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Risk Pathways among Traumatic Stress, Posttraumatic Stress Disorder Symptoms, and Alcohol and Drug Problems: A Test of Four Hypotheses

Moira Haller and Laurie Chassin

Arizona State University

Abstract

The present study utilized longitudinal data from a community sample ($n = 377$; 166 trauma-exposed; 54% males; 73% non-Hispanic Caucasian; 22% Hispanic; 5% other ethnicity) to test whether pre-trauma substance use problems increase risk for trauma exposure (high-risk hypothesis) or PTSD symptoms (susceptibility hypothesis), whether PTSD symptoms increase risk for later alcohol/drug problems (self-medication hypothesis), and whether the association between PTSD symptoms and alcohol/drug problems is due to shared risk factors (shared vulnerability hypothesis). Logistic and negative binomial regressions were performed in a path analysis framework. Results provided the strongest support for the self-medication hypothesis, such that PTSD symptoms predicted higher levels of later alcohol and drug problems, over and above the influences of pre-trauma family risk factors, pre-trauma substance use problems, trauma exposure, and demographic variables. Results partially supported the high-risk hypothesis, such that adolescent substance use problems had a marginally significant unique effect on risk for assaultive violence exposure but not on overall risk for trauma exposure. There was no support for the susceptibility hypothesis. Finally, there was little support for the shared vulnerability hypothesis. Neither trauma exposure nor preexisting family adversity accounted for the link between PTSD symptoms and later substance use problems. Rather, PTSD symptoms mediated the effect of pre-trauma family adversity on later alcohol and drug problems, thereby supporting the self-medication hypothesis. These findings make important contributions to better understanding the directions of influence among traumatic stress, PTSD symptoms, and substance use problems.

Keywords

posttraumatic stress disorder; substance use disorders; comorbidity; self-medication

Exposure to traumatic events is surprisingly common. Approximately 61% of men and 51% of women experience at least one traumatic event during their lifetimes (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Traumatic events may lead to the development of not only posttraumatic stress disorder (PTSD), but also alcohol and drug problems. Rates of

PTSD among adults with substance use disorders (SUDs) range from 14 to 60 percent (see Hien, Cohen, & Campbell, 2005). Studies of adolescents with SUDs indicate rates of PTSD ranging up to 20 percent (Deykin & Buka, 1997). Such high rates of comorbidity suggest that traumatic stress and trauma-related symptomatology may play a role in the etiology of SUDs or vice versa.

Several pathways may underlie the link between PTSD and SUDs. First, the “high-risk hypothesis” states that substance use or abuse may increase risk for exposure to a traumatic event by placing individuals in high risk situations (e.g., Windle, 1994) or by impairing detection of danger cues in the environment (Davis, Stoner, Norris, George, & Masters, 2009). Second, the “susceptibility hypothesis” states that substance use or abuse may increase risk for developing PTSD among individuals who are exposed to a traumatic event (Chilcoat & Breslau, 1998a). For instance, substance use problems may interfere with the ability to effectively manage negative emotions resulting from the traumatic event, may increase anxiety and arousal levels due to substance withdrawal symptoms (Stewart, Pihl, Conrod, & Doniger, 1998), or may facilitate avoidance and lack of processing of trauma material (Kaysen et al., 2011). Third, the “self-medication hypothesis” states that individuals may use substances in order to cope with symptoms of posttraumatic stress (e.g., Chilcoat & Breslau, 1998b; Reed et al., 2007). Finally, the “shared vulnerability hypothesis” (Stewart & Conrod, 2003) states that shared risk factors may account for both PTSD and alcohol/drug problems, such that PTSD and SUDs are not causally related once shared risk factors are taken into account.

Although these hypotheses are presented separately, they are not mutually exclusive and may be integrated into a larger, developmental model of PTSD-SUD comorbidity. For instance, pre-existing adolescent substance use problems may not only increase individuals’ risk for trauma exposure (high-risk hypothesis), but may also make it more likely that they will turn to alcohol/drugs to cope with subsequent PTSD symptoms (self-medication hypothesis); this increased substance use may further exacerbate PTSD symptoms. The present study examines evidence for each of the four pathways that have been proposed to underlie PTSD-SUD comorbidity.

The High-Risk and Susceptibility Hypotheses

The high-risk and susceptibility hypotheses both suggest that SUDs causally influence risk for PTSD. Retrospective and prospective studies examining these hypotheses have had mixed findings. Some studies show that substance use problems increase risk for trauma exposure but not PTSD (Bromet, Sonnega, & Kessler, 1998; Kilpatrick, Acierno, Resnick, Saunders, & Best, 1997), others show that substance use problems increase risk for PTSD but not trauma exposure (Acierno et al., 1999), and others show that substance use problems do not increase risk for either trauma exposure or PTSD after controlling for other risk factors (Chilcoat & Breslau, 1998a). Moreover, studies examining onset patterns of PTSD and SUDs in adults tend not to support the high-risk or susceptibility hypotheses, instead indicating that PTSD more often precedes than follows SUD onset (see Stewart & Conrod, 2003). This lack of compelling empirical support has led some researchers to conclude that it is unlikely that substance use problems causally influence risk for trauma exposure or

PTSD, especially when considered among other risk factors (e.g., Chilcoat & Breslau, 1998a; Stewart & Conrod, 2003).

However, little prospective research has examined relations between *adolescent* substance use problems and risk for trauma exposure or PTSD. This limitation is important, given that risk for trauma exposure—particularly assaultive violence exposure—peaks between the ages of 16 and 20 (Breslau et al., 1998). It is currently unclear what role adolescent substance use problems may play in this heightened risk period for assaultive violence exposure. Because adolescent substance use problems may reflect a tendency to engage in particularly risky behavior, they may be more likely to result in trauma exposure or PTSD compared to adult substance use problems. Although retrospective data indicate that adolescents with SUDs are at two to five times the risk for experiencing a traumatic event (risk for exposure to an event involving violence is even higher), and at four to nine times the risk for developing PTSD (Deykin & Buka, 1997; Giaconia et al., 2000; Kilpatrick et al., 2000) than those without a SUD, prospective studies are needed to disentangle the direction of influence. In addition, studies that capture pre-trauma risk stemming from subclinical levels of substance use problems are also needed, given that adolescent substance use problems, even if not meeting diagnostic threshold, may create meaningful risk for trauma exposure, PTSD, and post-trauma substance use problems. The current study addresses these limitations by testing the prospective effects of pre-trauma adolescent substance use problems on risk for later trauma exposure (as well as risk for assaultive violence exposure, specifically) and PTSD symptoms.

