Disentangling the Directions of Influence among Trauma Exposure, Posttraumatic

Stress Disorder Symptoms, and Alcohol and Drug Problems

by

Moira Haller

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Laurie Chassin, Chair Mary Davis Armando Piña Jenn-Yun Tein

ARIZONA STATE UNIVERSITY

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ABSTRACT

The present study utilized longitudinal data from a high-risk community sample (n= 377; 166 trauma-exposed; 54% males; 52% children of alcoholics; 73% non-Hispanic/Latino Caucasian; 22% Hispanic/Latino; 5% other ethnicity) to test a series of hypotheses that may help explain the risk pathways that link traumatic stress, posttraumatic stress disorder (PTSD) symptomatology, and problematic alcohol and drug use. Specifically, this study examined whether pretrauma substance use problems increase risk for trauma exposure (the high-risk hypothesis) or PTSD symptoms (the susceptibility hypothesis), whether PTSD symptoms increase risk for later alcohol/drug problems (the self-medication hypothesis), and whether the association between PTSD symptoms and alcohol/drug problems is due to shared risk factors (the shared vulnerability hypothesis). This study also examined the roles of gender and ethnicity in these pathways.

A series of logistic and negative binomial regressions were performed in a path analysis framework. A composite pre-trauma family adversity variable was formed from measures of family conflict, family life stress, parental alcoholism, and other parent psychopathology. Results provided the strongest support for the self-medication hypothesis, such that PTSD symptoms predicted higher levels of later alcohol and drug problems among non-Hispanic/Latino Caucasian participants, over and above the influences of pre-trauma family adversity, pretrauma substance use problems, trauma exposure, and demographic variables. Results partially supported the high-risk hypothesis, such that adolescent

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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, as pre-trauma adolescent substance use problems did not significantly influence risk for PTSD diagnosis/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 substance use problems.

These results add to a growing body of literature in support of the selfmedication hypothesis. Findings extend previous research by showing that PTSD symptoms may influence the development of alcohol and drug problems over and above the influence of trauma exposure itself, preexisting family risk factors, and baseline levels of substance use.

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Disentangling the Directions of Influence among Trauma Exposure, Posttraumatic

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Traumatic life events are well-known to have pervasive, long-lasting effects on both adolescent and adult functioning. Traumatic events may lead to the development of not only posttraumatic stress disorder (PTSD), but also a range of other internalizing and externalizing psychopathologies, particularly substance use disorders (SUDs; Breslau, Davis, Peterson, & Schultz, 2000; Breslau, Davis, & Schultz, 2003; Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). PTSD (Boscarino, 2006) and alcohol and drug abuse (Mokdad, Marks, Stroup, & Gerberding, 2004) are associated with increased risk for mortality and other adverse health outcomes, with annual costs estimated to be \$45 billion for PTSD (Kessler, 2000), \$185 billion for alcohol abuse (Harwood, 2000), and \$143 billion for drug abuse (Office of the National Drug Control Policy, 2001). Findings from the National Comorbidity Survey indicate that the prevalence of alcohol and drug disorders is approximately 35% and 29% (respectively) among individuals with PTSD, compared to 24% and 11% (respectively) among individuals without PTSD (Kessler et al., 1995). These individuals with PTSD-SUD comorbidity are at elevated risk for poor health and psychosocial impairments, including unemployment, homelessness, HIV, poor response to treatment, briefer abstinence periods after substance abuse treatment, and additional psychiatric comorbidities (Back, Dansky, Coffey, Saladin, Sonne, & Brady, 2000; Brown, Stout, & Mueller, 1996; Brown & Ouimette, 2003; Read, Brown, & Kahler, 2004).

Given the high prevalence and extensive public health implications of PTSD-SUD comorbidity, it is important to understand the mechanisms that link traumatic stress and PTSD symptomatology to problematic alcohol and drug use. However, these mechanisms remain poorly understood in part because very few longitudinal, community-based studies contain measures of both pre- and posttrauma functioning. The current study utilized longitudinal data from a high-risk community sample of children of alcoholics and demographically-matched controls (e.g., Chassin, Rogosch, & Barerra, 1991) to test a series of hypotheses that may explain the association between PTSD and problematic substance use. Specifically, this study examined whether problem substance use increases risk for trauma exposure or PTSD, whether PTSD increases risk for problem substance use, and/or whether the association between PTSD and problem substance use is due to shared risk factors. This study also tested the role of gender and ethnicity in these pathways.

This review of the literature begins with an overview of research issues pertinent to traumatic stress and PTSD. Next, various theories about the pathways that may link PTSD and SUDs are critically reviewed. This is followed by a discussion of gender and ethnicity as potential moderators of these pathways. Finally, the aims of this study and its potential contributions to the literature are described.

Trauma Exposure and PTSD

PTSD is diagnostically unique from most mental disorders in its requirement of a precipitating traumatic event. In addition to experiencing a

traumatic event, an individual must respond to the event with intense fear, helplessness, and horror in order to be diagnosed with PTSD (American Psychiatric Association, 2000). The resulting symptomatology falls into three broad categories: re-experiencing symptoms, avoidance and numbing symptoms, and physiological hyperarousal.

Although traumatic events were once thought of as rare, extraordinary events, research has shown that traumatic events are surprisingly common, with risk for exposure, particularly risk for assaultive violence, increasing sharply at age 15 and peaking between the ages of 16 to 20 (Breslau et al., 1998). In fact, the National Comorbidity Survey estimates that 60.7% of men and 51.2% of women experience at least one traumatic event during their lifetimes (Kessler et al., 1995). However, only a small proportion of trauma-exposed individuals— 20.4% of women and 8.2% of men—will actually develop PTSD (i.e., lifetime prevalence in the general population is 5% for men and 10.4% for women). Therefore, the vast majority of trauma-exposed individuals appear to successfully navigate traumatic events with little or no disruption in their normal functioning (Bonanno, Galea, & Bucciarelli, & Vlahov, 2007). Why some trauma-exposed individuals may not experience any negative post-trauma reactions at all, whereas others develop PTSD, SUDs, and/or other psychopathologies, is an important question.

Post-trauma adjustment appears to be determined by a complex interplay between characteristics of the traumatic event, preexisting vulnerability factors, and environmental or psychological resources more proximal to the traumatic

event (e.g., Silverman & La Greca, 2002). Early research on the negative effects of trauma exposure focused primarily on characteristics of the traumatic event; that is, its severity and type (e.g., Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993). For instance, risk for PTSD appears to be much higher for traumatic events involving assaultive violence (e.g., rape, physical assault) compared to other types of traumas (Kessler, 2000). However, trauma exposure variables appear to account for only a small proportion of the variance in PTSD symptoms (Yehuda & McFarlane, 1995).

Current theories also emphasize the role of preexisting vulnerabilities in determining post-trauma adjustment. These vulnerabilities include preexisting cognitive, emotional, and behavioral problems, as well as demographic variables and characteristics of the family environments (see meta-analyses by Brewin, Andrews, & Valentine, 2000, and Ozer, Best, Lipsey, & Weiss, 2003). These *distal* characteristics of the individual and his or her family environment appear to account for a limited but consistent proportion of the variance in PTSD ($r \approx .20$), whereas factors that are more *proximal* to the traumatic event, such as social support and coping skills, may be more strongly related to the development of PTSD ($r \approx .40$). However, even if the unique influences of distal risk factors on post-trauma outcomes are small, these factors may nonetheless play an important causal role in post-trauma maladjustment by affecting more proximal factors that directly influence post-trauma outcomes.

Preexisting individual and family risk factors may also influence risk for trauma exposure itself. Indeed, research indicates that trauma exposure is not randomly distributed in the general population (e.g., Breslau, 1998). Rather, there are numerous risk factors that may influence risk for trauma exposure, which may be either the same or different from the factors that increase vulnerability to the effects of that exposure. Moreover, the factors that increase risk for PTSD may be either the same or different from those that increase risk for alcohol and drug problems following traumatic stress. In order to better understand the etiology of PTSD and its association with problem substance use, it is important to differentiate among risk factors for trauma exposure, PTSD, and problematic alcohol and drug use.

Although measures of individual and family functioning obtained prior to trauma exposure are necessary for delineating causal relations among these variables, such measures are extremely rare in trauma and PTSD research. In fact, the vast majority of available studies are cross-sectional or retrospective, which are limited by both recall error and confirmation bias (Brewin et al., 2000). Moreover, the few prospective studies that exist consist largely of: (a) military studies using measures collected prior to deployment/combat exposure (e.g., Macklin et al., 1998; Pitman, Orr, Lowenhagen, Macklin, & Altman, 1991), (b) disaster studies using incidental data from research that was taking place when the disaster occurred (e.g., La Greca, Silverman, & Wasserstein, 1998; Kessler, Galea, Jones, & Parker, 2006; Parslow, Jorm, & Christensen, 2005), or (c) studies using measures of risk factors that were collected during early childhood (Breslau, Lucia, & Alvarado, 2006; Storr, Ialongo, Anthony, & Breslau, 2007).

These literatures have important limitations in terms of the generalizability of their findings. Research has indicated that the risk factors for PTSD in military samples may significantly differ from those in civilian samples (Brewin et al., 2000), thus suggesting that findings from military studies may not necessarily apply to the general population. In addition, unlike disasters (e.g., hurricanes, terrorist attacks), most traumatic events are not random and unrelated to a person's behavior; rather, there are many variables (e.g., substance use, externalizing behaviors) that are likely to influence one's risk for trauma exposure and how he or she responds afterwards. Similarly, given that risk for trauma exposure peaks during late adolescence/early adulthood (Breslau et al., 1998), studies of risk factors that are measured during early childhood do not provide a complete picture of how one's behavior influences his or her risk trauma exposure and post-trauma maladjustment. Studies of risk factors that are measured during adolescence may be more useful for understanding the risk mechanisms at play. Moreover, research on the comorbidity between PTSD and other disorders is often based on clinic samples, which tend to be biased because individuals with multiple disorders (rather than a single disorder) are more likely to seek treatment. Longitudinal, community-based studies that examine the complex mediated and moderated processes involved in risk for trauma exposure and post-trauma maladjustment are crucially needed.

Pathways to Account for the Link between PTSD and Problematic Alcohol and Drug Use

There are several potential pathways that may underlie the high rates of comorbidity 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). Alcohol and drug use may also increase risk for trauma exposure 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 make individuals more vulnerable to PTSD following exposure to trauma (Chilcoat & Breslau, 1998a). For instance, substance abuse may increase vulnerability for PTSD by interfering with the ability to effectively manage negative emotions resulting from trauma exposure or by increasing anxiety and arousal levels due to substance withdrawal symptoms (Stewart, Pihl, Conrod, & Doniger, 1998). Third, the "self-medication hypothesis" states that individuals may use substances in order to cope with symptoms of posttraumatic stress. Finally, the "shared vulnerability hypothesis" states that shared risk factors may account for both PTSD and alcohol/drug abuse.

Whereas the high-risk, susceptibility, and self-medication hypotheses suggest that PTSD and SUDs are causally related, the shared vulnerability hypothesis suggests that the co-occurrence of PTSD and SUDs is non-causal (see Chilcoat & Breslau, 1998a, for a review of causal criteria as they apply to the PTSD-SUD association). Numerous studies indicate that PTSD symptom severity is significantly associated with substance abuse severity, which demonstrates a "gradient of effect" among PTSD and SUDs and suggests the possibility of a causal relation (see Stewart & Conrod, 2003). However, studies examining the temporality among PTSD and SUDs have found that SUDs precede PTSD (Acierno, Resnick, Kilpatrick, Saunders, & Best, 1999; Cottler, Compton, Mager, & Spitznagel, 1992; Giaconia et al., 2000), and also that PTSD precedes SUDs (Chilcoat & Breslau, 1998b; Cornelius, Kirisci, Reynolds, Clark, Hayes, & Tarter, 2010; Kessler et al., 1995; see Stewart & Conrod, 2003, for review). Thus, there does not appear to be any clear answer regarding the direction of causality in the PTSD-SUD link, which may indicate that the association is better explained by a shared diathesis.

This study takes a closer look at evidence in support of each of the hypotheses that have been proposed to explain the PTSD-SUD link. Although these hypotheses are presented separately, it should be noted that they are not mutually exclusive. It is likely that more than one of these hypotheses are implicated in the PTSD-SUD link. For instance, pre-existing substance use problems may not only increase one's risk for trauma exposure (high-risk hypothesis), but may also make it more likely that he or sure will turn to alcohol or drugs to cope with PTSD symptoms that may result from said trauma exposure (self-medication hypothesis). Such bi-directional possibilities mean that multiple hypotheses may be supported and integrated into a larger, developmental model of PTSD-SUD comorbidity.

The High-Risk and Susceptibility Hypotheses

The high-risk and susceptibility hypotheses both suggest that SUDs causally influence risk for PTSD. Research indicates that alcohol, marijuana, and hard drug users are more likely to be physically and sexually assaulted compared to non-users, thus lending partial support to the high-risk hypothesis (Breslau, Davis, Andreski, & Peterson, 1991; Burnam et al., 1988; Cottler et al., 1992; Kessler et al., 1995). Moreover, retrospective data from the National Comorbidity Survey suggest that preexisting SUDs predict significantly increased risk for trauma exposure but not PTSD (Bromet, Sonnega, & Kessler, 1998). However, a longitudinal study of adult women found that drug use but not alcohol abuse was prospectively related to increased risk for future assault (Kilpatrick, Acierno, Resnick, Saunders, & Best, 1997), whereas results from their retrospective assessment indicated that a past history of alcohol abuse (and to a lesser extent, drug use) was associated with a current diagnosis of PTSD among participants who had been raped (Acierno et al., 1999).

Although the Kilpatrick et al. (1997) study provided tentative support for the high-risk hypothesis and the Acierno et al. (1999) study provided tentative support for the susceptibility hypothesis, longitudinal data from a communitybased sample of adults between the ages of 21 and 30 found that preexisting SUDs did not significantly increase risk for either trauma exposure or PTSD (Chilcoat & Breslau, 1998a). The Chilcoat and Breslau study, which included both men and women, also has the advantage of using both prospective and survival analytic strategies, which allow for a more detailed examination of the

temporal ordering among PTSD and SUDs. Therefore, there appears to be a lack of convergence in findings from studies investigating the high-risk and susceptibility hypotheses. The lack of compelling empirical support for these hypotheses has led some researchers to conclude that it is unlikely that substance use behaviors causally influence risk for trauma exposure or PTSD, especially when considered among other risk factors (e.g., Chilcoat & Breslau, 1998a; Stewart & Conrod, 2003). It is important to note, however, that even if substance use does not *cause* PTSD, it is likely that substance use contributes to the intensification of PTSD symptoms and to the maintenance of PTSD-SUD comorbidity (Brown, Stout, & Gannon-Riley, 1998; Stewart, 1996). In other words, individuals with PTSD-SUD comorbidity may experience an unrelenting cycle in which the symptoms of one disorder sustain the other.

One limitation to the literature on substance use and PTSD is that few studies have examined relations between *adolescent* substance use behaviors and risk for trauma exposure or PTSD, and that no known studies have examined these variables within a prospective design. This limitation is important because adolescent substance use problems, compared to adult substance use problems, may reflect a particularly high-risk behavior, and may thus be more likely to increase risk for trauma exposure. For instance, 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 at increased risk for trauma exposure. Moreover, adolescents who abuse alcohol or drugs are especially likely to associate with deviant peers who engage in delinquent behaviors (Barnow et al., 2004; Fergusson, Swain-Campbell, & Horwood, 2002), which may thereby increase their risk for trauma exposure (e.g., physical assault, being threatened with a weapon, etc.).

In addition, because of the gap between sensation-seeking tendencies and cognitive control systems during adolescence (Steinberg, 2008), adolescent substance users may be especially likely to engage in impulsive or reckless behaviors and poor decision-making when under the influence, thereby increasing their risk for trauma exposure compared to adult substance users. This heightened risk may be especially true of adolescents who, at such a young age, are already using substances to an extent that they experience consequences or dependence symptoms. For instance, adolescents who exhibit dependence on alcohol or drugs, compared to adolescents without dependence symptoms, may be at increased risk for trauma exposure because they may engage in dangerous activities (e.g., stealing, fighting, driving under the influence) during their efforts to procure these substances.

Adolescents with substance use problems, compared to adolescents without substance use problems, may be at particularly high risk for types of trauma involving assaultive violence (which are especially likely to result in PTSD). Indeed, retrospective data suggest that adolescents with SUDs are not only at 2 to 5 times the risk for experiencing trauma exposure compared to adolescents without trauma exposure, but are also at elevated risk for being exposed to traumatic events involving violence, such as physical and sexual assault and witnessing harm to others (Giaconia et al., 2000; Kilpatrick et al., 2000). Given that risk for trauma exposure and assaultive violence are especially high during mid-to-late adolescence (Breslau et al., 1998), it is important that future prospective studies clarify the role that adolescent substance use problems may play in this risk.

Retrospective research indicates that adolescents with SUDs are at 4 to 9 times the risk for developing PTSD compared to adolescents without SUDs (Deykin & Buka, 1997; Giaconia et al., 2000; Kilpatrick et al., 2000), suggesting that adolescent substance use problems may also increase risk for PTSD. Adolescents with substance use problems may be at increased risk for PTSD symptomatology following trauma exposure because they lack the emotion regulation and coping skills necessary for managing their reactions to the traumatic event. For instance, trauma exposure during adolescence may impair prefrontal cortical functioning (e.g., self-regulatory processes), which tends to already be impaired in adolescent substance abusers, such that the additive neurobiological dysregulation leads to increased risk for PTSD (e.g., Brady & Sinha, 2005). The additional dysregulation due to the effects of substance use or substance use withdrawal symptoms may further compound this risk. Given that adolescents tend to have poorer self-regulatory capacity than do adults, it is theoretically plausible that the effect of adolescent substance use problems on PTSD (among trauma-exposed adolescents) would be stronger compared to the effect of adult substance use problems on PTSD (among trauma-exposed adults). Interestingly, whereas studies with adult samples indicate that PTSD more often precedes than follows SUD onset (see Stewart & Conrod, 2003), studies that

examine the temporal patterns of onset between PTSD and SUDs with adolescent samples do not indicate any clear pattern of sequencing (Deykin & Buka, 1997; Giaconia et al., 2000; Giaconia, Reinherz, Paradis, & Stashwick, 2003; Perkonigg, Kessler, Storz, & Wittchen, 2000).

Future research that moves beyond examining patterns of onset among PTSD and SUDs is needed because this method does not capture pre-trauma risk stemming from premorbid levels of substance use and other preexisting risk factors. That is, adolescent substance use problems, even if not "clinically significant," may create meaningful risk for trauma exposure, PTSD, and posttrauma substance use problems. Although it is likely that substance use problems are both a risk factor for and a consequence of trauma exposure and/or PTSD, only prospective studies that control for baseline levels of substance use can disentangle these effects.

The Self-Medication Hypothesis

Although investigations of the high-risk and susceptibility hypotheses reveal a confusing pattern of results, there is a strong body of evidence in support of the self-medication hypothesis (Breslau, Davis, Peterson, & Schultz, 1997; Breslau et al., 2003; Chilcoat & Breslau, 1998b; Epstein, Saunders, Kilpatrick, & Resnick, 1998; Reed, Anthony, & Breslau, 2007; Shipherd, Stafford, &, Tanner, 2005; Ullman, Filipas, Townsend, & Starzynski, 2005; see also Hien, Cohen, & Campbell, 2005, for review). Theoretically, individuals with PTSD might use alcohol and drugs to induce sleep, reduce irritability, reduce concentration problems, reduce hypervigilance, and reduce excessive startle responses (Stewart, 1997). Indeed, in a review of both 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."

The self-medication hypothesis implies a mediating role for PTSD symptoms in the relation between trauma exposure and substance use problems. That is, trauma exposure may increase risk for SUDs to the extent that trauma exposure results in PTSD symptoms (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. For instance, using Cox proportional-hazard models, Chilcoat and Breslau (1998b) found that PTSD increased the risk for developing a drug disorder more than fourfold, whereas individuals who were exposed to a traumatic event but did not develop PTSD had no increase in risk relative to those without trauma exposure. Similarly, using both prospective and retrospective data, Breslau and colleagues (2003) found that trauma exposure without PTSD did not predict increased risk for SUDs, whereas PTSD predicted increased risk for future drug but not alcohol disorders. Other studies have similarly found PTSD is more likely to increase risk for future drug problems than alcohol problems (Driessen et al., 2008; Shipherd et al., 2005), even though alcohol disorders are more commonly comorbid with PTSD than are drug disorders. One exception comes from a retrospective study of a community

sample of women, which found that trauma-exposed women both with *and* without PTSD were at increased risk for *alcohol* disorders (Breslau et al., 1997). Nonetheless, it generally appears that PTSD is more strongly associated with substance use problems, particularly drug problems, than is trauma exposure per se.

However, studies comparing groups of people with PTSD to groups of people with trauma exposure who do not develop PTSD are unable assess the extent to which PTSD symptom severity matters in terms of risk for future substance use problems. Although studies have directly tested continuous measures of PTSD symptoms as mediators of the influence of trauma exposure on substance use problems and had significant findings (e.g., Epstein et al., 1998), these studies have not used appropriate methodology. That is, because one cannot have a valid measure of PTSD symptom severity without exposure to a traumatic event (i.e., trauma exposure is a prerequisite for assessing PTSD symptoms), PTSD cannot simply be tested as a mediator of the influence of trauma exposure. Future studies with alternative analytic strategies are needed to test the extent to which PTSD symptom severity influences risk for substance use problems, over and above the effects of trauma exposure. Future studies should also take into account the influence of shared risk factors for both PTSD symptoms and substance use problems. That is, even if PTSD symptoms influence risk for substance use problems over and above the influence of trauma exposure, the effect could be spurious if shared risk factors are not accounted for.

The Shared Vulnerability Hypothesis

The high prevalence of PTSD-SUD comorbidity suggests that PTSD and SUDs may share a common etiological diathesis. This shared diathesis may include preexisting individual and family risk factors, as well as shared genetic and environmental factors. Indeed, research suggests that genetic influences may account for about 30% of the variance in PTSD symptoms, even after accounting for differences in types of traumatic events (Stein, Jang, Taylor, Vernon, & Livesly, 2002). Behavioral genetic studies suggest that common genetic factors may account for the co-occurrence of PTSD with alcohol and drug disorders (Xian et al., 2000). Specifically, Xian and colleagues found that 15.3% of the genetic contribution to variance in PTSD liability was common to alcohol and drug disorders, whereas 20.0% was specific to PTSD.

Trauma exposure itself may be conceptualized as a shared environmental risk factor for both PTSD and SUDs. In other words, 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. The notion that trauma exposure might increase risk for SUDs regardless of PTSD grew from studies documenting high rates of SUDs among individuals exposed to traumatic events (e.g., Breslau et al., 2003). Indeed, a recent cross-sectional study of 34,160 adults from the National Epidemiological Survey of Alcohol and Related Conditions (NESARC) found that exposure to nearly any type of traumatic event predicted significantly elevated risk for having an alcohol use disorder (Fetzner, McMillan, Sareen, & Asmundson, 2011). Importantly, this study also found that individuals who were exposed to a traumatic event but did not develop PTSD were at significantly elevated risk for having an alcohol use disorder, suggesting that trauma exposure may increase risk for SUDs independent of PTSD.

According to the theory that trauma exposure may be a shared risk factor for multiple forms of psychopathology, whether a trauma-exposed individual develops PTSD, a SUD, or some other psychopathology, will depend on his or her preexisting vulnerabilities and biological predispositions (Friedman & Yehuda, 1995; Yehuda, McFarlane, & Shalev, 1998). Many different theories support the notion that traumatic life events may heighten or accentuate pathogenic traits that are present in the pre-trauma personality (Allport, Bruner, & Jandorf, 1941; Eberly, Harkness, & Engdahl, 1991; Miller, 2003). The development of PTSD may occur in individuals who are predisposed to biological hyper-responsiveness and thus experience further sensitizations in their stress response systems following trauma exposure (Yehuda et al., 1998). Individuals without this predisposition may experience a range of other stress responses that lead to other disorders, such as alcohol and/or drug problems. For instance, traumatic stress could increase risk for SUDs among individuals who are predisposed toward externalizing-spectrum behavior (a well-established risk factor for SUDs; see Zucker, 2006) by further exacerbating this tendency. Therefore, traumatic events may induce distinct diatheses for PTSD and SUDs, such that trauma exposure exerts direct effects on problem substance use, independent of PTSD. If this hypothesis were true, traumatic stress would be expected to directly predict

problematic alcohol and/or drug use, separate from its influence on PTSD. Alternatively, this direct effect would not be expected if other common risk factors account for the link between PTSD and alcohol/drug problems.

However, as was previously discussed, most studies show that traumaexposed 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). The lack of effect of trauma exposure itself on SUDs suggests that either PTSD and SUDs share a *common* diathesis (rather than distinct diatheses triggered by traumatic stress), or that PTSD directly influences the development of SUDs (i.e., self-medication). In a test of these alternative hypotheses, Reed and colleagues (2007) found that PTSD, but not trauma exposure without PTSD, predicted significantly increased risk for future drug disorders even after accounting for early life (i.e., measured at age 6) experiences and predispositions that are known risk factors for drug disorders, trauma exposure, and PTSD (e.g., conduct disorders and family socioeconomic status). Note all risk factors were measured at age 6; hence, this study was unable to examine the role of pre-trauma substance use in the PTSD-drug disorder link (this study did not examine risk for alcohol disorders). Replication is needed with other studies that contain pre-trauma measures of *adolescent* risk factors. Studies with pre-trauma measures that are assessed during adolescence and thus closer to the time that the traumatic event occurred will have the added advantage of contributing to a better understanding of not only how these risk factors influence

risk for post-trauma maladjustment, but also how these risk factors influence risk for trauma exposure itself.

There are a number of risk factors that trauma exposure, PTSD, and SUDs may share in common. A large body of research has shown that parental psychopathology, including SUDs, depression, and antisociality, may increase risk for offspring trauma exposure (Bromet et al., 1998; Koenen et al., 2002), PTSD (Brewin et al., 2000; Bromet et al., 1998; Ozer et al., 2003), and SUDs (Goodman & Gotlib, 1999; Merikangas et al., 1998; Sher, 1991; 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 (e.g., Dube et al., 2003), which may further increase risk for trauma exposure and post-trauma psychopathology (e.g., Brewin et al., 2000; Buka, Stichik, Birdthistle, & Earls, 2001; Deykin & Buka, 1997; Koenen, Moffitt, Poulton, Martin, & Caspi, 2007).

Adverse family environments may lead to post-trauma maladjustment by sensitizing individuals such that future stressors have more detrimental effects (Koenen et al., 2007). Indeed, animal research suggests that offspring who are reared under stressful conditions exhibit long-term changes in hypothalamic-pituitary-adrenal axis functioning (Heim & Nemeroff, 2002) and abnormal secretions of stress hormones (De Bellis, 2002). These changes to the body's stress response systems are linked with deficits in executive functioning, self-regulation, and impulse control, which may increase risk for both PTSD and SUDs following trauma exposure (De Bellis, 2002; Yehuda, 2002). Moreover,

when faced with a traumatic event, adolescents from adverse family environments are less likely to have the resources and supports necessary for effectively coping. Therefore, the familial backdrop against which trauma occurs is likely to be a key determinant of post-trauma functioning.

Importantly, adolescents who grow up in such adverse family environments are also more likely to display preexisting risk factors themselves (Koenen et al., 2002), which may further increase risk for trauma exposure and post-trauma maladjustment. Adolescents who grow up in high-conflict, highstress families are more likely to misuse alcohol and drugs (e.g., Guo, Hill, Hawkins, Catalano, & Abbott, 2002; Repetti, Taylor, & Seeman, 2002; Zhou et al., 2006), regardless of trauma exposure or PTSD. Therefore, in order to disentangle the directions of influence among traumatic stress, PTSD, and problematic substance use, it is important to control for both preexisting adolescent substance use and the confounding influence of the larger constellation of family adversity.

Because so few studies contain pre-trauma measures of risk factors, it is currently unclear whether trauma exposure and/or PTSD influence the development of alcohol and drug problems over and above the influence of preexisting family risk factors and baseline levels of substance use. Studies that test the extent to which trauma exposure and/or PTSD mediate the influence of preexisting risk factors on alcohol and drug problems will help to clarify whether the link between PTSD and problem substance use is causal or, instead, due to shared risk factors.

Potential Moderators of Risk Pathways

Gender

The fact that the majority of trauma-exposed individuals do not appear to experience major post-trauma maladjustment points to the importance of considering moderators of these mechanisms of risk. Given numerous gender differences in both the trauma/PTSD and substance use literatures, it is likely that gender may moderate these pathways. It is well-established that males are at elevated risk for alcohol and drug problems compared to females (e.g., Brady & Randall, 1999). It is also well-established that females are twice as likely as are males to develop PTSD during their lifetime, even though males are at greater risk for experiencing a traumatic event (Kessler et al., 1995; Kimerling, Ouimette, & Wolfe, 2002). These findings have held true in studies regardless of the population, type of assessment, age of participants, and other methodological variables (Tolin & Foa, 2006).

Some evidence suggests that substance use may be associated with greater risk for trauma exposure in females than in males (e.g., Windle, 1994). Therefore, it is likely that gender may moderate the influence of substance use problems on risk for trauma exposure (i.e., the high-risk hypothesis). However, is also possible that a *mediational* relation exists among gender, substance use problems, and trauma exposure, such that the influence of gender on trauma exposure is mediated by substance use problems. Given that males are more likely than are females to use/abuse alcohol and drugs (Chan, Neighbors, Gilson, Larimer, & Marlatt, 2007; Johnston, O' Malley, Bachman, & Schulenberg, 2009), and that males are also at greater risk for trauma exposure compared to females (Kessler et al., 1995), higher levels of substance use problems among males may be one mechanism through which the gender difference in trauma exposure occurs. This hypothesis has not been previously tested.

Many theories have been offered to explain females' greater vulnerability for PTSD. The most widely investigated explanation is that females are at greater risk for PTSD because they are more likely than are males to be exposed to traumatic events that are especially likely to result in PTSD, such as rape. However, it appears that rape has a particularly high probability of eliciting PTSD in both male and female victims, and that the gender difference in PTSD cannot be attributed to females' higher rates of rape or sexual assault (Breslau & Anthony, 2007). Research has also ruled out the possibility that the gender difference in the conditional risk of PTSD can be attributed to prior traumatic experiences or gender differences in reporting of traumatic events or PTSD symptoms (Breslau, 2009). Still, females appear to be at particularly high risk for developing PTSD following exposure to assaultive violence, although assaultive violence is more common among males (Breslau & Anthony, 2007).

