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Multi-level socioeconomic modifiers of the comorbidity of post-traumatic stress and tobacco, alcohol, and cannabis use: the importance of income

Henri M. Garrison-Desany[1], **Jacquelyn L. Meyers**[2], **Sarah D. Linnstaedt**[3], **Karestan C. Koenen**[4], **Stacey L. House**[5], **Francesca L. Beaudoin**[6,7], **Xinming An**[3], **Thomas C. Neylan**[8], **Gari D. Clifford**[9,10], **Tanja Jovanovic**[11], **Laura T. Germine**[12,13,14], **Kenneth A. Bollen**[15], **Scott L. Rauch**[12,14,16], **John P. Haran**[17], **Alan B. Storrow**[18], **Christopher Lewandowski**[19], **Paul I. Musey Jr.**[20], **Phyllis L. Hendry**[21], **Sophia Sheikh**[21], **Christopher W. Jones**[22], **Brittany E. Punches**[23,24], **Jose L. Pascual**[25,26], **Mark J. Seamon**[26,27], **Erica Harris**[28], **Claire Pearson**[29], **David A. Peak**[30], **Robert M. Domeier**[31], **Niels K. Rathlev**[32], **Brian J. O'Neil**[33], **Paulina Sergot**[34], **Steven E. Bruce**[14,35], **Samuel A. McLean**[36,37], **Christy A. Denckla**[1]

[1]Department of Social and Behavioral Sciences, Harvard T.H. Chan School of Public Health, 677 Huntington Avenue, Boston, MA 02115, USA

[2]Department of Psychiatry and Behavioral Sciences, State University of New York Downstate Medical Center, New York, NY 11203, USA

[3]Institute for Trauma Recovery, Department of Anesthesiology, University of North Carolina at Chapel Hill, Chapel Hill, NC 27559, USA

[4]Department of Epidemiology, Harvard T.H. Chan School of Public Health, Harvard University, Boston, MA 02115, USA

✉Henri M. Garrison-Desany hgarrisondesany@hsph.harvard.edu.

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[5]Department of Emergency Medicine, Washington University School of Medicine, St. Louis, MO 63110, USA

[6]Department of Epidemiology, Brown University, Providence, RI 02930, USA

[7]Department of Emergency Medicine, Brown University, Providence, RI 02930, USA

[8]Departments of Psychiatry and Neurology, University of California San Francisco, San Francisco, CA 94143, USA

[9]Department of Biomedical Informatics, Emory University School of Medicine, Atlanta, GA 30332, USA

[10]Department of Biomedical Engineering, Georgia Institute of Technology and Emory University, Atlanta, GA 30332, USA

[11]Department of Psychiatry and Behavioral Neurosciences, Wayne State University, Detroit, MI 48202, USA

[12]Institute for Technology in Psychiatry, McLean Hospital, Belmont, MA 02478, USA

[13]The Many Brains Project, Belmont, MA 02478, USA

[14]Department of Psychiatry, Harvard Medical School, Boston, MA 02115, USA

[15]Department of Psychology and Neuroscience and Department of Sociology, University of North Carolina at Chapel Hill, Chapel Hill, NC 27559, USA

[16]Department of Psychiatry, McLean Hospital, Belmont, MA 02478, USA

[17]Department of Emergency Medicine, University of Massachusetts Chan Medical School, Worcester, MA 01655, USA

[18]Department of Emergency Medicine, Vanderbilt University Medical Center, Nashville, TN 37232, USA

[19]Department of Emergency Medicine, Henry Ford Health System, Detroit, MI 48202, USA

[20]Department of Emergency Medicine, Indiana University School of Medicine, Indianapolis, IN 46202, USA

[21]Department of Emergency Medicine, University of Florida College of Medicine -Jacksonville, Jacksonville, FL 32209, USA

[22]Department of Emergency Medicine, Cooper Medical School of Rowan University, Camden, NJ 08103, USA

[23]Department of Emergency Medicine, Ohio State University College of Medicine, Columbus, OH 43210, USA

[24]Ohio State University College of Nursing, Columbus, OH 43210, USA

[25]Department of Surgery, Department of Neurosurgery, University of Pennsylvania, Philadelphia, PA 19104, USA

[26]Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA 19104, USA

[27]Division of Traumatology, Department of Surgery, Surgical Critical Care and Emergency Surgery, University of Pennsylvania, Philadelphia, PA 19104, USA

[28]Department of Emergency Medicine, Einstein Medical Center, Philadelphia, PA 19107, USA

[29]Department of Emergency Medicine, Wayne State University, Ascension St. John Hospital, Detroit, MI 48202, USA

[30]Department of Emergency Medicine, Massachusetts General Hospital, Boston, MA 02114, USA

[31]Department of Emergency Medicine, Trinity Health-Ann Arbor, Ypsilanti, MI 48197, USA

[32]Department of Emergency Medicine, University of Massachusetts Medical School-Baystate, Springfield, MA 01107, USA

[33]Department of Emergency Medicine, Wayne State University, Detroit Receiving Hospital, Detroit, MI 48202, USA

[34]Department of Emergency Medicine, McGovern Medical School at UTHealth, Houston, TX 77030, USA

[35]Department of Psychological Sciences, University of Missouri - St. Louis, St. Louis, MO 63121, USA

[36]Department of Emergency Medicine, University of North Carolina at Chapel Hill, Chapel Hill, NC 27559, USA

[37]Institute for Trauma Recovery, Department of Psychiatry, University of North Carolina at Chapel Hill, Chapel Hill, NC 27559, USA

Abstract

Purpose: Post-traumatic stress (PTS) symptoms are highly comorbid with substance use (i.e., alcohol, tobacco, and cannabis). Few studies have investigated potential individual-, household-, and neighborhood-level socioeconomic effect modifiers of this comorbidity in longitudinal analyses. We aim to examine interactions between this multi-level environment and PTS symptoms on future substance use behaviors.

Methods: Data were drawn from the Advancing Understanding of RecOvery afteR traumA (AURORA) study, including 2943 individuals who presented to the emergency department (ED) within 72 h of a traumatic event. Frequency of tobacco, alcohol, cannabis use, and PTS symptoms were reported at 6 timepoints. Mixed effect Poisson models, clustered by state, were used to generate incidence rate ratios (IRRs) substance use, both cross-sectionally and prospectively. Moderation analysis of PTS and substance use, stratified by household income and area deprivation index (ADI), was conducted using mixed effect models and parallel process growth curves.

Results: Significant associations were observed between PTS with tobacco, alcohol, and cannabis use frequency cross-sectionally, and for tobacco and alcohol and PTS exposure prospectively. Lower income ($P < 0.001$) and higher deprivation ($P < 0.001$) were associated with tobacco use, while higher income ($P < 0.001$) and less deprivation ($P = 0.01$) were associated with increased alcohol use. We found modest modification by household income for alcohol and tobacco, and little evidence of modification by neighborhood ADI.

Conclusions: Household income had greater evidence of effect modification for substance use, compared to neighborhood-level ADI. Our findings demonstrate that household indicators of socioeconomic status likely modify the relationship between PTS and substance use.

Keywords

Household income; Post-traumatic stress disorder; Alcohol; Tobacco; Neighborhood

Introduction

Traumatic events affect nearly 70% of people in their lifetime [1], and it is estimated, among those, the lifetime prevalence of post-traumatic stress (PTS) symptoms meeting clinical diagnostic threshold is 7.8% [2]. PTS symptoms are associated with a range of other behavioral health challenges, including increased substance use [3]. One systematic review, for example, found that up to 61.3% of those with PTSD reported alcohol misuse [4], and a meta-analysis found that 24.0% of individuals with PTSD currently smoked tobacco [5]. In literature, there is notable evidence that PTS symptoms lead to substance use behaviors [6]. However, there are cases of reverse directionality as well [7], and studies, primarily performed in treatment settings, have shown that PTS symptoms at baseline can influence future substance behaviors [8–11]. However, this relationship is also complicated by the impact of social and environmental factors. These risk factors can occur throughout many levels of the social-ecological landscape, compounding across the structural, community, and interpersonal levels and interacting with PTS symptoms to increase substance use beyond general population estimates.

