiles for each status, three statuses were characterized as low-symptom, moderate-symptom, and severe-symptom statuses, respectively. Across time points, severe- and moderate-symptom statuses decreased in prevalence, whereas the low-symptom status increased. All participants with the low-symptom status at baseline stayed in the same status after 1 month. Participants with the moderate-symptom status at baseline had a 56.5% probability of remaining at that status, a 41.6% probability of moving to the low-symptom status, and a 1.9% probability of moving to a severe-symptom status after 1 month. Further, participants with a severe-symptom status at baseline had a 74.3% probability of remaining at that status, a 25.6% probability of moving to the moderate-symptom status, and almost zero probability of moving to the low-symptom status after 1 month. From the one-to 2-month

follow-ups, the transition probabilities displayed high stability for latent statuses for all but one probability > 0.12: there was a 12.6% probability of moderate-symptom participants transitioning to low-symptom status.

Results from the conditional LTA model that characterize the effect of the covariates on the onset (status membership at baseline) are summarized in Table 3. Marginally (unadjusted), almost all of them presented a significant differentiation between latent statuses. Jointly (adjusting all the other covariates), the odds of being low-

symptom status compared to severe-symptom status were significantly lower for females (vs. males), non-whites (vs. whites), those with a total family income ≤ \$75,000 (vs. those with a total family income > \$75,000), and those with higher childhood trauma scores. Further, the odds of being moderate-symptom status compared to severe-symptom status were significantly lower for females (vs. males), individuals with less than a high school degree (vs. individuals with a high school degree or higher), those with a total family income ≤ \$75,000 (vs. those with a total family income > \$75,000), and those with higher childhood trauma scores.

Results that characterize the effect of covariates on the transitions are included in Table 4. From the baseline to 1-month follow up (Table 4), the odds of transitioning to the moderate-symptom status relative to staying at the severe-symptom status were significantly lower for non-whites (vs. whites) and those with higher childhood trauma scores, after adjusting for other covariates; furthermore, these odds were also lower for those living in a more deprived area, but were significant only when other covariates were not considered. The odds of transitioning to the low-symptom status relative to staying at the moderate-symptom status were significantly lower for those with higher childhood trauma scores, after controlling for other covariates; these odds were also significantly lower for non-whites (vs. whites), individuals with less than a high school degree (vs. individuals with a high school degree or higher), those with a total family income ≤ \$75,000 (vs. those with a total family income > \$75,000), and those living in a more deprived area, but this was applicable only when each covariate was not adjusted for the other. Further, the odds of transitioning to the low-symptom status relative to staying

TABLE 1. Baseline characteristics of the participants (n = 2557).

Variables	n (%) or mean ± standard deviation
Age, years	35.6 ± 13.07
Gender (n = 2555)	
Males	964 (37.7%)
Females	1591 (62.3%)
Race (n = 2545)	
Whites	859 (33.8%)
Non-whites	1686 (66.2%)
Education (n = 2556)	
Less than a high school degree	304 (11.9%)
A high school degree or higher	2252 (88.1%)
Total family income (n = 2264)	
≤ \$75,000	1956 (86.4%)
> \$75,000	308 (13.6%)
Childhood trauma ^a (range: 0-44; n = 2183)	9.5 ± 9.78
Area deprivation ^b (range: 0-100; n = 2471)	64.8 ± 27.58

^a An abbreviated 11-item version of the Childhood Trauma Questionnaire–Short Form was used to measure childhood trauma.

^b Area Deprivation Index national percentiles were used to measure area deprivation.

TABLE 2. The results of the unconditional LTA model with three latent statuses.

	Latent status		
	Low-symptom status	Moderate-symptom status	Severe-symptom status
Symptom indicator means (range of means) ^a			
Pain (0–10)	2.52 ± 0.16	5.48 ± 0.08	6.96 ± 0.10
Sleep discontinuity and nightmares (0–4)	0.67 ± 0.02	1.46 ± 0.04	2.54 ± 0.06
Avoidance and re-experience (0–4)	0.78 ± 0.03	1.89 ± 0.05	2.82 ± 0.05
Depression, anxiety, and hyperarousal (0–4)	0.68 ± 0.02	1.64 ± 0.05	2.72 ± 0.06
Somatic symptoms and mental fatigue (0–10)	2.35 ± 0.08	4.62 ± 0.10	6.85 ± 0.11
Latent status membership prevalence			
Time 1 (baseline)	18.6% (n = 475)	56.3% (n = 1439)	25.1% (n = 643)
Time 2 (1 month)	42.0% (n = 1074)	38.2% (n = 977)	19.8% (n = 506)
Time 3 (2 months)	46.8% (n = 1197)	35.3% (n = 902)	17.9% (n = 458)
Transition probabilities (rows for baseline, columns for 1 month)			
Low-symptom status	1.000	0.000	0.000
Moderate-symptom status	0.416	0.565	0.019
Severe-symptom status	0.001	0.256	0.743
Transition probabilities (rows for 1 month, columns for 2 months)			
Low-symptom status	1.000	0.000	0.000
Moderate-symptom status	0.126	0.874	0.000
Severe-symptom status	0.000	0.093	0.907

Note: Transition probabilities in bold font correspond to membership in the same latent status at both times.