Considering these findings together, it has been suggested that trauma exposure-related factors may account for only a small portion of the marked gender difference in risk for PTSD (Olff, Langeland, Draijer, & Gersons, 2007). Therefore, it is important to consider other explanations to explain the gender gap in PTSD. For example, given that rates of depression are higher among females than males, it has been proposed that gender differences in preexisting

internalizing symptomatology (i.e., depressive or anxiety disorders) may account for gender differences in PTSD. However, this explanation has also failed to garner substantial empirical support (Breslau, 2009; Olff et al., 2007). Psychobiological models indicate that females tend to have more sensitive dissociative stress response systems, which may contribute to greater peritraumatic dissociation—a known risk factor for PTSD (Olff et al., 2007). Although there are also numerous sex differences in HPA-axis functioning and other neurotransmitter and neurohormone systems that may contribute to the gender difference in PTSD, there is currently a lack of research on this issue (Olff et al., 2007).

Cognitive models of PTSD development suggest that females may be at greater risk for PTSD compared to males because females are more likely to blame themselves for the traumatic event, hold more negative views of themselves after trauma exposure, and to view the world as more dangerous after trauma exposure (Tolin & Foa, 2002). It is possible that females may be especially likely to blame themselves when substance use is involved in trauma exposure. For instance, females who are sexually assaulted when under the influence of alcohol may believe that is their own fault for putting themselves in a vulnerable position. This self-blame tendency may predispose female substance users to developing PTSD relative to male substance users. If this hypothesis were true, one would expect gender to moderate the influence of preexisting substance use on PTSD risk (i.e., the susceptibility hypothesis). This hypothesis awaits empirical investigation.

Given the lower rates of PTSD in males compared to females, it has been hypothesized that males may react to trauma exposure with externalizing behaviors and/or substance abuse, whereas trauma-exposed females tend to develop PTSD and other internalizing psychopathologies (e.g., Green et al., 1997). Although some research has shown that physically or sexually abused boys are more likely than physically or sexually abused females to display violent and aggressive behavior and to have conduct disorders (Darves-Bornoz, Choquet, Ledoux, Gasquet, & Manfrei, 1998; Livingston, Lawson, & Jones, 1993), this research is confounded by base rate differences in rates of psychopathology. In addition, Breslau and colleagues (2003) found that trauma-exposed men without PTSD were not at greater risk for SUDs compared to men without trauma exposure, which argues against the hypothesis that men tend to respond to traumatic experiences by abusing alcohol and drugs rather than developing PTSD. Although support for this hypothesis is currently lacking, future studies with both pre- and post-trauma measures may provide further insight into this issue by controlling for base rate gender differences in substance use.

Interestingly, although males are at higher risk for SUDs in the general population and have higher levels of lifetime comorbid PTSD and SUDs, it appears that females are at higher risk for developing SUDs that occur following PTSD onset (Stewart, Ouimette, & Brown, 2002). Said differently, females are more likely to experience the form of PTSD-SUD comorbidity in which PTSD occurs *first*. Therefore, PTSD appears to place women at higher risk for developing PTSD-SUD comorbidity compared to males. Both adolescent and
adult females appear to be more likely to start using substances excessively following trauma exposure compared to males (e.g., Deykin & Buka, 1997; Lipschitz, Grilo, Fehon, McGlashan, & Southwick, 2000; Stewart et al., 2002). Research has suggested that females are more likely to use emotion- and avoidance-focused coping strategies, as well as palliative behaviors like drinking, which places females at higher risk for developing both PTSD and SUDs relative to males (Olff et al., 2007).

Finally, there is also some evidence to suggest that females may be at greater risk for specifically developing *alcohol* problems following trauma exposure compared to males (Breslau et al., 1997; Breslau et al., 2003). As previously discussed (see section on the self-medication hypothesis), it generally appears that PTSD is more closely linked with the development of drug problems than alcohol problems. However, it is possible that the relatively weaker association between PTSD and future alcohol problems may be an artifact of gender moderation in this relation. In other words, trauma exposure and PTSD may increase risk for drug problems in both genders, but may increase risk for alcohol problems only for females after controlling for pre-trauma alcohol problems. Future studies are needed to test how the link between PTSD and both alcohol and drug problems may differ for males and females.

Ethnicity

Studies indicate a complex pattern of findings regarding ethnocultural differences in trauma exposure and PTSD. Within the United States, some studies have indicated that minority ethnicities are more likely to be exposed to traumatic

events compared to Caucasians, whereas others indicate either that there are no differences in rates of exposure or that these differences disappear after accounting for other risk factors (Bromet et al., 1998; Kessler et al., 1995; Pole, Gone, & Kulkarni, 2008). Although many studies also suggest that ethnic minority status is a risk factor for PTSD following trauma exposure, metaanalyses (Brewin et al., 2000) indicate that this increased risk for PTSD may be quite small (effect size= .05, which is below Cohen's (1988) threshold of .1 for a small effect size).

Hispanic/Latino ethnicity appears to be the exception to this trend. Strong evidence indicates that Hispanics/Latinos experience more severe PTSD symptoms and a higher probability of developing PTSD compared to other ethnicities across a multitude of trauma types (Galea et al., 2002; Kulka et al., 1990; Ortega & Rosenheck, 2000; Pole, Best, Metzler, & Marmar, 2005; Schnurr, Lunney, & Senqupta, 2004). Although Hispanics/Latinos also experience greater exposure to traumatic stress than do non-Hispanics/Latinos, it appears that various cultural, social, and religious factors, rather than differential exposure to traumatic stress, account for Hispanics'/Latinos' higher rates of PTSD (Kulka et al., 1990; Perilla, Norris, & Lavizzo, 2002; Pole et al., 2005). Indeed, research has found that greater peritraumatic dissociation, self-blame coping, greater wishful thinking, and perceived racism may account for Hispanics'/Latinos' increased vulnerability to PTSD (Pole et al., 2005). Increased tendency toward somatization among Hispanics/Latinos has also been posited to account for Hispanics'/Latinos' increased risk for PTSD (Pole et al., 2005), given that PTSD

symptoms are highly comorbid with somatic complaints (Andreski, Chilcoat, & Breslau, 1998; van der Kolk, Pelcovitz, Roth, & Mandel, 1996). It has been also theorized that acculturation, which has been shown to increase risk for a range of psychopathologies (Ortega, Rosenheck, Alegria, & Desai, 2000), may account for Hispanics'/Latinos' increased risk for PTSD (e.g., by diminishing traditional familial support systems). However, Ortega and Rosenheck (2000) did not find an association between level of acculturation and PTSD symptoms in a sample of Hispanic/Latino Vietnam War veterans. In fact, Marshall and Orlando (2002) found high levels of acculturation among Hispanics/Latinos were associated with *decreased* risk for peritraumatic dissociation, which may protect against risk for PTSD. Therefore, current research on the role of acculturation in Hispanics/Latinos' risk for PTSD is inconclusive.

In contrast to PTSD, research indicates that non-Hispanic/Latino Caucasians are more likely to use substances and are at greater risk for SUDs compared to Hispanics/Latinos and other minority ethnicities (Substance Abuse Mental Health Services Administration, 2007). Lower rates of substance use among Hispanics/Latinos may be due to protective factors that tend to be more concentrated in Hispanic/Latino ethnic groups, such as *familism* (Vega, Zimmerman, Gil, Warheit, & Apospori, 1993). Interestingly, higher levels of acculturation increase risk for substance use behaviors among Hispanic/Latino youth and adults, particularly females, (Gfroerer & De La Rosa, 1993; Caetano & Clark, 2003), suggesting that acculturation may erode some of these protective factors. Although it appears that Hispanics/Latinos are generally at lower risk for substance use problems than are non-Hispanic/Latino Caucasians, little research has examined whether there may be ethnocultural differences in the development of substance use problems in the context of trauma exposure and post-trauma adjustment.

Given that Hispanics/Latinos are at elevated risk for PTSD, one might hypothesize that trauma-exposed Hispanics/Latinos also are at increased risk for developing SUDs or other psychopathologies following a traumatic event. That is, Hispanics/Latinos may be at greater risk for a wide range of post-trauma psychopathologies, including SUDs. Alternatively, it is possible that non-Hispanic/Latino Caucasians, whose risk for PTSD is relatively lower than that of Hispanics/Latinos (Galea et al., 2002), are more likely to respond to a traumatic event by developing substance use problems rather than PTSD. If this latter hypothesis were true, trauma-exposed non-Hispanic/Latino Caucasians would have an elevated risk for developing substance use problems in the absence of PTSD, relative to unexposed non-Hispanic/Latino Caucasians (but this effect of trauma exposure without PTSD on substance use problems would be relatively weaker or nonexistent for Hispanics/Latinos). Note that these two hypotheses are in opposition to one another. Further research is needed to determine whether the types of negative outcomes that are most likely to occur following trauma exposure vary across ethnicity.

The tendency to self-medicate PTSD symptoms with alcohol or drugs may also vary across ethnicity. Indeed, a recent large (N= 43,093) epidemiological study found that Hispanics/Latinos and African Americans were less likely to use alcohol and drugs to self-medicate anxiety disorders other than PTSD (panic disorder, social phobia, specific phobia, and generalized anxiety disorder), whereas Caucasians and Native Americans were more likely to do so (Robinson, Sareen, Cox, & Bolton, 2009). Hispanics/Latinos are also significantly less likely to self-medicate *mood* disorders compared to other ethnicities (Bolton, Robinson, & Sareen, 2009). However, an additional study that specifically examined the use of alcohol and drugs to self-medicate PTSD symptoms did not find any differences in the tendency to self-medicate between Hispanics/Latinos and other ethnicities¹ (Leeies, Pagura, Sareen, & Bolton, 2010). Although these results tentatively suggest that Hispanics/Latinos may be somewhat less likely to self-medicate with alcohol and drugs compared to other ethnicities, it should be noted that each of these studies was cross-sectional, and so the directions of effects among PTSD, self-medication tendencies, and substance use behaviors could not be determined.

Together, these studies make it clear that Hispanic/Latino ethnicity is an important moderator to consider in models of post-trauma adjustment. More research is needed to determine whether the influence of PTSD symptoms on risk for substance use problems varies across ethnicity, particularly Hispanic/Latino ethnicity.

The Present Study

The purpose of the present study was to better understand the risk pathways that link trauma exposure, PTSD, and substance use problems. In

¹ Black non-Hispanics were significantly less likely to self-medicate PTSD symptoms compared to other ethnicities.

addition, this study aimed to understand the role of gender and ethnicity in these pathways. This study tested the following hypotheses, which are not mutually exclusive:

- High-risk hypothesis (Figure 1): This study tested whether adolescent substance use problems predict increased risk for trauma exposure or assaultive violence exposure over and above the influence of family risk factors and demographic predictors. Note that trauma exposure and assaultive violence exposure were examined as separate outcomes, given evidence that adolescents with substance use problems may be at particularly high risk for traumatic events involving assaultive violence (Giaconia et al., 2000; Kilpatrick et al., 2000). Adolescent substance use problems were expected to have a significant unique effect on trauma exposure, particularly assaultive violence exposure (see bolded path in Figure 1.1). Several specific hypotheses pertaining to the roles of gender and ethnicity within the high-risk hypothesis were also tested:
 - 1a. This study tested whether gender influences risk for trauma exposure or assaultive violence. It was hypothesized that males would be at significantly higher risk for trauma exposure and assaultive violence compared to females.
 - 1b. This study tested whether gender moderates the influence of adolescent substance use problems on risk for trauma exposure or assaultive violence exposure. It was hypothesized that

adolescent substance use problems would be a significantly stronger predictor of trauma exposure for females than for males. See dotted path in Figure 1.1.

- 1c. This study tested whether adolescent substance use problems mediate the influence of gender on risk for trauma exposure or assaultive violence exposure. It was hypothesized that males would exhibit higher levels of adolescent substance use problems, which, in turn, would increase risk for trauma exposure. See Figure 1.2.
- 1d. This study tested whether ethnicity influences risk for trauma exposure or assaultive violence. It was hypothesized that Hispanics/Latinos and ethnic minorities (i.e., non-Caucasians) would be at elevated risk for trauma exposure compared to Caucasians.
- 2. Susceptibility hypothesis (Figure 2): This study tested whether adolescent substance use problems predict increased risk for PTSD or PTSD symptoms among individuals exposed to trauma over and above the influence of family risk factors and demographic predictors (see bolded path in Figure 2). PTSD was examined as both a categorical variable (presence or absence or a PTSD diagnosis) and as a count variable (count of the number of PTSD symptoms). This approach addresses the individual's response to the traumatic event on a continuum of severity, as well as the categorical presence or absence

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of PTSD. The roles of gender and ethnicity within the susceptibility hypothesis were also tested:

- 2a. This study tested whether gender influences risk for PTSD or PTSD symptoms. It was hypothesized that females would be at higher risk compared to males.
- 2b. This study tested whether gender moderates the influence of pre-trauma substance use on risk for PTSD diagnosis/symptoms (i.e., the susceptibility hypothesis). It was hypothesized that substance use problems may make traumaexposed females especially susceptible to developing PTSD relative to trauma-exposed males. See dotted path in Figure 2.
- 2c. This study tested whether Hispanic/Latino ethnicity influences risk for PTSD diagnosis/symptoms. It was hypothesized that Hispanic/Latino participants would be at elevated risk for PTSD relative to non-Hispanic/Latino participants.
- 3. Self-medication hypothesis (Figures 3 and 4—each figure corresponds with a different analytic approach): This study tested whether PTSD predicts increased risk for future alcohol and/or drug problems over and above the influences of trauma exposure, pre-trauma substance use problems, demographic predictors, and family risk factors that are common to both PTSD and alcohol/drug problems. PTSD was examined as both a count variable (see Figure 3) and a categorical variable (see Figure 4) when testing this hypothesis. It was

hypothesized that PTSD diagnosis/symptoms would have a significant effect on future substance use problems, particularly drug problems, over and above the influences of trauma exposure, pre-trauma substance use problems, demographic predictors, and family risk factors (see path labeled H3 in Figures 3 and 4).

- 3a. This study tested whether gender moderated the influence of PTSD or PTSD symptoms on future alcohol and drug problems. It was hypothesized that females would be more likely to self-medicate symptoms of PTSD compared to males. Therefore, the unique influence of PTSD on future substance use problems—particularly alcohol problems—was expected to be significantly stronger for females than for males.
- 3b. This study tested whether ethnicity moderates the influence of PTSD or PTSD symptoms on substance use problems. Given a lack of conclusive research on this issue, no specific hypothesis was made regarding the direction of the moderated effect.
- 4. Shared vulnerability hypothesis (Figure 3 and 4—each figure corresponds with a different analytic approach): This study tested the extent to which shared risk factors increase risk for both PTSD and alcohol/drug problems. Specifically, this study examined a cluster of related family risk variables—family conflict, stress in the family environment, parental alcoholism, and other parent psychopathology—that are associated with increased risk for both PTSD and SUDs. Note

that these variables are likely to reflect a combination of both genetic and environmental risk. This study tested the extent to which preexisting family risk factors exert unique and direct effects on trauma exposure, PTSD symptoms, and adult substance use problems (see the three bolded paths in Figure 3 that stem from family adversity; Figure 4 is not relevant). This study also tested the extent to which the influence of preexisting family risk factors on adult alcohol and drug problems was mediated by trauma exposure and/or PTSD symptoms (the bolded paths in Figure 3 indicate paths that are involved in these meditational chains). It was hypothesized that PTSD symptoms would *partially* mediate the influence of preexisting family risk factors on future substance use problems.

This study also tested whether trauma exposure may be conceptualized as a shared risk factor for both PTSD and substance use problems, such that trauma exposure increases risk for alcohol or drug problems, independent of PTSD. If the *"trauma exposure as a shared risk factor hypothesis"* were true, individuals who were exposed to a traumatic event but did not develop PTSD would be at elevated risk for future substance use problems relative to unexposed individuals (see path labeled H4_{trauma as shared risk} in Figure 4). This hypothesis would also be supported if trauma exposure directly influenced future substance use problems over and above the influence of PTSD symptoms (see path labeled H4_{trauma as shared risk} in Figure 3).

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This study also tested whether gender and/or ethnicity moderate the trauma exposure as a shared risk factor hypothesis:

- 4a. This study tested whether gender moderates the direct
 influence of trauma exposure on problematic alcohol or drug
 use. It was hypothesized that trauma-exposed males without
 PTSD would be at higher risk for future alcohol and drug
 problems compared to unexposed males. This effect was not
 expected for females.
- 4b. This study conducted an exploratory analysis testing whether ethnicity moderates the influence of trauma exposure on problematic alcohol or drug use. Previous research has not examined how risk for post-trauma substance use problems may vary for Hispanics/Latinos and non-Hispanics/Latinos. There is theoretical rationale to predict that Hispanics/Latinos would be at elevated risk for post-trauma substance use problems relative to Caucasians, and also that Caucasians would be at higher risk for post-trauma substance use problems relative to Hispanics/Latinos. Given that these hypotheses are in direct opposition to one another, no specific hypothesis was made regarding the direction of the moderated effect.

These hypotheses were tested using data from a longitudinal study of familial alcoholism, which follows participants from early adolescence to adulthood and contains measures of both pre- and post-trauma functioning. By using a high-risk sample with elevated prevalence of risk factors, traumatic stress, and substance use problems, the present study was particularly well-suited for examining the hypothesized pathways.

Method

The Original Study

Participants. Participants for the current study were drawn from a larger longitudinal study of familial alcoholism across three generations (e.g. Chassin et al., 1991). The original study had 3 annual waves of data collection and 3 additional follow-ups separated by 5 years. At Wave 1 (1988), the total sample consisted of 454 "target" adolescents and their parents; 246 adolescents had at least one biological alcoholic parent who was also a custodial parent (COAs), and 208 adolescents were demographically matched controls without an alcoholic parent. Sample retention was excellent at all follow-ups, with 99% (*n*= 449) of the original targets interviewed at Wave 2, 98% (*n*= 444) interviewed at Wave 3, 90% (*n*= 407) interviewed at Wave 4, 91% (*n*=415) interviewed at Wave 5, and 90% (*n*= 409) interviewed at Wave 6.

Recruitment. COA families were recruited by using court records, health maintenance organization (HMO) wellness questionnaires, and community telephone surveys. Records from seven court systems were used to identify individuals who were convicted of driving while intoxicated between 1984 and 1988. To qualify for the study, individuals had to live in Arizona, be of non-Hispanic/Latino Caucasian or Hispanic/Latino ethnicity, and be born between 1926 and 1960. These court records were further examined for potential indicators of alcoholism, such as a blood alcohol content of at least .15 at the time of the arrest, prior alcohol-related arrests, a score of seven or higher on the Michigan Alcohol Screening Test (Selzer, 1971), or a diagnosis of probable

alcoholism by a substance abuse screening center. A total of 103 COA families were obtained by reviewing court records.

An additional 22 COA families were identified by reviewing HMO wellness questionnaire responses of members who joined a large between 1986 and 1988. New members who were arrested between 1984 and 1988 and met the aforementioned demographic criteria were examined for the following indications of alcoholism: consumption of 26 or more alcoholic drinks per week, reporting three or more alcohol-related social consequences, or self-labeling as an alcoholic. An additional 120 COA families were obtained by conducting community telephone surveys. Families who met demographic criteria to be in the study were assessed for alcoholism with the following criteria: attendance at Alcoholics Anonymous meetings, hospitalization for a drinking problem, or reporting that one's spouse had been an alcoholic. In addition, one COA family was referred by a local Veteran's Administration outpatient treatment program. Finally, 80 COA families were families who had originally been screened to be part of the demographically matched control group, but met diagnostic criteria for alcoholism.

Parental alcoholism was directly verified during a face-to-face structured diagnostic interview using the DIS-III (Robins, Helzer, Croughan, & Ratcliff, 1981), allowing for diagnoses of lifetime alcohol abuse or dependence to be made using DSM-III criteria. Interviews were conducted with the alcoholic parent unless they refused to participate, in which case he or she was diagnosed based on spousal report using the Family History-Research Diagnostic Criteria (FH-RDC, Endicott, Andreason, & Spitzer, 1975). In all, 219 biological fathers and 59 biological mothers met DSM-III diagnostic criteria for alcoholism.

Matched non-alcoholic families (matched on child's age, family composition, ethnicity, and socioeconomic status) were recruited by using reverse directories to find families living in the same neighborhoods as the COA families. The primary criterion for inclusion was that no biological or custodial parents met diagnostic criteria for a lifetime diagnosis of alcohol abuse or dependence according to the DSM-III or FH-RDC lifetime diagnosis of alcohol abuse or dependence. Seventeen families reported indicators of alcohol problems that were close to the diagnostic threshold, and were thus eliminated from the study in order to decrease the chance that the target parent would be diagnosed alcoholic later in the project.

Recruitment biases. There were two main sources of potential recruitment biases for the longitudinal study: selective contact with COA participants and refusal to participate in the study (Chassin, Barrera, Bech, & Kossak-Fuller, 1992). In order to assess the impact of selective contact, the court and HMO records of participants who were successfully contacted were compared to those who were not. Note that no archival data were available for the other participants. Based on t-test and chi square analyses, potential participants who were contacted did not differ from those who were not contacted with respect to blood alcohol level at time of arrest, number of prior alcohol-related arrests, self-labeling as alcoholic, or MAST scores. However, compared to contacted potential participants, those who were not contacted were significantly more

likely to be younger, from court sources of Hispanic/Latino ethnicity, unmarried, and were more likely to have a lower socioeconomic status (SES) rating associated with their residence.

The second source of potential recruitment bias was refusal to participate. Out of families screened by telephone contacts, 73% of COA families and 77% of control families participated. Individuals who refused to participate did not differ significantly from participants on age, sex, SES, or alcoholism indicators. However, people who refused to participate were significantly more likely to be Hispanic/Latino and married, but did not significantly differ from participants on age, sex, SES, or alcoholism indicators

Refusal bias for the control sample was estimated by comparing participating control families to the 91 potential families who provided demographic information during the initial phone screening but refused to participate. No differences were found in family composition or SES ratings of their residence. However, both mothers and fathers who refused to participate were more likely to be Hispanic/Latino (41% versus 18% for mothers and 40% versus 22% for fathers) than were those who agreed to be interviewed. For more information on possible bias in contact and recruitment samples, see Chassin et al. (1992).

Procedure. The Adolescent and Family Development Project was explained to families as a study supported by the National Institute on Drug Abuse that was designed to explore reasons why certain adolescents develop problems, such as alcohol and drug problems, whereas others do not. Although parental alcoholism was not mentioned as a selection criterion, participants were informed that they would be interviewed about drug and alcohol use.

After parents provided informed consent and adolescents provided assent, interviews were conducted either at the family's residence or at the Arizona State University campus. Trained interviewers used laptops to read items aloud to participants, who could either enter responses themselves using a laptop computer or respond verbally and allow interviewers to enter the data. To increase privacy of responses and avoid threats of contamination, family members were usually interviewed simultaneously in separate room. In addition, a Department of Health and Human Services Certificate of Confidentiality was used to emphasize confidentiality.

The Present Study

Participants. This study utilized data from Waves 1, 4 and 5 of the larger parent project. See Figure 5 for a summary of the measures used from each of these waves. At the 4th assessment (Wave 4; N=407), which occurred 7 to 10 years after the initial assessment, participants were asked about their history of trauma exposure and PTSD symptoms. Those who experienced a traumatic event were asked about their age when the event occurred. In order to ensure prospective prediction of trauma exposure, the current study excluded 29 participants who reported experiencing a traumatic event at an age that was earlier than their age at the initial Wave 1 interview. One participant who responded "don't know" when asked his or her age when the traumatic event occurred was also excluded. Participants (n=47) who were not interviewed at Wave 4 were also

excluded because it could not be determined if/when traumatic stress occurred, which is of central importance to hypothesis testing. Thus, this study contained data that preceded trauma exposure for all participants (*n*= 377; 54% male; 52% COAs; 73% non-Hispanic/Latino Caucasian; 22% Hispanic, 5% other ethnicity; mean age=13.2 at Wave 1, 20.4 at Wave 4, and 25.6 at Wave 5), thereby enabling prospective prediction of trauma exposure and problematic alcohol and drug use from preexisting risk factors. Because the 29 participants who reported trauma exposure prior to their age at Wave 1 were excluded from the sample, early patterns of repeated or chronic exposures to trauma are beyond the scope of this study and were not examined. Rates of trauma exposure (48.2%) and PTSD (10.1%) prior to excluding the 29 participants with early trauma exposure were slightly higher than those reported in similarly aged community samples (e.g., Giaconia, Reinherz, Silverman, & Pakiz, 1995), likely reflecting the high-risk nature of this sample.

Chi-square and t-test analyses compared differences on study variables (see Measures) between participants who were included in the present study (n= 377), compared to the 77 participants from the original Wave 1 sample (n=454) who were excluded from this subsample. As expected by exclusion of participants with early trauma, included participants were significantly more likely than were excluded participants to be children of non-alcoholic parents (χ^2 =6.65, p <.05) and to have lower levels of familial life stress (t = 2.16, p <.05) at the original data collection. However, included and excluded participants did not differ on gender, ethnicity, parental psychopathology other than alcoholism,

family conflict at the original data collection, adolescent substance use problems at the original data collection, adult (Wave 5) alcohol problems, or adult drug problems.

Measures

All measures used in the current study were obtained from the larger interview battery. See Figure 5 for depiction of measures at each wave. Descriptive statistics for all variables are displayed in Table 1. Correlations are displayed in Table 2. Note that the current study included several count variables. Because Pearson correlations are not valid for count variables², all count variables in Table 2 were log-transformed (log (count + 1)) before estimating correlations.

Adolescent gender. Gender was dummy coded such that "0" indicates male (n=202; 53.6%), and "1" indicates female (n=175; 46.4%).

Adolescent ethnicity. Adolescents were asked to pick the best description of their ethnicity from the following options: Caucasian (not Hispanic); Hispanic; Asian, Oriental or Pacific Islander; American Indian; Black or Afro-American; or Other. There were 275 (72.9%) non-Hispanic/Latino Caucasian participants, 84 (22.3%) Hispanic/Latino participants, and 18 (4.8%) participants of other ethnicities. A dichotomized adolescent ethnicity variable was computed such that "0" indicates non-Hispanic/Latino Caucasian (n= 275; 72.9%), and "1" indicates other ethnicities (n= 102; 27.1%). Note that 84 (82.4%) of these 102 participants

² Although commonly reported, Pearson-product moment correlations are inappropriate for count variables due to their limited range (non-negative integer values). Polychoric or polyserial correlations are also inappropriate because there is not an underlying continuous response variable formulation for count variables. Therefore, this study used log-transformations for count variables in order to approximate zero-order correlations with study variables. Using log-transformations is not the optimal method for analyzing count data; we use it only to provide a more accurate estimate of the bivariate associations between count variables and other study variables.

were Hispanic/Latino (most were Mexican-American). Note that for all hypotheses involving ethnicity, follow-up analyses dropped the18 participants who were of ethnicities other than Hispanic/Latino or non-Hispanic/Latino Caucasian in order to allow for a clearer examination of the effect of Hispanics/Latino versus Caucasian ethnicity.

Parent education. At Wave 1, parents reported on their highest level of education with the following response options: 1 = grade school, 2 = some high school, 3 = high school graduate, 4 = technical school, 5 = some college, 6 = college graduate, and 7 = graduate school/professional school. The current study used the highest education level achieved by either the mother or father as an indicator of socioeconomic status (SES) because previous studies have shown that it is the most sensitive and stable indicator of SES risk in adolescent health research (Krieger, Williams, & Moss, 1997; Williams & Collins, 1995).³ The highest level of parent education was grade school for 1.3% of participants, some high school for 6.4% of participants, a high school degree for 17.2% of participants, technical school for 7.2% of participants, and graduate school or professional school for 14.9% of participants.

³ Parental income at Wave 1 was also considered as a potential proxy for SES (note that parental income was significantly associated with parental education; r = .395, p < .001). However, zero-order correlations indicated that parent income was not significantly related to any study variables. The correlations between parent income and study variables were also examined separately by ethnicity and were again non-significant, with only one exception (parental income was correlated with adult alcohol problems for non-Hispanic/Latino Caucasian participants). In contrast, parent education was significantly associated with several study variables (see Table 2), and appeared to better capture risk associated with socioeconomic status. Therefore, parental education rather than parental income was tested as a potential covariate in the present study.

Adolescent substance use problems. Adolescent reported on consequences and dependence symptoms that they may have experienced due to their alcohol and drug use at Wave 1. The present study used 19 items to assess 14 different types of alcohol problems⁴ at Wave 1, collapsing across items within the same domain. See Appendix A for a list of substance use problems and corresponding items. Each problem was coded as 0 or 1, with a 1 indicating that the adolescent experienced the problem due to alcohol or drug use at some point in his/her lifetime. A summary count variable indicating the total number of adolescent substance use problems was computed.

Because of the young age of participants, the overall prevalence of substance use and substance use problems was generally low at Wave 1. There were 160 (42.4%) adolescents who reported ever drinking or using drugs at Wave 1, and 60 (15.9%) adolescents who reported experiencing at least one lifetime substance use problem. Given the low frequency of adolescents with high counts of substance use problems, analyses used an ordered categorical variable that was coded 0 if the adolescent reported no lifetime substance use problems (n=317; 84.1%), 1 if the adolescent reported 1 lifetime substance use problem (n=23; 6.1%), and 2 if the adolescent reported 2 or more lifetime substance use problems (n=37; 9.8%). Analyses modeled the effects of substance use problems, rather than substance use itself, because substance use problems were expected to be more prognostic of future risk for trauma exposure, PTSD, and adult substance

⁴ The term "problem" is used to denote either a consequence or dependence symptom due to substance use.

use problems.⁵ That is, adolescents who were using alcohol or drugs to such an extent that they were already experiencing social consequences or physical dependency symptoms were theorized to exhibit a high-risk substance use style that may place them at risk for trauma exposure, PTSD, and/or substance use problems. Moreover, modeling the effects of substance use problems allowed us to be longitudinally consistent when predicting adult alcohol and drug problems.