Drawing from Bronfenbrenner’s socioecological model, we conceptualize health outcomes embedded within multiple levels and influenced by factors ranging from the individual to the systemic domains [12]. For example, factors reflecting the microsystem, such as family characteristics including household income, and the exosystem, including broader resources such as neighborhood deprivation, can interact at the mesosystem and then influence individual health trajectories [13–15]. These multiple ecological levels can all affect socioeconomic status (SES), and there is empirical evidence of this complex relationship: prior studies have found associations with reduced SES and increased substance use behaviors, however these findings have varied considerably and have not demonstrated a clear linear relationship [16]. For instance, higher childhood SES has been shown to increase the risk of alcohol and cannabis use [17], while lower adult SES has been associated with increased tobacco use [18]. Socioeconomic status is also associated with differences in PTS symptoms [19, 20], and has demonstrated a moderating relationship between PTSD and other health outcomes, such as cardiovascular disease [21]. However, the moderating effect of SES between PTS and future substance use is understudied, and will be the basis of our study objectives.

Beyond individual SES, other environmental conditions are likely to affect this relationship. Changing legislative landscapes for substances, such as tobacco, alcohol, and cannabis, may moderate potential effects. For example, the availability of legal cannabis in some states but not others may lead to moderation due to geography [22]. At the more granular level, stress-diathesis theory suggests that compounding traumas over time then increases the risk of PTS and related sequelae [23], and there is evidence that the number of traumatic events one experience can be affected by neighborhood and environment. Areas with higher levels of reported crimes have more events that could be considered traumatic [24, 25]. Similarly, interpersonal violence has been shown to have different geographic patterns and risk factors based on urbanicity versus rurality [26, 27]. The influence of these higher-level effects of state and neighborhood on traumatic events and PTS symptoms should be considered in conjunction with household factors, which we aim to further investigate.

Understanding the role of the multi-level SES is critical for addressing substance use among communities exposed to traumatic events. A critical appraisal of these compounding stressors (such as neighborhood deprivation and household SES) is necessary to guide treatment and address preventable health disparities. To address these gaps in knowledge, we will use a large sample of individuals presenting to an emergency room within 72 h of a traumatic event, allowing us to investigate PTS symptom associations with incident substance use.

Objectives

(1) We will investigate the association of PTS with cross-sectional and prospective alcohol, tobacco, and cannabis use in this sample. (2) We will examine household income as a modifier of the relationship between PTS symptoms and substance use. (3) We will examine neighborhood deprivation as a modifier of the relationship between PTS and substance use, independent of the effect of income. And (4) we will test whether household income and neighborhood deprivation interact with one another to then modify the relationship between PTS and substance use.

Methods

Study sample

The Advancing Understanding of RecOvery afteR traumA (AURORA) cohort has been previously described in detail [28]. In brief, AURORA was a prospective cohort comprised of nearly 3000 individuals who present at one of 29 participating emergency departments (ED) within 72 h of experiencing an index traumatic event. Adults aged 18 to 75 years of age were excluded if they were administered general anesthesia at the time, experienced long bone fractures, significant hemorrhage, solid organ injury, or were not alert or oriented at the time of enrollment. Individuals were followed for one year, with follow-up at week 2, week 8, month 3, month 6, and month 12. Data for this analysis included a self-reported questionnaire at these timepoints. We included participants who responded to the three substance use questions and the PTSD scale.

Measures

Post-traumatic stress symptoms were assessed using the self-report PTSD checklist for DSM-5 (PCL-5) [29]. Participants were asked to rate on a 5-point scale severity of symptoms experienced in the 30 days prior to the timepoint, except week 2, which include the two weeks prior only. Scores were summed, with a range of 0 to 80. The PCL-5 was administered at ED enrollment, weeks 2 and 8, months 3, 6, and 12 after the index event. When binarizing, likely PTSD was defined as 33 or greater, based on the validated cut point for the PCL-5 for US Adults; unlikely PTSD was defined as less than 33 out of 80 [29].

Substance use was defined across three primary outcomes: tobacco, alcohol, and cannabis. These were ascertained as a count of frequency of use in the last 30 days using the PhenX Toolkit [30]. We focused on tobacco, alcohol, and cannabis due to their full legal or semi-legal status, which are subject to differing state laws and regulations.

Covariates

We defined social factors at the individual, household, and community level. Individual factors included participant age, gender identity (defined as cisgender men, cisgender women, and transgender/non-binary people), race/ethnicity (defined as non-Hispanic Black, non-Hispanic White, Hispanic, other non-Hispanic race/ethnicities), participant marital status (defined as never married, married, separated/divorced, and widowed/other), and participant education (defined as not attending high school, attending only high school, attending college, and attending graduate school).

Effect modifiers

Household income was originally collected as a categorical variable ("What is your best estimate of your total family income from all sources, before taxes, in the last calendar year?") with levels: < = \$19,000/year; > \$19,000 and < = \$35,000/year; > \$35,000 and < = \$50,000/year; > \$50,000 and < = \$75,000/year; > \$75,000 and < \$100,000/year, > = \$100,000/year, and "did not know." We collapsed household income initially as: < = \$35,000/year, > \$35,000 and < = \$75,000/year, > \$75,000 and "did not know." In our stratification analysis, in the interest of preserving power, we further collapsed categories into two strata: household income of > \$50,000/year and < = \$50,000/year.

At the community-level, we used data from the Area Deprivation Index (ADI) 2019 [31, 32] that was merged with census tract, determined based on self-reported address, for both national percentile and state-specific decile. The ADI is a multi-faceted index reflecting neighborhood average income, education, employment, and housing quality. We report the ADI as scaled within each state as state-specific decile, as well as the total national percentile. When using the state decile, we clustered our models at the state level.

Statistical methods

We generated descriptive statistics and examined the longitudinal trends of substance use frequency and PCL-5 symptoms graphically. We mapped substance use and PCL-5 symptom measures across the United States by state to highlight potential geographic trends in both our exposures and outcomes. We report the state-specific correlations between any substance use and likely PTSD diagnosis, along with the number of respondents per state, in supplement.

We used generalized estimating equations (GEE) to account for the longitudinal nature of our data, and estimate trends over time for both the dependent and independent variables. We determined autocorrelation as exchangeable via graphs and autocorrelation coefficient, and estimated GEE models controlling for socioenvironmental covariates. For Objective 1, we analyzed the effects of PTS on substance use, and the impact of potential confounding due to area deprivation, using generalized linear mixed effect models (GLMEM), with clustering at the state-level and random intercepts for individuals over time. GLMEM were used to account for the state-level clustering when using the state-specific ADI. This generates estimates of the conditional effect based on state membership, which is a more granular investigation of the effect of ADI. This model, however, had challenges with converging, and therefore we used the GEE and the national ADI ranking (without state-clustering) to generate marginal effects in instances when the GLMEM did not converge.

These models were specified both cross-sectionally (e.g., PTS symptoms and substance use behaviors were reported in the same time period) including the baseline report of the prior 30 days before the index traumatic event, as well as longitudinally by lagging the exposure to determine associations with prospective substance use following PTS symptoms. This lagging was such that sociodemographic variables from ED baseline were carried forward, PTS symptoms were ascertained at week 2 following the index trauma, week 8, month 3 and month 6, and substance use outcomes were ascertained at week 8, month 3, month 6, and month 12, resulting in a model with 4 timepoints. These models used the quasipoisson distribution and estimated the incidence rate ratios when exponentiated.

We then conducted a stratified analysis to investigate moderation by individual household income and national neighborhood ADI using GEEs and then fit parallel process latent growth curves to model PTS and substance use, as convergence was not achieved including a state-level random intercept. For Objective 2, stratifying by a binarized income variable (< = $50,000/year and > $50,000/year), we tested associations between PCL-5 symptoms and tobacco, alcohol, and cannabis use in our GEE models. These models estimated the marginal change in continuous use (quantity, frequency, quantity-frequency) for each substance. For Objective 3, we stratified by national ADI by the median value, to examine differences in the relationship between PCL-5 and substance use also using GEEs. These models estimated the marginal change in continuous use for each substance. We also conducted a sensitivity analysis stratifying by the 25th and 75th percentile of ADI (e.g., omitting the middle 50%) to compare extremes, however this analysis was underpowered for the continuous outcome, and therefore substance use was binarized and logistic GEEs are reported in supplement.