^a Symptom indicator means constrained to be equal at baseline, 1 month, and 2 months.

TABLE 3. Estimates for covariates predicting status membership at baseline.

	OR (95% CI)	AOR (95% CI)
Low-symptom status (vs. severe-symptom status)		
Females (vs. males)	0.32* (0.24, 0.42)	0.37* (0.27, 0.52)
Non-whites (vs. whites)	0.55* (0.42, 0.73)	0.69* (0.47, 0.99)
Less than a high school degree (vs. a high school degree or higher)	0.46* (0.29, 0.71)	0.75 (0.44, 1.28)
Total family income $\leq$ \$75,000 (vs. total family income $>$ \$75,000)	0.18* (0.11, 0.29)	0.32* (0.19, 0.54)
Childhood trauma	0.32* (0.25, 0.41)	0.35* (0.27, 0.46)
Area deprivation	0.73* (0.63, 0.83)	0.94 (0.78, 1.13)
Moderate-symptom status (vs. severe-symptom status)		
Females (vs. males)	0.65* (0.52, 0.83)	0.10* (0.55, 0.93)
Non-whites (vs. whites)	0.94 (0.75, 1.18)	1.12 (0.85, 1.48)
Less than a high school degree (vs. a high school degree or higher)	0.63* (0.46, 0.86)	0.67* (0.46, 0.97)
Total family income $\leq$ \$75,000 (vs. total family income $>$ \$75,000)	0.33* (0.21, 0.51)	0.42* (0.26, 0.69)
Childhood trauma	0.63* (0.57, 0.70)	0.64* (0.61, 0.76)
Area deprivation	0.83* (0.74, 0.94)	0.91 (0.79, 1.04)

Note: AOR represents the odds ratio after controlling all other covariates in the model. Note that *p*-values are not reported for the test statistics in the conditional LTA model and the evaluation of statistically significant differences (*) were made based on the CIs.

Abbreviations: AOR, adjusted odds ratio; CI, confidence interval; OR, odds ratio.

TABLE 4. Estimates for covariates predicting transitions.

	OR (95% CI)	AOR (95% CI)
Estimates from baseline to 1 month		
Severe-symptom status to moderate-symptom status (vs. staying in severe-symptom status)		
Females (vs. males)	1.30 (0.72, 2.34)	1.14 (0.62, 2.11)
Non-whites (vs. whites)	0.42* (0.25, 0.69)	0.43* (0.25, 0.73)
Less than a high school degree (vs. a high school degree or higher)	0.54 (0.23, 1.27)	0.84 (0.37, 1.95)
Total family income $\leq$ \$75,000 (vs. total family income $>$ \$75,000)	0.93 (0.32, 2.63)	1.22 (0.43, 3.42)
Childhood trauma	0.77* (0.63, 0.94)	0.75* (0.60, 0.92)
Area deprivation	0.76* (0.60, 0.97)	0.81 (0.61, 1.07)
Moderate-symptom status to low-symptom status (vs. staying in moderate-symptom status)		
Females (vs. males)	0.76 (0.56, 1.02)	0.69* (0.49, 0.98)
Non-whites (vs. whites)	0.54* (0.40, 0.72)	0.74 (0.52, 1.06)
Less than a high school degree (vs. a high school degree or higher)	0.52* (0.32, 0.84)	0.69 (0.38, 1.27)
Total family income $\leq$ \$75,000 (vs. total family income $>$ \$75,000)	0.58* (0.38, 0.89)	0.86 (0.53, 1.39)
Childhood trauma	0.65* (0.55, 0.78)	0.68* (0.57, 0.82)
Area deprivation	0.76* (0.67, 0.88)	0.89 (0.75, 1.06)
Estimates from 1 month to 2 months		
Severe-symptom status to moderate-symptom status (vs. staying in severe-symptom status)		
Females (vs. males)	13.47 [†] (0.03, 5332.65)	8.20 [†] (0.05, 759.38)
Non-whites (vs. whites)	1.28 (0.28, 5.79)	0.87 (0.20, 3.75)
Less than a high school degree (vs. a high school degree or higher)	0.32 (0.02, 4.16)	0.00 [†] (0.00, 0.00)
Total family income $\leq$ \$75,000 (vs. total family income $>$ \$75,000)	1.10 [†] (0.04, 33.93)	0.87 (0.08, 9.84)
Childhood trauma	0.81 (0.51, 1.27)	0.64 (0.38, 1.05)
Area deprivation	0.84 (0.50, 1.39)	1.03 (0.55, 1.92)
Moderate-symptom status to low-symptom status (vs. staying in moderate-symptom status)		
Females (vs. males)	1.32 (0.63, 2.80)	1.55 (0.59, 4.06)
Non-whites (vs. whites)	0.76 (0.38, 1.50)	0.85 (0.36, 1.98)
Less than a high school degree (vs. a high school degree or higher)	0.28 (0.04, 1.83)	0.11 (0.00, 87.77)
Total family income $\leq$ \$75,000 (vs. total family income $>$ \$75,000)	0.31* (0.13, 0.72)	0.35* (0.14, 0.87)
Childhood trauma	0.81 (0.55, 1.21)	0.81 (0.52, 1.26)
Area deprivation	0.97 (0.72, 1.31)	1.04 (0.71, 1.54)

Note: AOR represents the odds ratio after controlling all other covariates in the model. Note that *p*-values are not reported for the test statistics in the conditional LTA model and the evaluation of statistically significant differences (*) were made based on the CIs. Odds of transitioning between severe-symptom and low-symptom status could not be calculated across all time points due to the low sample size and are not presented. As transitions among statuses are much less frequent from one to 2 months after trauma exposure, some odds ([†]) could not be accurately estimated.