Parental alcoholism. Parents' lifetime DSM-III diagnoses of alcohol disorder (abuse or dependence) were assessed at Wave 1 by direct interview using the Diagnostic Interview Schedule (CDIS, Version 3; Robins et al., 1981). For non-interviewed parents, lifetime alcoholism diagnoses were established using Family History-Research Diagnostic Criteria (FH-RDC, Version 3; Endicott et al., 1975) based on spouse's report. Parental alcoholism was coded 1 for participants who had at least one biological parent with an alcohol disorder who was also a custodial parent (n=194; 51.5%) and 0 for those with no biological or custodial parents with an alcohol disorder (n=183; 48.5%).

Other parental psychopathology. Parents' lifetime DSM-III diagnoses of affective disorder (major depression or dysthymia) and antisocial personality disorder (ASPD) were assessed at Wave 1 by direct interview using the CDIS-III (Robins et al., 1981). Parental anxiety disorders (panic disorder, agoraphobia,

⁵ Additional analyses modeled the effects of frequency of binge drinking in the past year, frequency of getting drunk on alcohol in the past year, and frequency of marijuana use in the past year (the most commonly used illegal drug in this study) on risk for trauma exposure or assaultive violence exposure. Similar to the main analyses using the adolescent substance use problems variable, neither binge drinking nor getting drunk on alcohol 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. Frequency of getting drunk on alcohol and frequency of marijuana use had marginally significant unique effects on risk for assaultive violence exposure.

generalized anxiety disorder, social phobia, simple phobia, and PTSD) were not assessed at Wave 1, but were assessed at Wave 4 using DSM-III-R criteria via the DIS-III-R (Robins, Helzer, Cottler, & Golding, 1989). Because parents reported their age of onset of symptoms, it was possible to determine whether onset of anxiety disorders occurred prior to Wave 1. Parents who met criteria for simple phobia but no other anxiety disorder diagnoses were not considered to have an anxiety disorder. A dichotomous summary variable was computed to indicate whether the participant had at least one parent who met criteria for an affective disorder (n= 52; 13.8%), anxiety disorder (n= 111; 29.4%), or ASPD (n= 28; 7.4%) at Wave 1. This variable was coded "1" for the 149 (39.5%) adolescents who had a parent with one of these disorders at Wave 1, and was coded "0" for the 228 (60.5%) adolescents who had no parents with any of these disorders.

Adolescent's family conflict. Self- and parent-report of family conflict during the past 3 months was assessed at Wave 1 using Bloom's (1985) Family Process Scale (BFPS). The BFPS has been widely used in research and its psychometric properties have been well established (see Bloom & Naar, 1994, for a review). Adolescents and parents reported on 5 items. However, one of these items assessed the extent to which family members hit each other, and was not used in the present study in order to avoid potential overlap with the measure of trauma exposure. The 4 items (range: 1-5) that were used in the present study assessed the extent to which family members fought a lot, got so angry they threw things, lost their tempers, and criticized each other. Cronbach's alpha was .62 for adolescent report, .63 for mother report, and .63 for father report. Adolescent-

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reported family conflict was significantly correlated with both mother-reported (r=.33, p < .001) and father-reported family conflict (r=.37, p < .001). Mother-reported and father-reported family conflict were also significantly correlated (r=.46, p < .001). Cronbach's alpha for the three reports (adolescents', mothers', and fathers') of family conflict was .66, suggesting that it may be appropriate to combine the three reports into a composite variable. In order to simplify analyses by using a single measure of family conflict, a composite family conflict score was computed by averaging adolescents', mothers', and fathers' reports of family conflict. When one of the parents was not interviewed, only the interviewed parent's report and the adolescent's report were averaged. The average family conflict score was 2.74 (*SD*= .60). High scores indicate high levels of family conflict.

Adolescent's familial life stress. At Wave 1, parents and adolescents reported the occurrence of stressful events in the adolescent's life during the past 3 months. Items were adapted from the General Stressful Life Events Schedule for Children (GLESC; Sandler, Ramirez, & Reynolds, 1986) and the Children of Alcoholics Stressful Life Events Schedule (COALES; Roosa, Sandler, Gehring, Beals, & Cappo, 1988). All items were previously rated as negative and uncontrollable life events from the perspective of the adolescent. Computing Cronbach's alpha for these events is inappropriate because the occurrence of many of the events assessed are assumed to be independent.

Parents and adolescents reported on a total of 15 events from the GLESC and 10 events from the COALES. Adolescents also reported on 4 additional

items about events involving their peers, but these items were also excluded from the present study because they do not pertain to the family environment. The purpose of the family stress variable was to capture familial stress that could potentially increase risk for PTSD or SUDs. It is important that this measure is distinct from traumatic stress, instead reflecting the backdrop against which traumatic stress may occur and thereby increase risk for PTSD or SUDs. Therefore, two items from the GLESC (whether the adolescent had been the victim of a crime⁶, and whether the adolescent had suffered a serious physical illness or injury) were not used for the present study because they could potentially reflect events that are perceived as traumatic. Moreover, only 2 items from the COALES were used in the present study because the other 8 items overlap with the measures of either parental alcoholism (you saw your mom or dad drunk; you saw your mom or dad drunk in public; you took care of your mom or dad when they were drunk) or family conflict (your mom and dad argued in front of you; mom or dad criticized things you've done well; your mom said bad things about your dad; your dad said bad things about your mom; your mom or dad screamed, shouted or broke things). Thus, parents and adolescents reported on a total of 15 events that were examined in the present study as reflecting familial life stress. Please see Appendix B for a list of these items. The present study coded items as having occurred if any reporter (adolescent, mother, or father) indicated that the event had taken place within the past three months (each

⁶ The item assessing whether the adolescent was a victim of a crime was also removed for the additional reason that it does not directly reflect stress in the family environment. Being a victim of a crime may be unrelated to stress in the family environment.

event could only be counted once). A summary count variable indicating the number of the number of stressful life events that the adolescent experienced was computed. On average, adolescents were exposed to 3.20 stressful events (*SD*= 2.36; range: 0-11).

Late adolescent/early-adult trauma exposure and PTSD. The computerized Diagnostic Interview Schedule (CDIS-III-R; Robins et al., 1989) was used to assess participants' lifetime exposure to trauma and consequent PTSD symptoms at Wave 4. DSM-III-R criteria were used to establish diagnoses of PTSD. Participants reported on up to 3 traumatic events, and were asked which one was the worst. In accordance with DSM-III-R criteria, events such as divorce or the natural death of a loved one were not considered to be qualifying

events that trauma-exposed participants reported experiencing separately for males and females (for participants who were exposed to more than one traumatic event, the event that was reported to be the worst is presented). For each traumatic event that was reported, 17 symptoms of PTSD were assessed.

traumas. Table 3 presents descriptive information on the types of traumatic

A number of summary variables were derived from the Wave 4 assessment of trauma exposure and PTSD symptomatology that were used in the present study. First, a dichotomous variable was created to indicate whether or not participants had experienced at least one traumatic event (n= 166; 44.0%). Second, a dichotomous variable was computed to indicate whether participants were exposed to a traumatic event that involved assaultive violence, such that analyses predicted risk for assaultive violence exposure compared to risk for not

being exposed to trauma or being exposed to other types of traumatic events. Rape, physical assault, and being threatened with a weapon were considered events that involved assaultive violence, based on methods used by other researchers (e.g., Breslau et al., 1998). There were 72 participants (19.1% of the overall sample and 43.4% of the trauma-exposed sample) who reported at least one traumatic event involving assaultive violence. Third, a trichotomous variable was created to indicate which of the following 3 categories participants belonged to: no trauma exposure (n=211; 56.0%), trauma exposure without PTSD (n=135; 35.8%), or trauma exposure with PTSD (n=31; 8.2%). Therefore, 18.7% of the trauma-exposed sample developed PTSD. Fourth, a dichotomous variable was computed to indicate whether or not trauma-exposed participants met criteria for PTSD. The term "PTSD diagnosis" is used throughout this document to refer to this variable. Fifth, a count variable was computed that indicated the total number of PTSD symptoms for whichever event the participant had the highest number of symptoms (mean= 5.41 symptoms, SD= 4.11). The term "PTSD symptoms" is used throughout this document to refer to this variable.

On average, less than 3 years (mean= 2.65, SD = 1.70; range: .1-7.97) elapsed between the time of the most recent traumatic event and the assessment of PTSD, thereby minimizing the likelihood of recall errors or bias. Age at exposure to the worst traumatic event (mean =17.40, SD= 1.93) was examined as a potential covariate in the present study. Time elapsed between age at Wave 4 (i.e., when trauma and PTSD were assessed) and age at exposure to the most recent traumatic event was also tested as a covariate. Finally, time elapsed between age at Wave 5 (i.e., when adult alcohol and drug problems were assessed) and age at most recent exposure was tested as a covariate when predicting adult alcohol and drug problems. Note that all age-related covariates were tested separately given that they are highly collinear with one another.

Adult alcohol and drug problems. Similar to the measure of adolescent substance use problems, participants reported on their consequences and dependence symptoms due to alcohol and drug use at Wave 5 (i.e., adulthood). There were 3 substance use problems assessed at Wave 5 that were not assessed during adolescence for a total of 17 substance use problems. Parallel to the adolescent measure, items within the same domain were collapsed in order to avoid double-counting the same type of substance use problems. See Appendix A for a list of problems and corresponding items. Follow-up questions assessed the recency of each consequence or dependence symptom separately for alcohol and drugs. Response options for recency probes were as follows: within the past 3 months, within the past year, 1-2 years ago, 2-5 years ago, more than 5 years ago, or never. These items regarding the recency of each alcohol or drug problem were used to determine whether each problem occurred within the past two years at Wave 5, separately for alcohol and drugs. Note that the two-year timeframe allowed for prospective prediction of adult alcohol and drug problems from PTSD symptoms.

Analyses employed summary count variables indicating the total number of adult alcohol problems and the total number of adult drug problems (separately) experienced in the past two years at Wave 5. Analyses examined risk for alcohol and drug *problems* based on evidence that PTSD symptoms are more closely linked with problematic substance use behaviors than with substance use alone (Ouimette, Read, Wade, & Tirone, 2010). Adult alcohol and drug problems were examined separately, given that research suggests that trauma and PTSD may have differential relations with alcohol versus drugs (Coffey, Read, & Norberg, 2008; Driessen et al., 2008). At Wave 5, 44% of interviewed participants experienced at least one alcohol disorder symptom in the past year, and 17% of interviewed participants experienced at least one drug disorder symptom in the past year. Please see page 58 for a discussion of analytic issues pertaining to these outcome variables.

Data Analytic Strategy

Analyses for the present study were conducted in MPlus version 6.11 (Muthén & Muthén, 1998-2011) using a path analysis framework. An alpha level of .05 was used to determine significance (hypothesized effects that were significant at p < .10 were considered marginally significant and interpreted with caution). It should be noted that all data for the present study were independent and unclustered. Logistic regression was used to examine dichotomous dependent variables. For count dependent variables (PTSD symptoms, alcohol problems, and drug problems), a series of analyses were conducted prior to estimating the final models in order to determine whether Poisson, negative binomial, zero-inflated Poisson, or zero-inflated negative binomial regression was most appropriate. For definitions and comparisons between these different methods for modeling skewed count data, please see the subsection of the Results section titled "Determining the Appropriate Analytic Method for Count Dependent Variables."

Because of the sample selection procedures, all participants have complete data on all variables except for adult alcohol and drug problems, which were assessed at Wave 5. Of the 377 participants who were included in the present study, there were 29 participants who were not interviewed at Wave 5. Full information maximum likelihood estimation was used to account for missing data for these 29 participants, which provides unbiased parameter estimates when missingness at random is assumed (Schafer & Graham, 2002).

Preliminary analyses. Preliminary analyses were conducted for all hypotheses in order to determine which covariates, covariate by predictor interactions, and predictor by predictor interactions to include in the primary models. Non-hypothesized predictor by predictor and covariate by predictor interactions were retained if they had a significant unique effect at a *p*-value that controlled the false discovery rate (FDR), as described by Benjamini and Hochberg (1995). The FDR approach controls the expected proportion of falsely rejected null hypotheses (i.e., Type 1 errors). It is less conservative than the Bonferroni approach and has greater power to find truly significant results, while maintaining adequate control of Type 1 error rates (Shaffer, 1995). FDR-adjusted *p*-values (sometimes referred to in the literature as *q*-values) were computed using the "FDR" option under the "PROC MULTTEST" procedure in SAS. Non-hypothesized interactions that had an FDR-adjusted *p*-value less than .05 in preliminary analyses were included in the main analyses. However, in order to

avoid confusing the reader, standard *p*-values were reported for these interactions once they were included in the main analyses.

Gender and ethnicity were considered predictors rather than covariates due to their relevance to hypothesis testing, and were thus included in all models despite significance in preliminary testing. Note that hypothesized interactions were tested separately subsequent to testing the primary models (an alpha level of .05 was used to test hypothesized interactions). Parent education (SES), age, age at trauma exposure, and time since trauma exposure were tested as potential covariates.⁷ Again, note that age-related covariates were tested in separate models. Nonessential multicollinearity was reduced by centering (calculating deviation scores from the mean) all continuous predictors before computing interaction terms (Cohen, Cohen, West, & Aiken, 2003). All binary variables were coded using 0 and 1 dummy codes. Interactions were tested and probed using simple slope analyses as recommended by Aiken and West (1991).

Data reduction for family risk factors. When examining the directions of influence among trauma exposure, PTSD, and problematic alcohol and drug use, the present study sought to control for the confounding influence of adversity in the family environment. There were four variables reflecting family adversity in the present study: parental alcoholism, other parental psychopathology, family

⁷ Parent education (SES) and age were tested as covariates for all hypotheses (age at trauma exposure and time since trauma exposure were not relevant). Age at trauma exposure and time since trauma exposure were tested as potential covariates for all hypotheses other than the high-risk hypothesis.

conflict, and familial life stress.⁸ These variables likely reflect a combination of genetic and environmental risk that may predispose one to develop both PTSD and SUDs. That is, these variables may be shared risk factors for PTSD and SUDs. Because it was of theoretical interest to simply control for the larger influence of family adversity, rather than to examine the unique influences of these separate family risk factors, analyses employed a composite measure of "family adversity." This composite measure consisted of factor scores that were derived from confirmatory factor analysis of the four family risk factors, as described below.

Mediational analyses. When testing mediational hypotheses, MacKinnon, Lockwood, and Williams' (2004) "product of the coefficients" method will be used. 95% asymmetric confidence intervals were computed using MacKinnon, Fritz, Williams, and Lockwood's (2007) PRODCLIN program in order to test the significance of the indirect effect (MacKinnon, Lockwood, & Williams, 2004). In the present study (n=377), power was sufficient (> .8; Cohen, 1988) to detect significant mediation for small-moderate (B=.26) 'a' and 'b' paths (Fritz & MacKinnon, 2007).

Regression diagnostics. All data were checked for out of range values prior to analysis. In addition, regression diagnostics were used when available to examine the potential influence of outliers and influential cases on regression

⁸ Parent education, which is used as an indicator of SES in the present study, was not included in the family adversity factor analysis for both qualitative and quantitative reasons. Lower levels of parent education—as a proxy for low SES—are not necessarily reflective of adversity in the family environment; thus parent education is conceptually distinct from the other family risk factors. Moreover, zero-order correlations showed that parent education was unrelated to parent psychopathology, family conflict, and familial life stress (see Table 2).

models. Cook's D (Cook, 1977) and DFBETAS (see Cohen et al., 2003) were used to identify influential cases. Cook's D is a measure of the influence of an observation on the parameter estimates. DFBETAS is a measure of standardized change in the regression coefficient when a case is deleted. Cook's D and DFBETAS values greater than the absolute value of one indicate influential cases in a moderate sized dataset (Neter, Wasserman, & Kutner, 1989; Pregibon, 1981).

Results

Creating Family Adversity Factor Scores

Zero-order correlations were examined in order to determine whether the four family risk factors (parental alcoholism, other parental psychopathology, family conflict, and familial life stress) could be appropriately analyzed as a composite "family adversity" variable. All correlations among family risk factors were significant (see Table 2; ps < .001).

Next, a one-factor confirmatory factor analysis (CFA) was conducted using MPlus. Parental alcoholism and other parental psychopathology were specified as categorical variables, familial life stress was specified as a count variable⁹, and family conflict was specified as a continuous variable. The maximum likelihood estimator with robust standard errors (MLR) was used. With this estimator, logistic regressions are estimated for categorical factor indicators, Poisson regressions are estimated for count factor indicators, and ordinary least squares (OLS) regressions are estimated for continuous factor indicators. The factor variance was fixed at 1 and factor loadings were estimated for all 4 indicators. The resulting "family adversity" factor scores were employed in subsequent analyses. Note that MPlus computes continuous factor scores using an iterative procedure when there are categorical and count indicators.

⁹ Additional CFAs were estimated while specifying familial life stress as: (1) a continuous variable with a normal distribution, and (2) a count variable with a negative binomial distribution. However, the loglikelihood value, Bayesian Information Criterion (BIC), and Akaike Information Criterion (AIC) were best when familial life stress was specified as a count variable with a Poisson distribution. See the section "Determining the Appropriate Analytic Method for Count Dependent Variables" for further discussion of modeling issues pertinent to count variables.

Chi-square test statistics and related fit indices are not available for CFAs that include count variables. The unstandardized¹⁰ factor loadings for parental alcoholism, other parental psychopathology, family conflict, and familial life stress on the "family adversity" factor were .97, .72, .45, and .49, respectively (all ps < .001). Because these factor loadings are on different metrics due to indicators' varying distributions, the corresponding t-statistics (estimate/standard error) are also presented in order to allow the reader to make comparisons across indicators: 5.02 (parental alcoholism), 4.14 (other psychopathology), 11.50 (family conflict), and 14.87 (familial stress).

Zero-order correlations between the factor scores and study variables are presented in Table 2. Zero-order correlations with parental alcoholism, other parent psychopathology, family conflict, and familial life stress were .49, .38, .90, and .78, respectively). Note that family adversity was significantly associated with trauma exposure (r=.21, p < .001), PTSD symptoms (r=.26, p=.001), adult alcohol problems (r=.13, p=.019), and adult drug problems (r=.16, p=.002). Thus, this variable appeared to appropriately capture shared risk for these outcomes and was used in subsequent analyses.

Determining the Appropriate Analytic Method for Count Dependent

Variables

Analyses for the present study consisted of three dependent variables that were counts: PTSD symptoms, alcohol problems, and drug problems. Count variables consist of non-negative integers, which tend to be positively skewed and

¹⁰ Unstandardized factor loadings are reported because standardized factor loadings are not relevant for count variables, which do not have variances.

better approximated by a Poisson or negative binomial distribution rather than a normal distribution (see Hilbe, 2007; Long & Freese, 2006). When count variables are overdispersed (variance > mean), negative binomial regression is more appropriate than Poisson regression. Negative binomial models include a dispersion (i.e., residual) parameter that allows for independent specification of the mean and variance. When this dispersion parameter equals zero, the model reduces to the simpler Poisson model. A likelihood-ratio test can be used to compare the Poisson to the negative binomial model because these models are nested (the negative binomial model simply includes an additional dispersion parameter).

Although negative binomial models may account for excess zeros to some extent, when the major source of overdispersion is due to a preponderance of zero counts, zero-inflated Poisson regression (ZIP) or zero-inflated negative binomial regression (ZINB) may be more appropriate. Zero-inflated models are latent class (i.e., mixture) models that distinguish between cases that can only have zero counts and cases that can have the full range of outcomes from zero on up. Therefore, these models simultaneously estimate a logistic regression, which indicates the probability of being unable to assume any value except for zero (e.g., probability of being a non-drinker), as well as a negative binomial or Poisson regression, which indicates the frequency in which the outcome occurs for cases that are able to assume the full range of counts (e.g., frequency of alcohol problems among drinkers). If data are overdispersed even after accounting for zero-inflation, a ZINB model will more accurately reproduce the data compared
to a ZIP model (Long & Freese, 2006). A likelihood ratio test can also be used to compare the nested ZIP and ZINB models. However, the likelihood ratio test cannot be used to compare ZIP to Poisson or ZINB to negative binomial because these models are not nested (Long & Freese, 2006).

A series of analyses were conducted in order to determine which method of model estimation was most appropriate for each count dependent variable. Alcohol problems (56% zeros) and drug problems (81% zeros) exhibited an overabundance of zeros, whereas PTSD symptoms did not (7.8%). Therefore, only Poisson and negative binomial regression were considered for the analysis of PTSD symptoms. PTSD symptoms exhibited evidence of overdispersion (variance > mean), suggesting that negative binomial regression may be more appropriate than Poisson regression. Indeed, the negative binomial model yielded a significant dispersion parameter ($\theta = .32$, p < .001), and the loglikelihood test comparing the negative binomial and Poisson models was also significant ($\chi^2(1)=$ 92.25, p < .001). Therefore, negative binomial regression was used in the main analyses to predict PTSD symptoms.

In order to determine the appropriate method of estimating risk for adult alcohol and drug problems, separate Poisson, negative binomial, ZIP, and ZINB models were examined. When testing these models, two different approaches were used. The first approach (Approach 1) tested trauma exposure and PTSD symptoms as mediators of the influences of family adversity, pre-trauma substance use problems, gender, and ethnicity on risk for alcohol or drug problems. The second approach (Approach 2) examined whether risk for alcohol or drug problems varied among three groups: participants without trauma exposure, participants with trauma exposure but no PTSD, and participants with PTSD. These groups were compared using two dummy variables. Family adversity, pre-trauma substance use problems, gender, and ethnicity were included as predictors. More detailed information about model specification is included when describing the primary models. The purpose of these preliminary analyses was simply to determine which method of model estimation would be most appropriate for the primary models. For each approach, analyses were conducted separately for alcohol and drug problems. Table 4 presents a summary of fit statistics for Approach 1, and Table 5 presents a summary of fit statistics for Approach 2.

Results favored the negative binomial and the ZINB models over the ZIP and Poisson models, respectively.¹¹ Moreover, the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC), which can be used to compare non-nested models (lower values indicate a better fit), indicated a clear improvement in model fit for the negative binomial model compared to the ZIP model for both alcohol and drug problems. However, the AIC and BIC showed little difference between the negative binomial and ZINB models. In the Approach 1 analyses, the AIC and BIC showed slightly better fit for the ZINB model compared to the negative binomial model for alcohol problems, but the BIC, which places greater value on parsimony compared to the AIC, showed

¹¹ The negative binomial model was a significantly better fit than the Poisson model for both alcohol and drug problems, indicating that these variables were overdispersed. For both alcohol problems and drug problems, the ZINB model was a significantly better fit than the ZIP model.

slightly better fit for the negative binomial model compared to the ZINB model for drug problems. In the Approach 2 analyses, the BIC showed the best fit for the negative binomial model for both alcohol and drug problems.

Therefore, it appears that the dispersion parameter in the negative binomial model sufficiently accounted for both zero-inflation and overdispersion in counts without the need for a two-class model. Given that the more parsimonious negative binomial model appeared to adequately reproduce the observed data, and that it was not of substantive interest to model risk for substance use versus risk for substance use problems as two separate processes, negative binomial regression was determined to be the optimal method of estimating risk for both alcohol and drug problems. Thus, negative binomial regression was used to predict these outcomes in the subsequent analyses.

Zero-Order Correlations among Study Variables

Prior to estimating the primary models, the zero-order correlations among study variables were examined (see Table 2). As previously described, count variables were log-transformed prior to estimating zero-order correlations because Pearson correlations may not be appropriate for count variables (Cohen et al., 2003). As expected, males were more likely to be exposed to a traumatic event than were females (r= -.15, p < .05), but trauma-exposed females exhibited significantly higher levels of PTSD symptoms (r= .35, p < .001) and were significantly more likely to be diagnosed with PTSD (r= .26, p < .01) than were trauma-exposed males. Males exhibited significantly higher levels of adult alcohol (r= -.21, p < .001) and drug (r= -.12, p < .01) problems than did females.

Non-Hispanic/Latino Caucasian participants were significantly less likely to be exposed to a traumatic event than were ethnic minority participants (r= .11, p < .05). However, among participants exposed to trauma, ethnic minority and non-minority participants did not significantly differ in risk for PTSD or PTSD symptoms. There were no ethnic differences in adult alcohol or drug problems.

Children of alcoholics exhibited significantly higher levels of adolescent substance use problems (r= .22, p < .001), adult alcohol problems (r= .18, p < .001), and adult drug problems (r= .18, p < .001) compared to children of non-alcoholics. Children of alcoholics were also at marginally higher risk for trauma exposure (r= .09, p = .08). Among trauma-exposed participants, children of alcoholics exhibited marginally higher levels of PTSD symptoms compared to children of non-alcoholics (r=.15, p =.06), although they did not significantly differ in risk for actual PTSD diagnoses.

Zero-order correlations indicated that trauma-exposed participants were at significantly higher risk for adult alcohol problems (r=.12, p < .05) 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 (pr=.19, p < .05), and drug (r=.19, p < .05) problems.

High-Risk Hypothesis

Model specification and preliminary analyses. Separate logistic regressions¹² tested the influence of adolescent substance use problems on risk for trauma exposure and risk for assaultive violence exposure (Hypothesis 1; see bold path in Figure 1.1), over and above the influence of family adversity and demographic predictors (gender and ethnicity). Note that both trauma exposure and assaultive violence exposure were dichotomous variables. These analyses were performed using the entire sample (n= 377). Parameter estimates were obtained using maximum likelihood estimation.

Four predictors were included in analyses: adolescent substance use problems, family adversity, gender, and ethnicity. Age at the time of the Wave 4 interview (i.e., when trauma exposure was assessed) and parent education were tested as covariates. Preliminary analyses were conducted to determine which covariates, covariate by predictor interactions, and predictor by predictor interactions to include in the final models. FDR-adjusted *p*-values were used for these preliminary analyses. No covariates had significant (i.e., FDR-adjusted *p* < .05) main effects or interactions with predictors, and were thus trimmed from the primary analyses. However, there was a significant non-hypothesized gender by ethnicity interaction predicting trauma exposure (*B*= -1.658, FDR-adjusted *p*=

¹² Dependent variables in logistic regression are on the logit scale. Therefore, regression coefficients indicate the linear increase in the logit for a one unit increase in the predictor. Coefficients can be exponentiated in order to obtain odds ratios (ORs), which indicate the amount by which the odds of being in the group coded 1 are multiplied for each one unit increase in the independent variable.

.030, *OR*: .19), which was entered into the primary analysis in a separate block in order to allow examination of main effects prior to including the interaction term.

Final models. The final models testing the influence of adolescent substance use problems on risk for trauma exposure and assaultive violence exposure (Hypothesis 1) are presented in Tables 6 and 7, respectively. Note that regression diagnostics did not reveal any problematic influential cases for either model.

The primary goal for the high-risk hypothesis was to test whether adolescent substance use significantly influenced risk for trauma exposure or assaultive violence exposure (i.e., the bolded path in Figure 1 was tested for significance). Results showed that the unique effect of adolescent substance use problems on risk for trauma exposure was non-significant (B=.17, p=.35, OR: 1.18; see Table 6). However, the unique effect of adolescent substance use problems on risk for assaultive violence exposure was marginally significant (B=.38, p = .051, OR: 1.46; see Table 7). Therefore, results suggested that adolescent substance use problems may increase risk for forms of trauma that involve assaultive violence. Note that family adversity had a significant unique effect on risk for both trauma exposure (B= .46, p= .001, OR: 1.58; see Table 6 Block 2) and assaultive violence exposure (B= .48, p= .004, OR: 1.61; see Table 7). If family adversity were not included in analyses, the influence of adolescent substance use problems on risk for trauma exposure would have been marginally significant (B= .33, p= .055, OR: 1.39), and the influence of adolescent substance use problems on risk for assaultive violence exposure would have been significant (B=.53, p=.003, OR: 1.71). Thus, results support the importance of including family adversity as an importance confounding variable when testing the high-risk hypothesis.

Gender and ethnicity effects. Results from analyses testing the influence of gender and ethnicity within the high-risk hypothesis are presented below.

Hypothesis 1a. This study included gender as a predictor when testing the high-risk hypothesis in order to test whether males were at significantly higher risk for trauma exposure and assaultive violence compared to females. Note that gender was dummy coded 0 for males and 1 for females. Results indicated that gender had a significant unique main effect on risk for trauma exposure (B=-.61, p=.005, OR: .54; see Table 6 Block 1), such that males were at significantly greater risk for trauma exposure than were females. However, after entering the significant interaction between gender and ethnicity (B= -1.32, p= .009, OR: .27; see Table 6 Block 2), results showed that the effect of gender was only significant for ethnic minority participants. In order to examine how the effect of gender differed by ethnicity, simple slope analyses were conducted. Results indicated that ethnic minority males were at significantly greater risk for trauma exposure than were ethnic minority females (B= -1.58, p < .001, OR: .21), whereas male and female non-Hispanic/Latino Caucasians did not differ in their risk for trauma exposure (B= -.26, p= .31, OR: .77).

As for the assaultive violence exposure analysis, gender did not have a significant effect over and above the influence of the other predictors (B= -.40, p= .15, OR: .67; see Table 7).

Hypothesis 1b. This study also tested whether gender moderated the influence of adolescent substance use problems on trauma exposure or assaultive violence exposure. This hypothesis was tested by entering the interaction between gender and adolescent substance use problems into each model (see dotted path in Figure 1.1). There was no evidence that gender moderated the influence of adolescent substance use problems on either trauma exposure (B= - .03, p= .94, OR: .98) or assaultive violence exposure (B= .43, p= .25, OR: 1.54). Note that these interactions are not reported in any tables.

Given the significant interaction between gender and ethnicity when predicting trauma exposure, a three-way interaction among ethnicity, gender, and adolescent substance use problems was also tested. However, a model testing the three-way interaction term would not converge because there were only four Hispanic/Latino females and only nine Hispanic/Latino males who had at least one substance use problem (i.e., were in the groups coded 1 or 2 on the adolescent substance use problems variable), and none of the four females were exposed to a traumatic event, whereas all nine of the males were exposed to a traumatic event. Therefore, it was not possible to draw any conclusions with these data regarding a potential three-way interaction among gender, ethnicity, and adolescent substance use problems when predicting risk for trauma exposure.

Hypothesis 1c: This study examined whether adolescent substance use problems may mediate the influence of gender on risk for trauma exposure or assaultive violence exposure (see Figure 1.2). Given the lack of effect of adolescent substance use problems on risk for trauma exposure, this hypothesis

was only tested with assaultive violence as the outcome variable. An additional path analysis specified paths from gender to adolescent substance use problems, and from adolescent substance use problems to assaultive violence exposure (family adversity and ethnicity were included as predictors, and age at Wave 1 was included as a covariate for adolescent substance use problems). Gender did not have a significant unique effect on risk for adolescent substance use problems (B= -.19, p= .57, OR: .83). Therefore, there was no evidence that adolescent substance use problems mediated the influence of gender on risk for assaultive violence exposure.