We then used parallel process latent growth curves to more accurately model the changing trajectories between the development of PTS symptoms and substance use behaviors over time. We allowed both PTS symptoms and substance use to have a random intercept and for each of these intercepts to influence the slope of the other. This was completed with the lagged version, allowing for prospective assessment of substance use. Coefficients were standardized, and we adjusted for sociodemographic coefficients in both the intercepts and slopes. We compared fit statistics of the parallel processes including the income variable as a moderator, the ADI ranking as a moderator, and both as moderators in the same model. Fit statistics included log likelihood, Akaike’s Information Criterion (AIC), and the Bayesian

Information Criterion (BIC). The models required numerical integration for fitting, which do not produce the Tucker-Lewis Index or Comparative Fit Index.

For Objective 4, we examined the inclusion of both ADI and income as moderators in the GEE models and using parallel process modeling. In the GEE models, this was done both individually (ADI x PTS in model A, income x PTS in model B), then with both moderators in the same model (ADI x PTS and income x PTS), and finally with a 3-way interaction with PTS (ADI x income x PTS). These model variations are reported in supplement.

We also completed multigroup analysis of the parallel process models to examine any modification between ADI and income. First with (1) high ADI/low ADI, (2) high income/low income/did not report, and (3) both ADI and income defined as: high ADI and high income, high ADI and low income, high ADI and not reported income, low ADI and high income, low ADI and low income, low ADI and not reported income. These models were conducted using the Poisson distribution, with Monte Carlo integration to generate log incidence rate ratios.

We used an alpha threshold of 0.05 and generated 95% confidence intervals as appropriate. GEE models were conducted in R 4.2.3 [33]. Parallel process models were conducted in MPLUS [34].

Missingness

We assessed missingness and determined it was likely to be missing at random (MAR) using graphical methods and hypothesis testing with whether a variable was missing (yes/no) and our primary measures. Income missingness was significantly higher than other variables (nearly 15%) and therefore we maintained "Did not report" as a category. We conducted multiple imputation by chained equations for 20 datasets across 30 iterations each. All model estimates were pooled using Rubin's rules [35].

Results

In our analytic sample, we had 2943 participants (Table 1), among whom 1099 (37.3%) reported PCL-5 symptoms at baseline that would indicate likely PTSD diagnosis, defined as total scores of 33 or higher out of 80. We report enrollment and missingness across the waves in Supplemental Fig. 1 and Supplemental Table 1, respectively. There was a similarly expected sex ratio among the likely PTSD group, with the majority being cisgender female (70.2%, N = 772) (χ^2 p-value < 0.001). The majority of participants, regardless of likely PTSD diagnosis, reported any vehicle collision as the index traumatic event (76.4%, N = 2247). Most participants also reported past-month alcohol use (61.7%, N = 1815) though this did not differ by likely PTSD diagnosis at baseline (χ^2 p-value = 0.10). A large proportion also reported tobacco use (35.5%, N = 1046) and cannabis use (29.2%, N = 858), though neither of these differed by likely PTSD diagnosis (χ^2 p = 0.06 and 0.90 respectively). We also report the ADI distribution by income level to demonstrate the variability of household income across ADI strata (Supplemental Fig. 2). We also report the distribution of exposure and outcomes across all states (Supplemental Fig. 3) and their state-specific correlation (Supplemental Table 2).

Objective 1

In our primary models assessing the association between PCL-5 symptoms and incidence rate of tobacco, alcohol, and cannabis frequency (Table 2), PCL-5 symptoms were highly associated across these substances when controlling for sociodemographic covariates in the cross-sectional model. Higher ADI (e.g., greater neighborhood deprivation) was significantly associated with increased tobacco use (IRR: 1.02, 95% CI: 1.01, 1.03), with reduced alcohol consumption (IRR: 0.994, 95% CI: 0.989, 0.999), and with increased cannabis used (IRR: 1.01, 95% CI: 1.00, 1.02). Income was also highly associated with tobacco, alcohol, and cannabis use. Compared with individuals making > $35,000–< $75,000 annually, those making ≤ $35,000/year had a 26% higher rate of tobacco frequency in the last 30 days (95% CI: 1.16, 1.37). Those making ≥ $75,000/year had a higher risk of alcohol drinking frequency (IRR: 1.16, 95% CI: 1.08, 1.23), and for cannabis use, making ≤ $35,000/year had a 9% higher incidence of cannabis frequency (95% CI: 1.02, 1.17). We also used linear mixed effect models to examine the relationship with prospective substance use under the lagged model (Supplemental Table 3). The linear model was used because neither the Poisson, negative binomial, nor quasipoisson converged with the lagged model. There, tobacco showed associations with state ranked ADI (Coef: 0.11, 95% CI: 0.01, 0.22) but not with alcohol or cannabis (Supplemental Table 4). We conducted a sensitivity analysis to examine if there are potential confounding effects from ADI on the association between income and substance use behaviors by removing ADI from the model and comparing the change in the association estimates (Supplemental Table 5). We found a notable difference in the estimates and control for ADI in subsequent models including income.

Objective 2

In our models stratified by low/high household income (Fig. 1, Table 3), we found that, among individuals with low household income, there were highly significant associations between PTS symptoms and tobacco, alcohol, and cannabis ($p < 0.01$), with tobacco showing the greatest association (change in frequency: 0.03, 95% CI: 0.01, 0.04). In the high-income strata, there was no significant association between PCL-5 and alcohol or cannabis outcomes ($p = 0.051$ and $p = 0.41$, respectively). We repeated this analysis with the prospective exposure, and found significant associations with alcohol in the low income strata (change in frequency: 0.01, 95% CI: 0.003, 0.02), which were no longer significant in the high income strata (change in frequency: 0.007, 95% CI: − 0.01, 0.03), and a similar pattern for tobacco [change in frequency: 0.02 (95% CI; 0.001, 0.04) vs. 0.01 (95% CI: − 0.02, 0.04)].

Objective 3

When stratifying by national ADI ranking (Table 4), we found that PCL-5 symptoms were associated with cannabis use among low ADI ($p = 0.03$) but not high ADI ($p = 0.11$) strata, but these estimates had similar effect sizes and overlapping confidence intervals (Low ADI coefficient: 0.01, 95% CI: − 0.002, 0.02 vs. high ADI coefficient: 0.02, 95% CI: 0.003, 0.04, respectively). Associations were nearly the same for alcohol and tobacco between the high and low ADI, both in point estimate and significance across the strata. Given well-documented associations between gender and substance use behaviors [36–38], and gender and PTS symptoms [39–41], we also conducted a sensitivity analysis stratifying by gender to examine differences in our main effect based on this influential factor. We found no difference in direction of effect or effect size between cisgender men and women, or significant interaction though estimates for cis-gender men were more often significant compared to cisgender women (Supplemental Table 6). This demonstrates that our findings are likely robust to the effects of gender in our sample. Race/ethnicity did not show associations with substance use in our sample when controlling when ADI and income (Table 2), and therefore we did not pursue stratification.

To reflect the longitudinal trajectory of PTS symptoms and using tobacco, alcohol, and cannabis over time, we conducted parallel process models (Supplemental Fig. 4). There were statistically significant associations for the intercept of PTS symptoms and the slope of each substance use category. For tobacco, the standardized coefficient using the loglinear model was 0.134 (SE: 0.007) for alcohol frequency, the standardized coefficient was 0.032 (SE: 0.006), and for cannabis use frequency, the standardized coefficient was 0.086 (SE: 0.008). Fit statistics of the parallel process latent growth curve models are reported in Supplemental Table 7.

