Posttraumatic Stress Disorder and Alcohol Dependence in Young Women*

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ABSTRACT. Objective: The aim of the current study is to characterize the relationship between posttraumatic stress disorder (PTSD) and alcohol dependence (AD) in women, distinguishing PTSD-specific influences on AD from the contribution of co-occurring psychiatric conditions and from the influences of trauma more generally. **Method:** Trauma histories and DSM-IV lifetime diagnoses, including PTSD and AD, were obtained via telephone interview from 3,768 female twins. Based on PTSD status and trauma history, participants were categorized as no trauma (43.7%), trauma without PTSD (52.6%), or trauma with PTSD (3.7%). Cox proportional hazards regression analyses were conducted using trauma/PTSD status to predict AD, first adjusting only for ethnicity and parental problem drinking, then including conduct disorder, major depressive disorder, regular smoking, and cannabis abuse. **Results:** Before accounting for psychiatric covariates, elevated

rates of AD were evident in both trauma-exposed groups, but those with PTSD were at significantly greater risk for AD than those without PTSD. This distinction was no longer statistically significant when psychiatric covariates were included in the model, but both trauma-exposed groups continued to show elevated odds of developing AD compared with the no trauma group. **Conclusions:** The elevated rates of AD in women who have experienced trauma are not accounted for in full by psychiatric conditions that commonly co-occur with AD and trauma exposure. The greater likelihood of developing AD in the subset of trauma-exposed individuals who develop PTSD may reflect higher levels of distress and/or higher rates of psychopathology associated with traumas that lead to PTSD rather than PTSD-specific influences. (*J. Stud. Alcohol Drugs*, 71, 810-818, 2010)

ONE IN EIGHT WOMEN develops posttraumatic stress disorder (PTSD) over her lifetime, approximately twice the percentage of men who meet criteria for a lifetime PTSD diagnosis (Breslau et al., 1998; Kessler et al., 1995; Resnick et al., 1993), and studies based on community samples have consistently demonstrated an association between PTSD and alcohol use disorders (AUDs) in women (Breslau et al., 1997; Danielson et al., 2009; Kessler et al., 1997; Perkonig et al., 2000). For example, using data from the National Comorbidity Survey, Kessler and colleagues (1995) found that the prevalence of alcohol abuse/dependence in women with PTSD was double that in women without the disorder: 27.9% versus 13.5%. Evidence for elevation in the rate of AUDs in women who develop PTSD is compelling, but the source of the co-occurrence of the two conditions is not yet clearly established. One possibility is that traumatic experiences leading to the development of PTSD confer risk broad-

ly for psychopathology, with AUDs and PTSD being two of many potential negative outcomes of such experiences.

Numerous studies have documented the association of AUDs with history of child maltreatment (Anda et al., 2002; Dinwiddie et al., 2000; Fergusson et al., 1996a; Wilsnack et al., 1997) and physical and sexual assault in adulthood (Kilpatrick et al., 1997, 2003). It is important to note that although among traumatic events sexual assault has one of the highest conditional probabilities for developing PTSD, only 30%-50% of sexual assault victims develop the disorder (Breslau et al., 1998; Hapke et al., 2006; Resnick et al., 1993), suggesting that elevated risk for AUDs may not be limited to the subset of women who meet PTSD criteria. Investigations aimed at determining the specificity of risk conferred by PTSD compared with the effects of trauma more generally on alcohol-related problems have produced mixed findings. In their longitudinal study of young adults, Danielson et al. (2009) found that PTSD contributed additional variance to women's risk for past-year alcohol problems after accounting for sexual assault, physical assault, and witnessing violence, but sexual assault remained an independent predictor. By contrast, in Epstein et al.'s (1998) study of AUDs in victims of childhood rape, PTSD symptoms fully mediated the association between rape and alcohol problems. Yet another pattern of findings was reported by Kilpatrick et al. (1997) based on an adolescent sample: physical and sexual assault predicted past-year AUDs, but PTSD did not account for any additional variance. Breslau and colleagues

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have addressed the issue by comparing rates of AUDs in individuals who did not report any trauma exposure with those reporting trauma exposure in the absence of PTSD, in addition to those meeting PTSD criteria. In one such study making use of both retrospective and prospective data, analyses using prospective data revealed no association between alcohol abuse/dependence and either PTSD or trauma exposure in the absence of PTSD. However, analyses based on retrospective data revealed trauma in the absence of PTSD to be a significant predictor of alcohol abuse/dependence in women (Breslau et al., 2003). These findings contrasted with results from their work with an all-female sample, in which both trauma-exposed groups (with and without PTSD) had elevated rates of alcohol abuse/dependence compared with women who had no trauma exposure (Breslau et al., 1997). Thus, whether PTSD confers additional risk for AUDs in trauma-exposed individuals remains an open question.