Abbreviations: AOR, adjusted odds ratio; CI, confidence interval; OR, odds ratio.

at the moderate-symptom status were significantly lower for females (vs. males) after considering all covariates. From the one-to 2-month follow-ups (Table 4), the odds of transitioning to the low-symptom status relative to staying at the

moderate-symptom status were significantly lower for those with a total family income $\leq$ \$75,000 (vs. those with a total family income $>$ \$75,000), after adjusting for other covariates.

DISCUSSION

In this study, we identified three APNS symptom statuses (low-, moderate-, and severe-symptom status) based on APSN symptoms and examined transitions among these statuses during the first 2 months after trauma exposure. The survivors with low-symptom status after trauma retained this status over time. While most survivors with severe- or moderate-symptom status also maintained their status, some were more likely to follow a recovery trajectory by transitioning to a lesser symptom status, particularly within the first month. While those in the moderate-symptom status were most likely to transition, probabilities of transition varied based on the survivor's characteristics.

First, non-whites were significantly less likely to transit from severe-to moderate-symptom status than whites, implying that clinicians must closely monitor non-white survivors with an initial severe-symptom burden. These results match prior studies that found more severe and chronic posttraumatic symptoms (e.g., PTSD, anxiety) among African Americans and Latino populations, compared to whites (11–13). Interestingly, the disparity remained after accounting for other important covariates. Himle et al. (32) claim that socioeconomic backgrounds or psychiatric histories partially account for disparate trauma outcomes among different races/ethnicities, and researchers must consider sociocultural contexts and racial stressors as salient factors that affect these outcomes among non-white survivors. Examples include acculturation, institutional discrimination in healthcare (e.g., misattribution or dismissal of symptoms reported by non-whites, provider bias), chronic stress, or unequal access to healthcare—all of which contribute to racial/ethnic disparities in mental health recovery (13, 33, 34). Although these factors could not be directly discussed in this study, future studies should aim to precisely delineate the mechanisms through which non-whites experience more elevated APNS status than whites.

After controlling for other characteristics, females not only exhibited higher APNS symptoms than males at baseline but were also less likely to transition from moderate-to low-symptom status. It is well documented that females have a significantly higher risk of developing APNS after trauma than males (8, 9). Kornfield et al. (10) argue that this discrepancy is a result of the differences in biology, for instance, the regulation of risk and resilience responses by sex hormones, while others name differences in the appraisal of trauma, coping style, social support, or different life stressors (35). Our study contributed to the literature that gender also acts as a susceptibility factor in APNS recovery in the acute post-trauma window. Additional studies on key variables that may complicate recovery in females are necessary.

Likewise, higher level of childhood trauma is independently associated with higher probability of being in a

more severe symptom status at baseline and also decreases the likelihood of transitioning to a less severe symptom status (i.e., from both severe-to moderate-symptom status and from moderate-to low-symptom status). This implies that cumulative childhood trauma exerts an additive and detrimental impact on the course of the psychological sequelae of later trauma, independent of current socioeconomic conditions. That is, childhood trauma might represent a long-lasting vulnerability factor. Our findings corroborate recent neurological studies that demonstrate the association of childhood trauma with PTSD after adult trauma (11, 12). Xie et al. (12) shed light on thalamic contributions to posttraumatic dysfunction (generally, thalamus alterations are linked to a range of stress-related processing changes, e.g., incorrect integration of trauma-related sensory inputs, overconsolidation of traumatic memory) and reported that accumulated childhood trauma are inversely related to whole thalamus volumes within the first 2 weeks after adult trauma.

Of note is that those living in a more deprived area were less likely to transit from moderate-to low-symptom status. This finding is crucial as, to date, the field of trauma research has primarily focused on person-level risk factors associated with APNS and has not addressed the upstream-level contextual factors that may “set the stage” for APNS risk or make trauma recovery difficult (13). Living in a deprived area increases the likelihood of experiencing socioeconomic-based life stressors, such as exposure to violence, environmental insecurities, lack of access to health care, or low-quality nutrition (36). In traumatically-injured adults, such stressors may inherently alter an individual's neurocognitive functioning beyond factors traditionally assessed (e.g., income) and thus may alter the trajectory of the psychological disorder (9). Recently, Tomas et al. (8) and Webb et al. (10) found that brain regions crucial for recognizing and processing negative stimuli are susceptible to the effects of area-level socioeconomic factors, and changes to key brain regions may explain why those living in disadvantaged area are at a heightened risk of PTSD. However, after considering other person-level covariates, area deprivation did not significantly predict transitions among symptom statuses. This may imply that, although area deprivation alone may strongly influence trauma recovery, its influence is intrinsically intertwined with individual disadvantages. Webb et al. (9) also noted that the stress due to socioeconomic factors at both personal- and contextual-level concurrently get “under the skin” of patients and impact trauma outcomes. Moving forward, research would benefit from adopting more nuanced and intersectional approaches to make more meaningful conclusions about the complex interplay between multi-level variables and their influence on post-trauma disorders.