The Self-Medication Hypothesis

In contrast to the conflicting results from investigations of the high-risk and susceptibility hypotheses, there is a strong body of evidence supporting the self-medication hypothesis (Breslau, Davis, Peterson, & Schultz, 1997; Breslau, Davis, & Schultz, 2003; Chilcoat & Breslau, 1998b; Reed et al., 2007; Shipherd, Stafford, & Tanner, 2005; Ullman, Filipas, Townsend, & Starzynski, 2005; see also Hien et al., 2005, for review). Indeed, in a review of retrospective and prospective studies on PTSD-SUD comorbidity, Stewart and Conrod (2003, p. 37) summarized that “PTSD has been shown to develop before the SUD in the large majority of comorbid cases in retrospective studies, and PTSD has been shown to increase risk for SUDs in prospective studies.” Theoretically, individuals with PTSD may use alcohol and drugs to reduce irritability, concentration problems, hyperarousal, etc., raising risk for the development of SUDs.

The self-medication hypothesis implies a mediating role for PTSD symptoms in the relation between trauma exposure and substance use problems (Stewart, 1996). Support for the mediating role of PTSD comes from studies demonstrating that individuals who develop PTSD appear to be at higher risk for SUDs than do individuals who are exposed to a traumatic event but do *not* develop PTSD (Chilcoat & Breslau, 1998b; Breslau et al., 1998). However, these studies do not examine the extent to which PTSD symptom *severity* influences risk for future substance use problems. To address this limitation, the current study simultaneously estimated the unique effects of both PTSD symptoms and trauma exposure on future substance use problems in order to test the extent to which PTSD

symptom severity influences risk for substance use problems, controlling for the effects of trauma exposure itself, as well as shared risk factors for PTSD and SUDs.

The Shared Vulnerability Hypothesis

The high prevalence of PTSD-SUD comorbidity suggests that PTSD and SUDs may share a common etiological diathesis, including both environmental and genetic factors. There are a number of family-related risk factors that trauma exposure, PTSD, and SUDs may share in common. For instance, parental psychopathology has been shown to increase risk for offspring trauma exposure (Bromet et al., 1998; Koenen et al., 2002), PTSD (Brewin, Andrews, & Valentine, 2000; Bromet et al., 1998), and SUDs (Zhou, King, & Chassin, 2006). Importantly, parental psychopathology is also associated with other familial risk factors, such as higher levels of family conflict and higher levels of stress (Dube et al., 2003), which may further increase risk for trauma exposure and post-trauma psychopathology (Brewin et al., 2000; Deykin & Buka, 1997; Koenen, Moffitt, Poulton, Martin, & Caspi, 2007). Research suggests that individuals who grow up in adverse family environments may be sensitized to the effects of future stressors (Koenen et al., 2007), thus placing them at risk for post-trauma maladjustment. Adolescents from adverse family environments may also be less likely to have the resources and supports necessary for effectively coping with a traumatic event. Therefore, the familial backdrop against which trauma occurs is likely to be a key determinant of post-trauma functioning. Yet, the extent to which pre-trauma adversity in the family environment is a shared risk factor for PTSD and later substance use problems is currently unclear because so few studies contain pre-trauma measures of family risk factors. The present study thus examines how pre-trauma family adversity may influence risk for PTSD symptoms and/or substance use problems in order to better understand the role of family risk factors in PTSD-SUD comorbidity.

In addition to directly increasing risk for PTSD and substance use problems, it is also possible that adolescent family adversity may indirectly influence risk for post-trauma substance use problems by increasing risk for PTSD symptoms (i.e., PTSD symptoms may mediate the influence of preexisting family adversity on later alcohol and drug problems). Although finding that PTSD symptoms mediate the influence of preexisting family adversity on later alcohol and drug problems would support the self-medication hypothesis rather than the shared vulnerability hypothesis, such findings would nonetheless highlight preexisting family adversity as an important developmental antecedent in the PTSD-SUD link.

In order to test whether family adversity during adolescence accounts for the link between PTSD symptoms and post-trauma substance use problems (i.e., the shared vulnerability hypothesis), it is important to control for preexisting substance use problems that may have already been present and that co-occurred with adolescent family adversity. Indeed, adolescents who grow up in adverse (i.e., high-conflict, high-stress) family environments are also more likely to misuse alcohol and drugs (e.g., Repetti, Taylor, & Seeman, 2002; Zhou et al., 2006), regardless of trauma exposure or PTSD. Thus, in order to disentangle the directions of influence among traumatic stress, PTSD, and problematic substance use, both preexisting adolescent substance use problems and the confounding influence of the larger constellation of family adversity must be accounted for.

Trauma exposure itself may be conceptualized as a shared environmental risk factor for PTSD and SUD (Yehuda, McFarlane, & Shalev, 1998). Although traumatic events are most often associated with PTSD, they may also precipitate SUDs independent of their effects on PTSD, such that PTSD-SUD comorbidity reflects the co-occurrence of distinct diatheses. For instance, individuals who are predisposed to biological hyper-responsiveness may experience further sensitizations in their stress response systems following trauma exposure and may thus develop PTSD (Yehuda et al., 1998), whereas individuals with other predispositions may experience a range of other stress responses that lead to other disorders, such as SUDs. If this hypothesis were true, traumatic stress would be expected to directly predict substance use problems, separate from its influence on PTSD. Alternatively, a direct effect of traumatic stress on problematic alcohol/drug use (separate from the effects of PTSD) would not be expected if other common risk factors account for the link between PTSD symptoms and alcohol/drug problems. Previous tests of these hypotheses show that trauma-exposed individuals who do not develop PTSD are *not* at increased risk for subsequent onset of SUDs, but those who develop PTSD are (Breslau et al., 2003; Chilcoat & Breslau, 1998b; Reed et al., 2007; see Fetzner, McMillan, Sareen, & Asmundson, 2011 for an exception). However, the “trauma exposure as a shared risk factor” hypothesis has not yet been tested using analytic strategies other than comparing risk for onset of clinical SUDs among individuals with PTSD to individuals with trauma exposure who do not have PTSD. Therefore, the current study tested the effect of trauma exposure on later substance use problems, while controlling for a count of the number of PTSD symptoms that participants endorsed.