Hypothesis 1d: Ethnicity was included as a predictor when testing the high-risk hypothesis in order to test whether ethnic minorities were at elevated risk for trauma exposure or assaultive violence while controlling for other risk factors. Note that ethnicity was dummy coded 0 for non-Hispanic/Latino Caucasians and 1 for minority ethnicities. The unique main effect of ethnicity on risk for trauma exposure was marginally significant (B= .46, p= .06, OR: 1.58; see Table 6 Block 1), such that ethnic minority participants were at marginally higher risk for trauma exposure than were non-Hispanic/Latino Caucasian participants. However, after entering the significant interaction between gender and ethnicity, results showed that the effect of ethnicity was only significant for males (B= -1.32, p= .009, OR: .27; see Table 6 Block 2). That is, the influence of ethnicity on risk for trauma exposure for females was .27 times the influence of ethnicity for males. Simple slope examining how the effect of ethnicity differed by gender indicated that ethnic minority males were at significantly greater risk

for trauma exposure than were non-Hispanic/Latino Caucasian males (B= 1.09, p= .002, OR: 2.99), whereas there was no difference in risk for trauma exposure between non-Hispanic/Latino Caucasian and ethnic minority females (B= -.22, p= .53, OR: .80).

An additional analysis dropped the 18 participants who were of ethnicities other than Hispanic/Latino or non-Hispanic/Latino Caucasian in order to allow for a clearer examination of whether Hispanics/Latinos, specifically, were at greater risk for trauma exposure. Results were consistent. Specifically, the main effect of ethnicity showed that Hispanics/Latinos were at marginally higher risk for trauma exposure than were non-Hispanic/Latino Caucasians (B= .47, p= .07, OR: 1.60). Moreover, Hispanic/Latino males were at significantly higher risk for trauma exposure than were non-Hispanic/Latino Caucasian males (B= 1.16, p= .002, OR: 3.20), whereas female Hispanics/Latinos and non-Hispanic/Latino Caucasians did not differ in their risk for trauma exposure (B= -.25, p= .52, OR: .78).

In contrast to the trauma exposure model, results from the assaultive violence exposure model indicated that ethnicity did not have a significant unique effect on risk for assaultive violence exposure over and above the other predictors (B= .16, p= .58, OR: 1.18). Results were consistent after dropping the 18 participants who were of ethnicities other than Hispanic/Latino or non-Hispanic/Latino Caucasian (B= .23, p= .47, OR: 1.26).

Susceptibility Hypothesis

Model specification and preliminary analyses. Logistic regression was used to test the influence of pre-trauma adolescent substance use problems on risk for PTSD (dichotomous variable) over and above the influence of family adversity and demographic predictors (gender and ethnicity) among participants who were exposed to a traumatic event (Hypothesis 2; see Figure 2). Similarly, negative binomial¹³ regression was used to test the influence of adolescent substance use problems on risk for PTSD symptoms (count variable) over and above the influence of family adversity and demographic predictors among participants who were exposed to a traumatic event. Because these analyses were performed using only participants who were exposed to a traumatic event (*n*= 166), results indicate the *conditional* risk for developing PTSD symptoms or a PTSD diagnosis (i.e., risk among those exposed to a traumatic event). The maximum likelihood estimator was used for the logistic regression, and the maximum likelihood estimator with robust standard errors was used for the negative binomial regression. The MLR estimator is recommended for count outcomes because it may provide some protection against model misspecification.

¹³ Dependent variables in negative binomial regression are count variables. Negative binomial regression coefficients indicate the log of the expected count as a function of the independent variable, i.e. how much the log of the expected count of the dependent variable is expected to change for a 1 unit increase in the predictor. Just as logistic regression coefficients can be exponentiated in order to obtain odds ratios, negative binomial regression coefficients can be exponentiated in order to obtain incidence rate ratios (*IRRs*). The IRR indicates the multiplicative extent to which the log of the expected count of the dependent variable is expected to increase or decrease for a 1 unit change in the independent variable. For instance, an IRR of 1.50 means that for every one unit increase in the predictor, there is a 50 percent increase in the dependent variable.

Four predictors were included in analyses: adolescent substance use problems, family adversity, gender, and ethnicity. Parent education, age at trauma exposure, age at Wave 4 (when PTSD symptoms were assessed), and time since trauma exposure (time elapsed between age at Wave 4 and age at exposure) were tested as covariates. Preliminary analyses were conducted to determine which covariates, covariate by predictor interactions, and predictor by predictor interactions to include in the final model. Again, note that FDR-adjusted *p*-values were used for these preliminary analyses. Continuous predictors and covariates were re-centered with respect to the means for the 166 trauma-exposed participants prior to computing interaction terms. Separate preliminary analyses tested the effects of age at Wave 4, age at trauma exposure, and time elapsed since trauma exposure, given that these measures overlap with one another. No covariates had significant main effects over and above the other predictors, and were thus trimmed from the primary analyses. There were no significant predictor by predictor or covariate by predictor interactions. Therefore, the final models did not include any non-hypothesized covariates or interaction terms.

Final models. The final models testing the influence of adolescent substance use problems on conditional risk for PTSD diagnosis and PTSD symptoms are presented in Tables 8 and 9, respectively. Regression diagnostics did not reveal any problematic influential cases for either model.

Results indicated that adolescent substance use problems did not significantly influence risk for PTSD diagnosis (B= .39, p= .19, OR: 1.48; see Table 8) or PTSD symptoms (B= .07, p= .41, *IRR*: 1.07; see Table 9) over and

above the influences of family adversity, gender, and ethnicity.¹⁴ Therefore, results indicate that adolescent substance use problems do not increase susceptibility for developing PTSD (dichotomous variable) or PTSD symptoms (count variable) among participants exposed to a traumatic event over and above the influence of correlated adversity in the family environment.¹⁵ Note that family adversity had a significant unique effect on conditional risk for both PTSD (B= .85, p= .008, OR: 2.35; see Table 8) and PTSD symptoms (B= .26, p =.001, IRR: 1.30; see Table 9).

Gender and ethnicity effects. Results from analyses testing the influence of gender and ethnicity within the susceptibility hypothesis are presented below.

Hypothesis 2a. Gender was included as a predictor when testing the susceptibility hypothesis in order to test whether females were at higher risk for PTSD or PTSD symptoms compared to males (Hypothesis 2a). Gender had a significant unique effect on risk for both PTSD diagnosis (B= 1.61, p= .001, OR: 4.98; see Table 8) and PTSD symptoms (B= .55, p < .001, *IRR*: 1.73; see Table 9), such that females were at greater risk compared to males. Specifically,

¹⁴ Both analyses were underpowered to detect significant effects of these magnitudes. Post-hoc power analyses indicated that achieved power was approximately .34 in the analysis predicting PTSD diagnosis and approximately .39 in the analysis predicting PTSD symptoms (however, the latter power analyses tested whether the influence of adolescent substance use problems on risk for PTSD diagnosis or PTSD symptoms was significant prior to including family adversity in the model (i.e., significant over and above the effects of gender and ethnicity). Indeed, adolescent substance use problems had a significant effect on risk for PTSD (B=.64, p=.02, OR: 1.88) and a marginally significant effect on risk for PTSD symptoms (B=.14, p=.065, IRR: 1.15) over and above the effects of gender and ethnicity. These results highlight the importance of considering family adversity as a third variable in models of PTSD-substance use problems. The fact that the effect of adolescent substance use problems becomes non-significant when family adversity is included in the model indicates that it is family adversity, rather than substance use problems themselves, that may increase risk for developing PTSD or PTSD symptoms.

trauma-exposed females were at nearly five times the risk for PTSD than were trauma-exposed males.

Hypothesis 2b. Subsequent to the main analyses, the interaction between gender and adolescent substance use problems was entered into each model in order to test whether gender moderated the influence of adolescent substance use problems on risk for PTSD or PTSD symptoms (Hypothesis 2b). This interaction was non-significant in both the model predicting PTSD diagnosis (B= .61, p= .32, *OR*: 1.84), as well as the model predicting PTSD symptoms (B= .16, p= .24, *IRR*: 1.17). Because these interactions were non-significant, they are not reported in Tables 8 or 9. Therefore, there was no evidence that gender moderated the influence of adolescent substance use problems on risk for PTSD diagnosis or PTSD symptoms.

Hypothesis 2c. Ethnicity was initially included as a predictor when testing the susceptibility hypothesis. Ethnicity did not significantly influence risk for PTSD (B= .52, p= .26, OR: 1.69; see Table 8) but had a marginally significant unique effect on risk for PTSD symptoms (B= .20, p= .091, *IRR*: 1.22; see Table 9), such that ethnic minority participants exhibited marginally higher levels of PTSD symptoms than did non-Hispanic/Latino Caucasian participants.

In order to test whether Hispanics/Latinos were at greater risk for PTSD or PTSD symptoms compared to non-Hispanic/Latino Caucasians, an additional analysis was conducted that excluded the 10 trauma-exposed participants who were not of Caucasian or Hispanic/Latino ethnicity.¹⁶ Results showed that Hispanics/Latinos were at significantly greater risk for both PTSD (B= 1.01, p= .04, OR: 2.75) and PTSD symptoms (B= .30, p= .011, *IRR*: 1.35) than were non-Hispanic/Latino Caucasians.

Self-Medication Hypothesis

To test the self-medication hypothesis (Hypothesis 3), we examined whether PTSD symptoms or PTSD diagnosis increased risk for future alcohol or drug problems over and above the influences of trauma exposure, pre-trauma substance use problems, gender, ethnicity, and familial risk that is common to both PTSD and alcohol/drug problems (i.e., family adversity). Two different analytic approaches were used. However, note that the self-medication and shared vulnerability hypotheses were both tested using the same models. Therefore, the description of the two analytic approaches that follows is relevant to both the selfmedication and shared vulnerability hypotheses.

The first approach (Approach 1; see Figure 3) simultaneously examined the separate influences of trauma exposure (binary variable) and PTSD symptoms (count variable) on risk for future alcohol and drug problems. The second approach (Approach 2; see Figure 4) compared risk for future alcohol and drug problems among participants who were not exposed to trauma, participants who were exposed to a traumatic event but *did not* develop PTSD, and participants who were exposed to a traumatic event and *did* develop PTSD. For Approach 2,

¹⁶ There were 18 "other ethnicity" (ethnicities other than Hispanic/Latino or Caucasian) participants in the total sample of 377. However, in the trauma-exposed sample of 166, there were 10 "other ethnicity" participants.

two dummy coded variables were created in order to examine whether risk for alcohol and drug problems varied among the three groups because it was not possible to test group membership (a nominal variable¹⁷) as a mediator.

Both approaches controlled for pre-trauma substance use, family adversity, gender, and ethnicity. For both approaches, separate models were conducted using either adult alcohol problems or adult drug problems as the outcome variable. Negative binomial regression was used to predict alcohol and drug problems (see previous section titled "Determining the Appropriate Analytic Method for Count Dependent Variables"). 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. Note that these models required numerical integration.¹⁸ It should also be noted that chi-square and other related fit statistics are not available for models with count variables because means, variances, and covariances are not sufficient statistics for model estimation for count dependent variables.

Preliminary analyses tested for significant covariates, predictor by predictor interactions, and covariate by predictor interactions. Parent education,

¹⁷ Modeling a nominal variable as a mediator is an unresolved methodological issue. If a nominal variable was treated a mediator, the nominal mediating variable would be treated as continuous when predicting the dependent variable, which would be incorrect given that nominal variables have no order.

¹⁸ Numerical integration is required when using maximum likelihood estimation to predict categorical and/or count dependent variables. When models require numerical integration and there is missing data on mediators, Monte Carlo integration is required in order to allow for the fact that the dimensions on integration may vary for individuals due to missing data. Therefore, Monte Carlo integration was used for Approach 1 analyses due to missing data on PTSD symptoms for participants who were not exposed to a traumatic event. Note that FIML is still being used to account for missing data under Monte Carlo integration.

age at the assessment of alcohol and drug problems (Wave 5), age at trauma exposure, and time since trauma exposure (time elapsed between age at the most recent trauma exposure and age at the assessment of alcohol and drug problems) were tested as covariates. FDR-adjusted *p*-values were used for preliminary analyses. More detailed information about model specification and results of preliminary analyses are presented separately for each approach.

Approach 1.

Model specification and preliminary analyses. Path analyses examined the influences of trauma exposure (binary variable) and PTSD symptoms (count variable) on risk for future alcohol and drug problems. 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). Therefore, this analysis was performed using the entire sample (n = 377), but part of the model (i.e., the paths testing the influence of PTSD symptoms) only applied to participants who were exposed to a traumatic event (n=166). Rather than conducting separate analyses for trauma exposure and PTSD symptoms, the advantage of this model is that it simultaneously estimated the effects of both trauma exposure and PTSD symptoms on the substance use outcome variable (either alcohol problems or drug problems). Therefore, this model enabled examination of whether PTSD symptoms had a significant effect on future alcohol and drug problems over and above the effects of trauma exposure, as well

as pre-trauma substance use problems, family adversity, and demographic predictors.

Trauma exposure was specified as a categorical variable. PTSD symptoms, alcohol problems, and drug problems were specified as count variables with negative binomial distributions. Paths were specified from trauma exposure and PTSD symptoms to the substance use problems (either alcohol or drugs) outcome variable. The residual covariance between trauma exposure and PTSD symptoms was estimated in order to allow for the fact that they may share predictors other than those specified in the model. Family adversity, gender, and ethnicity were included as predictors of trauma exposure, PTSD symptoms, and adult alcohol/drug problems. Given that the results from Hypothesis 1 showed that there was a significant interaction between gender and ethnicity when predicting risk for trauma exposure, this interaction was also included as a predictor of trauma exposure in these analyses. In addition, a path was specified from adolescent substance use problems to adult alcohol/drug problems in order to establish temporal precedence and control for pre-trauma substance use problems. Paths were also specified from adolescent substance use problems to trauma exposure and PTSD symptoms.¹⁹ Preliminary analyses showed that there was a significant effect of time since trauma exposure on risk for alcohol problems (B= -.23, FDR-adjusted p= .004, IRR: .80). There was also a significant interaction between family adversity and gender in the model predicting risk for

¹⁹ Although results from the high-risk and susceptibility hypotheses indicated that the adolescent substance use problems did not significantly influence risk for trauma exposure or PTSD symptoms, these paths were included in the Approach 1 models for theoretical purposes.

drug problems (B= 1.13, FDR-adjusted p= .004, *IRR*: 3.10). These effects were retained in the final models. All other covariate effects and interactions were non-significant in preliminary analyses and were not further considered.

Final models. The model testing the main effect of PTSD symptoms on risk for future alcohol problems is presented in Tables 10. Results showed that PTSD symptoms had a significant unique effect on future adult alcohol problems (B=.09, p=.003, IRR: 1.10; see Table 10) over and above the effects of trauma exposure, time since trauma exposure, pre-trauma substance use problems, family adversity, gender, and ethnicity. Note that the time since trauma exposure covariate indicated that participants who had been exposed to trauma more recently were at significantly greater risk for alcohol problems (B=.23, p=.001, IRR: .80; see Table 11).

The model testing the main effect of PTSD symptoms on risk for future drug problems is presented in Tables 11. Results showed that PTSD symptoms also had a significant unique effect on future adult drug problems (B= .09, p= .042, *IRR*: 1.10; see Table 11) over and above the effects of trauma exposure, pre-trauma substance use problems, ethnicity, family adversity, gender, and the interaction between gender and family adversity.²⁰

²⁰ In order to differentiate between risk for using alcohol/drugs (i.e., probability of being a nonuser) and risk for developing alcohol/drug problems among those who use alcohol/drugs, followup analyses tested the influence of trauma exposure and PTSD symptoms on alcohol and drug problems using zero-inflated Poisson (ZIP) regression. Model specification was the same as described above. The ZIP model simultaneously estimates a logistic regression predicting the probability of being unable to assume any value except for zero (i.e., probability of being a nondrinker/user), as well as a Poisson regression predicting the frequency in which the outcome occurs for cases that are able to assume the full range of counts (including 0 for those who use substances without experiencing any problems). Results from the ZIP models were consistent with those described above. PTSD symptoms had a significant unique effect on risk for alcohol

Gender and ethnicity as moderators of PTSD symptoms. Additional analyses were conducted to test whether gender or ethnicity moderated the influence of PTSD symptoms on risk for future alcohol or drug problems.

Hypothesis 3a. Gender was initially included as a covariate, with results indicating that males were at significantly greater risk for both alcohol (B= -1.03, p < .001, *IRR:* .36; see Table 10) and drug problems (B= -1.36, p < .001, *IRR:* .26; see Table 11) than were females. An additional analysis tested the interaction between gender and PTSD symptoms in order to examine whether gender moderated the influence of PTSD symptoms on risk for future alcohol or drug problems. The interaction between gender and PTSD symptoms (B= -.04, p= .35, *IRR:* .96) and drug problems (B= -.01, p= .82, *IRR:* .99). Therefore, there was no evidence that the effect of PTSD symptoms on alcohol and drug problems varied for males and females. Because these interactions were non-significant, they were trimmed from the final models and not presented in any tables.

Hypothesis 3b. An additional analysis tested the interaction between ethnicity and PTSD symptoms in order to test whether ethnicity moderated the risk of PTSD symptoms on risk for future alcohol or drug problems. In the model

problems among those who drink (B=.10, p < .001, *IRR*: 1.11). PTSD symptoms also had a significant unique effect on risk for drug problems among those who use drugs (B=.076, 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 = .111, *OR*: .92). Therefore, these analyses demonstrate that PTSD symptoms significantly increase risk for alcohol and drug problems among those who use alcohol and drugs. Note that trauma exposure did not have a significant unique effect on risk for alcohol problems among those who drink (B=.02, p = .89, *IRR*: 1.02), risk for drug problems among those who use drugs (B=-.02, p = .956, *IRR*: .99), the probability of being a non-drinker (B=-.31, p = .26, *OR*: .73), or the probability of being a non-drug user (B=-.18, p = .54, *OR*: .83).

predicting alcohol problems, ethnicity significantly interacted with PTSD symptoms (B= -.10, p= .02, *IRR*: .90), such that the influence of PTSD symptoms on alcohol problems was significant for non-Hispanic/Latino Caucasians (B= .11, p= .001, *IRR*: .1.12) but not for minority ethnicities (B= .02, p= .52, *IRR*: 1.02). , the influence of PTSD symptoms on alcohol problems for minority ethnicities was .90 times (i.e., 10% lower) the influence of PTSD symptoms on alcohol problems for non-Hispanic/Latino Caucasians. In the model predicting drug problems, there was a marginally significant interaction between ethnicity and PTSD symptoms (B= -.15, p= .06, *IRR*: .86), such that the effect of PTSD symptoms on drug problems was significant for non-Hispanic/Latino Caucasians (B= .11, p= .04, *IRR*: 1.11) but not for minority ethnicities (B= .02, p= .67, *IRR*: 1.02). That is, the influence of PTSD symptoms on drug problems for minority ethnicities was .86 times (i.e., 14% lower) the influence of PTSD symptoms on drug problems for non-Hispanic/Latino Caucasians.

In order to allow comparisons specifically between Hispanics/Latinos and non-Hispanic/Latino Caucasians, analyses were repeated while excluding the 18 participants who were of ethnicities other than Hispanic/Latino or non-Hispanic/Latino Caucasian. These results are presented in Table 12 (alcohol problems as the dependent variable) and Table 13 (drug problems as the dependent variable). Similar to the results reported above, the interaction between ethnicity and PTSD symptoms was significant in the analysis predicting alcohol problems (B= -.10, p= .033, *IRR*: .91; see Table 12). Probing of this interaction showed that the influence of PTSD symptoms on alcohol problems was significant for non-Hispanic/Latino Caucasians (B=.10, p=.002, IRR: 1.11; see Table 12) but not for Hispanics/Latinos (B = .03, p = .45, IRR: 1.03; see Table 12). Specifically, the influence of PTSD symptoms on alcohol problems for minority ethnicities was .91 times (i.e., 9% lower) the influence of PTSD symptoms on alcohol problems for non-Hispanic/Latino Caucasians. The interaction between ethnicity and PTSD symptoms was marginally significant in the analysis predicting drug problems (B= -.14, p= .055, *IRR*: .87; see Table 13). Probing of this interaction showed that the influence of PTSD symptoms on drug problems was significant for non-Hispanic/Latino Caucasians (B= .10, p= .047, *IRR*: 1.11; see Table 13) but not for Hispanics/Latinos (B= .02, p= .617, *IRR*: 1.02; see Table 13). Specifically, the influence of PTSD symptoms on drug problems for minority ethnicities was .87 times (i.e., 13% lower) the influence of PTSD symptoms on drug problems for non-Hispanic/Latino Caucasians. In sum, it appears that PTSD symptoms may increase risk for future alcohol and drug problems for non-Hispanic/Latino Caucasians, but not Hispanics/Latinos.

Partial correlations examined the relations between PTSD symptoms and alcohol/drug problems while controlling for gender. These partial correlations were examined separately for trauma-exposed Hispanics/Latinos and non-Hispanic/Latino Caucasians in order to make sure that the results from these complex analyses were consistent with the bivariate associations. The partial correlations were consistent with the results reported above. Controlling for gender, PTSD symptoms were significantly associated with both alcohol (pr= .25, p =.010) and drug (pr= .26, p =.007) problems for trauma-exposed non-

Hispanic/Latino Caucasians. However, for trauma-exposed Hispanics/Latinos, PTSD symptoms were not significantly associated with either alcohol problems (pr=.14, p=.40) or drug problems (pr=.02, p=.92) while controlling for gender.

Approach 2.

Model specification and preliminary analyses. Additional analyses examined how risk for future alcohol and drug problems varied among three groups: participants without trauma exposure (n=211), participants with trauma exposure who did not meet diagnostic criteria for a PTSD diagnosis (n=135), and participants with trauma exposure who met diagnostic criteria for PTSD (n=31). Analyses were conducted using the full sample (n=377). Two dummy coded variables were created in order to examine whether risk for alcohol and drug problems varied among the three groups. The no trauma exposure group served as the reference group such that the first dummy variable examined risk for alcohol and drug problems for participants with trauma exposure but no PTSD relative to those without trauma exposure, and the second dummy variable examined risk for alcohol and drug problems for participants with PTSD relative to participants without trauma exposure. In addition to these dummy variables, analyses also included the following predictors: family adversity, pre-trauma substance use problems, gender, and ethnicity.

Results from preliminary analyses testing for covariate effects, predictor by predictor interactions, and covariate by predictor interactions were consistent with those from Approach 1. Results showed that time since trauma exposure was a significant covariate for the model predicting alcohol problems (B= -.24, FDR-adjusted p < .001, *IRR*: .79), and that there was a significant family adversity by gender interaction in the model predicting drug problems (B= 1.17, FDR-adjusted p= .004, *IRR*: 3.23). All other covariate effects and interactions were non-significant in preliminary analyses and were not further considered.

Final models. The final models testing the influence of PTSD diagnosis on risk for future alcohol and drug problems are presented in Tables 14 and 15, respectively. The risk for adult alcohol problems was marginally elevated in participants with PTSD compared to participants without trauma exposure (B=.62, p=.09, *IRR*: 1.86; see Table 14). Note that this marginal effect was found while controlling for the influences of trauma exposure without PTSD, family adversity, gender, ethnicity, pre-trauma (adolescent) substance use problems, and time since trauma exposure. However, recoding of the dummy variables such that the group with trauma exposure but no PTSD served as the reference group revealed that risk for alcohol problems was not significantly elevated in participants who were exposed to trauma and developed PTSD compared to participants who were exposed to trauma and did not develop PTSD (B=.50, p=.15, *IRR*: 1.65).

The risk for adult drug problems was not significantly elevated in participants with PTSD compared to participants who were not exposed to trauma (B=.49, p=.41, IRR: 1.64; see Table 15), over and above the effects of trauma exposure without PTSD, family adversity, gender, ethnicity, pre-trauma (adolescent) substance use problems, and the interaction between gender and family adversity. Moreover, recoding of the dummy variables such that the group with trauma exposure but no PTSD served as the reference group revealed that risk for drug problems was not significantly elevated in participants who were exposed to trauma and developed PTSD compared to participants who were exposed to trauma and did not develop PTSD (B=.28, p=.64, *IRR*: 1.33).

Gender and ethnicity as moderators of PTSD diagnosis.

Hypothesis 3a. An additional analysis tested the interaction between gender and PTSD diagnosis in order to examine whether gender moderated the influence of PTSD diagnosis on risk for future alcohol or drug problems. The interaction was non-significant for both alcohol problems (B=-.25, p= .70, *IRR*: .78) and drug problems (B=-.44, p= .70, *IRR*: .65). Therefore, there was no evidence that the influence of PTSD on alcohol and drug problems was significantly different for males and females. Because these interactions were non-significant, they were trimmed from the models and omitted from Tables 14 and 15.

Hypothesis 3b. An additional analysis tested the interaction between ethnicity and PTSD diagnosis in order to examine whether ethnicity moderated the risk of PTSD on risk for future alcohol or drug problems. In the model predicting alcohol problems, ethnicity significantly interacted with PTSD (B= -1.29, p= .037, *IRR*: .27), such that the influence of PTSD on alcohol problems was significant for non-Hispanic/Latino Caucasians (B= 1.036, p= .017, *IRR*: 2.82) but not for minority ethnicities (B= -.26, p= .60, *IRR*: .80). Specifically, the influence of PTSD diagnosis on alcohol problems for minority ethnicities was .27 times (i.e., 73% lower) the influence of PTSD diagnosis on alcohol problems for non-Hispanic/Latino Caucasians. In the model predicting drug problems, ethnicity significantly interacted with PTSD (B= -3.17, p < .001, *IRR*: .04), such that the influence of PTSD on drug problems was marginally significant for non-Hispanic/Latino Caucasians (B= 1.08, p= .075, *IRR*: 2.94) and significant in the opposite direction for minority ethnicities (B= -2.09, p= .001, *IRR*: .12). Specifically, the influence of PTSD diagnosis on drug problems for minority ethnicities decreased by a factor of .04 (i.e., 96%) compared to the influence of PTSD diagnosis on drug problems for non-Hispanic/Latino Caucasians. Note that these interactions were omitted from Tables 14 and 15, which tested only the main effect of PTSD.

In order to allow comparisons specifically between Hispanics/Latinos and non-Hispanic/Latino Caucasians, additional analyses excluded the 18 participants who were of ethnicities other than Hispanic/Latino or non-Hispanic/Latino Caucasian. Results are presented in Tables 16 and 17. The interaction between ethnicity and PTSD diagnosis was significant in the model predicting alcohol problems (B=-1.26, p= .04, *IRR*: .28; see Table 16) such that the influence of PTSD diagnosis on alcohol problems decreased by a factor of .28 (72%) for Hispanics/Latinos compared to non-Hispanic/Latino Caucasians. In fact, probing this interaction revealed that PTSD diagnosis significantly influenced risk for alcohol problems for non-Hispanic/Latino Caucasians (B=.97, p= .02, *IRR*: 2.65; see Table 16) but not for Hispanics/Latinos (B=-.30, p= .55, *IRR*: .74; see Table 16). That is, Caucasians who were exposed to trauma and developed PTSD (n=19) were at significantly elevated risk for adult alcohol problems compared to Caucasian participants who were not exposed to trauma (n=163). Again, note that this effect was significant over and above the influences of trauma exposure without PTSD, family adversity, gender, ethnicity, pre-trauma (adolescent) substance use problems, and time since trauma exposure. However, further analysis after recoding the dummy variables to compare participants with PTSD to participants who were exposed to trauma but did not develop PTSD showed that Caucasian participants who developed PTSD following trauma exposure (n =19) and Caucasian participants who were exposed to trauma but did not develop PTSD (n =93) did not significantly differ in their risk for adult alcohol problems (B=-.38, p =.43, *IRR*: .68).

The interaction between PTSD and ethnicity was also significant in the model predicting drug problems (B=-3.27, p < .001, *IRR*: .04; see Table 17) such that the influence of PTSD diagnosis on drug problems for Hispanics/Latinos decreased by a factor of .04 (i.e., 96%) compared to the influence of PTSD diagnosis on drug problems for non-Hispanic/Latino Caucasians. Probing this interaction revealed that PTSD diagnosis had a marginally significant unique effect on risk for drug problems for non-Hispanic/Latino Caucasians (B=1.09, p= .07, *IRR*: 2.98; see Table 17), such that non-Hispanic/Latino Caucasian participants who developed PTSD following trauma exposure were at nearly three times the risk for adult drug problems compared to non-Hispanic/Latino Caucasian participants without trauma exposure (again, while holding all other variables in the model constant). Surprisingly, results from probing the PTSD diagnosis by ethnicity interaction for Hispanics/Latinos showed that PTSD diagnosis significantly decreased risk for drug problems for Hispanics/Latinos.

(*B*=-2.18, *p*= .001, *IRR*: .11; see Table 17). That is, Hispanic/Latino participants who developed PTSD following trauma exposure (n = 11) were at significantly lower risk for adult drug problems compared to Hispanic/Latino participants without trauma exposure (n = 40), holding all other variables in the model constant. Further analysis after recoding the dummy variables to compare participants with PTSD to participants who were exposed to trauma but did not develop PTSD also revealed that Hispanic/Latino participants who developed PTSD following trauma exposure (n = 11) were at significantly lower risk for adult drug problems compared to Hispanic/Latino participants who were exposed to trauma but did not developed PTSD following trauma exposure (n = 11) were at significantly lower risk for adult drug problems compared to Hispanic/Latino participants who were exposed to trauma but did not develop PTSD (n = 33; B = -2.44, p < .001, *IRR*: .09).

In order to examine the consistency of this finding with descriptive statistics, the mean number of drug problems for Hispanics/Latinos in each of the three groups was examined. The mean number of drug problems was .27 (*SD*= .64) for Hispanics/Latinos who were exposed to trauma and developed PTSD (n = 11), .96 (*SD*= 2.03) for Hispanics/Latinos with trauma exposure but did not develop PTSD (n = 33), and .82 (*SD*= 2.04) for Hispanics/Latinos without trauma exposure (n = 40). These descriptive findings are consistent with the results from the main analyses, suggesting that Hispanics/Latinos with PTSD may be at lower risk for drug problems compared to Hispanics/Latinos who were not exposed to a traumatic event, as well as Hispanics/Latinos who were exposed to a traumatic event but did not develop PTSD.