Similarly, other factors such as race, education, and family income predicted survivors' transition from moderate-to low-symptom status, but their significance was not evident after accounting for other covariates. This

acknowledges that various SEP factors may have affected each other to shape the recovery trajectories of APNS. For instance, in Remigio-Baker et al.'s study (30), racial/ethnic disparities in the recovery of depression and neuro-behavioral symptoms varied by education level among service members who received rehabilitation treatment for concussion.

Transitions among statuses are much less frequent from one to 2 months after trauma exposure. Family income was a strong and independent factor that affected survivors' transition from moderate-to low-symptom status during this period, highlighting the role of financial resources in shaping the course of APNS symptomatology in the acute post-trauma window. Although more investigations are needed to assess why income was particularly associated with symptom recovery during this period, the findings corroborate prior works that found income to be a solid predictor of posttraumatic resilience (27, 37, 38). Indeed, individuals from high-income households can afford systems to reduce the consequences of traumatic events while simultaneously mitigating their financial impacts. However, limited income may impede survivors' access to recovery-supporting services, such as physical rehabilitation (39).

Limitations

Our study warrants several limitations. First, most of our participants were admitted because of motor vehicle collisions. Therefore, this study's results may primarily reflect the outcomes of this specific traumatic event; thus, they should be interpreted with caution. The distinct manifestations and dynamics of APNS transition associated with different trauma types may not be sufficiently captured if the individuals are studied as one group. Therefore, future studies should explore these differences based on trauma type. Such insights will enable the formulation of tailored prevention interventions for APNS. Second, it is possible that different subgroups may emerge depending on the symptomology required for study participation, which further underscores the importance of expanding research samples to include survivors with a wide diversity of APNS experiences. Third, the sample size was not stable across all time points, and our analytic approach accounted for the movement of participants between statuses by including all measurements contributed. Assessment of missing data would need to disentangle which survivors with which level of trauma experience or risk factors missed a measurement wave, which was not possible to account for in this analysis. Fourth, due to the model requirement for the LTA with covariates approach, some of the predictors at ordinal level or with multiple nominal level categories had to be recoded as binary. For instance, due to small sample size across ethno-racial groups, we were compelled to coerce our participants into non-white and white categories, which resulted in a loss of granularity. Although simplified predictors might be easier to obtain in clinical

practice, the results necessitate cautious interpretation. Fifth, our findings related to childhood trauma should be interpreted cautiously, as the score used in the scale weighs all trauma categories equally, neglecting interindividual differences in manifestations. Recent attention has emphasized the importance of assessing the severity, timing, and chronicity of each experience. For instance, earlier evidence demonstrated that compared to no history of previous trauma exposure, a history of two or more traumatic events, especially when involving assaultive violence in childhood, increased the risk that a traumatic adulthood event would lead to PTSD fivefold (40). Sixth, this study carefully defined equity-relevant characteristics, including gender, race, education, family income, childhood trauma, and area deprivation. These were meticulously strategized using all relevant data available within the AURORA dataset. Future researchers should also consider factors within the healthcare system that potentially contribute to the post-trauma variability of APNS trajectories, such as medical contact and visits for psychological or psychiatric treatment. Additionally, other contextual factors not captured by the ADI, such as healthcare and insurance coverage, access to transportation, and residential stability, may also be valuable in enhancing our understanding of the disparities in trauma outcomes. Finally, the proportions of women, non-whites (especially African Americans), and low-income individuals in the AURORA study were significantly higher than their representation in the general US population. This sampling bias potentially threatens the external validity of this study's findings, which should thus be generalized with caution.

Despite the limitations, our study is meaningful as it examines a period of significant symptom fluctuation before potential chronicity sets in. The early post-injury phase is characterized by "fluid symptom dynamics," where the reciprocal exacerbation among various APNS can significantly influence the trajectory from acute to chronic states. Our study focuses on this pivotal period among trauma survivors, aiming to identify these dynamic symptom transitions early on, as they can potentially alter the course toward chronicity. This insight may be invaluable for informing future research to develop targeted treatment strategies during the most crucial times of symptom development.

Clinical Implications

Our analysis highlights the crucial need for healthcare providers to recognize the dynamic trajectories of APNS in trauma survivors, particularly within the first 2 months following the trauma exposure. Early identification and close monitoring of high-risk groups—such as females, non-whites, and individuals with a history of childhood trauma—are crucial. Additionally, it is important to acknowledge that socioeconomic factors, like lower income, can significantly affect recovery paths. By

understanding these dynamics, healthcare providers can design and implement tailored interventions that address individual vulnerabilities and broader socioeconomic challenges, potentially mitigating long-term impacts and reducing health disparities among trauma survivors. Considering that trauma survivors presenting to the ED form a large high-risk population, timely administration of appropriate interventions could have a significant public health benefit, potentially reducing mortality, morbidity, and long-term disability across populations.

CONCLUSIONS

This study advances our current knowledge about the onset and dynamic transitions of APNS within the first 2 months after trauma exposure. The observed dynamic prognosis of APNS and associated risk factors can help develop effective preventive interventions for APNS and inform efforts to mitigate health disparities among trauma survivors.