Objective 4

To investigate how ADI and income may interact with one another to further modify the relationship, we first disaggregated based on ADI and income category. We found that 20.3% (n = 598) had low ADI and low income, 47.5% (n = 1399) had high ADI and low income, 10.6% (n = 313) had low ADI and high income, 9.2% (n = 271) had high ADI and high income, with similarly 4.2% (n = 124) had low ADI and did not report their income, while 8.1% (n = 238) had high ADI and did not report their income. Using the GEE model with both income and ADI included, there was no evidence of a 3-way interaction with PTS symptoms, or a two-way interaction between ADI and income (Supplemental Tables 8, 9, 10, 11, 12, 13). Using the multi-group parallel process model, there was no significant difference in the multigroup parameters.

Sensitivity analyses

We mapped baseline characteristics at the state level based on the proportion of likely PTSD diagnosis (Supplemental Fig. 2, Panel A), self-reported use of any tobacco in the past 30 days (Panel B), any alcohol use in the past 30 days (Panel C), and any cannabis use (Panel D). Due to small sample size, some states had rates of 100%. It was possible for participants to attend recruitment sites in a state different from their state of residence, therefore there were 29 recruitment sites, and 33 states represented. By state, most correlation estimates for tobacco and PTSD were about 0.15 (range: −0.01, 0.24) and were statistically significant (Supplemental Table 1). Alcohol had a wider range in point estimates, though none of the estimates except Michigan (0.05, 95% CI: 0.01, 0.10) and Missouri (0.06, 95% CI: 0.02, 0.10) were statistically significant, those these were quite modest associations. Cannabis also showed a slight correlation by state, with the highest being from Connecticut (0.45, 95% CI: 0.16, 0.67), though this had a small sample size (n = 8).

We estimated the association between income and ADI associated with PTS symptoms, using linear mixed effect models at each timepoint (Supplemental Table 3). We found that both individual income and ADI state ranking were highly associated in the cross-sectional model. Individuals in households ≤ $35,000/year had a 3.13-point increase in PCL-5 symptoms (95% CI: 1.54, 4.73) compared to those making between $35,000 and $75,000. Similarly, those making ≥ $75,000/year had a reduced incidence rate of −3.58 point change (95% CI: − 5.88, − 1.28). Controlling for individual income, the ADI state ranking decile also increased PCL-5 scores by 0.18 points (0.004, 0.37) for each decile.

Discussion

Our objective was to understand how income and neighborhood deprivation affect the relationship between PTS symptoms and substance use behaviors. We found that having lower income and living in neighborhoods with fewer resources were associated with tobacco and cannabis use, while higher income and less deprivation were associated with alcohol use over time. We found evidence that both household income and neighborhood deprivation modify the association of PTS symptoms and cannabis use cross-sectionally though not prospectively. Only household income showed some suggestive evidence of modifying the association of PTS symptoms and prospective tobacco and alcohol use, though this did not yield statistical significance in the interaction term.njj. We demonstrate that deprivation and income do not have a uniform association with these substances. These findings characterize the heterogeneity of the relationship between PTS symptoms and substance use behaviors, and have implications for tailoring interventions across different populations following a traumatic event.

Both individual income and ADI showed associations with PTS symptoms in Objective 1, which comports with prior literature [42]. Individuals in high deprivation areas may have an increased exposure to traumatic events and therefore risk of traumatic stress symptoms. There is a large body of evidence that traumatic events, such as motor vehicle crashes [43], and childhood injury [44] have geographic variability. Childhood traumatic events and a higher number of traumatic events in adulthood are both associated with lower SES [45, 46], which may then create a higher exposure load and risk of PTS symptoms when later traumatic events occur [47, 48]. The ADI variable may be capturing a mixture of both geographic associations and socioeconomic status, resulting in some of this moderating effect. The additional stress associated with lack of resources, which can affect both quality of life and mental health, may further compound the independent effects of low household income [23]. Notably, despite ADI having multiple variables considered in its index besides average income (including education, employment, and housing quality), only income had the most pronounced and consistent effects, and neither education nor race/ethnicity showed major associations with substance use after adjustment in our sample.

Tobacco and cannabis also demonstrated associations with reduced income and higher deprivation (e.g., increased use with increased ADI). In the prospective analysis, only tobacco showed associations with lower income and higher deprivation, suggesting that while income and deprivation may be associated with future tobacco use, this was unlikely to affect later cannabis use when controlling for prospective PTS symptoms and other sociodemographic factors. Cannabis, when stratifying in Objectives 2 and 3, demonstrated a paradoxical effect when comparing income and ADI; among lower deprivation (higher resource) areas, there was a positive association between PTS symptoms and cannabis use, but among lower incomes, there was an increased association of PTS symptoms and cannabis use. For cannabis in particular, given the proliferation of state-licensed dispensaries, those in less deprived areas may have access to purchase cannabis easily, with increased branding and normalization of use among upper socioeconomic classes. While individuals with lower incomes may use cannabis more often, there may also be a continued reckoning with the historic War on Drugs among lower socioeconomic classes [49, 50]. This may contribute to the bifurcation between neighborhood- and household-level factors. More work, potentially using qualitative or mixed methods, should investigate the nuances between area and income for this population.

When including both income and ADI as moderators in the association between PTSD and substance use outcomes, per Objective 4, there was no significant evidence that they interacted with one another beyond what was captured in the main effects. This suggests that they likely influence substance use behaviors independently. Given the complexity of these models, including when fitting with the parallel process, greater sample sizes to increase power may yield different results, as there were few individuals who reported high area deprivation but high income. More nuanced methods, such as qualitative interviews, may better describe the specific challenges individuals in discrepancy income/deprivation households may face. For example, some qualitative studies have described feelings of hopelessness related to community violence, and substance use being considered a socially acceptable coping mechanism to traumatic events compared to engaging with a historically stigmatizing or hostile medical system for care [51–53]. However, how this is impacted by neighborhood versus household characteristics separately remains a less investigated area.

Finally, it is notable that we did not have clear associations with self-reported race/ethnicity when controlling for income and ADI. Prior literature has demonstrated a consistent trend that Black Americans and Latinx Americans experience a higher prevalence of PTSD compared to White Americans [54–56]. These studies did not control for the ADI, which captures not only poverty or racial make-up of a census tract, but other markers of deprivation. These studies, therefore, may overstate the association of race/ethnicity with PTSD given the correlation of individual race/ethnicity with components of the ADI. This would result in confounding when ADI or similar neighborhood-level variables are not adjusted for in models. Other reasons we may not see this association is our sample may be due to selection bias, as our sample was not nationally representative but rather recruited trauma-exposed adults from EDs [55, 57, 58]. Given only certain types of traumatic events are likely to result in attending an ED, the type of traumatic event may confound prior studies due to the traumatic event type being associated with self-reported race/ethnicity. Studies have demonstrated that different racial/ethnic groups are more likely to experience certain traumatic events compared to others, and selection from the ED may effectively condition on certain traumatic event types and reduce this association.

Our study had a number of limitations, primarily that toxicological data was not available, and we therefore relied on self-reported past 30-day use of substances for our outcome classification. This may be subject to social desirability bias, whereby individuals are hesitant to report their substance use, and our estimates may be biased towards the null as a result [59]. We also had longitudinal data, allowing us to ascertain substance use beyond what individuals felt comfortable disclosing in the ED. The subsequent waves, however, were subject to attrition, with fewer participants continuing by the end of the study compared to the beginning. While MICE did estimate the responses for these individuals, it remains an estimate and we were not able to directly observe all outcomes at each timepoint. Our cohort also has many motor vehicle accidents as the index event, which may lead to our findings not being generalizable to other trauma-exposed populations. Populations that, for example, are primarily related to natural disasters may not have the same risk of substance use at baseline. Our methods for assessing income were also limited; other measures of income beyond self-report may more accurately capture different income sources or additional income not perceived as part of the household, such as gift funds or undocumented income (e.g., “gigs”). We did not investigate specific dimensions of PTS symptoms, but maintained the full scale, thereby reducing potential nuances between symptom types. We also stratified our analyses by categorizing continuous ADI or grouping income levels, which may further obfuscate more subtle between-group differences. Additionally, our null effects in the interaction models, especially the three-way interactions, may reflect being underpowered rather than a true null effect, and should be replicated in larger sample sizes. We believe our suggestive findings, however, offer an important exploration of the multi-faceted relationship between household income and neighborhood characteristics that may serve as the basis for future, larger studies. There may also be unmeasured confounders we did not include in our assessment, which could further impact the relationship of PTS symptoms and substance use. Finally, we acknowledge that there may not be a direct effect from these socioenvironmental factors, rather they may reflect some other unmeasured factor also associated with household income and area deprivation.