Shared Vulnerability Hypothesis

To test the shared vulnerability hypothesis (Hypothesis 4), we examined the extent to which shared familial risk factors, as represented by the composite family adversity variable, accounted for the relations among PTSD, alcohol problems, and drug problems. We also tested whether trauma exposure can be conceptualized as a shared risk factor, such that trauma exposure influences risk for alcohol or drug problems independent of PTSD. Note that the shared vulnerability hypothesis was tested using the same models that were used to test the self-medication hypothesis (see above).

Approach 1.

Model specification and preliminary analyses. See the description of model specification for Approach 1 under the self-medication hypothesis. See Figure 3 for a depiction of Approach 1.

Final models. The final models testing trauma exposure and PTSD symptoms as mediators of the influence of family adversity on future alcohol and drug problems are presented in Tables 10 and 11, respectively. The direct effects of trauma exposure on adult alcohol and drug problems were examined in order to test the "*trauma exposure as a shared risk factor hypothesis.*" Specifically, we examined whether trauma exposure had a unique effect on alcohol or drug problems over and above the effects of PTSD symptoms, family adversity, pre-trauma substance use problems, gender, and ethnicity. The unique effect of trauma exposure was non-significant in the model predicting alcohol problems (*B*= .17, *p*= .38, *IRR*: 1.19; see Table 10), as well as the model predicting drug

problems (B= .20, p= .55, *IRR*: 1.22; see Table 11).²¹ Therefore, trauma exposure did not exert unique effects on either alcohol or drug problems over and above PTSD symptoms; the effects of trauma exposure appeared to be fully accounted for by PTSD symptoms, such that trauma exposure only influences risk for future substance use problems to the extent that it is associated with higher levels of PTSD symptoms. Thus, there was no support for the trauma exposure as a shared risk factor hypothesis.

Next, the paths from family adversity to trauma exposure, PTSD symptoms, alcohol problems, and drug problems were examined in order to test the extent to which family adversity is a shared risk factor for these outcomes. Although family adversity had a significant effect on both trauma exposure and PTSD symptoms, its direct effect on alcohol problems (i.e., the "*c*" path in a meditational model) was non-significant (B= .01, p= .92, *IRR*: 1.01; see Table 10). The direct effect of family adversity on risk for drug problems was significant for females (B= 1.12, p < .001, *IRR*= 3.06; see Table 11) but not for males (B= -.01, p= .95, *IRR*: 99; see Table 11).

²¹ Follow-up analyses entered assaultive violence exposure into the model rather than trauma exposure in order to test whether assaultive violence exposure exerted unique effects on alcohol and drug problems over and above the influence of PTSD symptoms. These analyses were performed in two different ways: first, by recoding participants with trauma exposure not involving assaultive violence as 0 on the trauma variable and missing on the PTSD symptoms variable; and second, by deleting the 94 participants with trauma exposure not involving assaultive violence from the model. Results from both methods were consistent with those reported above. Assaultive violence exposure did not directly influence risk for either alcohol or drug problems over and above the effects of PTSD symptoms and the other predictors in the model. Moreover, there was no evidence that the unique effect of assaultive violence on risk for alcohol or drug problems was moderated by gender or ethnicity. Note that these results are not presented in the tables.

Finally, the extent to which trauma exposure or PTSD symptoms mediated the effect of family adversity on alcohol and drug problems was examined. Given that trauma exposure did not have a direct effect on risk for either alcohol or drug problems, there was no evidence that trauma exposure mediated the influence of pre-trauma family adversity. However, results showed that PTSD symptoms significantly mediated the effect of pre-trauma family adversity on both alcohol problems (95% CI= [.010, .038]) and drug problems (95% CI= [.001, .057]).

Gender and ethnicity as moderators of trauma exposure. Additional analyses were conducted to test whether gender or ethnicity moderated the influence of trauma exposure on risk for future alcohol or drug problems.

Hypothesis 4a. An additional analysis tested the interaction between gender and trauma exposure in order to test examine gender moderated the influence of trauma exposure on risk for future alcohol or drug problems. It was hypothesized that trauma-exposed males may be more likely to develop alcohol or drug problems following exposure compared to females, who are at greater risk for PTSD symptoms. If this hypothesis were true, gender would be expected to moderate the unique influence of trauma exposure on risk for future substance use problems such that trauma exposure exerts significant effects on substance use problems over and above PTSD symptoms for males but not females. However, results indicated that the interaction between gender and trauma exposure was non-significant for both alcohol problems (B= -.36, p= .35, IRR: .70) and drug problems (B= .01, p= .99, IRR: .1.01). Thus, there was no evidence that gender moderated the influence of trauma exposure. Because these interactions were non-significant, they were omitted from the tables.

Hypothesis 4b. An additional exploratory analysis tested the interaction between ethnicity and trauma exposure in order to examine whether ethnicity moderated the unique influence of trauma exposure on problematic alcohol or drug use. No specific hypothesis was made regarding the direction of the moderated effect. The interaction between ethnicity and trauma exposure was non-significant in the analysis predicting both alcohol problems (B= -.68, p= .10, *IRR:* .51) and drug problems (B= -1.06, p= .10, *IRR:* .35). Because these interactions were non-significant, they were omitted from the tables.

In order to make comparisons specifically between Hispanics/Latinos and non-Hispanic/Latino Caucasians, additional analyses were conducted with the 18 participants of other ethnicities were removed from analyses. Again, the interaction between ethnicity and trauma exposure was non-significant in the analysis predicting both alcohol problems (B= -.68, p= .14, IRR: .51) and drug problems (B= -1.03, p= .13, IRR: .36). Because these interactions were non-significant, they were omitted from the tables. However, it should be noted that analyses may have been underpowered to detect significant ethnicity by trauma exposure interactions with a small effect size.

In sum, there was no evidence that the unique effect of trauma exposure over and above PTSD symptoms varied across ethnicity.

Summary of shared vulnerability hypothesis findings: Approach 1. In summary, results did not support the shared vulnerability hypothesis, which

purports that the link between PTSD symptoms and substance use problems is due to shared risk factors and that there is no causal relation among PTSD symptoms and substance use problems—at least in terms of family adversity and trauma exposure as shared risk factors. Rather, the influences of both trauma exposure and family adversity on risk for future alcohol problems appeared to be fully mediated by PTSD symptoms. As for drug problems, the influence of trauma exposure on risk for future drug problems appeared to be fully mediated by PTSD symptoms. However, the influence of family adversity on risk for future drug problems appeared to be fully mediated by PTSD symptoms for males but only partially mediated by PTSD symptoms for females. Indeed, results showed that family adversity had a significant direct effect on females' but not males' risk for drug problems.

Approach 2.

Model specification and preliminary analyses. See the description of model specification for Approach 2 under the self-medication hypothesis. See Figure 4 for a depiction of Approach 2. Recall that it was not possible to test the extent to which trauma exposure with or without PTSD mediated the influence of pre-trauma family adversity because a nominal variable cannot be tested as a mediator. Nonetheless, it was possible to test whether family adversity had a unique effect on risk for future alcohol or drug problems while controlling for the effects of PTSD diagnosis and trauma exposure without PTSD (as well as pretrauma substance use problems, gender, and ethnicity).

Final models. The final models testing the influences of trauma exposure with and without PTSD on risk for future alcohol and drug problems are presented in Tables 14 and 15, respectively. The unique effect of trauma exposure in the absence of PTSD on alcohol and drug problems was examined in order to test the "trauma exposure as a shared risk factor hypothesis." The risk for adult alcohol problems was not elevated in participants exposed to trauma in the absence of PTSD compared to participants who were not exposed to trauma (B=.12, p=.53, IRR: 1.13), controlling for the effects of PTSD, family adversity, gender, ethnicity, pre-trauma substance use problems, time since trauma exposure. Similarly, the risk for adult drug problems was not elevated in participants exposed to trauma in the absence of PTSD compared to participants who were not exposed to trauma (B=.21, p=.53, IRR: 1.24), controlling for the effects of PTSD, family adversity, gender, ethnicity, pre-trauma substance use problems, and the interaction between family adversity and gender. Thus, there was no evidence for the trauma exposure as a shared risk factor hypothesis.

Next, the unique effect of family adversity on alcohol and drug problems was examined. Family adversity did not significantly influence risk for alcohol problems (B=.14, p= .23, IRR: 1.15) over and above the other variables in the model. A follow-up analysis was conducted without family adversity in order to examine the extent to which results change when adversity is not included in the model (i.e., family adversity may be an important covariate even if it does not have a significant unique effect on alcohol problems). Results showed a change in findings when family adversity was removed from the model. Specifically, the effect of the dummy variable comparing risk for alcohol problems between participants with PTSD and participants who were not exposed to trauma became significant (B= .72, p = .049, *IRR*: 2.06), whereas this effect was only marginally significant when family adversity was included in the model (B= .62, p = .091, *IRR*: 1.86). Therefore, even though the influence of pre-trauma family adversity on adult alcohol problems was non-significant, it appears that failing to account for this effect may inflate the influence of PTSD on alcohol problems.

In the model predicting drug problems, there was a significant interaction between gender and family adversity (B=1.17, p=.001, IRR: 3.21), such that family adversity predicted significantly higher levels of drug problems for females (B=1.27, p < .001, IRR: 3.58), but not for males (B=.10, p=.61, IRR: 1.11). A follow-up analysis showed that findings were unchanged when family adversity was removed from the model.

Gender and ethnicity as moderators of trauma exposure.

Hypothesis 4a. An additional analysis tested the interaction between gender and the dummy variable comparing those exposed to trauma without PTSD to those without trauma exposure in order to examine whether gender moderated the influence of trauma exposure without PTSD on risk for future alcohol or drug problems. It was hypothesized that the influence of trauma exposure without PTSD may be significant for males but not females. However, results indicated that the interaction between gender and the trauma exposure without PTSD dummy variable was non-significant for both alcohol problems (B= -.44, p= .24, IRR: .65) and drug problems (B= .12, p= .87, IRR: .1.12). Thus, there was no evidence that the effect of trauma exposure without PTSD was moderated by gender.

Hypothesis 4b. An additional exploratory analysis tested the interaction between ethnicity and the dummy variable comparing those exposed to trauma without PTSD to those without trauma exposure in order to examine whether ethnicity moderated the influence of trauma exposure without PTSD on problematic alcohol or drug use. No specific hypothesis was made regarding the direction of the moderated effect. The interaction between ethnicity and the trauma exposure without PTSD dummy variable was non-significant in the analysis predicting both alcohol problems (B= -.34, p= .40, *IRR*: .71) and drug problems (B= -.40, p= .54, *IRR*: .70).

In order to make comparisons specifically between Hispanics/Latinos and non-Hispanic/Latino Caucasians, the 18 participants of other ethnicities were removed from analyses. Again, the interaction between ethnicity and trauma exposure without PTSD was non-significant in the analysis predicting both alcohol problems (B= -.33, p= .46, IRR: .72) and drug problems (B= -.32, p= .65, IRR: .73). Thus, there was no evidence that the effect of trauma exposure without PTSD was moderated by ethnicity.

Summary of shared vulnerability hypothesis findings: Approach 2. In summary, the results from the Approach 2 analyses testing the shared vulnerability hypothesis were largely consistent with those from the Approach 1 analyses. Again, there was no support for trauma exposure as a shared risk factor. Moreover, there was a significant unique effect of family adversity on risk for
drug problems for females but not males, whereas the unique effect of family adversity on risk for alcohol problems was non-significant for both genders.

Adjusting Main Hypotheses for False Discovery Rate

The False Discover Rate approach was used to adjust for Type 1 error when testing the four primary hypotheses—the high-risk hypothesis (effect of adolescent substance use problems on risk for trauma exposure), the susceptibility hypothesis (effect of adolescent substance use problems on risk for PTSD), the self-medication hypothesis (effect of PTSD symptoms on risk for alcohol or drug problems), and the shared vulnerability hypothesis (trauma exposure and family adversity as shared risk factors for PTSD and alcohol and drug problems). All results were maintained, with the exception that the significant effect of PTSD symptoms on drug problems for non-Hispanic/Latino Caucasians became only marginally significant (FDR-adjusted p= .080). Note that the magnitudes of effect of PTSD symptoms on non-Hispanic/Latino Caucasians' risk for alcohol (IRR=1.12) and drug (IRR= 1.11) problems were approximately equal.

Discussion

The present study utilized longitudinal data from a high-risk community sample to test a series of hypotheses that may help to explain the risk pathways that link traumatic stress, PTSD symptomatology, and problematic alcohol and drug use. Specifically, this study tested whether pre-trauma substance use problems increase risk for trauma exposure (the high-risk hypothesis), whether pre-trauma substance use problems increase risk for PTSD among individuals who have been exposed to trauma (the susceptibility hypothesis), whether PTSD increases risk for the development of post-trauma alcohol or drug problems (the self-medication hypothesis), and whether shared risk factors account for both PTSD and alcohol/drug problems such that their link is non-causal (the shared vulnerability hypothesis). This study also examined the roles of gender and ethnicity in these mechanisms of risk.

A summary of results is presented in Table 18. Results provided the strongest support for the self-medication hypothesis, such that PTSD symptoms predicted higher levels of later alcohol and drug problems among non-minority (non-Hispanic/Latino Caucasian) 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 of effect (the influence of substance use problems on risk for trauma exposure or PTSD), the high-risk hypothesis was tentatively 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

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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. There was no support for the susceptibility hypothesis, as pre-trauma adolescent substance use problems did not significantly influence risk for PTSD diagnosis or 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. Each of these findings is explored in greater detail below.

High-Risk and Susceptibility Hypotheses

The present study is one of the few known studies to test whether adolescent substance use problems prospectively predicted increased risk for trauma exposure or PTSD. Importantly, the non-significant effect of adolescent substance use problems on risk for trauma exposure would have been marginally significant if pre-trauma family adversity were excluded from the model. The finding that preexisting family adversity had a significant influence on risk for trauma exposure, whereas as preexisting substance use problems did not, suggests that it is the high-risk family context within which problematic adolescent substance use is likely to co-occur (indeed, adolescent substance use problems and adolescent family adversity were significantly correlated) that increases risk for future trauma exposure, rather than problematic adolescent substance use itself. It is important for future studies to account for co-occurring family risk factors when examining individual risk factors for trauma exposure, particularly during adolescence. Failing to account for familial risk may lead to false conclusions about the extent to which associated individual behaviors lead to trauma exposure.

In contrast to the non-significant effect of adolescent substance use problems on risk for overall trauma exposure, adolescent substance use problems had a marginally significant effect on risk for *assaultive violence* exposure (i.e., events involving rape, physical assault or being threatened with a weapon, as opposed to other types of traumatic events-seeing someone hurt or killed, natural disaster, narrow escape from death/injury, sudden injury or accident, sudden death/injury of someone close, experiencing shock from other's experience, or other event), even after accounting for the significant influence of co-occurring family adversity. This finding suggests that adolescent substance use problems may place adolescents in dangerous situations where they are exposed to assaultive violence, and is consistent with retrospective data (Giaconia et al., 2000; Kilpatrick et al., 2000). For instance, adolescent substance abusers may be especially likely to be exposed to assaultive violence (e.g., physical assault; being threatened with a weapon) because they engage in dangerous activities while under the influence or during their efforts to obtain alcohol and drugs, or because they are more likely to associate with deviant peers who engage in delinquent behaviors (Barnow et al., 2004; Fergusson et al., 2002). Moreover, given that the average age at which pre-trauma adolescent substance use problems were measured in the present study was 13.2 years old, it is also 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. Understanding why substance use problems place adolescents at increased risk for assaultive violence is an important area for future research, given that assaultive violence exposure carries an especially high risk for developing PTSD compared to other types of traumatic events (Kessler, 2000).

With respect to risk for PTSD among trauma-exposed individuals (i.e., the susceptibility hypothesis, the present study found that adolescent substance use problems did not influence risk for PTSD or PTSD symptoms once pre-trauma family adversity was included in the model. However, it should be noted that the present study was underpowered to detect a small effect of adolescent substance use problems on PTSD. Similarly to the models predicting trauma exposure, adolescent substance use problems would have had a significant effect on risk for PTSD and a marginally significant effect on risk for PTSD symptoms over and above gender and ethnicity if family adversity were excluded from the models. The fact that the effect of adolescent substance use problems became nonsignificant when family adversity was included in the model indicates that it is family adversity, rather than substance use problems themselves, that increases risk for developing PTSD or PTSD symptoms. Thus, the present study does not support the theory that pre-trauma substance use problems increase susceptibility to PTSD by, for instance, disrupting the coping process and regulation of emotion in reaction to a traumatic event. Rather, the present study suggests that the stress

associated with parent psychopathology and a high-risk family environment has a much larger influence on one's risk for PTSD than does one's substance use history. For instance, trauma-exposed adolescents from adverse family environments may lack the safe context, resources, and social support needed to effectively cope with a traumatic event. Although previous retrospective data indicate that adolescents with substance use disorders are at 4 to 9 times the risk for developing PTSD compared to adolescents without substance use disorders (Deykin & Buka, 1997; Giaconia et al., 2000; Kilpatrick et al., 2000), such findings likely reflect the large body of risk factors associated with adolescent substance use disorders.

Although the present study contributes to our understanding of the extent to which early adolescent substance use problems are distal risk factors for later trauma exposure or PTSD, the timing of this study was unable to identify more proximal effects of substance use. The average time lag between age at the assessment of adolescent substance use problems and age at trauma exposure was approximately four years. Thus, the lack of evidence for the susceptibility hypothesis does not rule out the possibility that substance use immediately preceding or following a traumatic event may increase vulnerability for developing PTSD. Although measures of substance use problems that are close 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, the random nature of trauma exposure makes it nearly impossible to obtain such a measure.

The roles of gender and ethnicity. The present study also tested a series of hypotheses regarding the roles of gender and ethnicity within the high-risk and susceptibility hypotheses. Results from analyses predicting trauma exposure revealed a non-hypothesized gender by ethnicity interaction such that ethnic minority (predominantly Hispanic/Latino) males were at significantly greater risk for trauma exposure than were minority females, but male and female non-Hispanic/Latino Caucasians did not differ in their risk for trauma exposure. However, there was no evidence that substance use problems place females at greater risk for trauma exposure compared to males. Moreover, given the lack of gender difference in the prevalence of adolescent substance use problems, there was no evidence that greater prevalence of substance use problems among males contribute to the common finding in the literature that males are typically at greater risk for trauma exposure and assaultive violence than are females (Kessler et al., 1995). It should be noted, however, that because the present study assessed substance use problems in early adolescence—that is, when gender differences in substance use are unlikely to have yet developed (Johnston, O'Malley, Bachman, & Schulenberg, 2008)—the present study was poorly suited to test whether substance use problems mediate the influence of gender on trauma exposure. Future studies with measures of substance use problems in late adolescence or early adulthood will be better able to determine whether gender differences in substance use contribute to gender differences in trauma exposure.

Consistent with a large body of research documenting that females are at greater risk for PTSD compared to males (Tolin & Foa, 2006), trauma-exposed

females in the present study were at *five times* the risk for developing PTSD compared to trauma-exposed males, even after accounting for the effects of pretrauma family adversity, substance use problems, and ethnicity. This finding underscores the need to better understand why females are so much more vulnerable to PTSD than are males. We had hypothesized that one reason why females are more susceptible to PTSD may be because female substance abusers may be more likely to blame themselves for their traumatic event compared to male substance abusers, which would increase females' risk for PTSD. Given that gender did not moderate the influence of pre-trauma substance use problems on risk for PTSD, there was no evidence that this hypothesis was true. However, please note effect sizes for interaction terms in psychology tend to be small (Champoux & Peters, 1987), and that the present study was underpowered to detect the gender moderated effect of substance use problems on risk for PTSD. Studies with larger sample sizes and measures of substance use closer in time to the traumatic event will be better-suited to testing this hypothesis.

Finally, with respect to the role of ethnicity in risk for PTSD, the present study is consistent with previous findings that Hispanics/Latinos are at particularly high risk for developing PTSD compared to other ethnicities (Galea et al., 2002; Pole et al., 2005; Schnurr et al., 2004). Indeed, results indicated that trauma-exposed Hispanics/Latinos were at nearly three times the risk for PTSD than were trauma-exposed non-Hispanic/Latino Caucasians. Potential explanations for Hispanics/Latinos increased risk for PTSD include increased tendency toward somatization, greater peritraumatic dissociation, self-blame coping, and perceived racism (Ortega et al., 2000; Pole et al., 2005).

The Self-Medication and Shared Vulnerability Hypotheses

The present study adds to a growing literature in support of the selfmedication hypothesis, such that individuals may use alcohol and drugs to cope with PTSD symptoms (e.g., induce sleep, reduce irritability, reduce concentration problems, reduce hypervigilance, etc.). Results indicated that it is the extent to which one experiences PTSD symptoms that influences risk for future alcohol or drug problems, rather than the categorical presence or absence of clinically significant PTSD. Indeed, for every one unit increase in PTSD symptoms, non-Hispanic Latino Caucasians' risk for alcohol problems increased 12 percent and risk for drug problems increased 11 percent. However, PTSD *diagnosis* had only a marginally significant unique effect on risk for alcohol problems, and its effect on risk for drug problems was non-significant. Previous research suggests that PTSD diagnoses may represent arbitrary cutoff points rather than clinically meaningful dividing points (Cohen et al., 1998). Moreover, it is well-established that dichotomous variables have lower statistical power than do continuous variables (e.g., MacCallum, Zhang, Preacher, & Rucker, 2002). These studies, together with the results from the present study, suggest that the count measure of PTSD symptoms more accurately captures PTSD-related risk for future substance use problems than does the categorical measure of PTSD diagnosis.

Findings from the present study extend previous research on the selfmedication hypothesis in several important ways. First, this study accounted for the influence of pre-trauma, premorbid levels of substance use problems in the link between PTSD and later substance use problems. Previous research has typically examined patterns of onset among PTSD and substance use disorders, which ignores the role that subclinical levels of substance use may play in risk for both trauma exposure and post-trauma maladjustment. For example, it is likely that individuals who misuse alcohol and drugs prior to trauma exposure are also more likely to develop additional alcohol and drug problems in response to that exposure. By controlling for preexisting substance use problems, the present study rules out the possibility that the association between PTSD symptoms and later adult alcohol and drug problems is simply a continuation of substance use problems that were already present.

Second, the present study extends previous research on the selfmedication hypothesis by differentiating between the effects of traumatic stress and PTSD on future substance use problems. Few studies have recognized that trauma exposure may be a shared risk factor for both PTSD and substance use disorders, such that some individuals may develop PTSD in response to a traumatic event whereas others may develop substance use disorders in response to a traumatic event, depending on their predispositions (see Breslau et al., 2003; Yehuda et al., 1998). Therefore, the present study advances previous research by testing the separate effects of PTSD and traumatic stress on risk for later alcohol and drug problems. In contrast to findings from large epidemiological studies (Fetzner et al., 2011), results from analyses testing the effects of trauma exposure both with and without PTSD (i.e., Approach 2) indicated that trauma exposure in the absence of PTSD had no effect on alcohol or drug problems. Similarly, analyses simultaneously modeling the effects of PTSD symptoms and trauma exposure demonstrated that PTSD symptoms increased risk for alcohol and drug problems, but traumatic stress itself did not. Therefore, the present study does not support the theory that traumatic stress may influence alcohol and drug problems, independent of PTSD symptoms (i.e., the trauma exposure as a shared risk factor hypothesis). Given the lack of direct effect of trauma exposure on substance use problems, findings suggest that the influence of traumatic stress on future alcohol and drug problems is fully mediated by PTSD 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. Given that previous research has shown that family risk factors such as parental psychopathology, family conflict, and family stress may increase risk for both PTSD (Brewin et al., 2000) and substance use problems (Goodman & Gotlib, 1999; Zhou et al. 2006), this study tested whether preexisting family risk accounts for the PTSD-substance use link such that there is no causal relation between PTSD and substance use problems (i.e., the shared vulnerability hypothesis). Although zero-order correlations showed that family adversity during adolescence was significantly associated with trauma exposure, PTSD diagnosis and symptoms, alcohol problems, and drug problems, 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, the present study nonetheless suggests that preexisting family adversity may play an important role in the PTSD-substance abuse 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 among non-Hispanic/Latino Caucasians (see discussion of findings pertaining to ethnicity on page 106).

In summary, this is the first known longitudinal, community-based study to demonstrate that PTSD significantly influences the development of future alcohol and drug problems over and above the influence of trauma exposure itself, preexisting family risk factors, and baseline levels of substance use. It is important to note that the measures of alcohol and drug problems in the present study occurred approximately five years after the assessment of PTSD symptoms (and eight years after trauma exposure), thus providing strong evidence that PTSD symptoms have long-lasting effects on substance use problems. Although previous research has made it clear that substance abuse is a prevalent problem in the aftermath of trauma (Chilcoat & Menard, 2003; Vlahov et al., 2002), 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 recent study by Swendsen and colleagues (2010), which showed that PTSD diagnosis prospectively predicted onset of alcohol and drug dependence 10 years later.

Note that this study's finding that PTSD symptoms directly increased non-Hispanic/Latino Caucasians' 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 symptomatology. There are several methodological differences that may help explain the difference in results. 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, the present 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 (one year, rather than two years). 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 should be noted that it appears to be PTSD symptoms (e.g., hyperarousal, intrusive thoughts), specifically, that increase risk for substance use problems, rather than broader internalizing symptomatology (e.g., sad mood, decreased energy, feeling worthless) that is experienced during the aftermath of trauma.

The roles of gender and ethnicity. The primary finding with respect to the role of gender and ethnicity within the self-medication and shared vulnerability hypotheses is that PTSD symptoms increased risk for non-Hispanic/Latino Caucasians' alcohol and drug problems but not Hispanics/Latinos' alcohol and drug problems. Few previous studies have examined whether the link between PTSD and substance use problems varies by ethnicity. It is interesting that although Hispanics/Latinos were at elevated risk for PTSD compared to non-Hispanic Caucasians, this risk did not translate into higher levels of PTSD-related substance use problems for Hispanics/Latinos. In fact, for drug problems, there was evidence that PTSD may even have a *protective* effect on Hispanics/Latinos' risk for drug problems. Indeed, results indicated that Hispanic/Latino participants with PTSD were at 89 percent *lower* risk for adult drug problems compared to Hispanic/Latino participants without trauma exposure, and at 91 percent lower

risk for adult drug problems compared to Hispanic/Latino participants who were exposed to trauma but did not develop PTSD. However, replication of this finding is needed before definitive conclusions can be made regarding the tendency to self-medicate PTSD among Hispanics/Latinos due to the small sample size of the current study (there were 11 Hispanics/Latinos with PTSD, 33 with trauma exposure but no PTSD, and 40 without trauma exposure).

Consistent with our findings, cross-sectional data from large epidemiological studies indicate that Hispanics/Latinos may be less likely than other ethnicities to use substances to self-medicate mood (Bolton et al., 2009) and anxiety disorders other than PTSD (Robinson et al., 2009), although no differences were found with respect to PTSD, specifically (Leeies et al., 2010).²² Research also indicates that Hispanic/Latino adults in the U.S. are at one-fourth the risk for dual (co-occurring) diagnosis of substance and non-substance mental disorders, although U.S.-born Hispanics/Latinos are at greater risk for dual diagnoses than are foreign-born Hispanics/Latinos (Vega, Canino, Cao, & Alegria, 2009). Nonetheless, these studies, along with the present study (in which 96% of Hispanic/Latino participants were born in the U.S.), tentatively suggest that Hispanic/Latino ethnicity may be a protective factor in terms of a selfmedication pathway from PTSD to substance use problems.

Unfortunately, previous literature offers little to help understand *why* Hispanics/Latinos may be at lower risk for self-medication of PTSD symptoms.

²² Note that these studies defined self-medication using two dichotomous questions asking participants whether they ever drank or took medication or drugs to manage symptoms pertaining to whichever mood or anxiety disorder(s) they endorsed, and therefore do not directly map on to the self-medication mechanism tested in the present study.

As for substance use itself, previous research has found that Hispanic/Latino cultural norms against heavy substance use, proscribed gender roles (e.g., early marriage), religiosity, and an emphasis on the well-being of the family rather than individual interests (i.e., *familism*) are associated with lower rates of substance use disorders among Hispanics/Latinos, particularly Hispanic/Latino women (Canino, Vega, Sribney, Warner, & Alegria, 2008; Vega et al., 1993). Acculturation may erode these protective effects and thereby increase risk for Hispanics/Latinos' substance use, especially among women, as Hispanic/Latino cultures tend to be less accepting of female substance use compared to mainstream European-American culture (Caetano & Clark, 2003; De La Rosa, Holleran, Rugh, & MacMaster, 2005). It should be noted, however, that there were no mean differences in adult alcohol and drug problems between Hispanic/Latino participants and non-Hispanic/Latino Caucasian participants in our study, suggesting that ethnic/cultural factors were unlikely to have played a significant role in overall risk for substance use problems. Nonetheless, protective cultural factors have not been investigated within the context of protecting Hispanics/Latinos from the development of PTSD-related substance use problems. For instance, familism and cultural norms limiting substance use among women may make it less likely that Hispanics/Latinos turn to alcohol or drugs to cope with PTSD symptoms. Understanding why Hispanics/Latinos appear to be at lower risk for self-medicating PTSD symptoms may be an important avenue to identifying factors that protect against PTSD-related substance use problems.

Although the present study found that the effect of PTSD diagnosis/symptoms on future alcohol and drug problems varied across ethnicity, there was no evidence that the influence of trauma exposure itself (i.e., independent of PTSD) on substance use problems varied across ethnicity. Indeed, trauma exposure did not uniquely influence risk for alcohol or drug problems for participants of any ethnicity.

As for gender, there was no evidence that gender moderated the influence of either trauma exposure or PTSD diagnosis/symptoms on adult alcohol or drug problems. It was hypothesized that the influence of PTSD diagnosis/symptoms on substance use problems would be particularly stronger for females than for males. However, although males exhibited significantly higher overall levels of adult alcohol and drug problems compared to females, the tendency to abuse alcohol or drugs to self-medicate PTSD symptoms did not vary across gender. Thus, the finding in the literature that PTSD is more likely to onset prior to substance use disorder for females than for males with PTSD-SUD comorbidity (Stewart et al., 2002) does not appear to be attributable to a gender difference in the tendency to self-medicate PTSD. Please note, however, that the present study's small sample size limited the power to detect significant gender moderation.