A second possible explanation for the high comorbidity of PTSD and AUDs is the elevated rates of co-occurrence of AUDs and PTSD with other psychiatric disorders such as major depressive disorder (MDD), conduct disorder (CD), and other substance use disorders. The odds of meeting diagnostic criteria for PTSD in individuals with MDD ranges from 4 to 7 times that of those without MDD (Breslau et al., 1997; Fu et al., 2007; Hapke et al., 2006). MDD, in turn, is consistently associated with elevated rates of AUDs (Dawson et al., 2004; Grant et al., 2004; Hasin et al., 2007). Kessler et al. (1997) found that nearly half (48.5%) of women with alcohol dependence (AD) met MDD criteria. The strong association between PTSD and CD is also well documented (Fu et al., 2007; Koenen et al., 2002, 2005). According to National Comorbidity Survey data (Kessler et al., 1995), the prevalence of CD in individuals meeting diagnostic criteria for PTSD is nearly triple that of those without a PTSD diagnosis (15.4% vs. 5.9%). There is an extensive literature linking AUDs to CD (King et al., 2004; Kuperman et al., 2005; McGue et al., 2001) as well as smoking (Cardenal and Adell, 2000; Dawson, 2000; Grucza and Bierut, 2006) and cannabis abuse and dependence (Degenhardt et al., 2002; Grant and Pickering, 1998; Stinson et al., 2006). Evidence for elevated rates of cannabis and other illicit drug use disorders in individuals with PTSD is also strong (Breslau et al., 2003; Kilpatrick et al., 2000; Perkonig et al., 2000; Reed et al., 2007), with the odds of meeting criteria for illicit drug abuse or dependence ranging from two to four times that in those without PTSD (Breslau et al., 1997; Chilcoat and Breslau, 1998; Kilpatrick et al., 2000). Similar findings have been reported for nicotine dependence (Breslau et al., 2003; Perkonig et al., 2000).

Psychiatric disorders that frequently co-occur with PTSD are also associated with trauma more generally. Rates of MDD (Dinwiddie et al., 2000; Kendler et al., 2000; McCutcheon et al., 2009; Nelson et al., 2002), CD (Afifi et al., 2009; Fergusson et al., 1996a; Molnar et al., 2001), cannabis and

other illicit drug use disorders (Duncan et al., 2008; Kendler et al., 2000; Kilpatrick et al., 2000) and smoking (Al-Mamun et al., 2007; Anda et al., 1999; Nelson et al., 2006) are elevated in trauma-exposed populations. Thus, in examining the relationship between PTSD and alcohol-related outcomes, it is critical to adjust for the potential confounding effects of these commonly co-occurring conditions on the association of both PTSD and trauma exposure with AUDs.

The current study aims to characterize the relationship between PTSD and AD in women, distinguishing PTSD-specific influences on AD from the contribution of co-occurring psychiatric conditions and from the influences of trauma more generally. Toward this end, we follow Breslau et al. (1997, 2003) in contrasting rates of AD in women meeting PTSD criteria with rates in women who experienced trauma but did not develop PTSD, as well as women who did not endorse any traumatic events. By including psychiatric correlates of PTSD, trauma, and AD in our investigation of the PTSD-AD association, we extend the existing literature, which to date has rarely addressed the potential confounding effects of commonly co-occurring psychopathology on the relationship between PTSD and AUDs.