Conclusions

We found that household income showed some evidence of effect modification of the relationship between PTSD and alcohol and cannabis. We found evidence that ADI was associated with both PTS symptoms and substance use in our sample, but there was little evidence of effect modification. There was minimal evidence that gender modified the relationship between ADI, income, or PTS symptoms and their effects on substance use. These findings suggest that not all socioeconomic variables demonstrate the same level of association or modification with this comorbidity, and that income is the strongest moderator of the PTSD-SUD association in this sample.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data availability

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) https://doi.org/10.15154/nyb1-rm80.

References

1. Benjet C, Bromet E, Karam EG et al. (2016) The epidemiology of traumatic event exposure worldwide: results from the World Mental Health Survey Consortium. Psychol Med 46:327–343. 10.1017/S0033291715001981 [PubMed: 26511595]
2. Gradus JL (2017) Prevalence and prognosis of stress disorders: a review of the epidemiologic literature. Clin Epidemiol 9:251–260. 10.2147/CLEP.S106250 [PubMed: 28496365]
3. Conway KP, Compton W, Stinson FS, Grant BF (2006) Lifetime comorbidity of DSM-IV mood and anxiety disorders and specific drug use disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. J Clin Psychiatry 67:247–257. 10.4088/jcp.v67n0211 [PubMed: 16566620]
4. Debell F, Fear NT, Head M et al. (2014) A systematic review of the comorbidity between PTSD and alcohol misuse. Soc Psychiatry Psychiatr Epidemiol 49:1401–1425. 10.1007/s00127-014-0855-7 [PubMed: 24643298]
5. Pericot-Valverde I, Elliott RJ, Miller ME et al. (2018) Posttraumatic stress disorder and tobacco use: a systematic review and meta-analysis. Addict Behav 84:238–247. 10.1016/j.addbeh.2018.04.024 [PubMed: 29753221]
6. Hawn SE, Cusack SE, Amstadter AB (2020) A systematic review of the self-medication hypothesis in the context of posttraumatic stress disorder and comorbid problematic alcohol use. J Trauma Stress 33:699–708. 10.1002/jts.22521 [PubMed: 32516487]

7. Tripp JC, Worley MJ, Straus E et al. (2020) Bidirectional relationship of posttraumatic stress disorder (PTSD) symptom severity and alcohol use over the course of integrated treatment. Psychol Addict Behav 34:506–511. 10.1037/adb0000564 [PubMed: 32105112]
8. Davis JP, Diguiseppi G, De Leon J et al. (2019) Understanding pathways between PTSD, homelessness, and substance use among adolescents. Psychol Addict Behav 33:467–476. 10.1037/adb0000488 [PubMed: 31343198]
9. Hinojosa CA, Liew A, An X et al. (2023) Associations of alcohol and cannabis use with change in posttraumatic stress disorder and depression symptoms over time in recently trauma-exposed individuals. Psychol Med. 10.1017/S0033291723001642
10. Livingston NA, Lee DJ, Mahoney CT et al. (2021) Longitudinal assessment of PTSD and illicit drug use among male and female OEF-OIF veterans. Addict Behav 118:106870. 10.1016/j.addbeh.2021.106870 [PubMed: 33667852]
11. López-Castro T, Hu M-C, Papini S et al. (2015) Pathways to change: Use trajectories following trauma-informed treatment of women with co-occurring post-traumatic stress disorder and substance use disorders. Drug Alcohol Rev 34:242–251. 10.1111/dar.12230 [PubMed: 25735200]
12. Bronfenbrenner U (1977) Toward an experimental ecology of human development. Am Psychol 32:513–531. 10.1037/0003-066X.32.7.513
13. Galobardes B, Shaw M, Lawlor DA, Lynch JW (2006) Indicators of socioeconomic position (part 1). J Epidemiol Community Health 60:7–12. 10.1136/jech.2004.023531
14. Galobardes B, Shaw M, Lawlor DA, Lynch JW (2006) Indicators of socioeconomic position (part 2). J Epidemiol Community Health 60:95–101. 10.1136/jech.2004.028092 [PubMed: 16415256]
15. Krieger N, Chen JT, Waterman PD et al. (2003) Race/ethnicity, gender, and monitoring socioeconomic gradients in health: a comparison of area-based socioeconomic measures–the public health disparities geocoding project. Am J Public Health 93:1655–1671. 10.2105/ajph.93.10.1655 [PubMed: 14534218]
16. Karriker-Jaffe KJ (2011) Areas of disadvantage: a systematic review of effects of area-level socioeconomic status on substance use outcomes. Drug Alcohol Rev 30:84–95. 10.1111/j.1465-3362.2010.00191.x [PubMed: 21219502]
17. Patrick ME, Wightman P, Schoeni RF, Schulenberg JE (2012) Socioeconomic status and substance use among young adults: a comparison across constructs and drugs. J Stud Alcohol Drugs 73:772–782. 10.15288/jsad.2012.73.772 [PubMed: 22846241]
18. Hiscock R, Bauld L, Amos A et al. (2012) Socioeconomic status and smoking: a review. Ann N Y Acad Sci 1248:107–123. 10.1111/j.1749-6632.2011.06202.x [PubMed: 22092035]
19. El-Khodary B, Samara M, Askew C (2020) Traumatic events and PTSD among palestinian children and adolescents: the effect of demographic and socioeconomic factors. Front Psychiatry. 10.3389/fpsyt.2020.00004
20. Shiga T, Zhang W, Ohira T et al. (2021) Socioeconomic status, damage-related conditions, and PTSD following the Fukushimadaiichi nuclear power plant accident: the Fukushima Health Management Survey. Fukushima J Med Sci 67:71–82. 10.5387/fms.2020-24 [PubMed: 34456222]
21. Kim K, Tsai AC, Sumner JA, Jung SJ (2022) Posttraumatic stress disorder, cardiovascular disease outcomes and the modifying role of socioeconomic status. J Affect Disord 319:555–561. 10.1016/j.jad.2022.09.117 [PubMed: 36174781]
22. National Conference of State Legislatures (2023) State Medical Cannabis Laws. https://www.ncsl.org/health/state-medical-cannabis-laws. Accessed 2 Jun 2023
23. McKeever VM, Huff ME (2003) A diathesis-stress model of posttraumatic stress disorder: ecological, biological, and residual stress pathways. Rev Gen Psychol 7:237–250. 10.1037/1089-2680.7.3.237
24. Garo L, Allen-Handy A, Lewis CW (2018) Race, poverty, and violence exposure: a critical spatial analysis of African American Trauma Vulnerability and Educational Outcomes in Charlotte, North Carolina. J Negro Educ 87:246–269
25. Schuurman N, Hameed SM, Fiedler R et al. (2008) The spatial epidemiology of trauma: the potential of geographic information science to organize data and reveal patterns of injury and services. Can J Surg 51:389–395 [PubMed: 18841227]

26. Beyer K, Wallis AB, Hamberger LK (2015) Neighborhood environment and intimate partner violence: a systematic review. Trauma Violence Abuse 16:16–47. 10.1177/1524838013515758 [PubMed: 24370630]

27. Edwards KM (2015) Intimate partner violence and the rural–urban–suburban divide: myth or reality? A critical review of the literature. Trauma Violence Abuse 16:359–373. 10.1177/1524838014557289 [PubMed: 25477015]

28. McLean SA, Ressler K, Koenen KC et al. (2020) The AURORA study: a longitudinal, multimodal library of brain biology and function after traumatic stress exposure. Mol Psychiatry 25:283–296. 10.1038/s41380-019-0581-3 [PubMed: 31745239]

29. Weathers F, Litz B, Keane T et al. (2013) The PTSD checklist for DSM-5 (PCL-5)—standard [Measurement instrument].

30. Hamilton CM, Strader LC, Pratt JG et al. (2011) The PhenX Toolkit: get the most from your measures. Am J Epidemiol 174:253–260. 10.1093/aje/kwr193 [PubMed: 21749974]