The Present Study

The purpose of the present study was to better understand the risk pathways that link trauma exposure, PTSD, and alcohol and drug problems. Alcohol and drug problems were examined as separate outcomes based on previous studies that have found that traumatic stress and PTSD symptoms may have differential relations with alcohol versus drugs (e.g., Breslau et al., 2003; Haller & Chassin, 2012; Shipherd et al., 2005). Specifically, this study tested the following hypotheses (see Figure 1), which are not mutually exclusive:

1. *High-risk hypothesis:* Do adolescent substance use problems increase risk for trauma exposure or assaultive violence exposure over and above the influence of preexisting family risk factors and demographic predictors?
2. *Susceptibility hypothesis:* Do adolescent substance use problems increase risk for PTSD symptoms among individuals exposed to trauma over and above the influence of preexisting family risk factors and demographic predictors?
3. *Self-medication hypothesis:* Do PTSD symptoms increase risk for future alcohol and/or drug problems over and above the influences of trauma exposure itself, pre-trauma substance use problems, demographic predictors, and preexisting family risk factors that are common to both PTSD and alcohol/drug problems?
4. *Shared vulnerability hypothesis:* Do trauma exposure and/or adversity in the family environment account for the link between and substance use problems, such that PTSD symptoms and substance use problems are not related when these potential

shared risk factors are accounted for? By testing this hypothesis, this study also addresses a series of related questions: To what extent does adversity in the family environment increase risk for both PTSD and alcohol/drug problems? To what extent is the influence of preexisting family risk factors on adult alcohol and drug problems mediated by PTSD symptoms? Is trauma exposure a shared risk factor for both PTSD and substance use problems, such that trauma exposure increases risk for future alcohol or drug problems, independent of PTSD symptoms?¹

These hypotheses were tested using data from a longitudinal, community-based study of familial alcoholism, which is important given that most research on the overlap between PTSD and substance use problems consists of cross-sectional, retrospective, and clinic-based studies without preexisting measures of substance use and associated family risk factors. By using a high-risk sample with elevated prevalence of risk factors, trauma, and substance use problems, the present study was particularly well-suited for examining the hypothesized pathways.

Method

Participants

Participants ($N = 377$) were from a larger longitudinal study of familial alcoholism (Chassin, Barrera, Bech, and Kossak Fuller, 1992). The original study had three annual waves of data collection and three additional follow-ups separated by five years. The present study used data from Waves 1 (1988), 4 (≈ 1995), and 5 (≈ 2000). Both parents and adolescents were interviewed at each time point. At Wave 1, there were 454 “target” adolescents between the ages of 11 to 15 and their parents; 246 adolescents had at least one biological parent with an alcohol disorder who was also a custodial parent, and the remaining 208 adolescents were demographically-matched controls without any biological or custodial parents with an alcohol disorder. Sample retention ranged from 90 to 99% across waves.

Participants reported their history of trauma exposure and PTSD at Wave 4 (seven to ten years after the initial assessment). Forty-seven participants who were not interviewed at Wave 4 were excluded from the present study. Thirty participants who reported trauma exposure prior to Wave 1 were also excluded so that Wave 1 measures preceded trauma exposure for all participants. Thus, our final sample consisted of 377 participants (54% male; 52% children of parents with an alcohol disorder; 73% non-Hispanic Caucasian; 22% Hispanic, 5% other ethnicity; mean age=13.2 at Wave 1, 20.4 at Wave 4, and 25.6 at Wave 5).

Analyses examining differences between the 377 participants included in this study and the 77 excluded participants showed that excluded participants were more likely to be children of alcoholic parents ($\chi^2(1) = 6.65, p = .01$). However, included and excluded participants did not differ in gender, ethnicity, parental psychopathology other than alcoholism, family

¹Trauma exposure is by definition a risk factor for PTSD symptoms, but this study clarifies whether trauma exposure may also directly increase risk for future substance use problems.

conflict, family life stress, adolescent substance use problems, or adult alcohol or adult drug problems.

Recruitment and Procedure

Families with parental alcoholism were recruited using court DUI records, questionnaires from newly enrolled members of a large HMO, and community telephone surveys. Matched non-alcoholic families (matched on child's age, family composition, ethnicity, and SES) living in the same neighborhoods as the families with parental alcoholism were recruited via telephone surveys. Potential participants who were successfully contacted did not differ from those who were not contacted on available alcoholism indicators (e.g., blood alcohol level at time of arrest, number of prior alcohol-related arrests). See Chassin et al. (1992) for details. After parents provided informed consent and adolescents provided assent, interviews were conducted in person with computer-assisted interviews (parents and adolescents were interviewed in separate rooms), or via telephone (after verifying private conditions) for out-of-state families. All protocols were approved by the Arizona State University Institutional Review Board.

Measures

Adolescent substance use problems²—At Wave 1, adolescents reported on 14 (Cronbach's $\alpha = .86$) lifetime problems (e.g., receipt of complaints from family or friends) that they may have experienced due to alcohol or drug use. Items were adapted from Sher's (1987) questionnaire assessing substance use problems among college students. Given the low frequency of adolescents with high counts of substance use problems (only 42 percent of adolescents reported having ever used alcohol or drugs), analyses used a variable that was coded 0 if the adolescent reported no lifetime substance use problems ($n = 317$; 84.1%), 1 if the adolescent reported one lifetime substance use problem ($n = 23$; 6.1%), and 2 if the adolescent reported two or more lifetime substance use problems ($n = 37$; 9.8%).

Adolescent's family adversity—At Wave 1, family adversity was measured via a cluster of related family variables (family conflict, family stress, parental alcoholism, and other parent psychopathology) associated with trauma exposure, PTSD, and SUDs. These variables are likely to reflect a combination of both genetic and environmental risk. Correlations among the four family factors were all significant (see Table 1; $ps < .001$). To avoid multicollinearity problems, analyses employed a composite "family adversity" variable derived using factor scores (mean = 0.00; $SD = 0.84$, range: -1.91 to 2.36) from a one-factor confirmatory factor analysis. The family adversity factor score was significantly associated with trauma exposure, PTSD symptoms, adult alcohol problems, and adult drug problems (see Table 1). Thus, this variable appeared to appropriately capture shared risk for these outcomes.

²Analyses modeled the effects of substance use *problems*, rather than substance use itself, because problems were expected to be more prognostic of future risk for trauma exposure, PTSD, and adult substance use problems. That is, adolescents who were using substances to such an extent that they were already experiencing abuse or dependence symptoms were theorized to exhibit a high-risk substance use style that may place them at risk for trauma, PTSD, and/or substance use problems. Moreover, modeling the effects of substance use problems allowed us to be longitudinally consistent when predicting adult substance use problems.