AUTHOR AND ARTICLE INFORMATION

University of Arizona College of Nursing, Tucson, Arizona, USA (Lee); Department of Emergency Medicine, Washington University School of Medicine, St. Louis, Missouri, USA (House); Department of Epidemiology (Beaudoin) and Department of Emergency Medicine (Beaudoin), Brown University, Providence, Rhode, USA; Departments of Psychiatry and Neurology, University of California San Francisco, San Francisco, California, USA (Neylan); Department of Biomedical Informatics, Emory University School of Medicine, Atlanta, Georgia, USA (Clifford); Department of Biomedical Engineering, Georgia Institute of Technology and Emory University, Atlanta, Georgia, USA (Clifford); Department of Anesthesiology, Institute for Trauma Recovery, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA (Linnstaedt, An); Institute for Technology in Psychiatry, McLean Hospital, Belmont, Massachusetts, USA (Germine, Rauch); The Many Brains Project, Belmont, Massachusetts, USA (Germine); Department of Psychiatry, Harvard Medical School, Boston, Massachusetts, USA (Germine, Rauch); Department of Psychiatry, McLean Hospital, Belmont, Massachusetts, USA (Rauch); Department of Emergency Medicine, University of Massachusetts Chan Medical School, Worcester, Massachusetts, USA (Haran); Department of Emergency Medicine, Vanderbilt University Medical Center, Nashville, Tennessee, USA (Storow); Department of Emergency Medicine, Henry Ford Health System, Detroit, Michigan, USA (Lewandowski); Department of Emergency Medicine, Indiana University School of Medicine, Indianapolis, Indiana, USA (Musey); Department of Emergency Medicine, University of Florida College of Medicine - Jacksonville, Jacksonville, Florida, USA (Hendry, Sheikh); Department of Emergency Medicine, Ohio State University College of Medicine, Columbus, Ohio, USA (Punches); Ohio State University College of Nursing, Columbus, Ohio, USA (Punches); Department of Emergency Medicine, Oakland University William Beaumont School of Medicine, Rochester, Michigan, USA (Swor); Department of Emergency Medicine, Emory University School of Medicine, Atlanta, Georgia, USA (Hudak); Department of Surgery, Department of Neurosurgery (Pascual) and Perelman School of Medicine (Pascual, Seamon), University of Pennsylvania, Philadelphia, Pennsylvania, USA; Division of Traumatology, Surgical Critical Care and Emergency Surgery, Department of Surgery, University of Pennsylvania, Philadelphia, Pennsylvania, USA (Seamon); Department of Emergency Medicine, Einstein Medical Center, Philadelphia, Pennsylvania, USA (Harris); Department of Emergency Medicine, Wayne State University, Ascension St. John Hospital, Detroit, Michigan, USA (Pearson); Department of Emergency

Medicine, Massachusetts General Hospital, Boston, Massachusetts, USA (Peak); Department of Emergency Medicine, Harvard Medical School, Boston, Massachusetts, USA (Peak, Sanchez); Department of Emergency Medicine, Trinity Health-Ann Arbor, Ypsilanti, Michigan, USA (Domeier); Department of Emergency Medicine, University of Massachusetts Medical School-Baystate, Springfield, Massachusetts, USA (Rathlev); Department of Emergency Medicine, Wayne State University, Detroit Receiving Hospital, Detroit, Michigan, USA (O'Neil); Department of Emergency Medicine, McGovern Medical School at UTHealth, Houston, Texas, USA (Sergot); Department of Emergency Medicine, Brigham and Women's Hospital, Boston, Massachusetts, USA (Sanchez); Department of Psychological Sciences, University of Missouri - St. Louis, St. Louis, Missouri, USA (Bruce); Division of Biosciences, Ohio State University College of Dentistry, Columbus, Ohio, USA (Sheridan); Institute for Behavioral Medicine Research, OSU Wexner Medical Center, Columbus, Ohio, USA (Sheridan); Department of Anesthesiology (Harte) and Department of Internal Medicine-Rheumatology (Harte), University of Michigan Medical School, Ann Arbor, Michigan, USA; Department of Epidemiology, Harvard T.H. Chan School of Public Health, Harvard University, Boston, Massachusetts, USA (Koenen); Department of Health Care Policy, Harvard Medical School, Boston, Massachusetts, USA (Kessler); Department of Emergency Medicine (McLean) and Department of Psychiatry, Institute for Trauma Recovery (McLean), University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA; Duke University, School of Nursing, Durham, North Carolina, USA (Yang).

Send correspondence to Dr. Lee (cleec33@arizona.edu).

The investigators wish to thank the trauma survivors participating in the AURORA study. Their time and effort during a challenging period of their lives make our efforts to improve recovery for future trauma survivors possible. This project was supported by NIMH under U01MH110925, the US Army MPMC, One Mind, and The Mayday Fund. The content is solely the responsibility of the authors and does not necessarily represent the official views of any of the funders. Verily Life Sciences and Mindstrong Health provided some of the hardware and software used to perform study assessments. The Many Brains Project provided software for neurocognitive assessments. Data and/or research tools used in the preparation of this manuscript were obtained from the National Institute of Mental Health (NIMH) Data Archive (NDA). NDA is a collaborative informatics system created by the National Institutes of Health to provide a national resource to support and accelerate research in mental health. Dataset identifier(s): NIMH Data Archive Digital Object Identifier (DOI) 10.15154/3gtv-z090. This manuscript reflects the views of the authors and may not reflect the opinions or views of the NIH or of the Submitters submitting original data to NDA.