Finally, the present study also failed to support the hypothesis that males, whose risk for PTSD is typically lower compared to females (Kessler et al., 1995), may develop substance use problems in response to trauma rather than developing PTSD symptoms. Such vulnerability would be evidenced by a significant effect of trauma exposure on males' substance use problems, independent of PTSD. Given that trauma-exposed males without PTSD were not at higher risk for future alcohol and drug problems relative to unexposed males, there was no support for the notion that men respond to trauma by abusing alcohol and drugs. In fact, the influence of trauma exposure on future alcohol and drug problems appeared to be fully mediated by PTSD for both males and females. Because this theory also failed to garner support in a previous study by Breslau and colleagues (2003 with a larger sample size (n = 899), it is unlikely that the null findings in the present study can simply be attributed to a lack of statistical power.

Strengths, Limitations, and Conclusions

Several limitations to this study should be noted. First, some of the strongest predictors of posttraumatic maladjustment may be specific characteristics of the traumatic experience and the posttraumatic environment, such as the experience of shame, peritraumatic dissociation, and a sense of powerlessness (Ozer et al., 2003; Resick, Monson, & Rizvi, 2008; van der Kolk, 1994). Examining the role of such risk factors was beyond the scope of the present study. Examining how these more proximal factors may mediate or moderate the influence of preexisting vulnerabilities remains an important area for future research. Second, this study did not examine the influence of genes or gene-environment interactions, although it is likely that genetic factors may interact with risk and protective factors to influence posttraumatic outcomes. Future family and twin studies are needed to better elucidate genetic and environmental effects on PTSD-substance disorder comorbidity. Third, the sample size for the present study was relatively small and included a low prevalence of PTSD (8% of overall sample, or 18% of trauma exposed sample). Thus, replication of findings is needed in larger samples with greater prevalence of PTSD. Such studies will also have greater power to test gender and ethnicity as moderators of the hypothesized pathways.

Fourth, the present study did not examine shorter-term, more functional relations between PTSD and substance use behaviors, or the relations between specific clusters of symptoms and specific types of drugs, although it is likely that these relations exist (Stewart et al., 1998). Similarly, PTSD symptomatology was only assessed at one time point in the sample for this study, and thus this study was unable to examine reciprocal influences among PTSD symptoms and substance use problems. Future studies with shorter time lags and multiple assessments are needed to examine how PTSD symptoms and substance use problems reciprocally influence each other over time.

Fifth, research has highlighted the particularly deleterious effects that repeated or chronic exposure to trauma, particularly in the form of interpersonal violence or childhood abuse, may have on wide-ranging aspects of self-regulation and psychosocial functioning. This pervasive pattern of impairment associated with repeated victimization has been termed "Complex PTSD" or "Disorders of Extreme Stress Not Otherwise Specified" (DESNOS; Herman, 1992; Roth, Newman, Pelcovitz, van der Kolk, & Mandel, 1997). However, this study eliminated participants who experienced traumatic events prior to the first wave of data in order to ensure that prospective, pre-trauma measures of adolescent substance use problems and family adversity were available for all participants. Therefore, patterns of multiple, early trauma exposures and posttraumatic impairment were beyond the scope of the present study and were not examined.

Sixth, as previously noted, substance use problems were measured at a very young age in the present study. Future studies with measures of substance use problems later during adolescence and closer in time to the traumatic event may reveal larger effects of substance use problems on trauma exposure (highrisk hypothesis) or PTSD (susceptibility hypothesis). Seventh, because this study oversampled children of parents with alcohol disorders, the effects of family adversity on trauma exposure, PTSD diagnosis/symptoms, and substance use problems may have been magnified compared to the general population. Finally, although the present study failed to support the shared vulnerability hypothesis with respect to trauma exposure and family adversity, it should be noted that there may be many other third variables that may contribute to the association between PTSD and substance use problems.

Despite these limitations, there are a number of methodological strengths of the present study that allow it to make important contributions to existing research on the PTSD-substance use problems link. First, the present study is among the few longitudinal, community-based studies to test the directions of influence among trauma exposure, PTSD, and problematic alcohol and drug use. Indeed, the majority of studies on the overlap between PTSD and substance use problems consist largely of cross-sectional, retrospective, and clinic-based studies. Second, this is the first known study to examine how family functioning during adolescence prospectively influences risk for both trauma exposure and post-trauma adjustment. Among the few prospective community studies that do exist (e.g., Breslau et al., 2006; Reed et al., 2007; Storr et al., 2007; Storr, Schaeffer, Petras, Ialongo, & Breslau, 2009), there appear to be none with comprehensive measures of pre-trauma family risk factors during adolescence. Findings from the present study highlight family adversity during adolescence as a particularly important risk factor for trauma exposure, PTSD, and adult alcohol drug problems, alike. Moreover, results indicated that failing to account for family adversity would have led to an overestimation of the extent to which substance use problems influence risk for trauma exposure and PTSD.

Third, by accounting for pre-trauma adolescent substance use problems, the present study allows for strong inferences to be made regarding the direction of effect among PTSD symptoms and substance use problems. Indeed, the present study provides much stronger evidence for an effect of PTSD on substance use problems, than for an effect of substance use problems on PTSD. Fourth, the present study simultaneously estimated the effects of PTSD symptoms and traumatic stress on substance use problems, thereby allowing an examination of the extent to which traumatic stress may have unique effects on substance use problems independent of PTSD symptomatology. Previous studies examining the unique effects of trauma exposure have used a categorical approach, in which trauma-exposed individuals with or without PTSD were compared to individuals without trauma exposure. The present study is the first known study to test the effects of traumatic stress over and above subclinical levels of PTSD symptoms. The fact that trauma exposure failed to significantly influence risk for alcohol or drug problems while controlling for both clinical and subclinical levels of PTSD provides strong evidence that the effects of traumatic stress on substance use problems are fully mediated by PTSD symptoms. Moreover, by examining PTSD as both a categorical and count variable, the present study also demonstrated that even though the vast majority of trauma-exposed individuals do not meet criteria for PTSD (Kessler et al., 1995), 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.

Fifth, the composition of the sample for the present study enabled this study to contribute to a better understanding of the roles of gender and ethnicity in the risk mechanisms among trauma, PTSD, and substance use problems. Specifically, this sample was comprised of approximately half males and half females, whereas many studies in the trauma-substance use literature have been comprised of only males or only females (e.g., samples of male war veterans or female victims of sexual/physical assault). Although findings pertaining to gender as a moderator of the hypothesized pathways were mostly null (note that tests of gender moderation were far from significant, and it is therefore unlikely that null findings were due to low power), testing gender as a moderator allowed for conclusions to be made about the extent to which pathways vary across gender. Accounting for gender in the hypothesized pathways also increases the generalizability of the findings. Moreover, this study's sample was also comprised of predominantly non-Hispanic/Latino Caucasians and Mexican-Americans, and was thus ideal for examining Hispanic/Latino ethnicity as a moderator of risk mechanisms. Although there are important differences between Hispanics/Latinos and non-Hispanic/Latino Caucasians in both the PTSD and SUD literatures, little research has examined the potential influence of Hispanic/Latino ethnicity in the PTSD-SUD link. Indeed, this is the first known study to find that Hispanics/Latinos, who are typically at greater risk for PTSD compared to other ethnicities, appear to be significantly less likely to abuse substances to self-medicate PTSD symptoms compared to non-Hispanic/Latino Caucasians.

Finally, given that risk for trauma exposure peaks between the ages of 16 and 20 (Breslau et al., 1998), the timing of the present study, which followed participants from adolescence into adulthood (age 13 to 26, on average) contributes to our understanding of why risk for trauma exposure is so high during late adolescence/early adulthood. Indeed, results indicated that adolescent substance use problems may increase risk for being exposed to assaultive violence. 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.

In summary, the present study makes important contributions to disentangling the directions of influence among traumatic stress, PTSD symptoms, and substance use 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 substance use disorders. This study also provided support for adolescent substance use problems as a risk factor for assaultive violence exposure. Findings from the present study are thus consistent with the notion that multiple, non-mutually exclusive pathways may underlie the link between PTSD and substance use problems. Further research is needed to better understand how multiple risk mechanisms may interact with each other over time to influence the maintenance and course of PTSD and substance use symptoms across the lifespan.

The present study has implications for the prevention of substance use problems among individuals who present for treatment for PTSD. It is important that clinicians routinely assess clients' risk for using alcohol or drugs to selfmedicate PTSD symptoms, discuss the long-term dangers associated with selfmedication, and provide other means of coping with PTSD symptoms (e.g., relaxation training). Additionally, more formalized prevention efforts may be warranted. Future preventive interventions may target individuals with PTSD who are at risk for developing substance use problems (e.g., due to temperament or past substance use behavior).

This study also highlights the need to screen for and treat PTSD symptomatology among individuals who present with substance use problems. Importantly, research indicates a low detection rate of PTSD within addiction treatment centers because individuals with substance use problems often to do not report traumatic experiences and PTSD symptoms unless specifically asked (Kimerling, Trafton, & Nguyen, 2006; Reynolds et al., 2005). Moreover, it appears that individuals with concurrent PTSD symptoms and substance use problems are especially hard to treat, and do not optimally benefit from standard substance use disorder interventions (Mills, Teesson, Ross, Darke, & Shanahan, 2005; Norman, Tate, Anderson, & Brown, 2007; Ouimette, Finney, & Moos, 1999).

Although it is clear that co-occurring PTSD symptoms and substance use problems present unique treatment challenges, the extant literature on the treatment of PTSD-substance use disorder comorbidity is surprisingly small (for reviews, see van Dam, Vedel. Ehring. & Emmelkamp, 2012; Torchalla, Nosen, Rostam, & Allen, 2012). Understanding the development and treatment of cooccurring PTSD symptoms and substance use problems remains an important area for future research.

REFERENCES

- Acierno, R., Resnick, H., Kilpatrick, D. G., Saunders, B., & Best, C. L. (1999). Risk factors for rape, physical assault, and posttraumatic stress disorder in women: Examination of differential multivariate relationships. *Journal of Anxiety Disorders*, 13(6), 541-563.
- Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Thousand Oaks, CA, US: Sage Publications, Inc.
- Allport, G. W., Bruner, J. S., & Jandorf, E. M. (1941). Personality under social catastrophe: Ninety life-histories of the Nazi revolution. *Character & Personality; A Quarterly for Psychodiagnostic & Allied Studies, 10*, 1-22.
- American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders: Text Revision* (4th ed.). Washington, DC: Author.
- Andreski, P., Chilcoat, H., & Breslau, N. (1998). Post-traumatic stress disorder and somatization symptoms: A prospective study. *Psychiatry Research*, 79(2), 131-138. doi:10.1016/S0165-1781(98)00026-2
- Back, S., Dansky, B.S., Coffey, S.F., Saladin, M.E., Sonne, S., & Brady, K.T. (2000). Cocaine dependence with and without posttraumatic stress disorder: A comparison of substance use, trauma history and psychiatric comorbidity. *The American Journal on Addictions*, 9(1), 51-62.
- Barnow, S., Schultz, G., Lucht, M., Ulrich, I., Preuss, U-W., & Freyberger, H-J. (2004). Do alcohol expectancies and peer delinquency/substance use mediate the relationship between impulsivity and drinking behavior in adolescence? *Alcohol and Alcoholism*, 39, 213-219.
- Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: a practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society, Series B*, *57*(1), 289-300.
- Bloom, B. (1985). A factor analysis of self-report measure of family functioning. *Family Process, 24,* 225-239.
- Bloom, B. L., & Naar, S. (1994). Self-report measures of family functioning: Extensions of a factorial analysis. *Family Process*, *33*, 203–216.
- Bolton, J. M., Robinson, J., & Sareen, J. (2009). Self-medication of mood disorders with alcohol and drugs in the national epidemiologic survey on alcohol and related conditions. *Journal of Affective Disorders*, 115(3), 367-375. doi:10.1016/j.jad.2008.10.003

- Bonanno, G. A., Galea, S., Bucciarelli, A., & Vlahov, D. (2007). What predicts psychological resilience after disaster? the role of demographics, resources, and life stress. *Journal of Consulting and Clinical Psychology*, 75(5), 671-682.
- Boscarino, J.A. (2006). Posttraumatic stress disorder and mortality among US Army veterans 30 years after military service. *Annals of Epidemiology*, *16*, 248-58.
- Brady, K.T., & Randall, C.L. (1999). Gender differences in substance use disorders. *Psychiatric Clinics of North America*, 22(2), 241-252.
- Brady, K. T., & Sinha, R. (2005). Co-occurring mental and substance use disorders: The neurobiological effects of chronic stress. *The American Journal of Psychiatry*, 162(8), 1483-1493.
- Breslau, N. (2009). The epidemiology of trauma, PTSD, and other posttrauma disorders. *Trauma, Violence, & Abuse, 10*(3), 198-210.
- Breslau, N., & Anthony, J. C. (2007). Gender differences in the sensitivity to posttraumatic stress disorder: An epidemiological study of urban young adults. *Journal of Abnormal Psychology*, 116(3), 607-611.
- Breslau, N., Davis, G. C., Andreski, P., & Peterson, E. (1991). Traumatic events and posttraumatic stress disorder in an urban population of young adults. *Archives of General Psychiatry*, 48(3), 216-222.
- Breslau, N., Davis, G. C., Peterson, E. L., & Schultz, L. (1997). Psychiatric sequelae of posttraumatic stress disorder in women. Archives of General Psychiatry, 54(1), 81-87.
- Breslau, N., Davis, G. C., Peterson, E. L., & Schultz, L. R. (2000). A second look at comorbidity in victims of trauma: The posttraumatic stress disorder– major depression connection. *Biological Psychiatry*, 48(9), 902-909.
- Breslau, N., Davis, G. C., & Schultz, L. R. (2003). Posttraumatic stress disorder and the incidence of nicotine, alcohol, and other drug disorders in persons who have experienced trauma. *Archives of General Psychiatry*, 60(3), 289-294.
- Breslau, N., Kessler, R. C., Chilcoat, H. D., Schultz, L. R., Davis, G. C., & Andreski, P. (1998). Trauma and posttraumatic stress disorder in the community: The 1996 Detroit area survey of trauma. *Archives of General Psychiatry*, 55(7), 626-632.

- Breslau, N., Lucia, V. C., & Alvarado, G. F. (2006). Intelligence and other predisposing factors in exposure to trauma and posttraumatic stress disorder. *Archives of General Psychiatry*, 63(11), 1238-1245.
- Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal* of Consulting and Clinical Psychology, 68(5), 748-766.
- Bromet, E., Sonnega, A., & Kessler, R.C. (1998). Risk factors for DSM-III-R posttraumatic stress disorder: Findings from the National Comorbidity Survey. *American Journal of Epidemiology*, 147(4), 353-361.
- Brown, P.J., & Ouimette, P. (2003). Introduction. In P. Ouimette, & P. J. Brown (Eds.), *Trauma and substance abuse: Causes, consequences, and treatment of comorbid disorders*. (pp. 3-5). Washington, DC, US: American Psychological Association.
- Brown, P. J., Stout, R. L., & Gannon-Rowley, J. (1998). Substance use disorder-PTSD comorbidity: Patients' perceptions of symptom interplay and treatment issues. *Journal of Substance Abuse Treatment*, 15(5), 445-448.
- Brown, P. J., Stout, R. L., & Mueller, T. (1996). Posttraumatic stress disorder and substance abuse relapse among women: A pilot study. *Psychology of Addictive Behaviors*, 10(2), 124-128.
- Brown, T. A., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and lifetime comorbidity of the *DSM-IV* anxiety and mood disorders in a large clinical sample. *Journal of Abnormal Psychology*, *110*(4), 585-599.
- Buka, S. L., Stichick, T. L., Birdthistle, I., & Earls, F. J. (2001). Youth exposure to violence: Prevalence, risks and consequences. *American Journal of Orthopsychiatry*, 71(3), 298-310.
- Burnam, M. A., Stein, J. A., Golding, J. M., Siegel, J. M., Sorenson, S. B., Forsythe, A., et al. (1988). Sexual assault and mental disorders in a community population. *Journal of Consulting and Clinical Psychology*, 56(6), 843-850.
- Caetano, R., & Clark, C.L. (2003). Acculturation, alcohol consumption, smoking, and drug use among Hispanics. In K.M. Chun, P. Balls Organista, & G. Marín (Eds.), Acculturation: Advances in Theory, Measurement, and Applied Research (pp. 223-239). Washington, DC, US: American Psychological Association.

- Canino G, Vega W, Sribney W, Warner L, Alegría M. (2008). Social Relationships, Social Assimilation, and Substance use Disorder among Adult Latinos in the U.S. *Journal of Drug Issues*, *38*(1), 69-102.
- Cattell, R. B. (1966). The scree test for the number of factors. *Multivariate Behavioral Research, 1*, 245-276.
- Champoux, J. E., & Peters, W. S. (1987). Form, effect size and power in moderated regression analysis. *Journal of Occupational Psychology*, 60(3), 243-255.
- Chan, K., Neighbors, C., Gilson, M., Larimer, M. E., & Marlatt, G. A. (2007). Epidemiological trends in drinking by age and gender: Providing normative feedback to adults. *Addictive Behaviors*, 32, 967–976.
- Chassin, L., Barrera, M., Bech, K., & Kossak-Fuller, J. (1992). Recruiting a community sample of adolescent children of alcoholics: A comparison of three subject sources. *Journal of Studies on Alcohol*, *53*(4), 316-319.
- Chassin, L., Rogosch, F., & Barrera, M. (1991). Substance use and symptomatology among adolescent children of alcoholics. *Journal of Abnormal Psychology*, *100*(4), 449-463.
- Chilcoat, H. D., & Breslau, N. (1998a). Investigations of causal pathways between PTSD and drug use disorders. *Addictive Behaviors, 23*(6), 827-840.
- Chilcoat, H. D., & Breslau, N. (1998b). Posttraumatic stress disorder and drug disorders: Testing causal pathways. *Archives of General Psychiatry*, 55(10), 913-917.
- Chilcoat, H. D., & Menard, C. (2003). Epidemiological investigations:
 Comorbidity of posttraumatic stress disorder and substance use disorder.
 In P. Ouimette, & P. J. Brown (Eds.), *Trauma and substance abuse: Causes, consequences and treatment of comorbid disorders* (pp. 9–28).
 Washington, DC: American Psychological Publishing.
- Coffey, S. F., Read, J. P., & Norberg, M. M. (2008). Posttraumatic stress disorder and substance use disorder: Neuroimaging, neuroendocrine, and psychophysiological findings. In S. H. Stewart, & P. J. Conrod (Eds.), *Anxiety and substance use disorders: The vicious cycle of comorbidity.* (pp. 37-57). New York, NY, US: Springer Science + Business Media.
- Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences* (2nd ed.). Mahwah, NJ: Erlbaum.

- Cohen, J. A., & American Academy of Child & Adolescent Psychiatry, Work Group on Quality Issues, Washington, DC, US. (1998). Practice parameters for the assessment and treatment of children and adolescents with posttraumatic stress disorder. *Journal of the American Academy of Child & Adolescent Psychiatry Special Issue: Practice Parameters*, 37(10), 4S-26S.
- Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences* (3rd ed.). Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers.
- Cook, R.D. (1977). Detection of influential observations in linear regression. *Technometrics*, *19*, 15-18.
- Cottler, L. B., Compton, W. M., Mager, D., & Spitznagel, E. L. (1992).
 Posttraumatic stress disorder among substance users from the general population. *The American Journal of Psychiatry*, 149(5), 664-670.
- Cornelius, J.R., Kirisci, L., Reynolds, M., Clark, D.B., Hayes, J., & Tarter, R. (2010). PTSD contributes to teen and young adult cannabis use disorders. *Addictive Behaviors*, *35*(2), 91-94.
- Darves-Bornoz, J., Choquet, M., Ledoux, S., Gasquet, I., & Manfredi, R. (1998). Gender differences in symptoms of adolescents reporting sexual assault. *Social Psychiatry and Psychiatric Epidemiology*, 33(3), 111-117.
- Davis, K. C., Stoner, S. A., Norris, J., George, W. H., & Masters, N. T. (2009). Women's awareness of and discomfort with sexual assault cues: Effects of alcohol consumption and relationship type. *Violence Against Women*, 15, 1106-1125.
- De Bellis, M. D. (2002). Developmental traumatology: A contributory mechanism for alcohol and substance use disorders. *Psychoneuroendocrinology: Special Issue: Stress and Drug Abuse, 27*(1-2), 155-170.
- De La Rosa, M.R., Holleran, L.K., Rugh, D., & MacMaster, S.A. (2005). Substance abuse among U.S. Latinos: A review of the literature. *Journal* of Social Work Practice in the Addictions, 5, 1-20.
- Deykin, E. Y., & Buka, S. L. (1997). Prevalence and risk factors for posttraumatic stress disorder among chemically dependent adolescents. *The American Journal of Psychiatry*, 154(6), 752-757.
- Driessen, M., Schulte, S., Luedecke, C., Schaefer, I., Sutmann, F., Ohlmeier, M., et al. (2008). Trauma and PTSD in patients with alcohol, drug, or dual dependence: A multi-center study. *Alcoholism: Clinical and Experimental*

Research: Special Issue: Alcohol and Adolescent Brain Development, 32(3), 481-488.

- Dube, S.R., Felitti, V.J., Dong, M., Chapman, D.P., Giles, W.H., & Anda, R.F. (2003). Childhood abuse, neglect, and household dysfunction and the risk of illicit drug use: The adverse childhood experiences survey. *Pediatrics*, 111, 564-572.
- Eberly, R. E., Harkness, A. R., & Engdahl, B. E. (1991). An adaptational view of trauma response as illustrated by the prisoner of war experience. *Journal of Traumatic Stress*, 4(3), 363-380. doi:10.1002/jts.2490040305
- Endicott, J., Anderson, N., & Spitzer, R. L. (1975). *Family History Diagnostic Criteria*. New York Biometrics Research, New York Psychiatric Institute.
- Epstein, J. N., Saunders, B. E., Kilpatrick, D. G., & Resnick, H. S. (1998). PTSD as a mediator between childhood rape and alcohol use in adult women. *Child Abuse & Neglect*, 22(3), 223-234.
- Fergusson, D. M., Swain-Campbell, N. R., & Horwood, J. (2002). Deviant peer affiliations, crime and substance use: A fixed effects regression analysis. *Journal of Abnormal Child Psychology*, 39, 419-430.
- Fetzner, M.G., McMillan, K.A., Sareen, J., & Asmundson, G. (2011). What is the association between traumatic life events and alcohol abuse/dependence in people with and without PTSD? Findings from a nationally representative sample. *Depression and Anxiety, 28,* 632-638.
- Friedman, M.J., & Yehuda, R. (1995). Post-traumatic stress disorder and comorbidity: Psychobiological approaches to differential diagnosis. In M.J. Friedman, D.S. Charney, & A.Y. Deutch (Eds.), *Neurobiological and Clinical Consequences of Stress: From Normal Adaptation to Post-Traumatic Stress Disorder*. (pp. 429-445). Philadelphia, PA: Lippincott-Raven.
- Fritz, M.S., & MacKinnon, D.P. (2007). Required sample size to detect the mediated effect. *Psychological Science*, *18*, 233-239.
- Galea, S., Ahern, J., Resnick, H., Kilpatrick, D., Bucuvalas, M., Gold, J., et al. (2002). Psychological sequelae of the September 11 terrorist attacks in New York City. *The New England Journal of Medicine*, 346(13), 982-987.
- Gfroerer, J., & de la Rosa, M. (1993). Protective and risk factors associated with drug use among Hispanic youth. *Journal of Addictive Diseases*, *12*(2), 87-107. doi:10.1300/J069v12n02_07

- Giaconia, R. M., Reinherz, H. Z., Hauf, A. C., Paradis, A. D., Wasserman, M. S., & Langhammer, D. M. (2000). Comorbidity of substance use and posttraumatic stress disorders in a community sample of adolescents. *American Journal of Orthopsychiatry*, 70(2), 253-262.
- Giaconia, R. M., Reinherz, H. Z., Paradis, A. D., & Stashwick, C. K. (2003).
 Comorbidity of substance use disorders and posttraumatic stress disorder in adolescents. In P. Ouimette, & P. J. Brown (Eds.), *Trauma and substance abuse: Causes, consequences, and treatment of comorbid disorders*. (pp. 227-242). Washington, DC, US: American Psychological Association.
- Giaconia, R. M., Reinherz, H. Z., Silverman, A. B., & Pakiz, B. (1995). Traumas and posttraumatic stress disorder in a community population of older adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, 34(10), 1369-1380.
- Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review*, *106*(3), 458-490.
- Green, B. L., Kramer, T. L., Grace, M. C., Gleser, G. C., Leonard, A. C., Vary, M. G., et al. (1997). Traumatic events over the life span: Survivors of the buffalo creek disaster. In T. W. Miller (Ed.), *Clinical disorders and stressful life events*. (pp. 283-305). Madison, CT, US: International Universities Press, Inc.
- Guo, J., Hill, K. G., Hawkins, J. D., Catalano, R. F., & Abbott, R. D. (2002). A developmental analysis of sociodemographic, family and peer effects on adolescent illicit drug initiation. *Journal of the American Academy of Child & Adolescent Psychiatry*, 41, 838-845.
- Haller, M., & Chassin, L. (2012). The influence of PTSD symptoms on alcohol and drug problems: Internalizing and externalizing pathways. *Psychological Trauma: Theory, Research, Practice, and Policy*. Advance online publication.
- Harwood, H. (2000). Updating Estimates of the Economic Costs of Alcohol Abuse in the United States: Estimates, Update Methods, and Data. Report prepared by The Lewin Group for the National Institute on Alcohol Abuse and Alcoholism. Rockville, MD: U.S. Department of Health and Human Services.
- Heim, C., & Nemeroff, C. B. (1999). The impact of early adverse experiences on brain systems involved in the pathophysiology of anxiety and affective disorders. *Biological Psychiatry*, 46(11), 1509-1522.

- Herman, J. L. (1992). Complex PTSD: A syndrome in survivors of prolonged and repeated trauma. *Journal of Traumatic Stress*, 5(3), 377-391.
- Hien, D., Cohen, L., & Campbell, A. (2005). Is traumatic stress a vulnerability factor for women with substance use disorders? *Clinical Psychology Review*, 25, 813–823.
- Hilbe, J. M. (2007). *Negative binomial regression*. Cambridge, UK: Cambridge University Press.
- Johnston, L. D., O'Malley, P. M., Bachman, J. G., & Schulenberg, J. E. (2008). Monitoring the Future national results on adolescent drug use: Overview of key findings, 2007 (NIH Publication No. 08-6418). Bethesda, MD: National Institute on Drug Abuse.
- Johnston, L.D., O'Malley, P.M., Bachman, J.G., & Schulenberg, J.E. (2009).
 Monitoring the Future national survey results on drug use, 1975-2008:
 Volume II, College students and adults ages 19-50 (NIH Publication No. 09-7403). Bethesda, MD: National Institute on Drug Abuse.
- Kessler, R. C. (2000). Posttraumatic stress disorder: The burden to the individual and to society. *Journal of Clinical Psychiatry. Special Issue: Focus on Posttraumatic Stress Disorder, 61*(5), 4-14.
- Kessler, R.C., Galea, S., Jones, R.T. Parker, H.A. (2006). Mental illness and suicidality after Hurricane Katrina. *Bulletin of the World Health Organization* 84(12), 930-939.
- Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C.B. (1995). Posttraumatic stress disorder in the national comorbidity survey. *Archives of General Psychiatry*, 52(12), 1048-1060.
- Khantzian, E. J. (1985). The self-medication hypothesis of addictive disorders: Focus on heroin and cocaine dependence. *The American Journal of Psychiatry, 142*(11), 1259-1264.
- Kilpatrick, D.G., Acierno, R., Resnick, H.S., Saunders, B.E., & Best, C.L. (1997). A 2-year longitudinal analysis of the relationship between violent assault and substance use in women. *Journal of Consulting and Clinical Psychology*, 65, 834-847.
- Kilpatrick, D. G., Acierno, R., Saunders, B., Resnick, H. S., Best, C. L., & Schnurr, P. P. (2000). Risk factors for adolescent substance abuse and dependence: Data from a national sample. *Journal of Consulting and Clinical Psychology*, 68(1), 19-30.

- Kimerling, R., Ouimette, P., & Wolfe, J. (2002). *Gender and PTSD*. New York, NY, US: Guilford Press.
- Kimerling, R., Trafton, J. A., & Nguyen, B. (2006). Validation of a brief screen for posttraumatic stress disorder with substance use disorder patients. *Addictive Behaviors*, 31, 2074–2079.
- Koenen, K. C., Harley, R., Lyons, M. J., Wolfe, J., Simpson, J. C., Goldberg, J., et al. (2002). A twin registry study of familial and individual risk factors for trauma exposure and posttraumatic stress disorder. *Journal of Nervous and Mental Disease*, 190(4), 209-218.
- Koenen, K. C., Moffitt, T. E., Poulton, R., Martin, J., & Caspi, A. (2007). Early childhood factors associated with the development of post-traumatic stress disorder: Results from a longitudinal birth cohort. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences*, 37(2), 181-192.
- Krieger, N., Williams, D. R., & Moss, N. E. (1997). Measuring social class in US public health research: Concepts, methodologies and guidelines. *Annual Review of Public Health*, 18, 341–378.
- Kulka, R. A., Schlenger, W. E., Fairbank, J. A., Hough, R. L., Jordan, B. K., Marmar, C. R., et al. (1990). *Trauma and the Vietnam war generation: Report of findings from the National Vietnam Veterans Readjustment Study*. Philadelphia, PA, US: Brunner/Mazel.
- La Greca, A. M., Silverman, W. K., & Wasserstein, S. B. (1998). Children's predisaster functioning as a predictor of posttraumatic stress following Hurricane Andrew. *Journal of Consulting and Clinical Psychology*, 66(6), 883-892.
- Leeies, M., Pagura, J., Sareen, J., & Bolton, J. M. (2010). The use of alcohol and drugs to self-medicate symptoms of posttraumatic stress disorder. *Depression and Anxiety*, *27*(8), 731-736.
- Lipschitz, D. S., Grilo, C. M., Fehon, D., McGlashan, T. M., & Southwick, S. M. (2000). Gender differences in the associations between posttraumatic stress symptoms and problematic substance use in psychiatric inpatient adolescents. *Journal of Nervous and Mental Disease*, *188*(6), 349-356.
- Livingston, R., Lawson, L., & Jones, J. G. (1993). Predictors of self-reported psychopathology in children abused repeatedly by a parent. *Journal of the American Academy of Child & Adolescent Psychiatry*, *32*(5), 948-953.