31. Kind AJH, Buckingham WR (2018) Making neighborhood-disadvantage metrics accessible—the neighborhood atlas. N Engl J Med 378:2456–2458. 10.1056/NEJMp1802313 [PubMed: 29949490]

32. University of Wisconsin School of Medicine and Public Health (2019) Area Deprivation Index 2019

33. R Core Team (2023) R: A language and environment for statistical computing

34. Muthen LK, Muthen BO (2017) MPLUS user's guide, 8th edn. Muthen & Muthen, Los Angeles

35. Rubin DB (1987) Multiple imputation for nonresponse in surveys. John Wiley & Sons

36. Lev-Ran S, Le Strat Y, Imtiaz S et al. (2013) Gender differences in prevalence of substance use disorders among individuals with lifetime exposure to substances: results from a large representative sample. Am J Addict 22:7–13. 10.1111/j.1521-0391.2013.00321.x [PubMed: 23398220]

37. Evans EA, Grella CE, Washington DL, Upchurch DM (2017) Gender and race/ethnic differences in the persistence of alcohol, drug, and poly-substance use disorders. Drug Alcohol Depend 174:128–136. 10.1016/j.drugalcdep.2017.01.021 [PubMed: 28324815]

38. Higgins ST, Kurti AN, Redner R et al. (2015) A literature review on prevalence of gender differences and intersections with other vulnerabilities to tobacco use in the United States, 2004–2014. Prev Med 80:89–100. 10.1016/j.ypmed.2015.06.009 [PubMed: 26123717]

39. Hetzel-Riggin MD, Roby RP (2013) Trauma type and gender effects on PTSD, general distress, and peritraumatic dissociation. J Loss Trauma 18:41–53. 10.1080/15325024.2012.679119

40. Kimerling R, Weitlauf JC, Street AE (2021) Gender issues in PTSD. In: Handbook of PTSD: Science and practice, 3rd edn. The Guilford Press, New York, NY, US, pp 229–245

41. Olff M (2017) Sex and gender differences in post-traumatic stress disorder: an update. Eur J Psychotraumatol 8:1351204. 10.1080/20008198.2017.1351204

42. George G, Webb EK, Harnett N (2023) Neighborhood-level factors in the development and treatment of trauma and stress-related disorders. Curr Treat Options Psych 10:181–198. 10.1007/s40501-023-00300-x

43. Brown JB, Rosengart MR, Billiar TR et al. (2017) Distance matters: effect of geographic trauma system resource organization on fatal motor vehicle collisions. J Trauma Acute Care Surg 83:111–118. 10.1097/TA.0000000000001508 [PubMed: 28422905]

44. Waller AE, Baker SP, Szocka A (1989) Childhood injury deaths: national analysis and geographic variations. Am J Public Health 79:310–315. 10.2105/AJPH.79.3.310 [PubMed: 2916717]

45. Assari S (2020) Family socioeconomic status and exposure to childhood trauma: racial differences. Children 7:57. 10.3390/children7060057 [PubMed: 32503310]

46. Mock S, Arai S (2011) Childhood trauma and chronic illness in adulthood: mental health and socioeconomic status as explanatory factors and buffers. Front Psychol. 10.3389/fpsyg.2010.00246

47. Hughes K, Bellis MA, Hardcastle KA et al. (2017) The effect of multiple adverse childhood experiences on health: a systematic review and meta-analysis. Lancet Public Health 2:e356–e366. 10.1016/S2468-2667(17)30118-4 [PubMed: 29253477]

48. Sacchi L, Merzhvynska M, Augsburger M (2020) Effects of cumulative trauma load on long-term trajectories of life satisfaction and health in a population-based study. BMC Public Health 20:1612. 10.1186/s12889-020-09663-9 [PubMed: 33109171]
49. Bender SW (2013) Joint reform: the interplay of state, federal, and hemispheric regulation of recreational marijuana and the failed war on drugs. Alb Gov't L Rev 6:359
50. Swinburne M, Hoke K (2019) State Efforts to Creative an inclusive Marijuana Industry in the Shadow of the Unjust War on Drugs. J Bus Tech L 15:235
51. Mueller D, Bacalso E, Ortega-Williams A et al. (2021) A mutual process of healing self and healing the community: a qualitative study of coping with and healing from stress, adversity, and trauma among diverse residents of a midwestern city. J Community Psychol 49:1169–1194. 10.1002/jcop.22530 [PubMed: 33634881]
52. Opara I, Lardier DT, Metzger I et al. (2020) "Bullets Have no Names": a qualitative exploration of community trauma among black and Latinx youth. J Child Fam Stud 29:2117–2129. 10.1007/s10826-020-01764-8 [PubMed: 34475729]
53. Rich JA, Grey CM (2005) Pathways to recurrent trauma among young black men: traumatic stress, substance use, and the "Code of the Street." Am J Public Health 95:816–824. 10.2105/AJPH.2004.044560 [PubMed: 15855457]
54. McLaughlin KA, Alvarez K, Fillbrunn M et al. (2019) Racial/ethnic variation in trauma-related psychopathology in the United States: a population-based study. Psychol Med 49:2215–2226. 10.1017/S0033291718003082 [PubMed: 30378513]
55. Roberts AL, Gilman SE, Breslau J et al. (2011) 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 41:71–83. 10.1017/S0033291710000401 [PubMed: 20346193]
56. Kirkinis K, Pieterse AL, Martin C et al. (2021) Racism, racial discrimination, and trauma: a systematic review of the social science literature. Ethn Health 26:392–412. 10.1080/13557858.2018.1514453 [PubMed: 30165756]
57. Hatch SL, Dohrenwend BP (2007) Distribution of traumatic and other stressful life events by race/ethnicity, gender, SES and age: a review of the research. Am J Community Psychol 40:313–332. 10.1007/s10464-007-9134-z [PubMed: 17906927]
58. Lipsky S, Kernic MA, Qiu Q, Hasin DS (2016) Traumatic events associated with posttraumatic stress disorder: the role of race/ethnicity and depression. Violence Against Women 22:1055–1074. 10.1177/1077801215617553 [PubMed: 26620827]
59. Latkin CA, Edwards C, Davey-Rothwell MA, Tobin KE (2017) The relationship between social desirability bias and self-reports of health, substance use, and social network factors among urban substance users in Baltimore, Maryland. Addict Behav 73:133–136. 10.1016/j.addbeh.2017.05.005 [PubMed: 28511097]

