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Alcohol-Involved Assault and the Course of PTSD in Female Crime Victims

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Abstract

Although alcohol use has been associated with increased risk of victimization, little is known about how victim substance use at the time of assault may affect posttraumatic stress disorder (PTSD) symptom development. The present study is a longitudinal examination of substance use on PTSD symptom severity and course. A community sample of female crime victims ($n = 60$) were assessed within 5 weeks of sexual or physical assault with 3 and 6 month postassault follow-ups. Twenty-three participants had consumed alcohol or alcohol/drugs prior to the assault (38%) and 37 had consumed neither alcohol nor drugs. Analyses were conducted using hierarchical linear modeling. Participants who had consumed alcohol had lower initial intrusive symptoms, but their symptoms improved less over time.

Researchers are increasingly recognizing that a substantial proportion of assaults involve alcohol and may differ from assaults where alcohol is not involved (McCauley et al., 2009; Testa, Vanzile-Tamsen, & Livingston, 2004). Prevalence estimates range from half to three quarters of assaults (Abbey, Zawacki, Buck, Clinton, & McAuslan, 2004; Roy-Byrne et al., 2004). Little research has focused on how victim substance use affects posttraumatic stress disorder (PTSD) symptoms over time. The current research addresses these gaps by examining alcohol-involved assaults, crime characteristics, and PTSD.

In studies examining PTSD and substance use disorders, alcohol misuse has been associated with elevated avoidance and hyperarousal symptoms (Back, Sonne, Killeen, Dansky, & Brady, 2003; Read, Brown, & Kahler, 2004) and more severe and chronic symptoms (Kaysen et al., 2006; Zlotnick et al., 1999). However, these studies focused exclusively on consistent drinking behavior rather than on how alcohol use at the time of or just prior to an assault may affect the course of PTSD (Bisby, Brewin, Leitz, Curran, 2009).

Alcohol influences information processing, including encoding new information, and dampens startle reactivity (Bruce, Pihl, Mayerovitch, & Shestowsky, 1999; Curtin, Lang, Patrick, & Stritzke, 1998; Weitemier & Ryabinin, 2003). Alcohol consumption also appears to affect risk appraisal, by decreasing perceptions of risk for assault, concerns about injury, and perceptions of physiological reactivity during an assault (Clum, Nishith, & Calhoun, 2002; Davis, Stoner, Norris, George & Masters, 2009).

Three studies to date have examined the relationship between peritraumatic alcohol use and PTSD, with contradictory results (Kilpatrick et al., 2007; Littleton & Henderson, 2009; Maes, Delmeire, Mylle, & Altamura, 2001). In one study, individuals who were intoxicated at the time of a traumatic event had lower rates of PTSD 7 to 9 months later (Maes et al., 2001). Other studies found no differences in PTSD rates between substance-involved rapes and other rapes (Kilpatrick et al., 2007; Littleton & Henderson, 2009). In these studies, PTSD was assessed retrospectively, making it impossible to examine alcohol's role in the development of PTSD symptoms. The current study examines alcohol-involved assault on PTSD symptoms both within and across individuals over the first 6 months following a physical or sexual assault. We hypothesized alcohol-involved assault would be associated with lower perceptions of assault severity, hyperarousal, and intrusive symptoms.

METHOD

Participants

Female participants in the Seattle metropolitan area were recruited through flyers posted in the community (45.1%), hospitals (4.8%), health and human services (3.2%), and victim service agencies (4.8%), advertisements in local newspapers (35.5%), and word of mouth (8.0%). Flyers stated that the study was examining women's recovery from physical or sexual assault. Inclusion criteria consisted of experiencing a first-degree physical ($n = 38$; experienced injury or felt perpetrator was trying to kill/injure her) or sexual ($n = 22$; completed vaginal, oral, or anal penetrative assault) assault within 5 weeks of the scheduled assessment. Exclusion criteria included current delusions or psychosis and a lack of English literacy.