Both family conflict and familial life stress were measured via adolescent, mother, and father reports. Family conflict was a composite of all reports, each measured via four items (e.g., “we fought a lot in our family”) from Bloom’s (1985) Family Process Scale with responses ranging from *Strongly Disagree* to *Strongly Agree* (mean = 2.74; *SD* = 0.60; range: 1.33–4.38; Cronbach’s alphas were .62, .63, and .63 for adolescent, mother, and father report). Familial life stress was a count of 15 independent events (e.g., serious money troubles; mean = 3.20; *SD* = 2.36; range: 0–11) from the General Stressful Life Events Schedule for Children (Sandler, Ramirez, & Reynolds, 1986) and the Children of Alcoholics Stressful Life Events Schedule (Roosa, Sandler, Gehring, Beals, & Cappel, 1988). Parent alcoholism (DSM-III abuse or dependence) was measured via parents’ self-reports on the Computerized Diagnostic Interview Schedule (CDIS-III; Robins, Helzer, Croughan, & Ratcliff, 1981), or via spousal report for non-interviewed parents using Family History-Research Diagnostic Criteria, Version 3 (Endicott, Andreason, & Spitzer, 1975; 51.5% of adolescents had at least one biological parent with an alcohol disorder who was also a custodial parent). Other parent psychopathology (DSM-III affective, anxiety, or antisocial personality disorder) was measured via parents’ self-reports on the CDIS-III (39.5% of adolescents had a parent with one of these disorders).

Late adolescent/early-adult trauma exposure and PTSD symptoms—At Wave 4, the computerized Diagnostic Interview Schedule (CDIS-III-R; Robins, Helzer, Cottler, & Golding, 1989) was used to assess participants’ lifetime exposure to trauma and PTSD symptoms using DSM-III-R criteria. Participants reported on up to three traumatic events and 17 PTSD symptoms (118 [71%] participants reported one event; 33 [9%] participants reported two events, and 15 [4%] participants reported three events). On average, less than 3 years (mean = 2.65, *SD* = 1.70) elapsed between the time of the traumatic event and the assessment of PTSD. Among trauma-exposed participants (*n* = 166; 44%; mean age at exposure = 17.3 years.), 72 (43%) experienced at least one event involving assaultive violence (rape, physical assault or being threatened with a weapon), whereas 94 (57%) experienced other types of events (seeing someone hurt or killed, natural disaster, narrow escape from death/injury, sudden injury/accident, sudden death/injury of someone close, experiencing shock from other’s experience, or other event). See Haller and Chassin (2012) for rates of each type of event. Analyses employed a dichotomous measure of trauma exposure, and a count variable that indicated the total number of PTSD symptoms for whichever event produced the highest number of symptoms (mean = 5.41 symptoms, *SD* = 4.11). Thirty-one participants (19% of trauma-exposed) met criteria for PTSD.

Adult alcohol and drug problems—At Wave 5³, participants reported on 17 problems (e.g., failed attempts to cut down) due to alcohol and drug use (note that three substance use problems were assessed at Wave 5 that were not assessed during adolescence). Follow-up questions assessed the recency of each problem separately for alcohol and drugs.⁴ Analyses employed count variables indicating the total number of adult alcohol problems and drug problems (separately) experienced in the past two years at Wave 5. The two-year timeframe

³This study examined risk for substance use problems at Wave 5 rather than Wave 4 (when trauma/PTSD was assessed) to provide a prospective test of PTSD symptoms on future substance use problems. Analyses controlled for Wave 1 (pre-trauma) substance use problems when examining risk for Wave 5 substance use problems.

allowed for prospective prediction of adult alcohol and drug problems from PTSD symptoms. Cronbach's α was .85 for alcohol and .91 for drugs. Participants who drank at Wave 5 (82%) reported drinking on average more than "5 times in the past year" but less than "1–3 times a month." Twenty-nine percent of participants reported using drugs at Wave 5, with marijuana being the most commonly used drug. At Wave 5, 44% of interviewed participants experienced at least one alcohol problem in the past two years (mean = 1.54, SD = 2.58, range: 0–13), and 19% experienced at least one drug problem in the past two years (mean = 0.83, SD = 2.33, range: 0–13).

Data Analytic Strategy

All analyses were conducted in MPlus version 6.11 (Muthén & Muthén, 1998–2011). Models were estimated using the maximum likelihood estimator with robust standard errors (MLR) in order to ensure robustness against heteroscedasticity, non-normality, and model misspecification. Full information maximum likelihood estimation was used to account for missing data for 29 participants who were not interviewed at Wave 5.⁵

Figure 1 presents a simplified depiction of model estimation and those paths that are relevant to hypothesis testing. Adult alcohol problems and adult drug problems were examined in separate models. Each model included three endogenous variables: trauma exposure (binary), PTSD symptoms (count variable), and adult alcohol/drug problems (count variables). Logistic regression was used to predict trauma exposure, and negative binomial regression⁶ was used to predict PTSD symptoms and adult alcohol/drug problems. Incidence rate ratios (*IRRs*) are presented for count outcomes (e.g., an *IRR* of 1.10 means that for every one unit increase in the predictor, there is a 10 percent increase in the dependent variable). Because PTSD symptoms are conditional upon trauma exposure, data were specified as missing on the count measure of PTSD symptoms for participants who were not exposed to a traumatic event (i.e., those coded 0 on the binary trauma exposure variable).⁷ Paths were specified from adolescent family adversity, adolescent substance use problems, gender (coded 0 for males and 1 for females), and ethnicity (coded 0 for non-Hispanic Caucasians and 1 for other ethnicities) to each endogenous variable. Paths were also specified from trauma exposure and PTSD symptoms to adult alcohol/drug problems. The residual covariance between trauma exposure and PTSD symptoms was estimated to

⁴The assessment of drug-related problems referred to drugs in general rather than a specific class of drugs ("how recently have you used a drug enough so that that you felt like you needed or depended on it?"). Before beginning these questions, the interviewer stated "When we ask you about drug use we do NOT mean medicines that were given to you by your doctor. We want to know about your use of drugs that were not prescribed by your doctor."

⁵Analyses were repeated after deleting the 29 participants who were missing data at Wave 5 ($n = 348$). Results were unchanged. All findings pertaining to hypothesis testing were identical to those presented below.

⁶Preliminary analyses tested whether Poisson, zero-inflated Poisson (ZIP), negative binomial, or zero-inflated negative binomial regression was the most appropriate method of model estimation for each count dependent variable. Results showed the best fit for the negative binomial models. However, in order to differentiate between risk for using alcohol/drugs and risk for developing alcohol/drug problems among those who use alcohol/drugs, follow-up analyses predicted alcohol and drug problems using ZIP regression. Results were consistent with those presented below. PTSD symptoms had significant unique effects on risk for alcohol problems among those who drink ($B = .10, p < .001, IRR: 1.11$) and risk for drug problems among those who use drugs ($B = .08, p = .001, IRR: 1.08$). However, PTSD symptoms did not significantly influence the probability of being a non-drinker ($B = -.02, p = .770, OR: .98$) or the probability of being a non-drug user ($B = -.08, p = .11, OR: .92$). Therefore, these analyses PTSD symptoms significantly increase risk for alcohol and drug problems among those who use alcohol and drugs.