Method

Participants

The sample consisted of 3,768 female twins from the Missouri Adolescent Female Twin Study (MOAFTS), a longitudinal study of alcohol-related problems and associated psychopathology in female adolescents and young adults. Twins born to Missouri-resident parents between July 1, 1975 and June 30, 1985 were identified through birth records and recruited from 1995 to 1999 using a cohort-sequential design. Cohorts of 13-, 15-, 17-, and 19-year-old female twin pairs and their families were ascertained in the first two years, with new cohorts of 13-year-old twins and their families added in the subsequent two years. Parental diagnostic interviews were completed by at least one parent in 78% of families eligible for participation. (See Heath et al., 2002 for additional details on ascertainment). Wave 3 retest interviews were conducted with a subset of Wave 1 participants approximately two years after Wave 1 interviews were completed. (Data were not drawn from Wave 2 because Wave 2 assessments did not cover all domains of interest in the current study and only referenced experiences from the previous 24 months.) All twins from the target cohort (excluding those who had withdrawn from the study or whose parents requested that the family not be recontacted) were contacted for Wave 4 interviews, which were conducted from 2002 to 2005. PTSD was assessed only in Wave 4, but other relevant data (including history of alcohol use and trauma exposure) were collected in Waves 1 and 3 as well. For those individuals who had participated in prior waves of

data collection (78% of Wave 4 participants), data from these interviews were incorporated and used to establish lifetime status for a given risk factor or outcome. Mean age at the time of Wave 4 interviews was 21.7 years ($SD = 2.7$). Fifteen percent of the sample self-identified as African-American, the remainder as non-Hispanic White, reflective of the ethnic composition of Missouri.

Procedure

Data were collected via telephone by trained interviewers. At baseline, a screening to determine zygosity of the twin pair was conducted with one of the twins' parents. Parents who agreed to participate were scheduled for diagnostic interviews. Verbal consent was obtained prior to the start of interviews. Interviews with the twins were conducted after obtaining verbal consent (and, for those younger than age 18, the consent of parents). The same protocol was followed in Waves 3 and 4.

Assessment battery

Data were collected through an interview modified for telephone administration from the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA/SSAGA-II; Bucholz et al., 1994; Hesselbrock et al., 1999), an instrument designed to assess AUDs and related psychiatric conditions. The SSAGA was used to obtain history of trauma exposure, gather information on alcohol and other substance use, and obtain DSM-IV diagnoses (American Psychiatric Association, 1994), including PTSD and AD.

Trauma checklist. Traumatic event exposure was queried in Waves 1, 3, and 4 with a standard trauma checklist. Respondents were asked if they had experienced any of the following events: (a) fire, flood, or natural disaster; (b) life-threatening accident; (c) witnessing someone being badly injured or killed; (d) rape; (e) sexual molestation; (f) physical abuse as a child; (g) serious physical attack or assault; (h) serious neglect as a child; and (i) being threatened with a weapon, held captive, or kidnapped. Age at first experience of the event was queried for all endorsed events.

Additional child maltreatment and sexual assault questions. In an effort to capture as many cases as possible, child maltreatment and sexual assault questions were also included, under different wording, in the early home environment and sexual maturation sections of the Wave 1, 3, and 4 interviews. These items are listed in Table 1. For those individuals who responded positively only to those questions querying abuse and neglect between ages 6 and 13, age at trauma onset was estimated at 9 years. Refusals were counted as missing.

Trauma status. Trauma history was coded positive if participants endorsed one or more items on the traumatic event checklist or if they met criteria for child physical abuse or neglect or sexual assault based on the additional child maltreatment and sexual assault questions in any wave of data collection. (Although some individuals who endorsed child maltreatment or sexual assault questions did not report the corresponding events on the Wave 4 trauma checklist, 79.5% of participants meeting criteria for lifetime trauma exposure endorsed at least one event on the checklist and were therefore assessed for PTSD.) Age at first trauma exposure was

TABLE 1. Additional questions used to determine sexual assault and CPAN status shown by section of the interview

Sexual assault
Sexual maturation
1. Has anyone ever forced you to have sexual intercourse?
Parental discipline and early childhood experiences
2. Before you turned 16, was there any sexual contact between you and any family member like a parent or step-parent, grandfather, etc.? By sexual contact I mean their touching your sexual parts, your touching their sexual parts, or intercourse.
3. Before you turned 16, was there any sexual contact between you and anyone who was 5 or more years older than you (<i>other than a family member</i>)?
CPAN
Parental discipline and early childhood experiences
When you were 6 to 13...
1. When you did something wrong, how often were you hit with a belt or stick or something like that by your mother figure/father figure? ^a
2. How often did your mother figure/father figure punish you so hard that you hurt the next day? ^a
3. Were you ever physically injured or hurt on purpose by an adult (e.g., having broken bones or burns)? ^b
4. What was the usual way in which your parents punished or disciplined you?
Non-physical harsh (e.g., lock in closet, deprive of food)
Physical harsh (e.g., use weapon, kick)

Notes: CPAN = child physical abuse and neglect. ^aPositive if frequency reported as "often"; ^bpositive if occurred before age 16.

coded as the earliest age at onset for any of the qualifying traumatic events.