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

- Dr. Neylan has received research support from NIH, VA, and Rainwater Charitable Foundation, and consulting income from Jazz Pharmaceuticals.
- In the last 3 years, Dr. Clifford has received research funding from the NSF, NIH and LifeBell AI, and unrestricted donations from AliveCor Inc, Amazon Research, the Center for Discovery, the Gates Foundation, Google, the Gordon and Betty Moore Foundation, MathWorks, Microsoft Research, Nextsense Inc, One Mind Foundation, the Rett Research Foundation, and Samsung Research. Dr. Clifford has financial interest in AliveCor Inc and Nextsense Inc. He also is the CTO of MindChild Medical and CSO of LifeBell AI and has ownership in both companies. These relationships are unconnected to the current work.
- Dr. Germine receives funding from the National Institute of Mental Health (R01 MH121617) and is on the board of the Many Brains

Project. Dr. Germine's family also has equity in Intelrad Medical Systems, Inc.

- Dr. Rauch reports grants from NIH during the conduct of the study; personal fees from SOBP (Society of Biological Psychiatry) paid role as secretary, other from Oxford University Press royalties, other from APP (American Psychiatric Publishing Inc.) royalties, other from VA (Veterans Administration) per diem for oversight committee, and other from Community Psychiatry/Mindpath Health paid board service, including equity outside the submitted work; other from National Association of Behavioral Healthcare for paid Board service; other from Springer Publishing royalties; and Leadership roles on Board or Council for SOBP, ADAA (Anxiety and Depression Association of America), and NNDC (National Network of Depression Centers).
- Dr. Sheikh has received funding from the Florida Medical Malpractice Joint Underwriter's Association Dr. Alvin E. Smith Safety of Healthcare Services Grant; Allergan Foundation; the NIH/NIA-funded Jacksonville Aging Studies Center (JAX-ASCENT; R33AG05654); and the Substance Abuse and Mental Health Services Administration (1H79TI083101-01); and the Florida Blue Foundation.
- Dr. Harte has no competing interests related to this work, though in the last 3 years he has received research funding from Aptinix and Arbor Medical Innovations, and consulting payments from Aptinix, Heron Therapeutics, and Eli Lilly.
- Dr. Koenen's research has been supported by the Robert Wood Johnson Foundation, the Kaiser Family Foundation, the Harvard Center on the Developing Child, Stanley Center for Psychiatric Research at the Broad Institute of MIT and Harvard, the National Institutes of Health, One Mind, Anonymous Philanthropy, LLC, and Cohen Veterans Bioscience. She has been a paid consultant for the US Department of Justice and Covington Burling, LLP over the last 3 years. She has been a paid external reviewer for the Chan Zuckerberg Foundation, the University of Cape Town, and Capita Ireland. She has had paid speaking engagements in the last 3 years with the American Psychological Association, European Central Bank, Sigmund Freud University—Milan, Cambridge Health Alliance, and Coverys. She receives royalties from Guilford Press and Oxford University Press.
- In the past 3 years, Dr. Kessler was a consultant for Cambridge Health Alliance, Canandaigua VA Medical Center, Holmusk, Partners Healthcare, Inc., RallyPoint Networks, Inc., and Sage Therapeutics. He has stock options in Cerebral Inc., Mirah, PYM, and Roga Sciences.
- Dr. McLean served as a consultant for Walter Reed Army Institute for Research and for Arbor Medical Innovations.

As AURORA consists of anonymized, de-identified data, this study was exempted by the University of Arizona Institutional Review Board. It does not require any type of approval.

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.

© 2024 The Author(s). Psychiatric Research and Clinical Practice published by Wiley Periodicals LLC on behalf of American Psychiatric Association.

Received February 15, 2024; revised August 7, 2024; accepted August 13, 2024.

REFERENCES

1. Kessler RC. Posttraumatic stress disorder: the burden to the individual and to society. *J Clin Psychiatry*. 2000;64:14.