- Long, J.S., & Freese, J. (2006). *Regression analysis for categorical dependent variables using Stata.* (2nd ed.). College Station, TX: Stata Press.
- Macklin, M. L., Metzger, L. J., Litz, B. T., McNally, R. J., Lasko, N. B., Orr, S. P., et al. (1998). Lower precombat intelligence is a risk factor for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 66(2), 323-326.
- MacKinnon, D. P., Fritz, M. S., Williams, J., & Lockwood, C. M. (2007). Distribution of the product confidence limits for the indirect effect: Program PRODCLIN. *Behavior Research Methods*, 39, 384-389.
- MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate Behavioral Research*, 39(1), 99-128.
- Marshall, G.N., & Orlando, M. (2002). Acculturation and peritraumatic dissociation in young adult survivors of community violence. *Journal of Abnormal Psychology*, 111(1), 166-174.
- Merikangas, K., Stolar, M., Stevens, D., Goulet, J., Preisig, M., Fenton, B., et al. (1998). Familial transmission of substance use disorders. *Archives of General Psychiatry*, 55, 973-979.
- Miller, M. W. (2003). Personality and the etiology and expression of PTSD: A three-factor model perspective. *Clinical Psychology: Science and Practice*, *10*(4), 373-393. doi:10.1093/clipsy/bpg040
- Mills, K. L., Teesson, M., Ross, J., Darke, S., & Shanahan, M. (2005). The costs and outcomes of treatment for opioid dependence associated with posttraumatic stress disorder. *Psychiatric Services*, 56, 940–945. doi: 10.1176/appi.ps.56.8.940.
- Mokdad, A.H., Marks, J.S., Stroup, D.F., & Gerberding, J.L. (2004). Actual causes of death in the United States, 2000. *Journal of the American Medical Association*, 291(10), 1238-1245.
- Muthén, B.O. & Muthén, L.K. (1998-2011). Version 6.11, Computer Software and Manual. Los Angeles, CA: Muthén and Muthén.
- Neter, J. Wasserman, & Kutner, M.H. (1989). *Applied linear regression analysis*. Homewood, IL: Irwin.
- Norman, S. B., Tate, S. R., Anderson, K. G., & Brown, S. A. (2007). Do trauma history and PTSD symptoms influence addiction relapse context? *Drug and Alcohol Dependence*, *90*, 89–96. doi:10.1016/j.drugalcdep.2007.03.002.

- Office of the National Drug Control Policy. (2001). *The Economic Costs of Drug Abuse in the United States, 1992-1998.* Washington, DC: Executive Office of the President (Publication No. NCJ-190636).
- Olff, M., Langeland, W., Draijer, N., & Gersons, B. P. R. (2007). Gender differences in posttraumatic stress disorder. *Psychological Bulletin*, 133(2), 183-204.
- Ortega, A. N., & Rosenheck, R. (2000). Posttraumatic stress disorder among Hispanic Vietnam veterans. *The American Journal of Psychiatry*, 157(4), 615-619. doi:10.1176/appi.ajp.157.4.615
- Ortega, A. N., Rosenheck, R., Alegría, M., & Desai, R. A. (2000). Acculturation and the lifetime risk of psychiatric and substance use disorders among Hispanics. *Journal of Nervous and Mental Disease, 188*(11), 728-735. doi:10.1097/00005053-200011000-00002
- Ouimette, P. C., Finney, J. W., & Moos, R. H. (1999). Two-year posttreatment functioning and coping of substance abuse patients with posttraumatic stress disorder. *Psychology of Addictive Behaviors*, 13, 105–114. doi:10.1037/0893-164X.13.2.105.
- Ouimette, P., Read, J. P., Wade, M., & Tirone, V. (2010). Modeling associations between posttraumatic stress symptoms and substance use. *Addictive Behaviors*, *35*(1), 64-67.
- Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin*, 129(1), 52-73.
- Parslow, R.A., Jorm, A.F., & Christensen, H. (2005). Associations of pre-trauma attributes and trauma exposure with screening positive for PTSD: analysis of a community based study of 2085 young adults. *Psychological Medicine*, 36, 387-395.
- Perilla, J. L., Norris, F. H., & Lavizzo, E. A. (2002). Ethnicity, culture, and disaster response: Identifying and explaining ethnic differences in PTSD six months after Hurricane Andrew. *Journal of Social and Clinical Psychology*, 21(1), 20-45.
- Perkonigg, A., Kessler, R. C., Storz, S., & Wittchen, H. (2000). Traumatic events and post-traumatic stress disorder in the community: Prevalence, risk factors and comorbidity. *Acta Psychiatrica Scandinavica*, 101(1), 46-59.
- Pitman, R.K., Orr, S.P., Lowenhagen, M.J., Macklin, M.L., & Altman, B. (1991). Pre-Vietnam contents of posttraumatic stress disorder veterans' service medical and personnel records. *Comprehensive Psychiatry*, 32, 416-422.
- Pole, N., Best, S. R., Metzler, T., & Marmar, C. R. (2005). Why are Hispanics at greater risk for PTSD? *Cultural Diversity and Ethnic Minority Psychology*, 11(2), 144-161.
- Pole, N., Gone, J. P., & Kulkarni, M. (2008). Posttraumatic stress disorder among ethnoracial minorities in the United States. *Clinical Psychology: Science and Practice*, 15(1), 35-61.
- Pregibon, D. (1981). Logistic Regression Diagnostics. Annals of Statistics, 9, 705–724.
- Read, J.P., Brown, P.J., & Kahler, C. W. (2004). Substance use and posttraumatic stress disorders: Symptom interplay and effects on outcome. *Addictive Behaviors, 29*, 1665-1672.
- Reed, P. L., Anthony, J. C., & Breslau, N. (2007). Incidence of drug problems in young adults exposed to trauma and posttraumatic stress disorder: Do early life experiences and predispositions matter? *Archives of General Psychiatry*, 64(12), 1435-1442.
- Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin, 128*, 330-366.
- Resick, P. A., Monson, C. M., & Rizvi, S. L. (2008). *Posttraumatic stress disorder*. New York, NY, US: Guilford Press.
- Resnick, H. S., Kilpatrick, D. G., Dansky, B. S., Saunders, B. E., & Best, C. L. (1993). Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. *Journal of Consulting and Clinical Psychology*, *61*(6), 984-991.
- Reynolds, M., Mezey, G., Chapman, M., Wheeler, M., Drummond, C., & Baldacchino, A. (2005). Co-morbid post-traumatic stress disorder in a substance misusing clinical population. *Drug and Alcohol Dependence*, 77, 251–258.
- Robins, L.M., Helzer, J.E. Cottler, L., & Golding, E. (1989). National Institute of Mental Health Diagnostic Interview Schedule, version III, revised. St. Louis, Washington University.
- Robins, L. N., Helzer, J. E., Croughan, J. L., & Ratcliff, K. S. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history,

characteristics, and validity. *Archives of General Psychiatry*, 38(4), 381-389.

- Robinson, J., Sareen, J., Cox, B. J., & Bolton, J. (2009). Self-medication of anxiety disorders with alcohol and drugs: Results from a nationally representative sample. *Journal of Anxiety Disorders*, 23(1), 38-45. doi:10.1016/j.janxdis.2008.03.013
- Roosa, M. W., Sandler, I. N., Gehring, M., & Beals, J. (1988). The children of alcoholics life-events schedule: A stress scale for children of alcoholabusing parents. *Journal of Studies on Alcohol*, 49(5), 422-429.
- Roth, S., Newman, E., Pelcovitz, D., van der Kolk, B., & Mandel, F. S. (1997). Complex PTSD in victims exposed to sexual and physical abuse: Results from the DSM-IV field trial for posttraumatic stress disorder. *Journal of Traumatic Stress*, 10(4), 539-555.
- Sandler, I., Ramirez, R., & Reynolds, K. (1986, August). Life stress for children of divorce, bereaved, and asthmatic children. Paper presented at the annual meeting of the American Psychological Association, Washington DC.
- Satorra, A. (2000). Scaled and adjusted restricted tests in multi-sample analysis of moment structures. In Heijmans, R.D.H., Pollock, D.S.G. & Satorra, A. (Eds.), *Innovations in multivariate statistical analysis. A Festschrift for Heinz Neudecker* (pp.233-247). London: Kluwer Academic Publishers.
- Schafer, J. & Graham, J. (2002). Missing data: Our view of the state of the art. *Psychological Methods*, *7*, 147–177.
- Schnurr, P.P., Lunney, C.A., & Senqupta, A. (2004). Risk factors for the development versus maintenance of posttraumatic stress disorder. *Journal* of Traumatic Stress, 17, 85-95.
- Selzer, M. L. (1971). The Michigan Alcoholism Screening Test: The quest for a new diagnostic instrument. *American Journal of Psychiatry*, 127(12), 1653-1658.
- Shaffer, J.P. (1995). Multiple hypothesis testing. *Annual Review of Psychology*, 46, 561-584.
- Shedler, J., & Block, J. (1990). Adolescent drug use and psychological health. *American Psychologist, 45,* 612-630.
- Sher, K. J. (1991). *Children of alcoholics: A critical appraisal of theory and research*. Chicago, IL, US: University of Chicago Press.

- Shipherd, J. C., Stafford, J., & Tanner, L. R. (2005). Predicting alcohol and drug abuse in Persian Gulf War veterans: What role do PTSD symptoms play? *Addictive Behaviors*, 30(3), 595-599.
- Silverman, W. K., & La Greca, A. M. (2002). Children experiencing disasters: Definitions, reactions, and predictors of outcomes. In A. M. La Greca, W. K. Silverman, E. M. Vernberg & M. C. Roberts (Eds.), *Helping children cope with disasters and terrorism*. (pp. 11-33). Washington, DC, US: American Psychological Association.
- Stein, M. B., Jang, K. L., Taylor, S., Vernon, P. A., & Livesley, W. J. (2002). Genetic and environmental influences on trauma exposure and posttraumatic stress disorder symptoms: A twin study. *The American Journal of Psychiatry*, 159(10), 1675-1681.
- Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review, 28,* 78-106.
- Stewart, S. H. (1996). Alcohol abuse in individuals exposed to trauma: A critical review. *Psychological Bulletin*, *120*(1), 83-112.
- Stewart, S.H. (1997). Trauma memory and alcohol abuse: Drinking to forget? In J.D. Read & D.S. Lindsay (Eds.), *Recollections of Trauma: Scientific Evidence and Clinical Practice* (pp. 461-467). New York: Plenum.
- Stewart, S. H., & Conrod, P. J. (2003). Psychosocial models of functional associations between posttraumatic stress disorder and substance use disorder. In P. Ouimette, & P. J. Brown (Eds.), *Trauma and substance abuse: Causes, consequences, and treatment of comorbid disorders.* (pp. 29-55). Washington, DC, US: American Psychological Association.
- Stewart, S. H., Ouimette, P., & Brown, P. J. (2002). Gender and the comorbidity of PTSD with substance use disorders. In R. Kimerling, P. Ouimette & J. Wolfe (Eds.), *Gender and PTSD*. (pp. 232-270). New York, NY, US: Guilford Press.
- Stewart, S. H., Pihl, R. O., Conrod, P. J., & Dongier, M. (1998). Functional associations among trauma, PTSD and substance-related disorders. *Addictive Behaviors*, 23(6), 797-812.
- Storr, C. L., Ialongo, N. S., Anthony, J. C., & Breslau, N. (2007). Childhood antecedents of exposure to traumatic events and posttraumatic stress disorder. *The American Journal of Psychiatry*, 164(1), 119-125.
- Storr, C. L., Schaeffer, C. M., Petras, H., Ialongo, N. S., & Breslau, N. (2009). Early childhood behavior trajectories and the likelihood of experiencing a

traumatic event and PTSD by young adulthood. *Social Psychiatry and Psychiatric Epidemiology*, 44(5), 398-406.

- Substance Abuse and Mental Health Services Administration. (2007) Results from the 2006 National Survey on Drug Use and Health: National Findings (Office of Applied Studies, NSDUH Series H-32, DHHS Publication No. SMA 07-4293). Substance Abuse and Mental Health Services Administration, Rockville, MD.
- Swendsen, J., Conway, K. P., Degenhardt, L., Glantz, M., Jin, R., Merikangas, K. R., et al. (2010). Mental disorders as risk factors for substance use, abuse and dependence: Results from the 10-year follow-up of the National Comorbidity Survey. *Addiction*, 105, 1117–1128. doi:10.1111/j.1360-0443.2010.02902.x.
- Tolin, D. F., & Foa, E. B. (2002). Gender and PTSD: A cognitive model. In R. Kimerling, P. Ouimette & J. Wolfe (Eds.), *Gender and PTSD*. (pp. 76-97). New York, NY, US: Guilford Press.
- Tolin, D. F., & Foa, E. B. (2006). Sex differences in trauma and posttraumatic stress disorder: A quantitative review of 25 years of research. *Psychological Bulletin*, 132(6), 959-992.
- Torchalla, I., Nosen, L, Rostam, H., & Allen, P. (2012). Integrated treatment programs for individuals with concurrent substance use disorders and trauma experiences: A systematic review and meta-analysis. *Journal of Substance Abuse Treatment*, *42*. 65-77.
- Ullman, S. E., Filipas, H. H., Townsend, S. M., & Starzynski, L. L. (2005). Trauma exposure, posttraumatic stress disorder and problem drinking in sexual assault survivors. *Journal of Studies on Alcohol*, 66(5), 610-619.
- van Dam, D., Vedel, E., Ehring, T., & Emmelkamp, P.M.G. (2012) Psychological treatments for concurrent posttraumatic stress disorder and substance use disorder: A systematic review. *Clinical Psychology Review*, 32, 202-214.
- van der Kolk, B. (1994). The body keeps the score: Memory and the evolving psychobiology of PTSD. *Harvard Review of Psychiatry*, 1, 253-265.
- van der Kolk, B. A., Pelcovitz, D., Roth, S., & Mandel, F. S. (1996). Dissociation, somatization, and affect dysregulation: The complexity of adaption to trauma. *The American Journal of Psychiatry*, *153*(Suppl), 83-93.
- Vega, W, Canino, G, & Alegria, M. (2009). Prevalence and Correlates of Dual Diagnoses in U.S. Latinos. *Drug Abuse and Dependence*, 100(1-2), 32-38.

- Vega, W.A., Zimmerman, R., Gil, A., Warheit, G.J., & Aprospori, E. (1993). Acculturation strain theory: Its application in explaining drug use behavior among Cuban and other Hispanic youth. In. M. De La Rosa & J. Recio Adrados (Eds.) *Drug Abuse Among Minority Youth: Advances in Research and Methodology* (NIH Publication No. 93-3479). National Institute on Drug Abuse Research Monograph Series. National Institute on Drug Abuse: Bethesda, MD.
- Vlahov, D., Galea, S., Resnick, H., Ahern, J., Boscarino, J. A., Bucuvalas, M., et al. (2002). Increased use of cigarettes, alcohol, and marijuana among Manhattan, New York residents after the September 11th terrorist attacks. *American Journal of Epidemiology*, 155, 988–996.
- Williams, D. R., & Collins, C. (1995). US socioeconomic and racial differences in health: Patterns and explanations. *Annual Review of Psychology*, 21, 349– 386.
- Windle, M. (1994). Substance use, risky behaviors, and victimization among a US national adolescent sample. *Addiction*, *89*(2), 175-182.
- Xian, H., Chantarujikapong, S. I., Scherrer, J. F., Eisen, S. A., Lyons, M. J., Goldberg, J., et al. (2000). Genetic and environmental influences on posttraumatic stress disorder, alcohol and drug dependence in twin pairs. *Drug and Alcohol Dependence*, 61(1), 95-102.
- Yehuda, R. (2002). Post-traumatic stress disorder. New England Journal of Medicine, 346, 108-115.
- Yehuda, R., & McFarlane, A. C. (1995). Conflict between current knowledge about posttraumatic stress disorder and its original conceptual basis. *The American Journal of Psychiatry*, 152(12), 1705-1713.
- Yehuda, R., McFarlane, A. C., & Shalev, A. Y. (1998). Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event. *Biological Psychiatry*, 44(12), 1305-1313.
- Zhou, Q., King, K. M., & Chassin, L. (2006). The roles of familial alcoholism and adolescent family harmony in young adults' substance dependence disorders: Mediated and moderated relations. *Journal of Abnormal Psychology*, 115, 320-331.
- Zucker, R.A. (2006). Alcohol use and the alcohol use disorders: A developmental biopsychosocial systems formulation covering the life course. In D. Cicchetti & D. Cohen (Eds), *Developmental Psychopathology, Volume 3: Risk, Disorder, and Adaptation (2nd ed.)* (pp. 620-656). Willey: Hoboken, NJ.

Table 1Descriptive Statistics of Study Variables

Categorical variables	Frequency						
Adolescent gender	202 (53.6%)	males					
	175 (46.4%)	175 (46.4%) females					
Adolescent ethnicity	102 (27.1%)	102 (27.1%) Hispanic/other					
	275 (72.9%)	non-Hisp	oanic Cauca	sian			
Parental alcoholism	183 (48.5)%	children	of non-alco	holics			
	194 (51.5%)	children	of alcoholic	s			
Other parental psychopathology	228 (60.5) no	o other ps	sychopathol	ogy			
	149 (39.5%)	other psy	chopatholo	gy			
Adolescent substance use			-				
problems	317 (84.1%)	no proble	ems				
	23 (6.1%) or	ne probler	n				
Late adalassent/serly adult	37 (9.8%) tw	o or mor	e problems				
trauma exposure	211 (56.0%)	no traum	a exposure				
	166 (44 0%)	trauma e	xposure				
Late adolescent/early adult	100 (11.070)		nposare				
assaultive violence exposure	305 (80.1%)	no expos	sure to assau	ltive violence			
	72 (19.1%) e	exposure t	to assaultive	e violence			
Late adolescent/early adult PTSD diagnosis	346 (91.8%)	no PTSF)				
1 15D diugnosis	31 (8 2%) P						
Late adolescent/early adult	51 (0.270)1	150					
trauma/PTSD status	211 (56%) n	o trauma	exposure				
	135 (35.8%)	exposure	without PT	SD			
	31 (8.2%) PT	ГSD					
Continuous and Count Variables	Mean (SD)	Min.	Max	Skewness	Kurtosis		
Adolescent family conflict	2.74 (.60)	1.33	4.38	.22	25		
Adolescent familial life stress	3.20 (2.36)	0.00	11.00	.90	.58		
Adolescent family adversity			• • •	• -			
factor score	.25 (.62)	-1.91	2.36	.26	25		
PTSD symptoms ^a	5.41 (4.11)	.00	16.00	.69	469		
Adult alcohol problems ^b	1.54 (2.58)	.00	13.00	2.16	4.60		
Adult drug problems ^b	.83 (2.33)	.00	13.00	3.37	11.40		

Note. n=377.

^a *n*=166 (trauma exposed only).

^b n=348 due to missing data at Wave 5.

Table 2 Zero-Order Correlations

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	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	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. $\dagger p < .10$, $\ast p < .05$, $\ast p < .01$, $\ast \ast p < .001$. n = 377. All count variables were log-transformed prior to estimating zero-order correlations. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos and other ethnicities. an = 166 (trauma exposed only). bn = 348 due to missing data at Wave 5.

Traumatic Event	Females	Males	Total
Rape	23.8%	0%	9.0%
Physical Assault	11.1%	8.7%	9.6%
Saw someone hurt or killed	14.3%	19.4%	17.5%
Natural disaster	0%	1.9%	1.2%
Threatened with a weapon	3.2%	27.2%	18.1%
Narrow escape from death/injury	1.6%	10.7%	7.2%
Sudden injury or accident	17.5%	15.5%	16.3%
Sudden death/injury of someone close	6.3%	5.8%	6.0%
Other personal shock	0%	1.0%	.6%
Shock from other's experience	22.2%	9.7%	14.5%
Experienced assaultive violence	42.1%	43.7%	43.4%
Meet lifetime criteria for PTSD	31.7%	10.7%	18.7%

Table 3	
Types of Events among Participants Exposed to	Trauma

Note. n=166 (211 participants were not exposed to a traumatic event). Percentages provided for types or events are based on the event participants considered to be the worst. Percentages for assaultive violence and PTSD are based on all events (up to 3) that adolescent reported on. Note that there were no participants who reported military combat.

Comparison of Fit Statistics for Poisson, Negative Binomial, ZIP, and ZINB Models Predicting Alcohol and Drug Problems: Approach 1

Alcohol Models	Parameters	Loglikelihood	AIC	BIC
Poisson	19	-1362.61	2763.23	2837.94
ZIP	20	-1244.46	2528.92	2607.56
Negative binomial	20	-1223.14	2486.29	2564.93
ZINB	21	-1218.68	2479.36	2561.94
Drug Models	Parameters	Loglikelihood	AIC	BIC
Poisson	19	-1176.81	2391.63	2467.34
ZIP	20	-1013.70	2067.394	2146.04
Negative binomial	20	-994.17	2028.34	2106.99
ZINB	21	-991.99	2025.98	2108.56

Note. ZIP= Zero-Inflated Poisson. ZINB= Zero-Inflated Negative Binomial. AIC= Akaike Information Criterion. BIC= Bayesian Information Criterion.

Comparison of Fit Statistics for Poisson,	Negative	Binomial,	ZIP,	and	ZINB	Models	Predicting
Alcohol and Drug Problems: Approach 2	?						

Alconol and Drug Prob	lems: Approach 2			
Alcohol Models	Parameters	Loglikelihood	AIC	BIC
Poisson	7	-761.83	1537.65	1564.62
ZIP	8	-587.15	1190.30	1221.12
Negative binomial	8	-549.33	1114.67	1145.48
ZINB	9	-546.52	1111.04	1145.71
Drug Models	Parameters	Loglikelihood	AIC	BIC
Poisson	7	-630.41	1274.82	1301.78
ZIP	8	-346.41	708.82	739.63
Negative binomial	8	-319.71	655.42	686.24
ZINB	9	-319.27	656.53	691.20

Note. ZIP= Zero-Inflated Poisson. ZINB= Zero-Inflated Negative Binomial. AIC= Akaike Information Criterion. BIC= Bayesian Information Criterion.

Block 1—Results prior to including gender x ethnicity interaction (main effects only)							
Variable	В	SE	Odds Ratio				
Intercept	15	.17	.87				
Adolescent substance use problems	.20	.18	1.22				
Family adversity	.44**	.14	1.55				
Female gender	61**	.22	.54				
Ethnicity	.46†	.24	1.58				
Block 2-Results after including gender by ethnicity in	iteraction						
Intercept	29	.18	.75				
Adolescent substance use problems	.17	.18	1.18				
Family adversity	.46**	.14	1.58				
Female gender	26	.25	.77				
Ethnicity	1.09**	.35	2.99				
Gender x ethnicity ^a	-1.32**	.51	.27				

High-Risk Hypothesis: Results of Logistic Regression Predicting Risk for Trauma Exposure

Note. $\dagger p = .061$, *p < .05, **p < .01, ***p < .001. n = 377. B = Unstandardized regression coefficient. SE = Standard error. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos and other ethnicities. Note that odds ratios for categorical versus continuous predictors are not directly comparable.

^aPreliminary analyses indicated a significant interaction between gender and ethnicity. Including this interaction changes the interpretation of the gender and ethnicity coefficients.

Ехроѕите			
Variable	В	SE	Odds Ratio
Intercept	-1.50***	.21	.22
Adolescent substance use problems	.38†	.19	1.46
Family adversity	.48**	.17	1.61
Female gender	40	.28	.67
Ethnicity	.16	.30	1.17

 Table 7

 High-Risk Hypothesis: Results of Logistic Regression Predicting Risk for Assaultive Violence

 Exposure

Note. $\dagger p = .051$, $\ast p < .05$, $\ast p < .01$, $\ast \ast p < .001$. n = 377. B = Unstandardized regression coefficient. SE = Standard error. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos and other ethnicities. Note that odds ratios for categorical versus continuous predictors are not directly comparable.

Diagnosis			
Variable	В	SE	Odds Ratio
Intercept	-2.90***	.47	.06
Adolescent substance use problems	.39	.30	1.48
Family adversity	.85**	.32	2.35
Female gender	1.61***	.46	4.98
Ethnicity	.52	.46	1.69

Table 8Susceptibility-Risk Hypothesis: Results of Logistic Regression Predicting Risk for PTSDDiagnosis

Note. *p < .05, **p < .01, ***p < .001. n = 166. B = Unstandardized regression coefficient. SE = Standard error. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos and other ethnicities. Note that odds ratios for categorical versus continuous predictors are not directly comparable.

Variable	В	SE	Incidence Rate
			Ratio
Intercept	1.28***	.10	3.60
Adolescent substance use problems	.07	.08	1.07
Family adversity	.26**	.08	1.30
Female gender	.55***	.11	1.73
Ethnicity	.20†	.12	1.22

 Table 9. Susceptibility-Risk Hypothesis: Results of Negative Binomial Regression Predicting Risk

 for PTSD Symptoms

Note. $\dagger p = .091$, $\ast p < .05$, $\ast \ast p < .01$, $\ast \ast \ast p < .001$. n = 166. B = Unstandardized regression coefficient. SE = Standard error. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos and other ethnicities. Note that incidence rate ratios for categorical versus continuous predictors are not directly comparable.

Predictor	Tra	uma Expos	ure	РТ	SD Sympto	ms	Adult	Alcohol Pr	oblems
						Incidence			Incidence
			Odds			Rate			Rate
	В	SE	Ratio	В	SE	Ratio	В	SE	Ratio
Family adversity	.54**	.17	1.72	.26**	.08	1.30	.01	.12	1.01
Female gender	32 ^a	.30	.73	.54***	.11	1.72	-1.03***	.22	.36
Ethnicity	1.32 ^b **	.42	3.73	.20†	.12	1.22	.08	.21	1.08
Adolescent substance use problems	.21	.21	1.23	.07	.08	1.07	.29†	.16	1.34
Gender x ethnicity	-1.60**	.60	.21						
Time since trauma exposure							23**	.07	.80
Trauma exposure							.17	.20	1.19
PTSD Symptoms ^c							.09**	.03	1.10

Table 10Results of Adult Alcohol Problems Model: Approach 1

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Note. $\dagger p < .10$, *p < .05, **p < .01, ***p < .001. n = 377. B = Unstandardized regression coefficient. SE = Standard error. Logistic regression was used for

trauma exposure, negative binomial regression was used for PTSD symptoms, and negative binomial regression was used for drug problems. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos 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= .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= -.26, SE= .43, p =. 55, OR= .77).

^c Note that additional analyses indicated that ethnicity significantly interacted with PTSD symptoms (B= -.10, p= .02, *IRR*: .90), such that the influence of PTSD symptoms on alcohol problems was significant for non-Hispanic/Latino Caucasians (B= .11, p= .001, *IRR*: 1.12) but not for minority ethnicities (B= .02, p= .52, *IRR*: 1.02).

Predictor	Tra	uma Expos	ure	РТ	SD Sympto	ms	Adult	Drug Pro	blems
						Incidence			Incidence
			Odds			Rate			Rate
	В	SE	Ratio	В	SE	Ratio	В	SE	Ratio
Family adversity	.54**	.17	1.72	.26**	.08	1.30	01 ^c	.24	.99
Female gender	32 ^a	.30	.73	.54***	.11	1.71	-1.36***	.39	.26
Ethnicity	1.32 ^b **	.42	3.73	.20†	.12	1.22	10	.33	.91
Adolescent substance use problems	.21	.21	1.23	.07	.08	1.07	.47†	.27	1.60
Gender x ethnicity	-1.60**	.60	.20						
Gender x family adversity							1.13**	.35	3.10
Trauma exposure							.20	.05	1.22
PTSD Symptoms ^d							.09*	.05	1.10

Table 11Results of Adult Drug Problems Model: Approach 1

Note. $\dagger p < .10$, $\ast p < .05$, $\ast p < .01$, $\ast \ast p < .001$, n = 377. B = Unstandardized regression coefficient. SE = Standard error. Logistic regression was used for

trauma exposure, negative binomial regression was used for PTSD symptoms, and negative binomial regression was used for drug problems. Adolescent

 $\stackrel{\bullet}{\sim}$ trauma exposure, negative binomial regression was used for PTSD symptoms, and negat

^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= .15).

⁶ 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= -.26, SE= .43, p =. 55, OR= .77).

^c Due to the interaction between gender and family adversity interaction, 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).

^d Note that additional analyses indicated that there was a marginally significant interaction between ethnicity and PTSD symptoms (B= -.15, p= .06, IRR: .86), such that the effect of PTSD symptoms on drug problems was significant for non-Hispanic/Latino Caucasians (B= .11, p= .04, IRR: 1.11) but not for minority ethnicities (B= .02, p= .67, IRR: 1.02).

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Results of Adult Alcohol Problems Model with Ethnicity by PTSD Symptoms Interaction, Excluding 18 "Other Ethnicity" Participants (Approach 1; Hypothesis 3b)

Predictor	Tra	uma Expos	ure	РТ	SD Sympto	oms	Adult	Alcohol Pr	oblems
			Odds			Incidence Rate			Incidence Rate
	В	SE	Ratio	В	SE	Ratio	В	SE	Ratio
Family adversity	.48**	.17	1.62	.33***	.08	1.40	01	.13	.99
Female gender	32 ^a	.30	.73	.50***	.11	1.64	-1.08***	.23	.34
Ethnicity	1.40 ^b **	.45	4.06	.31**	.12	1.36	.35	.27	1.42
Adolescent substance use problems	.19	.22	1.20	.09	.08	1.10	.32†	.17	1.38
Gender x ethnicity	-1.69**	.65	.19						
Time since trauma exposure							26**	.07	.77
Trauma exposure							.18	.21	1.19
PTSD Symptoms							.10 ° **	.03	1.11
PTSD Symptoms x ethnicity							10*	.05	.91

Note. $\dagger p < .10$, $\ast p < .05$, $\ast \ast p < .01$, $\ast \ast \ast p < .001$. n = 359. B = Unstandardized regression coefficient. SE = Standard error. Logistic regression was used for trauma exposure, negative binomial regression was used for PTSD symptoms, and negative binomial regression was used for drug problems. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos.

^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 Hispanic/Latino participants was significant (B= -2.01, SE= .58, p < .001, OR= .13).

^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= -.28, SE= .47, p =. 54, OR= .75).

^c Due to the interaction between PTSD symptoms and ethnicity, the coefficient presented for PTSD symptoms indicates the effect of gender for Caucasians only. The effect of PTSD symptoms on alcohol problems for Hispanic/Latino participants was non-significant (B= .03, SE = .04, p= .45, *IRR*: 1.03).