Posttraumatic stress disorder. All respondents endorsing one or more traumatic events in the trauma checklist administered at Wave 4 ($n = 1,684$; 44.5% of the sample) were asked which of the events they experienced was the most disturbing, then whether they had experienced “intense fear, helplessness, or horror” following that event (Criterion A for DSM-IV PTSD). This became the index event for which PTSD symptoms were assessed. For Criteria B, C, and D, diagnostic questions were administered only if the preceding criterion was met. Criterion B was met if one or more of the five possible re-experiencing symptoms were endorsed. Meeting Criterion C required endorsement of three or more of the seven possible avoidance symptoms. Criterion D was met if two or more of the five arousal symptoms were endorsed. A diagnosis of PTSD was given if Criteria A through D were met and respondents reported clinically significant distress or impairment and persistence of symptoms for 1 month or longer. Age at onset of PTSD was defined as the age at which any of the symptoms were first experienced for 1 month or longer. Mean age at PTSD onset was 13.8 years ($SD = 5.2$).

Alcohol dependence. AD was defined according to DSM-IV criteria (i.e., three or more of the seven possible AD symptoms occurring during the same 12-month period). Age at onset of AD was defined as the age at which full DSM-IV criteria for AD were first met. First positive report was used in cases in which more than one report was available. Mean age at AD onset in the sample was 17.8 years ($SD = 2.4$).

Other risk factors for alcohol dependence. Maternal and paternal alcohol-related problems/excessive drinking, regular smoking, CD, MDD, and cannabis abuse were assessed at Waves 1, 3, and 4. Parental alcohol-related problems were assessed with two questions: (a) “Has drinking ever caused your biological mother/father to have problems with health, family, job, or police, or other problems?” and (b) “Have you ever felt that your biological mother/father was an excessive drinker?” (Mother’s and father’s drinking were queried separately.) Endorsement of either item by the twin or her co-twin resulted in a positive value for history of problem alcohol use in that parent. Regular smoking was defined as smoking more than 20 cigarettes (lifetime) and smoking at least once a week for 3 weeks or longer in Waves 1 and 3. In Wave 4, the minimum quantity was also 21 cigarettes, but the minimum duration of weekly smoking was 2 months. (A lower threshold than the standard 100 cigarettes used with adult samples was chosen given the substantial number of participants who were younger than age 18 when smoking behaviors were first assessed. This intensity of smoking is associated with loss of control over smoking and nicotine dependence in young smokers [DiFranza et al., 2007].) Diagnoses of CD, MDD, and cannabis abuse were based on DSM-IV criteria. Endorsement at any wave of data collection resulted in a positive value for that risk factor. For regu-

lar smoking, MDD, and cannabis abuse, when more than one report was available, age at onset was derived from the first report. Age at onset of CD was not queried in the first wave of data collection and CD criteria were not re-assessed for all participants in Wave 4, resulting in a substantial number of cases missing age at onset of CD. Analyses were therefore based on a dichotomous CD variable.

Data analysis

Analyses were conducted with a three-level PTSD variable—PTSD, trauma without PTSD, and no trauma exposure—so that PTSD-specific effects on AD could be distinguished from those attributable to trauma exposure more generally. Distribution across the three levels was as follows: 3.7% met PTSD criteria, 52.6% reported trauma exposure but did not meet PTSD criteria, and the remaining 43.7% did not report experiencing any traumatic events. Prevalence of each of the relevant covariates is shown by the three-level PTSD variable in Table 2, along with rates of trauma exposure and PTSD by ethnicity.