2. Roberts AL, Gilman SE, Breslau J, Breslau N, Koenen KC. Race/ethnic differences in exposure to traumatic events, development of post-traumatic stress disorder, and treatment-seeking for post-traumatic stress disorder in the United States. *Psychol Med*. 2011;41(1):71–83. <https://doi.org/10.1017/S0033291710000401>
3. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Post-traumatic stress disorder in the national comorbidity survey. *Arch Gen Psychiatr*. 1995;52(12):1048–60.
4. McLean SA, Ressler K, Koenen KC, Neylan T, Germine L, Jovanovic T, et al. The AURORA Study: a longitudinal, multi-modal library of brain biology and function after traumatic stress exposure. *Mol Psychiatry*. 2020;25(2):283–96. <https://doi.org/10.1038/s41380-019-0581-3>
5. Byrne SP, Harpaz-Rotem I, Tsai J, Southwick SM, Pietrzak RH. Latent typologies of DSM-5 PTSD symptoms in US military veterans. *Psychiatr Res*. 2019;273:266–73. <https://doi.org/10.1016/j.psychres.2018.12.094>
6. Campbell SB, Trachik B, Goldberg S, Simpson TL. Identifying PTSD symptom typologies: a latent class analysis. *Psychiatr Res*. 2020;285:112779. <https://doi.org/10.1016/j.psychres.2020.112779>
7. Pietrzak RH, El-Gabalawy R, Tsai J, Sareen J, Neumeister A, Southwick SM. Typologies of posttraumatic stress disorder in the US adult population. *J Affect Disord*. 2014;162:102–6. <https://doi.org/10.1016/j.jad.2014.03.024>
8. Tomas CW, Webb EK, Bennett KP, Huggins AA, Fitzgerald JM, Miskovich TA, et al. Neighborhood socioeconomic disadvantage and the neurobiology of uncertainty in traumatically injured adults. *Biol Psychiatry Global Open Science*. 2022;2(3):263–71. <https://doi.org/10.1016/j.bpsgos.2022.02.006>
9. Webb EK, Weis CN, Huggins AA, Parisi EA, Bennett KP, Miskovich T, et al. Neighborhood disadvantage is associated with stable deficits in neurocognitive functioning in traumatically-injured adults. *Health Place*. 2021;67:102493. <https://doi.org/10.1016/j.healthplace.2020.102493>
10. Webb EK, Weis C, Bennett K, Huggins A, Parisi E, Miskovich T, et al. Neural impact of neighborhood disadvantage in traumatically-injured adults: a multi-nodal investigation. *Biol Psychiatry*. 2021;89(9):S103–4. <https://doi.org/10.1016/j.biopsych.2021.02.267>
11. Xie H, Shih CH, Lughmani MB, Chaudhry HA, Mattin M, Naimy G, et al. Thalamic volume in early weeks after adult trauma is related to both preceding adverse childhood experience and subsequent PTSD development. *Biol Psychiatry*. 2021;89(9):S188. <https://doi.org/10.1016/j.biopsych.2021.02.477>
12. Xie H, Huffman N, Shih CH, Cotton AS, Buehler M, Brickman KR, et al. Adverse childhood experiences associate with early post-trauma thalamus and thalamic nuclei volumes and PTSD development in adulthood. *Psychiatry Res Neuroimaging*. 2022;319:111421. <https://doi.org/10.1016/j.pscychresns.2021.111421>
13. Hruska B, Pacella-LaBarbara ML, Castro IE, George RL, Delahanty DL. Incorporating community-level risk factors into traumatic stress research: adopting a public health lens. *J Anxiety Disord*. 2022;86:102529. <https://doi.org/10.1016/j.janxdis.2022.102529>
14. Ulirsch JC, Weaver MA, Bortsov AV, Soward AC, Swor RA, Peak DA, et al. No man is an island: living in a disadvantaged neighborhood influences chronic pain development after motor vehicle collision. *Pain*. 2014;155(10):2116–23. <https://doi.org/10.1016/j.pain.2014.07.025>
15. Shalev AY, Freedman S, Peri T, Brandes D, Sahar T, Orr SP, et al. Prospective study of posttraumatic stress disorder and depression following trauma. *Am J Psychiatr*. 1998;155(5):630–7. <https://doi.org/10.1176/ajp.155.5.630>
16. Koren D, Arnon I, Lavie P, Klein E. Sleep complaints as early predictors of posttraumatic stress disorder: a 1-year prospective study of injured survivors of motor vehicle accidents. *Am J*