⁷We did not believe it was advisable to assume that participants without trauma exposure had zero PTSD symptoms because it is feasible that non-trauma exposed individuals may have similar symptoms (e.g., sleep disturbances, irritability, feeling detached or estranged from others) that are not trauma-induced.

allow for the fact that they may share predictors other than those specified in the model. In addition to the primary models, a separate logistic regression was conducted in order to test whether adolescent substance use problems significantly increase risk for assaultive violence exposure (i.e., high-risk hypothesis), over and above family adversity, gender, and ethnicity.

Prior to conducting the main analyses, preliminary analyses tested for significant covariates (gender, ethnicity, parent education, age, age at trauma exposure, and time since trauma exposure), covariate by predictor interactions, and predictor by predictor interactions. Preliminary analyses indicated a significant effect of time since trauma exposure on risk for alcohol problems, an interaction between family adversity and gender when predicting risk for drug problems, an interaction between gender and ethnicity when predicting trauma exposure, and interactions between PTSD symptoms and ethnicity when predicting alcohol and drug problems. These effects were retained in the final models (as shown in Table 2). All other covariate effects and interactions were non-significant and were not further considered. However, gender and ethnicity were retained as covariates in all models, given numerous gender and ethnic differences in both the trauma/PTSD and SUDs literatures.

Results

Correlations among study variables are presented in Table 1. As expected, males were more likely to be exposed to a traumatic event than were females ($r = -.15, p = .003$), but trauma-exposed females exhibited higher levels of PTSD symptoms ($r = .35, p < .001$) than trauma-exposed males. Males exhibited higher levels of adult alcohol ($r = -.21, p < .001$) and drug ($r = -.12, p = .029$) problems than did females. Trauma-exposed participants were at significantly higher risk for adult alcohol problems ($r = .12, p = .024$) and at marginally higher risk for adult drug problems ($r = .09, p = .096$), than were participants who were not exposed to a traumatic event. Among trauma-exposed participants, PTSD symptoms were not significantly associated with adult alcohol problems ($r = .11, p = .198$) and were only marginally associated with adult drug problems ($r = .14, p = .089$). However, partial correlations revealed that after controlling for gender, there was a significant association between PTSD symptoms and both alcohol ($r = .22, p = .006$), and drug ($r = .21, p = .011$) problems.⁸ Gender was specified as a covariate in all analyses.

Table 2 presents results from the primary analyses. Results showed that the unique effect of adolescent substance use problems on risk for trauma exposure was non-significant (*high-risk hypothesis*; $B = 0.21, p = .33, OR = 1.23$), over and above pre-trauma family adversity, gender, and ethnicity. However, a separate logistic regression indicated that there was a small unique effect of adolescent substance use problems on risk for assaultive violence exposure ($B = 0.38, p = .051, OR = 1.46$; results not shown in table).⁹

⁸Males had more alcohol and drug problems than did females, whereas females had more PTSD symptoms than did males (see Table 1), thus obscuring the relation between PTSD symptoms and alcohol and drug problems.

⁹In order to examine the robustness of this finding, additional analyses modeled the effects of past-year frequency of binge drinking, getting drunk, and using marijuana, on risk for trauma exposure or assaultive violence exposure. Similar to the main analyses, neither binge drinking nor getting drunk predicted overall risk for trauma exposure. However, binge drinking had a significant unique effect on risk for assaultive violence exposure over and above family adversity, gender, and ethnicity ($B = 0.30, p = .032, OR = 1.34$). Frequency of getting drunk and marijuana use had marginally significant unique effects on risk for assaultive violence exposure.

In terms of the susceptibility hypothesis, adolescent substance use problems did not significantly increase susceptibility for developing PTSD symptoms ($B = 0.07$, $p = .40$, $IRR = 1.07$) among participants exposed to a traumatic event over and above the influence of correlated adversity in the family environment. Follow-up analyses showed that if family adversity were excluded from the model, adolescent substance use problems would have had significant effects on both trauma exposure ($B = 0.41$, $p = .045$, $OR = 1.50$) and PTSD symptoms ($B = 0.15$, $p = .04$, $IRR = 1.16$).¹⁰

In terms of the self-medication hypothesis, results showed that PTSD symptoms had a significant unique effect on future adult alcohol ($B = 0.09$, $p = .003$, $IRR = 1.10$) and drug problems ($B = 0.09$, $p = .042$, $IRR = 1.10$), over and above the effects of trauma exposure, pre-trauma substance use problems, family adversity, and covariates.¹¹ However, preliminary analyses indicated a significant interaction between ethnicity and PTSD symptoms in the model predicting alcohol problems ($B = -0.10$, $p = .02$, $IRR = 0.90$), and a marginally significant interaction in the model predicting drug problems ($B = -0.15$, $p = .06$, $IRR = 0.86$). Probing these interactions indicated that the influence of PTSD symptoms on both alcohol and drug problems was significant for non-Hispanic Caucasians but not for minority ethnicities (see note “c” in Table 2). In order to allow comparisons specifically between Hispanics and non-Hispanic Caucasians, analyses were repeated while excluding the 18 participants of other ethnicities. Results were consistent; the effect of PTSD symptoms on alcohol and drug problems was significant for non-Hispanic Caucasians but not for Hispanics.

Finally, we examined evidence for the *shared vulnerability hypothesis*. Because PTSD symptoms were significantly related to both alcohol and drug problems while accounting for family adversity and trauma exposure, this hypothesis was not supported. We subsequently examined the paths from family adversity to trauma exposure, PTSD symptoms, alcohol problems, and drug problems in order to test the extent to which family adversity increased risk for these outcomes. Although family adversity had a significant effect on both trauma exposure and PTSD symptoms, its direct effect on alcohol problems (the “c” path in a mediational model) was non-significant ($B = 0.01$, $p = .92$, $IRR = 1.01$). Mediational analyses showed that PTSD symptoms significantly and fully mediated the effect of pre-trauma family adversity on alcohol problems (95% CI = [0.010, 0.038]), while controlling for gender, ethnicity, and pre-trauma substance use problems. As for drug problems, the direct effect of family adversity on risk for drug problems was significant for females ($B = 1.12$, $p < .001$, $IRR = 3.06$) but not for males ($B = -0.01$, $p = .95$, $IRR = 0.99$). Thus, while PTSD symptoms significantly mediated the influence of family adversity on drug problems (95%

¹⁰Additional analyses tested whether adolescent substance use problems or binge drinking interacted with two indices of trauma severity—number of traumatic events and type of trauma (assaultive violence exposure vs. other types of events)—to increase susceptibility to developing PTSD symptoms. There were no significant interactions.