The association of PTSD with AD was examined within a survival analysis framework, using no trauma exposure as the reference group. A Cox proportional hazards model was chosen to conduct multivariate regression with time-to-event data, as not all participants had passed through the age of risk for AD. PTSD/trauma exposure was modeled as a time-varying covariate, so that only PTSD (or trauma) that occurred before AD onset counted toward risk for AD. Regular smoking, MDD, and cannabis abuse were also modeled as time-varying covariates. This was done by creating a “person-year” data set using SAS Version 8.2 (SAS Institute Inc., Cary, NC), in which each line of data represented a single year of life for every individual. For cases that were positive for a given covariate, that covariate was coded as absent in each year up to the age of onset for that condition and present from that year onward. Ethnicity, maternal alcohol-related problems, paternal alcohol-related problems,

TABLE 2. Family history, ethnicity, and psychiatric risk factors by trauma/PTSD status*

Variable	PTSD ($n = 138$)	Trauma without PTSD ($n = 1,981$)	No trauma ($n = 1,649$)
Maternal alcohol-related problems	34.8%	16.8%	8.4%
Paternal alcohol-related problems	63.8%	39.7%	27.8%
Major depressive disorder	73.9%	30.5%	13.8%
Conduct disorder	16.3%	6.2%	1.9%
Regular smoking	62.3%	40.8%	29.0%
Cannabis abuse	13.0%	5.0%	1.9%
Distribution by ethnicity			
African-American	7.0%	68.6%	24.4%
White	3.1%	49.9%	47.0%

Notes: PTSD = posttraumatic stress disorder.

*All chi-square tests of association were statistically significant at the $p < .05$ level.

and CD were treated as time-invariant variables. Age at the time of AD report was included in the models to adjust for potential retrospective reporting bias resulting from the use (in some cases) of AD diagnoses derived from earlier data collections than the one in which PTSD was assessed. For those participants who met AD criteria ($n = 455$), this was calculated as the age at the time of interview in which AD diagnosis was first given. For the remaining cases, age at AD report was set equal to age at the time of Wave 4 interview (i.e., when the absence of AD was reported). The age range was broken into three approximately equal groups: 13-19, 20-22, and 23-29 years, represented in the models by two dummy variables, with ages 20-22 as the comparison group. Analyses were conducted in Stata Version 9.2 (StataCorp, 2007), using the Huber-White correction to adjust for the non-independence of observations in twin pairs.

Analyses began with the construction of a base model that included maternal alcohol-related problems, paternal alcohol-related problems, ethnicity, and dummy variables representing age at AD report, in addition to the two PTSD/trauma status variables (PTSD and trauma without PTSD) to predict AD. Additional covariates were then introduced one at a time into the base model. Covariates were retained if the hazard ratios were statistically significant. After testing each individually, all statistically significant covariates were combined into a single model to determine each of their unique contributions to AD risk in the context of the other covariates.

The proportional hazards assumption that risk remains constant over time was assessed using the Grambsch and Therneau test of the Schoenfeld residuals (Grambsch and Therneau, 1994). There were no violations in the base model, but the proportional hazards assumption was violated for ethnicity and CD in the second model. To adjust for the violation, the period of risk for onset of AD was split into six empirically derived subdivisions: up to age 7, 8-11, 12-15, 16-19, 20-23, and 24-29, and terms for the interaction of ethnicity and of CD with all six periods of risk were created. Inclusion of the interaction between ethnicity and the period of risk for ages 20-23 and the interaction between CD and the period of risk for ages 8-11 in the model resulted in a non-significant outcome in the proportional hazards assumption tests. (The addition of the CD $\times$ ages 8-11 interaction term also resulted in a non-significant hazard ratio for the main effect of CD.)

Results

Family history of problem drinking, ethnicity, and psychiatric risk factors by trauma exposure/PTSD

The proportion of respondents reporting maternal and paternal drinking problems and the prevalence of CD, MDD, regular smoking, and cannabis abuse are shown by trauma

status in Table 2, along with the rates of endorsement of trauma and PTSD by ethnicity. All tests of association yielded chi-square values that exceeded the minimum values for significance at the $p < .05$ level. Rates of the risk factors were uniformly highest in the PTSD group, followed by the trauma without PTSD group and the no trauma exposure group. More than 13% of women with PTSD, for example, met cannabis abuse criteria compared with 5% of women in the trauma without PTSD group, and fewer than 2% who had no history of trauma exposure. As seen in Table 2, rates of trauma exposure and PTSD diagnosis differed markedly by ethnicity. Whereas three fourths of African-American women endorsed some form of trauma over their lifetimes, trauma exposure was reported by just over half of White women. Similarly, compared with White women, more than twice as many African-American women met PTSD criteria (7.0% vs. 3.1%).