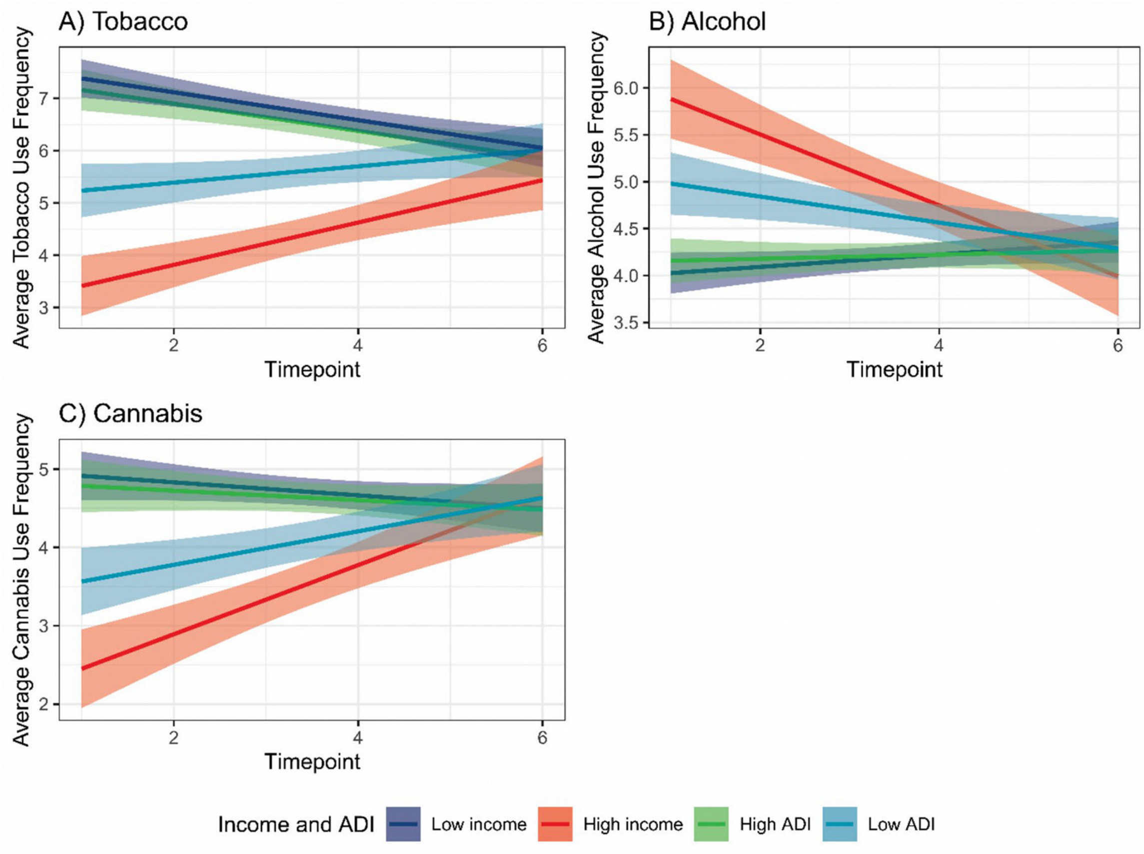

Fig. 1.

Substance use at six timepoints post-traumatic event, stratified by income and state-specific area deprivation index. "Low income" was determined as self-reported household income less than $50 k/year, "high income" was determined as self-reported household income greater or equal to $50 k/year, and "No report income" was determined as not reporting any household income. "High ADI" was determined as state-specific area deprivation index percentile greater than the 60th percentile, while "Low ADI" was determined as state-specific area deprivation index percentile less than or equal to the 60th percentile. Time 1 was equivalent to baseline emergency department visit, time 2 was 2 weeks post-traumatic event, time 3 was 8 weeks, time 4 was 3 months, time 5 was 6 months, and time 6 was 12 months

Table 1

Sociodemographic Information and Prevalent Substance Exposure Stratified By Likely PTSD Diagnosis at Emergency Department Recruitment Visit

	No PTSD N = 1844	Likely PTSD N = 1099	Overall N = 2943	P-value
Age-Mean (SD)	34.8 (13.1)	37.7 (13.5)	34.9 (11.3)	< 0.001
Median (Min,Max)	31.5 (18.0, 74.0)	34.0 (18.0, 73.0)	32 (18, 74)	
Race/Ethnicity				
Hispanic	207 (11.2%)	135 (12.3%)	342 (11.6%)	< 0.001
Non-Hispanic Black	969 (52.5%)	489 (44.5%)	1458 (49.5%)	
Non-Hispanic Other	67 (3.6%)	44 (4.0%)	111 (3.8%)	
Non-Hispanic White	593 (32.2%)	427 (38.9%)	1020 (34.7%)	
Missing	8 (0.4%)	4 (0.4%)	12 (0.4%)	
Gender Identity				< 0.001
Male	795 (43.1%)	325 (29.6%)	1120 (38.1%)	
Female	1043 (56.6%)	772 (70.2%)	1815 (61.7%)	
Transgender	2 (0.1%)	2 (0.2%)	4 (0.1%)	
None	3 (0.2%)	0 (0%)	3 (0.1%)	
Missing	1 (0.1%)	0 (0%)	1 (0.0%)	
Marital status				0.002
Divorced	252 (13.7%)	198 (18.0%)	450 (15.3%)	
Married	375 (20.3%)	233 (21.2%)	608 (20.7%)	
Never married	1175 (63.7%)	636 (57.9%)	1811 (61.5%)	
Widowed	30 (1.6%)	27 (2.5%)	57 (1.9%)	
Missing	12 (0.7%)	5 (0.5%)	17 (0.6%)	
Education				< 0.001
No HS	11 (0.6%)	4 (0.4%)	15 (0.5%)	
Some/Finished HS	1243 (67.4%)	661 (60.1%)	1904 (64.7%)	
Some/Finished College	476 (25.8%)	331 (30.1%)	807 (27.4%)	
Grad	108 (5.9%)	100 (9.1%)	208 (7.1%)	
Missing	6 (0.3%)	3 (0.3%)	9 (0.3%)	
Household income				< 0.001
≤ $35 k/year	986 (53.5%)	658 (59.9%)	1644 (55.9%)	

	No PTSD N = 1844	Likely PTSD N = 1099	Overall N = 2943	P-value
> $35 k and < $75 k/year	349 (18.9%)	220 (20.0%)	569 (19.3%)	
≤ $75 k/year	224 (12.1%)	144 (13.1%)	368 (12.5%)	
Did not report	285 (15.5%)	77 (7.0%)	362 (12.3%)	
ADI rank[1] (National, percentile)				0.13
Mean (SD)	64.7 (27.8)	63.1 (27.7)	64.1 (27.8)	
Median (range)	67 (2, 100)	65 (2, 100)	66 (2, 100)	
Missing	71 (3.9%)	25 (2.3%)	96 (3.3%)	
ADI rank[1] (state, decile)				
Mean (SD)	6.84 (2.97)	6.69 (2.94)	6.79 (2.96)	0.19
Median (range)	8 (1, 10)	7 (1, 10)	8 (1, 10)	
Missing	71 (3.9%)	25 (2.3%)	96 (3.3%)	
Traumatic events				0.84
Assault	174 (9.5%)	113 (10.3%)	288 (9.8%)	
Collision	1414 (76.7%)	833 (75.8%)	2247 (76.4%)	
Fall	136 (7.4%)	77 (7.0%)	213 (7.2%)	
Other	118 (6.4%)	75 (6.8%)	193 (6.6%)	
Missing	1 (0.1%)	1 (0.1%)	2(0.1%)	
Tobacco use				0.06
None	1205 (65.3%)	679 (61.8%)	1884 (64.0%)	
Any	632 (34.3%)	414 (37.7%)	1046 (35.5%)	
Missing	7 (0.4%)	6 (0.5%)	13 (0.4%)	
Alcohol use				0.10
None	721 (39.1%)	395 (35.9%)	1116 (37.9%)	
Any	1116 (60.5%)	699 (63.6%)	1815 (61.7%)	
Missing	7 (0.4%)	5 (0.5%)	12 (0.4%)	
Cannabis use				0.90
None	1290 (70.0%)	774 (70.4%)	2064 (70.1%)	
Any	539 (29.2%)	319 (29.0%)	858 (29.2%)	
Missing	15 (0.8%)	6 (0.5%)	21 (0.7%)	

P-values were determined by Student's T-test for age and area deprivation index. Likely PTSD was defined as having a PTSD Checklist for the DSM-5 (PCL-5) score of 33 and above, based on the validated threshold for US adults [29]. Unlikely PTSD was defined as having a PCL-5 score of less than 33, with range from 0 to 80

[1] ADI: Area deprivation index, as reported in 2019 by census tract

Table 2

Poisson mixed effect models of substance use, clustered by state with ranking by area deprivation index using cross-sectional modeling at six timepoints using mixed effect models