¹¹Additional analyses tested whether PTSD symptoms mediated the influence of trauma severity (as indicated by type of trauma or number of traumas) on substance use problems among the 166 trauma-exposed participants. PTSD symptoms did not significantly mediate the effect of type of trauma, as type of trauma did not have a significant unique effect on risk for PTSD symptoms ($B = 0.11$, $p = .35$, $IRR = 1.11$). PTSD symptoms fully mediated the influence of number of traumas on risk for alcohol problems. For drug problems, the effect of PTSD symptoms on drug problems was marginally significant ($B = 0.12$, $p = .057$, $IRR = 1.12$) after accounting for the significant effect of number of traumas on drug problems ($B = 0.57$, $p = .04$, $IRR = 1.76$). In sum, these analyses supported the self-medication hypothesis, as PTSD symptoms influenced risk for alcohol and drug problems even when controlling for these indices of trauma severity.

CI= [0.001, 0.057]), there appeared to be full mediation for males but only partial mediation for females.

With respect to the theory that trauma exposure itself may be conceptualized as a shared risk factor, trauma exposure did not have a unique effect on either alcohol ($B= 0.17$, $p= .38$, $IRR= 1.19$) or drug problems ($B= 0.20$, $p= .55$, $IRR= 1.22$).

Discussion

The present study tested a series of hypotheses to help explain the risk pathways that link traumatic stress, PTSD symptomatology, and alcohol and drug problems. Results provided the strongest support for the self-medication hypothesis, such that PTSD symptoms predicted higher levels of later alcohol and drug problems participants, over and above the influences of pre-trauma family adversity, pre-trauma substance use problems, trauma exposure, and demographic variables. As for the reverse direction (the influence of substance use problems on risk for trauma exposure or PTSD), the high-risk hypothesis was partially supported but only with respect to trauma exposure that involved assaultive violence. That is, pre-trauma adolescent substance use problems did not significantly influence overall risk for trauma exposure over and above the influence of pre-trauma family adversity, but did have a marginally significant unique effect on risk for assaultive violence exposure. Moreover, pre-trauma binge drinking was significantly associated with increased risk of assaultive violence exposure. There was no support for the susceptibility hypothesis, as pre-trauma adolescent substance use problems did not significantly influence risk for PTSD symptoms over and above the influence of pre-trauma family adversity. Finally, there was little support for the shared vulnerability hypothesis. Neither trauma exposure nor preexisting family adversity accounted for the link between PTSD symptoms and later alcohol and drug problems. Findings are explored in greater detail below.

High-Risk and Susceptibility Hypotheses

The present study is among the first to test whether *adolescent* substance use problems prospectively predict increased risk for trauma exposure or PTSD symptoms. Importantly, the non-significant effect of adolescent substance use problems on risk for both trauma exposure and PTSD would have been significant if pre-trauma family adversity were excluded from the model. This finding suggests that it is the high-risk family context within which problematic adolescent substance use occurs that may increase risk for future trauma exposure and PTSD symptoms, rather than adolescent substance use problems themselves. Trauma-exposed adolescents from adverse family environments may lack the safe context, resources, and social support needed to effectively cope with a traumatic event. These results highlight the importance of considering family adversity as an important contextual risk factor in models of PTSD-SUD risk in order to avoid making false conclusions about the about the extent to which associated individual behaviors lead to trauma exposure and post-trauma maladjustment. Although previous retrospective data indicate that adolescents with SUDs are at greatly elevated risk for both trauma exposure and PTSD compared to adolescents without SUDs (Deykin & Buka, 1997; Giaconia et al., 2000; Kilpatrick et al.,

2000), such findings likely reflect the large body of risk factors associated with adolescent SUDs.

In contrast to the non-significant effect of adolescent substance use problems on risk for overall trauma exposure, adolescent substance use problems did have a marginally significant effect on risk for assaultive violence exposure (events involving rape, physical assault or being threatened with a weapon), even after accounting for the significant influence of co-occurring family adversity. Further, post-hoc analysis showed that adolescent *binge drinking* significantly increased risk for exposure to assaultive violence, over and above the effect of family adversity. These findings are important given that risk for assaultive violence exposure, which carries an especially high risk for developing PTSD compared to other types of traumatic events (Kessler et al., 1995), is especially high during late adolescence/early adulthood (Breslau et al., 1998). Adolescent substance misuse, such as binge drinking, may be one factor driving this risk. Importantly, this finding suggests that programs to prevent adolescent substance abuse may have the added benefit of reducing assaultive violence exposure, thus also reducing risk for PTSD.

There are several reasons why substance use problems and binge drinking may place adolescents at risk for assaultive violence exposure. Risky substance use, such as binge drinking, may impair judgment and one's ability to discern danger cues in the environment. Moreover, compared to adults, adolescents may be more likely to use substances outside of the home in order to avoid adult supervision, which may place them in dangerous situations. Adolescents may also engage in unsafe activities while under the influence or during their efforts to obtain alcohol and drugs. In addition, adolescent substance abusers are especially likely to associate with deviant peers who engage in delinquent behaviors (Fergusson, Swain-Campbell, & Horwood, 2002), which may thereby increase their risk for assaultive violence. Finally, given that the average age at which adolescent substance use problems were measured was 13.2 years old, it is possible that those individuals who experience substance use problems so early in life comprise a particularly high-risk group that is likely to engage in multiple risk behaviors (e.g., stealing, fighting, early initiation of sex), any of which may increase their risk for being exposed to violence. Indeed, a recent study found that adolescent boys who engaged in high-risk behaviors (i.e., alcohol use, drug use, and delinquent behavior) were at increased risk for exposure to physical assault and/or witnessed violence later in adolescence (Begle et al., 2011).

The Self-Medication and Shared Vulnerability Hypotheses

This study adds to a growing literature in support of the self-medication hypothesis, such that individuals may use alcohol and drugs to cope with PTSD symptoms and are thus at increased risk for substance use problems. Indeed, for each additional PTSD symptom, risk for alcohol and drug problems increased approximately 10 percent. Findings extend previous research on the self-medication hypothesis in several ways. First, this study accounted for the influence of preexisting, subclinical levels of substance use problems. Previous research has typically examined patterns of onset among PTSD and SUDs, which ignores the role that subclinical levels of substance use problems may play in risk for both trauma exposure and post-trauma maladjustment. Moreover, by including both pre- and

post-trauma measures of substance use problems, the present study allowed for inferences regarding the direction of effect.