Trauma exposure, PTSD, and alcohol dependence

Base model. Results from the first Cox proportional hazards regression analysis using the three-level trauma exposure/PTSD variable (no trauma, trauma without PTSD, and PTSD) to predict progression to AD are shown in Table 3. In this base model, adjustments were made for risk conferred by maternal and paternal histories of alcohol-related problems, age, and ethnicity. Trauma exposure—in the absence as well as in the presence of PTSD—was a strong predictor of AD. Compared with those who did not report experiencing any trauma, women in the trauma without PTSD group had nearly twice the odds of developing AD (HR = 1.85, CI [1.49, 2.31]). The elevation in risk for AD was even greater for those who developed PTSD (HR = 3.54, CI [2.33, 5.38]). The hazard ratio for PTSD was significantly higher than the hazard ratio for trauma without PTSD, $\chi^2(1) = 10.41$, $p < .01$, indicating that PTSD confers risk for AD beyond that attributable to the trauma alone. Family history (maternally and paternally transmitted risk) of alcohol-related problems was associated with elevated rates of AD as well. By contrast, African-American ethnicity was associated with substantially lower risk of AD (HR = 0.43, CI [0.29, 0.64]).

Model with covariates. Results from the second Cox proportional hazards regression analysis using PTSD/trauma status to predict progression to AD are also shown in Table 3. In addition to factors included in the base model, this model incorporated psychiatric covariates that produced significant hazard ratios in the context of the base model variables (all four that were tested; i.e., MDD, CD, regular smoking, and cannabis abuse). Even after adjusting for these additional factors, trauma status strongly predicted likelihood of developing AD. Hazard ratios for trauma without PTSD and for trauma with PTSD were somewhat attenuated compared with estimates in the base model, but they remained significant (1.50, CI [1.22, 1.89], and 2.19, CI [1.37, 3.50],

TABLE 3. Results of Cox proportional hazards regression analyses predicting alcohol dependence from trauma/PTSD status

Variable	Hazard ratio [95% CI]	
	Base model	Full model
PTSD	3.54 [2.33, 5.38]	2.19 [1.37, 3.50]
Trauma without PTSD	1.85 [1.49, 2.31]	1.50 [1.22, 1.89]
African-American ethnicity	0.43 [0.29, 0.64]	0.62 [0.41, 0.94]
Maternal alcohol-related problems	1.63 [1.26, 2.11]	1.11 [0.85, 1.45]
Paternal alcohol-related problems	1.68 [1.35, 2.10]	1.32 [1.06, 1.64]
Major depressive disorder	—	1.77 [1.43, 2.20]
Conduct disorder	—	0.93 [0.62, 1.42]
Regular smoking	—	3.55 [2.80, 4.50]
Cannabis abuse	—	2.03 [1.44, 2.86]

Notes: PTSD = posttraumatic stress disorder; CI = confidence interval.

respectively). However, in contrast with the base model, the hazard ratios for the two trauma groups did not differ significantly from each other, $\chi^2(1) = 2.64$, $p = .10$. As in the base model, paternal alcohol-related problems were associated with higher rates of AD and African-American ethnicity was associated with lower prevalence of AD, but in the context of the additional psychiatric covariates, maternal problem drinking no longer predicted AD. The most potent psychiatric predictor of AD was regular smoking, which was associated with a three-and-a-half-fold increase in likelihood of developing AD (HR = 3.55, CI [2.80, 4.50]). Cannabis abuse and MDD also contributed significantly to risk for AD, but (as noted in the Method section) after adjusting for proportional hazards violations, the hazard ratio for CD was no longer statistically significant.

Discussion

The current investigation builds on the evidence for elevated rates of AUDs in women who have experienced traumatic events—both leading to and in the absence of PTSD—and the high rates of co-occurrence of both AUDs and PTSD with CD, MDD, and misuse of other substances. It addresses the possible contribution of comorbid psychopathology and PTSD-specific versus more general effects of trauma on the association between PTSD and AD in young women. Our integration of the approach taken by Breslau et al. (1998, 2003), using both trauma without PTSD and no trauma groups as controls, with statistical adjustments for the possible confounding effects of family history of alcoholism and of psychiatric conditions that commonly co-occur with PTSD and AD (which is rarely seen in the literature), affords us a unique perspective on this complex relationship. Findings indicate that the relationship between trauma and AD is not explained in full by commonly co-occurring risk factors and psychopathology and that after accounting for comorbid psychiatric conditions, the relationship between trauma and AD is not significantly stronger in cases in which PTSD develops.