	Tobacco		Alcohol		Cannabis	
	Incidence Rate Ratio (95% CI)	**P-Value**	**Incidence Rate Ratio (95% CI)**	**P-Value**	**Incidence Rate Ratio (95% CI)**	**P-Value**
PTSD Checklist-5 Symptoms	1.003 (1.002, 1.003)	< 0.001	1.003 (1.002, 1.003)	< 0.001	1.001 (1, 1.002)	0.004
Time	1.004 (0.996, 1.011)	0.34	1.02 (1.02, 1.03)	< 0.001	1.02 (1.01, 1.02)	< 0.001
Marital status						
Never married	1.08 (0.98, 1.19)	0.11	1.05 (1, 1.11)	0.04	1.19 (1.1, 1.28)	< 0.001
Divorced	1.18 (1.06, 1.31)	0.003	0.99 (0.94, 1.05)	0.85	1.13 (1.03, 1.23)	0.007
Widowed/other	1 (0.79, 1.27)	0.996	1.07 (0.95, 1.22)	0.26	1.11 (0.92, 1.34)	0.29
Gender						
Cisgender men	1.35 (1.26, 1.44)	< 0.001	1.14 (1.1, 1.18)	< 0.001	1.26 (1.19, 1.33)	< 0.001
Transgender	1.63 (0.84, 3.16)	0.15	0.89 (0.63, 1.27)	0.53	2.17 (1.23, 3.83)	0.008
Race/ethnicity						
Hispanic	0.84 (0.51, 1.38)	0.49	0.9 (0.68, 1.17)	0.42	0.79 (0.52, 1.2)	0.27
Non-Hispanic Black	0.91 (0.55, 1.49)	0.71	0.92 (0.7, 1.2)	0.53	0.82 (0.54, 1.25)	0.36
Non-Hispanic Other	0.86 (0.51, 1.45)	0.57	0.85 (0.64, 1.13)	0.26	0.74 (0.48, 1.14)	0.17
Non-Hispanic White	1.08 (0.66, 1.77)	0.76	0.97 (0.74, 1.26)	0.81	0.84 (0.55, 1.27)	0.41
Age	0.999 (0.996, 1.002)	0.55	1 (0.999, 1.002)	0.92	0.992 (0.989, 0.994)	< 0.001
Income						
≤ $35 k	1.26 (1.16, 1.37)	< 0.001	0.97 (0.92, 1.01)	0.13	1.09 (1.02, 1.17)	0.01
≥ $75 k	0.86 (0.76, 0.96)	0.008	1.16 (1.08, 1.23)	< 0.001	0.95 (0.87, 1.05)	0.33
Did not report	1.16 (1.04, 1.31)	0.01	1.05 (0.99, 1.12)	0.13	1.13 (1.02, 1.24)	0.02
ADI State Ranking[1]	1.02 (1.01, 1.03)	< 0.001	0.994 (0.989, 0.999)	0.01	1.01 (1, 1.02)	0.03

Poisson mixed effect models allowed for clustering at the state level with an exchangeable correlation structure. “Cross-sectional” used here refers to post-traumatic stress symptoms (PTS) collected at a given timepoint regressed on tobacco, alcohol, and cannabis use at the same timepoint without any lag between independent and dependent variable for baseline, week 2, week 8, month 3, month 6, and month 12 following the index traumatic event. Time was coded 0 to 5 as a continuous scale based on data collection wave

[1] Area Deprivation Index (ADI) was reported as deciles within each state, with higher ADI relating to higher deprivation (less resources), and lower score relating to lower deprivation (more resources)

Table 3

Stratified analysis of PCL-5 measure of PTS symptoms and substance use behaviors among high and low income participants, estimated cross-sectionally and prospectively

Individual income stratification analyses[1]					
Outcome[2]	Low income		High income		*P*-value for the interaction term
	Change in frequency (95% CI)[4]	*P*-value	Change in frequency (95% CI)	*P*-value	
Cross-sectional					
Tobacco					
PCL-5[3]	0.03 (0.01, 0.04)	< 0.001	0.02 (0.001, 0.04)	0.04	0.50
Alcohol					
PCL-5	0.03 (0.02, 0.03)	< 0.001	0.02 (0.00, 0.03)	0.051	0.26
Cannabis					
PCL-5	0.02 (0.004, 0.03)	0.008	0.01 (−0.01, 0.03)	0.41	0.24
Prospective					
Tobacco					
PCL-5	0.02 (0.001, 0.04)	0.04	0.01 (−0.02, 0.04)	0.40	0.58
Alcohol					
PCL-5	0.01 (0.003, 0.02)	0.01	0.007 (−0.01, 0.03)	0.45	0.69
Cannabis					
PCL-5	0.01 (−0.005, 0.03)	0.16	0.01 (−0.02, 0.03)	0.50	0.60

GEE models were adjusted for all sociodemographic covariates: age, gender, race/ethnicity, education and marital status. The “change in frequency” refers to the interpretation of the Beta coefficient in these models, and the p-values correspond to the Wald test statistic for this Beta coefficient, comparing no change vs. a 1-point increase in the PCL-5. “Cross-sectional” used here refers to post-traumatic stress symptoms (PTS) collected at a given timepoint regressed on tobacco, alcohol, and cannabis use at the same timepoint without any lag between independent and dependent variable for baseline, week 2, week 8, month 3, month 6, and month 12 following the index traumatic event. “Prospective” refers to a lag between PTS symptoms (week 2, week 8, month 3, month 6) and tobacco, alcohol, and cannabis outcomes (week 8, month 3, month 6, month 12). The baseline timepoint was not included in this model as that may also reflect PTS symptoms before the index traumatic event

The interaction term refers to the model with the interaction term between the household income variable and the PCL-5 scores (e.g., income * PTS symptoms) as a direct test of a potential interaction effect

[1]“Low income” was defined as an annual household income of ≤ $50,000 per year, and “high income” was defined as an annual household income of > $50,000 per year

[2]Substance use was measured as using a substance in the past 30 days at each of the 6 timepoints, as estimated by linear generalized estimating equation (GEE) to account for the longitudinal data with exchangeable correlation structure

[3]PCL-5: Post-Traumatic Stress Disorder PTSD) Checklist - 5, computed as a raw sum of all items and scaled to increments of 10 (e.g., the raw score divided by 10)

Table 4

Stratified analysis of PCL-5 measure of PTS symptoms and substance use behaviors among high and low ADI participants, estimated cross-sectionally and prospectively

Area deprivation index stratification analyses[1]					P-value of the interaction term
Outcome[2]	High ADI (high deprivation)		Low ADI (low deprivation)		
	Change in Frequency (95% CI)	P-value	Change in Frequency (95% CI)	P-value	
Cross-sectional					
Tobacco					
PCL-5[3]	0.02 (0.01, 0.04)	0.001	0.03 (0.01, 0.04)	0.005	0.97
Alcohol					
PCL-5	0.02 (0.01, 0.03)	< 0.001	0.02 (0.01, 0.04)	0.003	0.84
Cannabis					
PCL-5	0.01 (−0.002, 0.02)	0.11	0.02 (0.003, 0.04)	0.03	0.18
Prospective					
Tobacco					
PCL-5	0.02 (−0.01, 0.05)	0.12	0.02 (−0.002, 0.04)	0.08	0.82
Alcohol					
PCL-5	0.01 (0.00, 0.02)	0.06	0.01 (−0.003, 0.03)	0.10	0.54
Cannabis					
PCL-5	0.01 (−0.01, 0.03)	0.29	0.01 (−0.03, 0.05)	0.55	0.55

GEE models were adjusted for all sociodemographic covariates: age, gender, race/ethnicity, education and marital status. “Cross-sectional” used here refers to post-traumatic stress symptoms (PTS) collected at a given timepoint regressed on tobacco, alcohol, and cannabis use at the same timepoint without any lag between independent and dependent variable for baseline, week 2, week 8, month 3, month 6, and month 12 following the index traumatic event. “Prospective” refers to a lag between PTS symptoms (week 2, week 8, month 3, month 6) and tobacco, alcohol, and cannabis outcomes (week 8, month 3, month 6, month 12). The baseline timepoint was not included in this model as that may also reflect PTS symptoms before the index traumatic event

[1] ADI: Area deprivation index, defined by national percentile ranking and binarized as “high ADI” as less than the 50th percentile, and “low ADI” as greater than the 50th percentile. A sensitivity analysis with the 25th and 75th percentile cutoffs were used (e.g., omitting the middle 50% of data) but that analysis was underpowered as a continuous outcome, and the binarized logistic regression is reported in supplement

[2] Substance use was measured as using a substance in the past 30 days at each of the 6 timepoints, as estimated by linear generalized estimating equation (GEE) to account for the longitudinal data with exchangeable correlation structure

[3] PCL-5: Post-Traumatic Stress Disorder PTSD) Checklist – 5, computed as a raw sum of all items and scaled to increments of 10 (e.g., the raw score divided by 10)