Interviews were conducted at the University of Washington Medical Center. The project was reviewed and approved by the University of Washington Institutional Review Board, informed consent was obtained from all research participants, and subjects were compensated for participation. Participants were assessed at three periods: the first 2 to 5 weeks postassault (Time 1), at 3 months postassault (Time 2), and at 6 months postassault (Time 3). Of participants assessed at Time 1 ($n = 60$), 83% returned for Time 2 ($n = 50$), and 80% returned for Time 3 ($n = 48$). Seventy-eight percent of participants were seen at all time points ($n = 47$).

The mean age of the sample was 35.6 years ($SD = 9.0$; range = 19–53). Seventy-eight percent had received at least a high school education ($n = 47$). Fifty-two percent of participants were single, 19% were married/cohabiting, 27% were divorced/separated, and 2% were widowed. Seventy percent earned less than \$10,000 annually. Twenty-nine percent were African American, 44% Caucasian, 5% were Asian/Pacific Islander, 14% identified as Native American, and 8% identified as other. Eleven percent of the participants identified as Hispanic/Latina.

Measures

A battery of interviews and self-report questionnaires were administered to participants, of which the following are relevant to this study.

The Clinician Administered PTSD Scale (CAPS; Blake et al., 1995) is a clinician-administered diagnostic interview that measures PTSD and has been found to have excellent psychometric properties. The CAPS was used to assess current PTSD symptom severity at each time point.

The Standardized Trauma Interview (Resick, Jordan, Girelli, & Hutter, 1988) is a clinician-administered structured interview yielding descriptive characteristics about the traumatic

experience. Relevant to the current study were questions regarding injuries sustained and victim appraisal of risk of injury or death (Kaysen, Morris, Rizvi, & Resick, 2005). Perceived threat was measured by summing two questions regarding perception of risk and certainty of harm. Internal consistency, as measured by coefficient alpha, was found to be .61 in our sample. Participants were also asked whether they had consumed alcohol or drugs prior to the assault and what type of substance was consumed.

Timeline follow-back (Sobell & Sobell, 1992) was used to assess alcohol use at every time point. It has good psychometric properties (Sobell, Brown, Leo, & Sobell, 1996). We examined the highest number of standard drinks consumed in the month prior to the first assessment as a covariate. This measure was selected given findings that higher peak drinking has been associated with higher PTSD symptom severity (Kaysen et al., 2007).

Data Analysis

Hierarchical linear modeling (HLM; Raudenbush & Bryk, 2002) was used to analyze change in PTSD symptoms over time following assault, moderated by alcohol-involved assault. PTSD symptom clusters were analyzed separately. The Level 1 model included the repeated-assessments of PTSD symptoms by time since the assault, and the Level 2 model included alcohol prior to the assault (dummy coded; non-alcohol-involved assault = 0, alcohol-involved assault = 1) with peak drinking and perceived severity as covariates. The primary interest focused on the cross-level interaction between alcohol-involved assault and days since the assault. Based on exploratory graphs of the data, the relationship between PTSD symptom clusters and days since the assault was nonlinear. We examined several possible relationships, including linear, quadratic, and natural log transformation. The log transformation yielded a superior fit to the data compared to linear and quadratic models, based on Akaike information criterion (AIC) and Bayesian information criterion (BIC).

The primary equation used in the analyses is as follows, using the composite equation:

$$\text{PTSD}_{it} = B_0 + B_1 (\text{AIA}_i) + B_2 (\text{LOGDAYS}_{it}) + B_3 (\text{AIA}_i * \text{LOGDAYS}_{it}) + B_4 (\text{PEAKDRINK}_i) + B_5 (\text{SEVERITY}) + r_{0i} + e_{it}$$

where i indexes individuals and t indexes time in days since the assault. A random intercept term (r_{0i}) modeled between participant variability in PTSD symptoms, and the Level-1 error term (e_{it}) captured the scatter of the individual data points around the subject-specific regression lines. Hierarchical linear modeling assumes error terms are normally distributed, homoscedastic, and conditionally independent (Raudenbush & Bryk, 2002).