Association of posttraumatic stress disorder and trauma with ethnicity and risk factors for alcohol dependence

Consistent with prior studies of trauma exposure and PTSD, elevated rates of parental problem drinking (Dube et al., 2001; Fergusson et al., 1996b; Sher et al., 1997), MDD (Breslau et al., 1997; Hapke et al., 2006; Kendler et al., 2000; McCutcheon et al., 2009), CD (Afifi et al., 2009; Fu et al., 2007; Kessler et al., 1995; Koenen et al., 2002), regular smoking (Al-Mamun et al., 2007; Breslau et al., 2003; Nelson et al., 2006), and cannabis abuse (Duncan et al., 2008; Kilpatrick et al., 2000; Nelson et al., 2006; Reed et al., 2007) were observed in women who had experienced traumatic events—both those who developed PTSD and those who did not—compared with women who did not report any trauma exposure. Much of the prior research in this area has involved comparing rates of co-occurring psychopathology and psychosocial risk factors in individuals with PTSD with those who do not meet diagnostic criteria, without regard to the trauma exposure status of individuals in the non-PTSD group or, conversely, comparing trauma-exposed with non-trauma-exposed individuals, without regard to the PTSD status of individuals in the trauma-exposed group. By making this distinction in our study, we were able to identify a pattern of increasing prevalence of these risk factors from the no trauma to the trauma without PTSD to the PTSD group, which was present for all of the factors examined. In addition, higher rates of both PTSD and trauma in the absence of PTSD were found in African-American women compared with White women, a finding that has been reported in a number of prior studies (Breslau et al., 1998, 2004; Rheingold et al., 2004; Stein et al., 2003).

Trauma exposure, posttraumatic stress disorder, and alcohol dependence

Results from our initial analysis examining risk for AD in women with PTSD versus those who were exposed to trauma but did not develop PTSD and those without a history of trauma indicated that women exposed to trauma—both with and without PTSD—were at elevated risk for AD. Furthermore, they indicated that women with PTSD were at significantly greater risk for developing AD than trauma-exposed women without PTSD, suggesting that there are PTSD-specific effects that act to increase risk for AD. This finding persisted after adjusting for family history of problem drinking and ethnicity (the latter being an important factor to consider given the higher rates of trauma and PTSD and the lower rates of AUDs in African-American vs. White women [Dawson et al., 2004; Falk et al., 2006; Harford et al., 2006]). These findings are closely aligned with those of Danielson et al. (2009), who found that PTSD conferred additional risk for AUDs after accounting for exposure to

physical and sexual assault and witnessing violence, but sexual assault remained an independent predictor of AUDs. These findings are also consistent with those from a study by Breslau and colleagues (1997), in which women's trauma histories were categorized as no trauma, trauma without PTSD, and trauma with PTSD, and elevated rates of alcohol abuse/dependence were found in both trauma-exposed groups. There are, however, as many studies reporting findings that conflict with results from our initial analysis (i.e., Breslau et al., 2003; Epstein et al., 1998; Kilpatrick et al., 1997) as there are with findings that support it. In an effort to identify possible mechanisms underlying the relationship of AD with trauma and PTSD, in our next stage of analysis we examined the potential confounding effects of co-occurring psychiatric conditions on these associations.

The inclusion of MDD, CD, regular smoking, and cannabis abuse in models predicting AD decreased estimates of risk for AD attributable to PTSD and to trauma without PTSD. However, the observed elevation in rates of AD remained significant for both trauma groups—a remarkable finding given the high degree of co-morbidity of AUDs with MDD (Grant et al., 2004; Hasin et al., 2007; Kessler et al., 1997), CD (King et al., 2004; Kuperman et al., 2005; McGue et al., 2001), regular smoking (Cardenal and Adell, 2000; Dawson, 2000; Grucza and Bierut, 2006) and cannabis abuse (Degenhardt et al., 2002; Grant and Pickering, 1998; Stinson et al., 2006). The evidence indicates that commonly co-occurring psychiatric conditions do not fully mediate the association between AD and either PTSD or trauma exposure more generally; traumatic experiences confer risk for AD that cannot be explained by shared risk factors. One frequently proposed model for this association is the tension-reduction hypothesis (Cappell and Greeley, 1987), also referred to as the “self-medication hypothesis,” which posits that elevated rates of problem drinking in trauma-exposed populations result from the use of alcohol to attempt to manage the negative affect associated with trauma (i.e., self-medicate), and in the case of PTSD, to manage symptoms as well. The self-medication model, which has found support in community-based studies (Epstein et al., 1998; Stewart et al., 1998; Ullman et al., 2005), offers a plausible source for the trauma-specific factor (or factors) that increase risk for alcohol-related problems.