Second, the present study differentiated between the effects of trauma exposure and PTSD symptoms on future substance use problems. Few studies have recognized that trauma exposure may be a shared risk factor for both PTSD and SUDs. The fact that trauma exposure failed to significantly influence risk for alcohol or drug problems while controlling for subclinical levels of PTSD provides strong evidence that the effects of traumatic stress on substance use problems are mediated by PTSD symptoms. Even though the vast majority of trauma-exposed individuals do not develop clinically-significant PTSD (Kessler et al., 1995), this study suggests that trauma exposure may nonetheless have meaningful effects on one's risk for future substance use problems to the extent that there are resultant posttraumatic symptoms.

Third, the present study advances previous research on the self-medication hypothesis by controlling for the confounding influence of preexisting adversity in the family environment. Findings highlight adolescent family adversity as an important risk factor for trauma exposure, PTSD, and adult substance use problems, alike. However, there was no evidence that family adversity accounted for the association between PTSD and either alcohol or drug problems. The influence of family adversity on alcohol problems was fully mediated by PTSD symptoms; the influence of family adversity on drug problems was fully mediated by PTSD symptoms for males but only partially mediated by PTSD symptoms for females. Although the effects of family adversity on alcohol and drug problems were generally indirect rather than direct, findings nonetheless suggest that preexisting family adversity plays an important role in the PTSD-SUD link. Indeed, results provided evidence for a causal chain, whereby family adversity increased risk for trauma exposure and PTSD symptoms, which in turn increased risk for later adult alcohol and drug problems. Thus, it appears that family adversity operates as an important contextual risk factor such that trauma-exposed individuals who grow up in adverse family environments are more likely to develop PTSD symptoms and later substance use problems.

Fourth, although previous research has made it clear that substance use problems are prevalent in the aftermath of trauma (Stewart & Conrod, 2003), the present study extends this knowledge by demonstrating that the effects of PTSD on substance use problems persist well into the future. This finding is consistent with a study by Swendsen and colleagues (2010), which showed that PTSD diagnosis prospectively predicted onset of alcohol and drug dependence 10 years later. Finally, the present study provided tentative evidence that the self-medication hypothesis may vary across ethnicity, such that PTSD symptoms increase risk for substance use problems for non-Hispanic Caucasians but not Hispanics. However, given the small sample size, replication of this finding is needed before definitive conclusions can be made.

This study's finding that PTSD symptoms directly increased risk for both alcohol and drug problems differs from a previous study using this same sample, which examined externalizing and internalizing symptoms as mediators of the influence of PTSD symptoms on alcohol and drug problems (Haller & Chassin, 2012). This previous study found that

PTSD symptoms directly influenced risk for adult drug problems, but PTSD symptoms only influenced risk for adult alcohol problems to the extent that PTSD symptoms increased early adult externalizing symptoms. Several methodological differences may help explain the difference in findings between the present study and the Haller and Chassin (2012) study. First, the present study included both individuals who were and were not exposed to trauma in its analysis, whereas the previous study included only trauma-exposed participants. Second, the present study accounted for family adversity, ethnicity, and trauma exposure, whereas the previous study did not. Third, this study used a count of alcohol/drug problems as its outcome variable, whereas the previous study used a composite of frequency of use and problems within a shorter timeframe (only one year). Thus, the outcome variable in the current study reflects a more severe measure of alcohol problems. It is possible that PTSD symptoms are more strongly related to *problematic* alcohol use than to alcohol use itself.

Despite these methodological differences, findings from the Haller and Chassin (2012) study have important implications for the present study. Haller and Chassin distinguished between a PTSD-specific self-medication mechanism, and a more generalized negative affect self-medication mechanism (e.g., Khantzian, 1985), such that individuals may use substances to reduce negative affect and other internalizing symptoms. Importantly, Haller and Chassin found that PTSD-related increases in internalizing symptoms did *not* significantly increase risk for either alcohol or drug problems. Thus, it appears to be PTSD symptoms, specifically, that increase risk for substance use problems, rather than broader internalizing symptomatology (e.g., sad mood, low energy, worthlessness) that is often experienced during the aftermath of trauma.

Limitations and Conclusions

Several study limitations should be noted. First, many factors outside the scope of this study (e.g., peritraumatic factors, genetic influences) may influence risk for trauma exposure and/or post-trauma adjustment. Although this study failed to support the shared vulnerability hypothesis with respect to trauma exposure and family adversity, many other shared risk factors may contribute to the association between PTSD and SUDs. Similarly, despite the lack of support for the susceptibility hypothesis, future studies of moderators may find that preexisting substance use problems may indeed increase risk for PTSD for certain individuals or under certain conditions. Second, it was not possible to examine reciprocal relations between PTSD symptoms and substance use problems over time because PTSD symptoms were assessed at only one time point. Third, findings may not generalize to those with very early trauma exposure, given that we excluded participants who experienced trauma prior to Wave 1. Fourth, adolescent substance use problems were measured at a very young age and, on average, four years prior to trauma exposure. Measures closer in time to the traumatic event will be better suited to testing the true extent to which preexisting substance use problems are a causal risk factor for trauma exposure and/or PTSD; however, the unpredictable timing of trauma exposure makes it nearly impossible to obtain such a measure.¹² Finally, trauma exposure and PTSD symptoms were assessed using DSM-III-R criteria rather than the current DSM-5 criteria.

In summary, this study is among the few longitudinal, community-based studies to test the directions of influence among trauma exposure, PTSD, and alcohol and drug problems. Results demonstrated that PTSD symptoms may have long-lasting effects on substance use problems, thereby highlighting PTSD symptomatology as an important etiological factor in the development of SUDs. Findings also indicated that family environments characterized by high levels of conflict, stress, and psychopathology may influence risk for post-trauma substance use problems by increasing the likelihood of developing PTSD symptoms following a traumatic event. Finally, this study also provided support for adolescent substance use problems and binge drinking as risk factors for assaultive violence exposure, which conveys an especially high risk for PTSD compared to other traumatic events (Kessler et al., 1995) and may thus increase the likelihood of post-trauma substance use problems. Findings are thus consistent with the notion that multiple, non-mutually exclusive pathways may underlie the link between PTSD and SUDs.