- Psychiatr. 2002;159(5):855–7. <https://doi.org/10.1176/appi.ajp.159.5.855>
17. Kobayashi I, Sledjeski EM, Spoonster E, Fallon WF, Jr., Delahanty DL. Effects of early nightmares on the development of sleep disturbances in motor vehicle accident victims. *J Trauma Stress*. 2008;21(6):548–55. <https://doi.org/10.1002/jts.20368>
 18. Afari N, Ahumada SM, Wright LJ, Mostoufi S, Golnari G, Reis V, et al. Psychological trauma and functional somatic syndromes: a systematic review and meta-analysis. *Psychosom Med*. 2014;76(1):2–11. <https://doi.org/10.1097/PSY.000000000000010>
 19. Pietrzak RH, Goldstein RB, Southwick SM, Grant BF. Prevalence and Axis I comorbidity of full and partial posttraumatic stress disorder in the United States: results from wave 2 of the national epidemiologic survey on alcohol and related conditions. *J Anxiety Disord*. 2011;25(3):456–65. <https://doi.org/10.1016/j.janxdis.2010.11.010>
 20. Cox BJ, Mota N, Clara I, Asmundson GJ. The symptom structure of posttraumatic stress disorder in the National Comorbidity Replication Survey. *J Anxiety Disord*. 2008;22(8):1523–8. <https://doi.org/10.1016/j.janxdis.2008.03.00920>
 21. United States Census Bureau. Income in the United States: 2021; 2022. <https://www.census.gov/library/publications/2022/demo/p60-276.html#:~:text=Highlights,and%20Table%20A%2D1>. Accessed 13 March 2023.
 22. Bernstein DP, Stein JA, Newcomb MD, Walker E, Pogge D, Ahluvalia T, et al. Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse Negl*. 2003;27(2):169–90. [https://doi.org/10.1016/S0145-2134\(02\)00541-0](https://doi.org/10.1016/S0145-2134(02)00541-0)
 23. Lanza ST, editors. Latent class and latent transition analysis: with applications in the social, behavioral, and health Sciences. New York: John Wiley and Sons; 2010.
 24. Lanza ST, Collins LM, Lemmon DR, Schafer JL. Proc LCA: a SAS procedure for latent class analysis. *Struct Equ Model*. 2007;14(4):671–94. <https://doi.org/10.1080/10705510701575602>
 25. Enders CK, Bandalos DL. The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Struct Equ Model*. 2001;8(3):430–57. https://doi.org/10.1207/S15328007SEM0803_5
 26. Alcántara C, Casement MD, Lewis-Fernández R. Conditional risk for PTSD among Latinos: a systematic review of racial/ethnic differences and sociocultural explanations. *Clin Psychol Rev*. 2013;33(1):107–19. <https://doi.org/10.1016/j.cpr.2012.10.005>
 27. Sibrava NJ, Bjornsson AS, Pérez Benítez ACI, Moitra E, Weisberg RB, Keller MB. Posttraumatic stress disorder in African American and Latinx adults: clinical course and the role of racial and ethnic discrimination. *Am Psychol*. 2019;74(1):101–16. <https://doi.org/10.1037/amp0000339>
 28. Hall-Clark B, Sawyer B, Golik A, Asnaani A. Racial/ethnic differences in symptoms of posttraumatic stress disorder. *Curr Psychiatr Rev*. 2016;12(2):124–38. <https://doi.org/10.2174/1573400512666160505150257>
 29. Himle JA, Baser RE, Taylor RJ, Campbell RD, Jackson JS. Anxiety disorders among African Americans, blacks of Caribbean descent, and non-Hispanic whites in the USA. *J Anxiety Disord*. 2009;23(5):578–90. <https://doi.org/10.1016/j.janxdis.2009.01.002>
 30. Remigio-Baker RA, Hungerford LD, Lee-Tauler SY, Bailie JM, Caswell M, Babakhanyan I, et al. Disparities in mental health symptoms recovery across race/ethnicity and education level following mild traumatic brain injury. *Dialogues Clin*. 2022;1:100048. <https://doi.org/10.1016/j.dialog.2022.100048>
 31. Helms JE, Nicolas G, Green CE. Racism and ethnoviolence as trauma: enhancing professional and research training. *Traumatology*. 2012;18(1):65–74. <https://doi.org/10.1177/1534765610396728>
 32. Holbrook TL, Hoyt DB, Stein MB, Sieber WJ. Gender differences in long-term posttraumatic stress disorder outcomes after major trauma: women are at higher risk of adverse outcomes than men. *J Trauma Acute Care Surg*. 2002;53(5):882–8. <https://doi.org/10.1097/01.TA.0000033749.65011.6A>
 33. Stein MB, Walker JR, Forde DR. Gender differences in susceptibility to posttraumatic stress disorder. *Behav Res Ther*. 2000;38(6):619–28. [https://doi.org/10.1016/S0005-7967\(99\)00098-4](https://doi.org/10.1016/S0005-7967(99)00098-4)
 34. Kornfield SL, Hantsoo L, Epperson CN. What does sex have to do with it? The role of sex as a biological variable in the development of posttraumatic stress disorder. *Curr Psychiatr Rep*. 2018;20(6):1–8. <https://doi.org/10.1007/s11920-018-0907-x>
 35. Hu J, Feng B, Zhu Y, Wang W, Xie J, Zheng X. Gender differences in PTSD: susceptibility and resilience In: Alvinus A, editors. Gender differences in different contexts. InTechOpen; 2017.
 36. Hunt JF, Buckingham W, Kim AJ, Oh J, Vogt NM, Jonaitis EM, et al. Association of neighborhood-level disadvantage with cerebral and hippocampal volume. *JAMA Neurol*. 2020;77(4):451–60. <https://doi.org/10.1001/jamaneurol.2019.4501>
 37. Bonanno GA, Galea S, Bucciarelli A, Vlahov D. What predicts psychological resilience after disaster? The role of demographics, resources, and life stress. *J Consult Clin Psychol*. 2007;75(5):671–82. <https://doi.org/10.1037/0022-006X.75.5.671>
 38. Aitken LM, Macfarlane B, Chaboyer W, Schuetz M, Joyce C, Barnett AG. Physical function and mental health in trauma intensive care patients: a 2-year cohort study. *Crit Care Med*. 2016;44(4):734–46. <https://doi.org/10.1097/CCM.0000000000001481>
 39. De Palma JA, Fedorka P, Simko LC. Quality of life experienced by severely injured trauma survivors. *AACN Clin Issues*. 2003;14:54–63.
 40. Breslau N, Chilcoat HD, Kessler RC, Davis GC. Previous exposure to trauma and PTSD effects of subsequent trauma: results from the Detroit Area Survey of Trauma. *Am J Psychiatr*. 1999;156(6):902–7. <https://doi.org/10.1176/ajp.156.6.902>