RESULTS

Twenty-one women reported they had consumed alcohol prior to the assault (33%); two had consumed alcohol and an illicit drug (3%). Thus, there were two comparison groups: one group of participants who had consumed alcohol or alcohol and drugs prior to the assault ($n = 23$) and one group who had not consumed either alcohol or drugs prior to the assault ($n = 37$). Of the alcohol-involved assault group, 59.1% had consumed 4 or more standard drinks prior to the assault (range: 1–17), defined as heavy episodic drinking for women (Wechsler, Dowdall, Davenport, & Rimm, 1995).

We examined differences between alcohol-involved and non-alcohol-involved assault participants across assault characteristics, demographics, and prior trauma history. There were no significant differences in education, prior child sexual abuse, or prior adult rape,

between alcohol-involved and non-alcohol-involved assault participants. There also were no differences in rates of physical versus sexual assault between alcohol-involved and non-alcohol-involved assault participants or relationship with the perpetrator (stranger, acquaintance, or an intimate partner). The majority of assaults were by current or ex-partners (74% of alcohol-involved assault, 54% of non-alcohol-involved assault), followed by stranger assaults (13% alcohol-involved assault, 29% non-alcohol-involved assault), and acquaintances (13% alcohol-involved assault, 17% non-alcohol-involved assault). No differences were observed between alcohol-involved assault and non-alcohol-involved assault participants in the number of days prior to each assessment, differential dropout, or number of injuries, although there were higher levels of perceived threat reported by non-alcohol-involved assault ($M = 3.94$, $SD = 2.55$) than alcohol-involved assault ($M = 2.26$, $SD = 2.17$) participants, $p < .01$. There was a significant difference in pretrauma peak drinks per drinking occasion between alcohol-involved assault and non-alcohol-involved assault groups, $F(1, 60) = 11.14$, $p < .01$.

Parameter estimates for the HLM results are presented in Table 1. Non-alcohol-involved assault participants had significantly more intrusive symptoms at the posttrauma assessment (i.e., they had higher intercepts) than did alcohol-involved assault participants. Over time the non-alcohol-involved assault participants had a significantly steeper dropoff in symptoms (i.e., they had steeper, negative slopes). For avoidance and hyperarousal symptoms, there were no group differences or differences in the slope of PTSD symptom improvement over time. Predicted regression lines for each PTSD symptom cluster are shown in Figure 1.

DISCUSSION

The present study examined the impact of victim alcohol use on initial reactions to a sexual or physical assault and on recovery over time. This study extends previous research in several ways. The research on alcohol-involved assault, a relatively new field of inquiry, has begun to address the role of alcohol-involved assault on assault severity, victim resistance behaviors, and on later substance use, but has not generally examined the potential impact of alcohol-involved assault on PTSD (Maes et al., 2001; McCauley et al., 2009).

Similar to the work of Clum and colleagues (2002), we did find differences in perceived assault severity, with the alcohol-involved assault group seeing the assault as less life threatening than the non-alcohol-involved assault group. Moreover, this difference occurred despite other indicators suggesting that the events were comparable (i.e., type of assault, injuries, and perpetrator relationship) thus replicating previous findings of similarities in assault severity regardless of victim intoxication (Testa, Livingston, Vanzile-Tamsen, & Frone, 2003). However, it is important to note other studies have found differing assault characteristics between alcohol-involved assault and non-alcohol-involved assault, such as whether assaults were acknowledged as such, victim resistance strategies, and perpetrator behaviors (Littleton, Grills-Tauchel, & Axsom, 2009; Ullman, Karabatsos, & Koss, 1999).