These findings have implications for preventing substance use problems among individuals who present for treatment for PTSD. Clinicians should routinely assess clients' risk for using alcohol or drugs to self-medicate PTSD symptoms, discuss long-term dangers associated with self-medication, and provide other means of coping. Findings also highlight the need to screen for and treat PTSD symptomatology among individuals who present with substance use problems. Research indicates a low detection rate of PTSD within addiction treatment centers because individuals with substance use problems often do not report traumatic experiences and PTSD symptoms unless specifically asked (Kimerling, Trafton, & Nguyen, 2006). Individuals with concurrent PTSD symptoms and SUDs are especially hard to treat, and do not optimally benefit from standard SUD interventions (Norman, Tate, Anderson, & Brown, 2007). Findings from the present study suggest that in addition to addressing the functional associations between PTSD symptoms and problematic substance use, resolving distress related to adversity in the family environment may also be a potentially important treatment target. Understanding the development and treatment of co-occurring PTSD symptoms and substance use problems remains an important area for research.

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Figure 1.

Simplified depiction of model estimation and paths relevant to hypothesis testing.

^a Note that Wave 4 measures reflect trauma exposure and PTSD symptoms that occurred *between* Waves 1 and 4 (average age at exposure was 17.3).

Zero-Order Correlations

Table 1

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Female gender	--												
2. Adolescent ethnicity	.03	--											
3. Parental education	-.07	-.27***	--										
4. Adolescent substance use problems	-.04	-.05	-.04	--									
5. Parental alcoholism	.02	.13*	-.11*	.22***	--								
6. Other parental psychopathology	-.03	-.03	-.06	.11*	.22***	--							
7. Adolescent family conflict	-.04	.11*	-.07	.26***	.28***	.19***	--						
8. Adolescent familial life stress	-.06	.12*	-.11*	.17***	.27***	.26***	.52***	--					
9. Adolescent family adversity factor score	-.05	.13*	-.11*	.27***	.49***	.38***	.90***	.78***	--				
10. Late adolescent/early adult trauma exposure	-.15**	.11*	-.01	.11*	.09†	.10*	.18***	.19***	.21***	--			
11. Late adolescent/early adult PTSD diagnosis ^a	.26**	.06	-.04	.14†	.05	.06	.24**	.21**	.25***	n/a	--		
12. Late adolescent/early adult PTSD symptoms ^a	.35***	.10	-.15*	.09	.16*	.15†	.20*	.23***	.26***	n/a	.56***	--	
13. Adult alcohol problems ^b	-.21***	.04	.03	.12*	.18***	-.02	.11†	.08	.13*	.12*	.04	.11	--
14. Adult drug problems ^b	-.12*	.02	-.05	.09†	.18***	.01	.11*	.16**	.16***	.09†	.03	.14†	.52***

Note.

†
 $p < .10$,*
 $p < .05$,**
 $p < .01$,***
 $p < .001$.^a $n = 377$. All count variables were log-transformed prior to estimating zero-order correlations. Adolescent ethnicity is coded 0 for non-Hispanic Caucasians and 1 for Hispanics and other ethnicities.^b $n = 166$ (trauma exposed only).^c $n = 348$ due to missing data at Wave 5.

Table 2

Results from Primary Analyses

Predictor	Outcomes							
	Trauma Exposure		PTSD Symptoms		Adult Alcohol Problems		Adult Drug Problems	
	<i>B</i> (<i>SE</i>)	Odds Ratio	<i>B</i> (<i>SE</i>)	Incidence Rate Ratio	<i>B</i> (<i>SE</i>)	Incidence Rate Ratio	<i>B</i> (<i>SE</i>)	Incidence Rate Ratio
Family adversity	0.55** (.17)	1.72	0.26** (.08)	1.30	0.01 (.12)	1.01	-0.01 ^d (.24)	0.99
Female gender	-0.32 ^d (.30)	0.73	0.54*** (.11)	1.72	-1.03*** (.22)	0.36	-1.36*** (.39)	0.26
Ethnicity	1.32 ^b ** (.42)	3.73	0.20 [†] (.12)	1.22	0.08 (.21)	1.08	-0.10 (.33)	0.91
Adolescent substance use problems	0.21 (.21)	1.23	0.07 (.08)	1.07	0.29 [†] (.16)	1.34	0.47 [†] (.27)	1.60
Gender x ethnicity	-1.60** (.60)	0.21						
Time since trauma exposure					-0.23** (.07)	0.80		
Gender x family adversity							1.13** (.35)	3.10
Trauma exposure					0.17 (.20)	1.19	0.20 (.05)	1.22
PTSD Symptoms ^c					0.09** (.03)	1.10	0.09* (.05)	1.10

Note.

[†] $p < .10$,

* $p < .05$,

** $p < .01$,

*** $p < .001$.

$n=377$. $n=166$ for PTSD symptoms. *B*= Unstandardized regression coefficient. *SE*= Standard error. Logistic regression was used for trauma exposure, and negative binomial regression was used for PTSD symptoms, alcohol problems, and drug problems. Adolescent ethnicity is coded 0 for non-Hispanic Caucasians and 1 for Hispanics and other ethnicities.

^a Due to the interaction between gender and ethnicity, the coefficient presented for gender indicates the effect of gender for Caucasians only. The effect of gender on trauma for minority ethnicity participants was significant ($B = -1.90$, $SE = .52$, $p < .001$, $OR = 0.15$).

^b Due to the interaction between gender and ethnicity, the coefficient presented for ethnicity indicates the effect of ethnicity for males only. The effect of ethnicity on trauma for females was non-significant ($B = -0.26$, $SE = .43$, $p = .55$, $OR = 0.77$).

^c Note that additional analyses indicated that ethnicity interacted with PTSD symptoms in the models predicting both alcohol ($B = -.10$, $p = .02$, $IRR = .90$) and drug problems ($B = -.015$, $p = .06$, $IRR = 0.86$). The influence of PTSD symptoms on alcohol problems was significant for non-Hispanic Caucasians ($B = 0.11$, $p = .001$, $IRR = 1.12$) but not for minority ethnicities ($B = 0.02$, $p = .52$, $IRR = 1.02$). The effect of PTSD symptoms on drug problems was significant for non-Hispanic Caucasians ($B = 0.11$, $p = .04$, $IRR = 1.11$) but not for minority ethnicities ($B = 0.02$, $p = .67$, $IRR = 1.02$).

^dDue to the interaction between gender and family adversity, the coefficient presented for family adversity indicates the effect of family adversity for males only. The effect of family adversity on drug problems for females was significant ($B = 1.12$, $SE = .26$, $p < .001$, $IRR = 3.06$).