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Results of Adult Drug Problems Model with Ethnicity by PTSD Symptoms Interaction, Excluding 18 "Other Ethnicity" Participants (Approach 1; Hypothesis 3b)

Predictor	Tra	uma Expos	ure	РТ	SD Sympto	oms	Adult	t Drug Pro	blems
	D	C.F.	Odds	D	<u>ar</u>	Incidence Rate	D	<u>CE</u>	Incidence Rate
	В	SE	Ratio	В	SE	Ratio	В	SE	Ratio
Family adversity	.48**	.17	1.62	.33***	.08	1.40	07 ^c	.30	.94
Female gender	32 ^a	.30	.73	.50***	.11	1.64	-1.40***	.39	.25
Ethnicity	1.40 ^b **	.45	4.05	.31**	.12	1.36	.36	.43	1.43
Adolescent substance use problems	.19	.22	1.20	.09	.08	1.10	.47	.31	1.60
Gender x ethnicity	-1.68**	.65	.19						
Gender x family adversity							1.20**	.38	3.33
Trauma exposure							.23	.37	1.26
PTSD Symptoms							.10* ^d	.05	1.11
PTSD Symptoms x ethnicity							14†	.07	.87

Note. $\dagger p < .10$, *p < .05, *p < .01, **p < .001. n = 359. B =Unstandardized regression coefficient. SE =Standard error. Logistic regression was used for trauma exposure, negative binomial regression was used for PTSD symptoms, and negative binomial regression was used for drug problems. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos.

^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 Hispanic/Latino participants was significant (B= -2.00, SE= .58, p < .001, OR= .14).

^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= -.28, SE= .47, p =. 55, OR= .76).

^c Due 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=1.14, SE=.26, p < .001, IRR=3.12).

^d Due to the interaction between PTSD symptoms and ethnicity, the coefficient presented for PTSD symptoms indicates the effect of gender for Caucasians only. The effect of PTSD symptoms on drug problems for Hispanic/Latino participants was non-significant (B= .02, SE = .04, p= .62, *IRR*: 1.02).

Results of Multi Mednoi 1 robients Mouel. Approach 2				
Variable	В	SE	Incidence Rate	
			Ratio	
Family adversity	.14	.11	1.15	
Female gender	79***	.19	.45	
Ethnicity	.16	.20	1.17	
Adolescent substance use problems	.31*	.16	1.37	
Time since trauma exposure	24**	.07	.79	
Exposed to trauma and no PTSD	.12	.19	1.13	
PTSD Diagnosis	.62†	.37	1.86	

Table 14Results of Adult Alcohol Problems Model: Approach 2

Note. $\dagger p = .091$, $\ast p < .05$, $\ast \ast p < .01$, $\ast \ast \ast p < .001$. n = 377. B = Unstandardized regression coefficient. SE = Standard error. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos and other ethnicities. Note that incidence rate ratios for categorical versus continuous predictors are not directly comparable.

Variable	В	SE	Incidence Rate
			Ratio
Family adversity	.10 ^a	.24	1.11
Female gender	-1.11	.33	.33
Ethnicity	04	.33	.96
Gender x family adversity	1.17**	.35	3.21
Adolescent substance use problems	.49	.26	1.63
Exposed to trauma and no PTSD	.21	.34	1.24
PTSD Diagnosis	.49	.59	1.64

Table 15Results of Adult Drug Problems Model: Approach 2

Note. *p < .05, **p < .01, ***p < .001. n = 377. B = Unstandardized regression coefficient. SE = Standard error. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos and other ethnicities. Note that incidence rate ratios for categorical versus continuous predictors are not directly comparable.

^aDue 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=1.27, SE=.26, p < .001, IRR=3.58).

Table 16

Variable	B	SE	Incidence Rate
			Ratio
Family adversity	.13	.12	1.14
Female gender	83***	.19	.44
Ethnicity	.35	.24	1.42
Adolescent substance use problems	.37*	.17	1.44
Time since trauma exposure	25**	.08	.78
Exposed to trauma and no PTSD	.09	.20	1.10
PTSD Diagnosis	.97 ^a *	.43	2.65
PTSD x ethnicity	-1.26*	.63	.28

Results of Adult Alcohol Problems Model with Ethnicity by PTSD Interaction, Excluding 18 "Other Ethnicity" Participants (Approach 2; Hypothesis 3b)

Note., *p < .05, **p < .01, ***p < .001. n = 359. B = Unstandardized regression coefficient. SE = Standard error. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos. Note that incidence rate ratios for categorical versus continuous predictors are not directly comparable.

^a Due to the interaction between PTSD and ethnicity, the coefficient presented for PTSD indicates the effect of gender for Caucasians only. The effect of PTSD symptoms on alcohol problems for Hispanic/Latino participants was non-significant (B = -.30, SE = .49, p = .55, IRR: .74).

Table 17

Variable	B	SE	Incidence Rate
			Ratio
Family adversity	.10 ^a	.26	1.10
Female gender	-1.16***	.32	.31
Ethnicity	.34	.36	1.40
Gender x family adversity	1.25***	.35	3.50
Adolescent substance use problems	.63	.27	1.87
Exposed to trauma and no PTSD	.26	.34	1.30
PTSD Diagnosis	1.09 ^b †	.61	2.98
PTSD x ethnicity	-3.27***	.87	.04

Results of Adult Drug Problems Model with Ethnicity by PTSD Interaction, Excluding 18 "Other Ethnicity" Participants (Approach 2; Hypothesis 3b)

Note. $\dagger p=.071$, *p<.05, **p<.01, ***p<.001. n=359. B= Unstandardized regression coefficient. SE= Standard error. Adolescent ethnicity is coded 0 for non-Hispanic/Latino Caucasians and 1 for Hispanics/Latinos. Note that incidence rate ratios for categorical versus continuous predictors are not directly comparable.

^a Due 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 = 1.27, SE = .26, p < .001, IRR = 3.58).

^b Due to the interaction between PTSD and ethnicity, the coefficient presented for PTSD indicates the effect of gender for Caucasians only. The effect of PTSD symptoms on drug problems for Hispanic/Latino participants was significant in the opposite direction (B= -2.18, SE = .66, p= .001, *IRR*: .11).

Table 18Summary of Results

Hypothesis	Finding		
1. High-risk hypothesis:	PARTIAL SUPPORT: Adolescent substance use		
Adolescent substance use	problems had a marginally significant unique effect on		
problems increase risk for trauma	risk for assaultive violence exposure ($B=.38$, $p=.051$,		
exposure or assaultive violence	OR: 1.46) but not on trauma exposure $(B=.17, p=.35, p=.35)$		
exposure, over and above family	<i>OR</i> : 1.18).		
adversity, gender and ethnicity.			
1a. Males are at greater risk for	MINIMAL SUPPORT: The unique effect of gender on		
trauma exposure and assaultive	trauma exposure was only significant for ethnic minority		
violence exposure compared to	participants, such that ethnic minority males were at		
females.	significantly greater risk for trauma exposure than were		
	ethnic minority females (B = -1.58, p < .001, OR : .21).		
	Gender did not have a significant unique effect on		
	assaultive violence exposure ($B =40$, $p = .15$, OR : .67).		
1b. Gender moderates the unique	NO SUPPORT: There was no evidence that gender		
influence of adolescent substance	moderated the influence of adolescent substance use $(P_{1}, Q_{2}) = 0.4$		
use problems on risk for trauma	problems on either trauma exposure ($B =03$, $p = .94$, OR :		
exposure of assaultive violence	($B = .45, p = .25, OR$) of assaultive violence exposure ($B = .45, p = .25, OR$)		
problems place females at greater	1.54).		
risk for exposure compared to			
males			
1c Adolescent substance use	NO SUPPORT: There was no evidence that adolescent		
problems mediate the influence of	substance use problems mediated the influence of gender		
gender on risk for trauma exposure	on risk for either trauma exposure or assaultive violence		
or assaultive violence exposure.	exposure.		
1d. Ethnic minority participants are	MINIMAL SUPPORT: The unique effect of ethnicity on		
at elevated risk for trauma exposure	trauma exposure was only significant for males such that		
or assaultive violence exposure	ethnic minority males were at significantly greater risk for		
compared to Caucasian participants.	trauma exposure than were Caucasian males ($B=1.09$, $p=$		
	.002, OR: 2.99). Ethnicity did not have a significant unique		
	effect on risk for assaultive violence exposure ($B=.16, p=$		
	.58, <i>OR</i> : 1.18).		
2. Susceptibility hypothesis:	NO SUPPORT: Adolescent substance use problems did		
Addrescent substance use	not significantly influence fisk for FTSD diagnosis ($D = 30$, $n = 10$, OP ; 1.48) or PTSD symptoms ($R = 07$, $n = 10$		
diagnosis/symptoms over and	41 IRR: 1 07) over and above the influences of family		
above the influences of family	adversity, gender, and ethnicity.		
adversity, gender and ethnicity.	adversity, gender, and connexty.		
2a. Females are at greater risk for	SUPPORTED: Gender had a significant unique effect on		
PTSD diagnosis/symptoms	risk for both PTSD diagnosis ($B = 1.61$, $p = .001$, OR: 4.98)		
compared to males.	and PTSD symptoms (B = .55, $p < .001$, IRR: 1.73), such		
-	that females were at greater risk compared to males.		
2b. Gender moderates the unique	NO SUPPORT: There was no evidence that gender		
influence of adolescent substance	moderated the influence of adolescent substance use		
use problems on risk for PTSD	problems on either PTSD diagnosis (B = .61, p = .32, OR :		
diagnosis/symptoms, such that	1.84) or PTSD symptoms (B = .16, p = .24, <i>IRR</i> : 1.17).		
substance use problems makes			
trauma-exposed females especially			
susceptible to developing PTSD			
relative to trauma-exposed males.			

2c. Hispanic/Latino participants are at elevated risk for PTSD diagnosis/symptoms compared to non-Hispanic/Latino Caucasian participants.	SUPPORTED: Hispanics/Latinos were at significantly greater risk for both PTSD (B = 1.01, p = .04, OR : 2.75) and PTSD symptoms (B = .30, p = .011, IRR : 1.35) than were non-Hispanic/Latino Caucasians.
3. <u>Self-medication hypothesis</u> : PTSD diagnosis/symptoms increase risk for adult alcohol and drug problems over and above the influence of trauma exposure, pre- trauma substance use problems, pre-trauma family adversity, gender, and ethnicity.	SUPPORTED <u>Method 1</u> : PTSD symptoms had a significant unique effect on risk for future adult alcohol problems (B = .09, p= .003, IRR: 1.10) and future adult drug problems (B = .09, p = .042, IRR: 1.10). <u>Method 2</u> : PTSD diagnosis had a marginally significant unique effect on risk for adult alcohol problems (B =.62, p= .09, IRR: 1.86) and a non-significant unique effect on risk for adult drug problems (B =.49, p = .41, IRR: 1.64).
3a. Gender moderates the unique influence of PTSD diagnosis/symptoms on future alcohol and drug problems such that females are more likely to self- medicate symptoms of PTSD compared to males.	NO SUPPORT <u>Method 1</u> : There was no evidence that gender moderated the influence of PTSD symptoms on either alcohol problems (B =04, p = .35, IRR: .96) or drug problems (B = 01, p = .82, IRR: .99). <u>Method 2</u> : There was no evidence that gender moderated the influence of PTSD diagnosis on either alcohol problems (B =25, p = .70, IRR: .78) or drug problems (B = 44, p = .70, IRR: .65).
3b. Ethnicity moderates the unique influence of PTSD diagnosis/symptoms on future alcohol and drug problems.	SUPPORTED Results presented below excluded the 18 participants of ethnicities other than Hispanic/Latino or non- Hispanic/Latino Caucasian. <u>Method 1:</u> There was a significant interaction between ethnicity and PTSD symptoms in the analysis predicting alcohol problems (B =10, p = .033, <i>IRR</i> : .91), such that the influence of PTSD symptoms on alcohol problems was significant for non-Hispanic/Latino Caucasians (B = .10, p= .002, <i>IRR</i> : 1.11) but not for Hispanics/Latinos (B = .03, p= .45, <i>IRR</i> : 1.03). There was a marginally significant interaction between ethnicity and PTSD symptoms in the analysis predicting drug problems (B =14, p = .055, <i>IRR</i> : .87), such that the influence of PTSD symptoms on drug problems was significant for non-Hispanic/Latino Caucasians (B = .10, p = .047, <i>IRR</i> : 1.11) but not for Hispanics/Latinos (B = .02, p = .617, <i>IRR</i> : 1.02). <u>Method 2:</u> There was a significant interaction between ethnicity and PTSD diagnosis in the analysis predicting alcohol problems (B =-1.26, p = .04, <i>IRR</i> : .28), such that PTSD significantly influenced risk for alcohol problems for non-Hispanic/Latino Caucasians (B =.97, p = .02, <i>IRR</i> : .74). There was a significant interaction between ethnicity and PTSD diagnosis in the analysis predicting diagnosis in the analysis predicting drug problems (B =-3.27, p < .001, <i>IRR</i> : .04), such that PTSD marginally <i>increased</i> risk for drug problems for non- Hispanic/Latino Caucasians (B =.109, p = .07, <i>IRR</i> : 2.98) and significantly <i>decreased</i> risk for drug problems for non- Hispanic/Latino Significant for drug problems for non- Hispanic/Latino Caucasians (B =-1.09, p = .07, <i>IRR</i> : 2.98) and significantly <i>decreased</i> risk for drug problems for Hispanic/Latinos (B =-2.18, p = .001, <i>IRR</i> : .11).

4. <u>Shared vulnerability</u>	NO SUPPORT FOR TRAUMA EXPOSURE AND
hypothesis: Shared risk factors	FAMILY ADVERSITY AS SHARED RISK
(trauma exposure or family	FACTORS
adversity) increase risk for both	<i>Method 1:</i> The unique effect of trauma exposure was
PTSD and alcohol and drug	non-significant both in the model predicting both
nrohlems, such that they have no	alcohol ($B=17$, $p=38$, $IRR \cdot 1$, 19) and drug problems
causal relation This study tasted	$(B = 20 \ n = 55 \ IRR \cdot 1.22)$ Thus there was no evidence
whether trauma avnosure	(D = .20, p = .55, TKK, 1.22). Thus, there was no evidence that trauma avansura madiated the influence of pro-
increases with for clockel and	that trauma exposure incutated the influence of pre-
Increases risk for alcohol and	trauma family adversity on addit alcohol of drug
drug problems, over and above	problems. Although family adversity had a significant
the effects of PISD symptoms,	effect on both trauma exposure and PISD symptoms,
family adversity, pre-trauma	its direct effect on alcohol problems was non-significant
substance use problems, gender,	(B=.01, p=.92, IRR: 1.01). The direct effect of family
and ethnicity. This study also	adversity on risk for drug problems was significant for
tested the extent to family	females (<i>B</i> = 1.12, <i>p</i> < .001, <i>IRR</i> = 3.06) but not for males
adversity exerts unique and direct	(B=01, p=.95, IRR: 99). PTSD symptoms significantly
effects on trauma exposure, PTSD	mediated the effect of pre-trauma family adversity on
symptoms, and adult alcohol and	both alcohol problems (95% CI= [.010, .038]) and drug
drug problems, as well as the	problems (95% CI= [.001, .057]).
extent to which the influence of	Method 2: Neither risk for adult alcohol problems
pre-trauma family adversity is	$\overline{(B=.12, p=.53, IRR: 1.13)}$ nor adult drug problems
mediated by trauma exposure	(B=.21, p=.53, IRR: 1.24), was elevated in participants
and/or PTSD symptoms	exposed to trauma in the absence of PTSD compared to
and/of 1 15D symptoms.	narticinants who were not exposed to trauma over and
	above the effects of DTSD family adversity gonder
	and atheniaity. Earning advansity did not have a
	and ethnicity. Family adversity did not have a
	significant unique influence risk for adult alcohol
	significant unique influence risk for adult alcohol problems (<i>B</i> =.14, <i>p</i> =.23, <i>IRR</i> : 1.15). Family adversity
	significant unique influence risk for adult alcohol problems (B =.14, p = .23, IRR: 1.15). Family adversity had a significant unique influence on risk for adult
	significant unique influence risk for adult alcohol problems (B =.14, p = .23, IRR: 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (B = 1.27, p < .001, IRR:
	significant unique influence risk for adult alcohol problems (B =.14, p = .23, IRR: 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (B = 1.27, p < .001, IRR: 3.58), but not for males (B = .10, p = .61, IRR: 1.11).
4a. Gender moderates the unique	significant unique influence risk for adult alcohol problems (B =.14, p = .23, <i>IRR</i> : 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (B = 1.27, p < .001, <i>IRR</i> : 3.58), but not for males (B = .10, p = .61, <i>IRR</i> : 1.11). NO SUPPORT
4a. Gender moderates the unique influence of trauma exposure on	significant unique influence risk for adult alcohol problems (B =.14, p = .23, IRR: 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (B = 1.27, p < .001, IRR: 3.58), but not for males (B = .10, p = .61, IRR: 1.11). NO SUPPORT <u>Method 1:</u> There was no evidence that gender moderated
4a. Gender moderates the unique influence of trauma exposure on future alcohol and drug problems	significant unique influence risk for adult alcohol problems (B =.14, p = .23, IRR: 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (B = 1.27, p < .001, IRR: 3.58), but not for males (B = .10, p = .61, IRR: 1.11). NO SUPPORT <u>Method 1</u> : There was no evidence that gender moderated the influence of trauma exposure on either alcohol
4a. Gender moderates the unique influence of trauma exposure on future alcohol and drug problems such that trauma-exposed males (but	significant unique influence risk for adult alcohol problems (B =.14, p = .23, IRR: 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (B = 1.27, p < .001, IRR: 3.58), but not for males (B = .10, p = .61, IRR: 1.11). NO SUPPORT <u>Method 1:</u> There was no evidence that gender moderated the influence of trauma exposure on either alcohol problems (B =36, p = .35, IRR: .70) or drug problems (B =
4a. Gender moderates the unique influence of trauma exposure on future alcohol and drug problems such that trauma-exposed males (but not females) are at higher risk for	significant unique influence risk for adult alcohol problems (B =.14, p = .23, IRR: 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (B = 1.27, p < .001, IRR: 3.58), but not for males (B = .10, p = .61, IRR: 1.11). NO SUPPORT <u>Method 1:</u> There was no evidence that gender moderated the influence of trauma exposure on either alcohol problems (B =36, p = .35, IRR: .70) or drug problems (B = .01, p = .99, IRR: 1.01).
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4a. Gender moderates the unique influence of trauma exposure on future alcohol and drug problems such that trauma-exposed males (but not females) are at higher risk for future alcohol and drug problems, independent of PTSD compared to	significant unique influence risk for adult alcohol problems (B =.14, p =.23, IRR : 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (B = 1.27, p < .001, IRR : 3.58), but not for males (B = .10, p = .61, IRR : 1.11). NO SUPPORT <u>Method 1</u> : There was no evidence that gender moderated the influence of trauma exposure on either alcohol problems (B =36, p = .35, IRR : .70) or drug problems (B = .01, p = .99, IRR : .1.01). <u>Method 2</u> : There was no evidence that gender moderated the effect of trauma exposure without PTSD on either
4a. Gender moderates the unique influence of trauma exposure on future alcohol and drug problems such that trauma-exposed males (but not females) are at higher risk for future alcohol and drug problems, independent of PTSD, compared to unexposed males	significant unique influence risk for adult alcohol problems (B =.14, p =.23, IRR: 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (B = 1.27, p < .001, IRR: 3.58), but not for males (B = .10, p = .61, IRR: 1.11). NO SUPPORT <u>Method 1:</u> There was no evidence that gender moderated the influence of trauma exposure on either alcohol problems (B =36, p = .35, IRR: .70) or drug problems (B = .01, p = .99, IRR: .1.01). <u>Method 2:</u> There was no evidence that gender moderated the effect of trauma exposure without PTSD on either alcohol problems (B =44, p = .24, IRR: .65) or drug
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4a. Gender moderates the unique influence of trauma exposure on future alcohol and drug problems such that trauma-exposed males (but not females) are at higher risk for future alcohol and drug problems, independent of PTSD, compared to unexposed males.	significant unique influence risk for adult alcohol problems (B =.14, p =.23, IRR : 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (B = 1.27, p < .001, IRR : 3.58), but not for males (B = .10, p = .61, IRR : 1.11). NO SUPPORT <u>Method 1</u> : There was no evidence that gender moderated the influence of trauma exposure on either alcohol problems (B =36, p = .35, IRR : .70) or drug problems (B = .01, p = .99, IRR : .1.01). <u>Method 2</u> : There was no evidence that gender moderated the effect of trauma exposure without PTSD on either alcohol problems (B =44, p = .24, IRR : .65) or drug problems (B = .12, p = .87, IRR : .1.12).
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 4a. Gender moderates the unique influence of trauma exposure on future alcohol and drug problems such that trauma-exposed males (but not females) are at higher risk for future alcohol and drug problems, independent of PTSD, compared to unexposed males. 4b. Ethnicity moderates the unique influence of trauma exposure on future alcohol and drug problems. 	significant unique influence risk for adult alcohol problems (<i>B</i> =.14, <i>p</i> =.23, <i>IRR</i> : 1.15). Family adversity had a significant unique influence on risk for adult drug problems for females (<i>B</i> = 1.27, <i>p</i> < .001, <i>IRR</i> : 3.58), but not for males (<i>B</i> = .10, <i>p</i> = .61, <i>IRR</i> : 1.11). NO SUPPORT <u>Method 1</u> : There was no evidence that gender moderated the influence of trauma exposure on either alcohol problems (<i>B</i> =36, <i>p</i> = .35, <i>IRR</i> : .70) or drug problems (<i>B</i> = .01, <i>p</i> = .99, <i>IRR</i> : .1.01). <u>Method 2</u> : There was no evidence that gender moderated the effect of trauma exposure without PTSD on either alcohol problems (<i>B</i> =44, <i>p</i> = .24, <i>IRR</i> : .65) or drug problems (<i>B</i> = .12, <i>p</i> = .87, <i>IRR</i> : .1.12). NO SUPPORT Results presented below excluded the 18 participants of ethnicities other than Hispanic/Latino or non- Hispanic/Latino Caucasian. <u>Method 1</u> : There was no evidence that ethnicity moderated the influence of trauma exposure on either alcohol problems (<i>B</i> =68, <i>p</i> = .14, <i>IRR</i> : .51) or drug problems (<i>B</i> = -1.03, <i>p</i> = .13, <i>IRR</i> : .36).
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n=377. Separate analyses tested the influence of adolescent substance use problems on risk for trauma exposure or assaultive violence exposure (see bold path). Additional analyses entered an interaction between gender and adolescent substance use problems (see dotted path in Figure 1.1). An additional analysis tested whether adolescent substance use problems mediate the influence of gender on risk for trauma exposure or assaultive violence exposure (Figure 1.2).





n=166. Separate analyses tested the influence of adolescent substance use problems on risk for PTSD diagnosis (dichotomous variable) and PTSD symptoms (count variable). Additional analyses entered an interaction between gender and adolescent substance use problems (see dotted path).



Figure 3. The Self-Medication and Shared Vulnerability Hypotheses: Approach 1 n=377. The self-medication and shared vulnerability hypotheses were tested using two different approaches. The first approach examined the influences of trauma exposure (dichotomous variable) and PTSD symptoms (count variable) on risk for adult alcohol or drug problems. Adult alcohol problems and adult drug problems were tested as the dependent variable in separate models. Additional analyses examined whether gender or ethnicity moderated the paths labeled H3_{self-medication} or H4_{trauma as shared risk}.



Figure 4. The Self-Medication and Shared Vulnerability Hypotheses: Approach 2 n=377. The self-medication and shared vulnerability hypotheses were tested using two different approaches. The second approach examined how risk for adult alcohol and drug problems varied among three different groups: (1) those who were exposed to trauma and developed PTSD, (2) those who were exposed to trauma. These groups were compared using two dummy variables. The group without trauma exposure served as the reference group. Adult alcohol problems and adult drug problems were tested as the dependent variable in separate models. Additional analyses examined whether gender or ethnicity moderated the bolded paths.



Figure 5. Summary of Measures Used in the Present Study

APPENDIX A

SUBSTANCE USE PROBLEMS

Adolescent Substance Use Problems

Response options for the Wave 1 items were "yes" or "no." The substance use problem was considered to be endorsed if the adolescent reported ever experiencing any of the corresponding items due to *either* alcohol *or* drug use in his or her lifetime.

Adult Alcohol or Drug Problems

Response options for the Wave 5 items were: within the past 3 months, within the past year, 1-2 years ago, 2-5 years ago, more than 5 years ago, or never. These recency probes were asked separately for alcohol and drug use. Separate alcohol and drug variables indicated whether the adult reported experiencing each problem due to their alcohol use within the past two years, or due to their drug use within the past two years.

Substance Use Problem	Items Used to Assess Substance Use Problem	Items Used to Assess Alcohol/Drug Problem.
	at Wave 1	separately, at Wave 5
	(Adolescence)	(Adulthood)
1. Problems at school or work due to substance use	 Did you ever get in trouble at school or work because of your ALCOHOL or DRUG USE Did you ever miss school or work because of your ALCOHOL or DRUG USE Did you ever have problems with your schoolwork or studying because of your 	 How recently did you get in trouble at school or work because of your ALCOHOL/DRUG USE How recently did you miss school or work because of your ALCOHOL/DRUG USE How recently did you have problems with your schoolwork or studying because of your ALCOHOL/DRUG USE

	 ALCOHOL or DRUG USE Did you ever lose a job or get kicked out of school because or your ALCOHOL or DRUG USE 	
2. Substance use led to accident or injury	• Did you ever have an accident or injury because of your ALCOHOL or DRUG USE	 How recently did you have an accident or injury because of your ALCOHOL/DRUG USE How recently has your ALCOHOL/DRUG use caused you to injure someone else
3. Problems with family or friends due to substance use	 Have you ever gotten complaints from your family because of your ALCOHOL or DRUG USE? Have you ever gotten complaints from your friends about your ALCOHOL or DRUG USE Did you ever have problems with your family or friends because of your ALCOHOL or DRUG USE 	 How recently did you get complaints from your family about your ALCOHOL/DRUG USE How recently did you get complaints from your friends about your ALCOHOL/DRUG USE
4. Physical fight due to substance use	• Did you ever get into a physical fight because of your ALCOHOL or DRUG USE	• How recently did you get into a physical fight because of your ALCOHOL/DRUG USE
5. Destroyed property due to substance use	• Did you ever destroy property because of your ALCOHOL or DRUG USE	• How recently did you destroy property because of your ALCOHOL/DRUG USE?
6. Legal problems due to substance use	• Did you ever get arrested because of your ALCOHOL or DRUG USE?	• How recently did you get arrested because of your ALCOHOL/DRUG USE?

7. Tolerance	• Have you ever found that you needed larger amounts of ALCOHOL or DRUGS to get an effect - or that you could no longer get high on the amount you used to use?	 How recently have you found that you needed larger amounts of ALCOHOL/DRUG to get an effect How recently have you found that you could no longer get high on the same amount of ALCOHOL/DRUG you used to use
8. Withdrawal symptoms	• Have you ever needed a DRINK or a DRUG (not counting caffeine) just after you'd gotten up - that is, before breakfast	 How recently have you had withdrawal symptoms because you stopped using ALCOHOL/DRUGS or cut down on your ALCOHOL/DRUGS use How recently have you needed a DRINK/DRUG (not counting caffeine) just after you'd gotten up - that is, before breakfast
9. Used larger amounts of substance than intended, or for longer period of time than intended	• Have you ever ended up using much larger amounts of ALCOHOL or DRUGS than you expected to when you began, or over more days than you intended to	 How recently have you ended up using much larger amounts of ALCOHOL/DRUGS than you expected to when you began How recently have you ended up using ALCOHOL/DRUGS over more days than you intended to when you began
10. Tried to cut down on substance use	• Have you ever tried to cut down on ALCOHOL or DRUGS but found that you couldn't	• How recently have you tried to cut down on ALCOHOL/DRUGS but found that you couldn't
11. Great deal of time is spent arranging to get substance or having it on mind	• Has there ever been a period when you spent so much time arranging to get ALCOHOL or DRUGS or having	• How recently has there been a period where you spent so much time arranging to get ALCOHOL/DRUGS or

	them on your mind so much that you had little time for anything else	having it on your mind so much that you had little time for anything else
12. Felt guilty about substance use	• Have you ever felt guilty about your DRINKING or DRUG USE	• How recently have you felt guilty about your DRINKING/DRUG USE
13. Passed out or fainted due to substance use	• Did you ever pass out or faint because of your ALCOHOL or DRUG USE)	• How recently did you pass out or faint because of your ALCOHOL/DRUG USE
14. Felt he/she needed or depended on substance	• Have you ever used ALCOHOL or DRUGS enough so that you felt like you needed it or depended on it	• How recently have you used ALCOHOL/DRUGS enough so that you felt like you needed it or depended on it
15. Neglected usual responsibilities due to substance use	None	• How recently has your ALCOHOL/DRUG use caused you to neglect some of your usual responsibilities
16. Substance use led to sexual situations later regretted	None	• How recently has your ALCOHOL use gotten you into sexual situations that you later regretted
17. Drove motor vehicle when under influence of substance	None	• How recently did you drive a car or other motor vehicle when you knew that you had too much ALCOHOL/DRUGS to drive safely

APPENDIX B

FAMILIAL LIFE STRESS ITEMS
The adolescent version is present here. The wording and order of the items differed between the parent and adolescent versions.

"Here is a list of things that happen to people, which of these happened to you in the past 3 months?"

- Your brother or sister has serious trouble (with the law, school, drugs, etc.).
- 2. Your brother or sister suffered a serious physical illness or injury.
- 3. Your mom or dad suffered a serious illness or injury.
- 4. Your mom or dad talked about having serious money troubles.
- 5. Your relatives said bad things about your mom or dad.
- 6. Your mom or dad fought or argued with your relatives.
- 7. People in your neighborhood said bad things about your mom or dad.
- 8. Your mom or dad acted badly in front of your friends.
- Your mom or dad forgot to do important things for you that they promised they would do (such as take you someplace or go to school or athletic activities).
- 10. Your mom or dad was arrested or sent to jail.
- 11. Your mom or dad lost their job.
- 12. A close family member died.
- 13. You changed schools because of a family move.
- 14. Your mom and dad got divorced or separated.

Your mom or dad spent one or more nights away from home when they should have been home.