The second major finding from the analysis including psychiatric covariates is that the difference in risk for AD between the PTSD and trauma without PTSD groups was no longer statistically significant (despite the higher point estimate for the PTSD group). Results indicate that the apparent PTSD-specific effects observed in the initial analysis could be accounted for by the relatively higher rates of co-occurring psychopathology in women who developed PTSD following a trauma compared with those who did not. The impact of the constellation of risk factors examined in this study on the association between trauma, PTSD, and

AD has been relatively unexplored in the literature, so we cannot make direct comparisons to studies using parallel methodology, but we propose two possible explanations for these patterns of findings. The first is simply that after accounting for a range of possible confounders, we no longer had sufficient statistical power to detect differences between the PTSD and trauma without PTSD group. The second explanation—which is not incompatible with the first—is that the observed difference in risk for AD (evident only at the trend level after adjustment for covariates) is a reflection of the greater distress associated with traumatic events that lead to PTSD. That is, more distressing events are associated with higher rates of psychopathology, including AD, but there is nothing specific to the experience of PTSD (e.g., the desire to suppress such PTSD symptoms as intrusive memories) that influences risk for alcohol-related problems. Exploratory analyses (data not shown)—in which the trauma without PTSD group was broken into (a) no items endorsed on Wave 4 checklist, (b) Criterion A assessed but not met, and (c) Criterion A met—revealed increasingly higher rates of AD across the three subgroups, providing preliminary support for our hypothesis.

Limitations

Certain limitations should be taken into account when interpreting findings from the current study. First, the prevalence of PTSD in our sample is lower than the rates reported in other population-based studies (Breslau et al., 1998; Kessler et al., 1995; Resnick et al., 1993). If this reflects underreporting, some participants with PTSD may have been inaccurately assigned to the trauma without PTSD group, which would have reduced the power to detect differences between the two trauma-exposed groups. Second, psychiatric and substance use data were gathered through retrospective assessments and are therefore subject to bias inherent in retrospective reporting methods, although statistical adjustments for age at the time of AD report minimized this bias with respect to AD diagnoses. Third, by design, the current study assessed risk for the development of AD following trauma exposure, so we cannot draw any inferences about the possible increase in risk for experiencing traumatic events consequent to the onset of AD. Finally, although integrating data from earlier waves of data collection and other sections of the interview with the Wave 4 trauma checklist enhanced accuracy in establishing trauma status, we encountered the same issue regarding determination of PTSD status that exists in studies making exclusive use of trauma checklists: only those individuals who chose an item from the trauma checklist were assessed for PTSD. In assigning individuals meeting criteria for trauma based on supplementary data to the trauma without PTSD group, we made the assumption (potentially erroneous in some cases) that these women did not meet PTSD criteria.

Future directions

Findings from the current study suggest several possible future directions for this line of research. First, given the possibility that varying levels of distress following traumatic events underlie the distinctions in AD risk between PTSD and trauma without PTSD, measures of trauma severity (e.g., chronicity of exposure) are important to include in future studies of the relationship between trauma exposure, PTSD, and alcohol misuse. Second, the generalizability of our findings to men is not known. Gender differences in rates of trauma exposure, PTSD, and conditional probability of developing PTSD following exposure to assaultive trauma (Breslau et al., 1998; Hapke et al., 2006; Kessler et al., 1995; Resnick et al., 1993) suggest that the interrelationships among trauma, PTSD, AD, and co-occurring psychiatric conditions in men may differ substantially from those in women, making this a particularly intriguing direction for future research. Third, future investigations focused on smoking and illicit drug-related outcomes would be highly informative with respect to the specificity of the role of co-occurring psychopathology in the association of trauma and PTSD with AD versus misuse of substances of abuse more generally. Finally, the investigation of common heritable influences on PTSD and AD (which our group is currently pursuing using this sample) may contribute to our understanding of the mechanisms underlying the association between the two disorders.

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