Alcohol-involved assault was associated with lower initial intrusive symptoms. Acute alcohol administration can dampen stress responses and impair the acquisition of fear memories (Faingold, N'Gouemo, & Riaz, 1998; Nomura & Matsuki, 2008), including intrusive memories in PTSD analogue studies (Bisby et al., 2009), which is consistent with lower perceived severity and lower initial intrusive symptoms endorsed by the alcohol-involved assault women in our sample. These findings highlight the importance of examining whether factors associated with initial symptoms differ from those associated with symptoms over time.

Overall, PTSD symptoms improved for both alcohol-involved assault and non- alcohol-involved assault groups. In our sample, the slope of recovery for intrusive symptoms in the alcohol-involved assault group was significantly less steep over time, suggesting that although these symptoms may initially be less severe in the alcohol-involved assault group, they may not remit as rapidly. This is consistent with animal studies suggesting alcohol is associated with reductions in extinction learning (Lattal, 2007; Nomura & Matsuki, 2008). Thus, alcohol may inhibit initial symptoms, but may be associated with reduced extinction of those learned associations over time (Stephens et al., 2005). Longer-term follow-up with a larger sample is necessary to examine whether these relationships continue.

It is important to consider the study's limitations. Although the sample size was adequate for testing the primary hypotheses, it did limit our ability to test further subgroup analyses such as the role of prior trauma exposure or ongoing trauma on the alcohol-involved assault and PTSD relationship. The small sample size also prevented us from examining whether there was an interaction between the role of alcohol and assault characteristics. Level of intoxication was not assessed during the study. It is possible that the inclusion of participants with low levels of intoxication may have alcohol and PTSD relationships that would have emerged had we focused on women with high blood alcohol levels.

Despite this limitation, this study builds upon the existing literature in a number of ways. By using an acute trauma sample and examining PTSD, this study was able to add to our knowledge of how substances like alcohol may affect the initial presentation of PTSD over time. In conclusion, the present study provides a unique perspective on the impact of alcohol on victim recovery. The results suggest that although alcohol consumed prior to or during an assault may dampen initial perceptions of assault severity and initial intrusive symptoms of PTSD, alcohol is also associated with reductions in symptom improvement over time.

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Figure 1. HLM slopes and intercepts for posttraumatic stress disorder (PTSD) symptoms as a function of the number of days since the index assault and whether the assault involved alcohol.

Table 1

Hierarchical Linear Modeling Results for PTSD Symptoms as a Function of the Log Transformed Number of Days Since Index Assault, Whether the Assault Involved Alcohol (Not Alcohol-Involved vs. Alcohol-Involved), and the Interaction of Those Two Terms, While Controlling for Perceived Threat and Peak Drinks Over Time

	Intrusive symptoms			Arousal symptoms			Avoidance symptoms		
	<i>B</i>	<i>B SE</i>	<i>t</i>	<i>B</i>	<i>B SE</i>	<i>t</i>	<i>B</i>	<i>B SE</i>	<i>t</i>
Intercept	42.76	3.75	11.40***	41.53	4.54	9.13***	39.12	5.55	7.05***
Alcohol-involvement	-24.81	6.79	-3.65***	-6.47	8.46	<1	-1.26	10.63	<1
Days (log transformed)	-6.36	0.87	-7.35***	-5.35	1.08	-4.93***	-4.40	1.13	-3.90***
Perceived threat	1.27	0.43	2.94**	1.74	0.45	3.83***	1.42	0.65	2.17*
Peak drinks	0.02	0.16	<1	0.20	0.19	1.06	0.36	0.26	1.38
Alcohol-Involvement × Days	5.59	1.47	3.79***	1.83	1.88	<1	0.85	2.12	<1

Note. *N* = 60. Alcohol-involvement was dummy coded (0 = *no*, 1 = *yes*). PTSD = Posttraumatic stress disorder.

*
p < .05.

**
p < .01.

p < .001.