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Sexual Revictimization, PTSD, and Problem Drinking in Sexual Assault Survivors

Sarah E. Ullman, PhD^{a,b}

^aUniversity of Illinois at Chicago, 1007 West Harrison Street, Chicago, Illinois 60607-7137, United States

Abstract

Posttraumatic stress disorder (PTSD) and problem drinking are common and often co-occurring sequelae experienced by women survivors of adult sexual assault, yet revictimization may mediate risk of symptoms over time. Structural equation modeling was used to examine data from a 3-wave panel design with a large ($N = 1,012$), ethnically diverse sample of women assault survivors to examine whether repeated sexual victimization related to greater PTSD and problem drinking. Structural equation modeling revealed that child sexual abuse was associated with greater symptoms of PTSD and problem drinking and intervening sexual victimization was associated with greater symptoms of PTSD and problem drinking at both 1 and 2 year follow-ups. We found no evidence, however, that PTSD directly influenced problem drinking over the long term or vice versa, although they were correlated at each timepoint. Revictimization during the study predicted survivors' prospective PTSD and problem drinking symptoms. Implications and recommendations for future research are discussed.

Keywords

sexual assault; child sexual abuse; revictimization; PTSD; problem drinking

1. Introduction

Posttraumatic stress disorder (PTSD) and problem drinking are common consequences of adult sexual assault (ASA) (Walsh et al., 2014). Some studies suggest that PTSD and problem drinking comorbidity can be explained by the self-medication model, which proposes that survivors drink to cope with PTSD symptoms (e.g., Cappell & Greeley, 1987).

^bCorrespondence should be addressed to Sarah E. Ullman at the above postal or email addresses, or by telephone (312-996-6679) or fax (312-996-8355). seullman@uic.edu.

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Contributors

Sarah E. Ullman designed the study and wrote the protocol, conducted literature searches, analyzed the data, and wrote the manuscript and approves of the final manuscript.

Conflict of Interest

The author declares she has no conflicts of interest.

Such drinking can relieve trauma-related distress, but may worsen PTSD symptoms (Volpicelli, Balaraman, Hahn, Wallace, & Bux, 1999) and even lead to chronic PTSD and problem drinking over time (Stewart, Pihl, Conrod, & Dongier, 1998). A test of this hypothesis in sexual assault victims showed that over 1 year sexual revictimization explained changes in PTSD and problem drinking over time (Najdowski & Ullman, 2009), however this finding has yet to be replicated or examined over a longer followup period.

In addition, having a history of child sexual abuse (CSA) is related to more severe PTSD and problem drinking in women ASA survivors (Ullman, Najdowski, & Filipas, 2009). Furthermore, CSA, PTSD, and problem drinking are all associated with risk of revictimization in ASA survivors (e.g., Ullman et al., 2009; Ullman & Najdowski, 2009). Drinking is specifically associated with alcohol-involved sexual assaults rather than forcible assaults (Littleton & Ullman, 2013; Messman-Moore et al., 2013; Testa et al., 2003). The strong association between past victimization and risk for revictimization may be explained by PTSD and problem drinking behaviors that women develop in response to their victimization experiences. Ullman et al. (2009) found that ASA victims with CSA histories reported having more arousal, reexperiencing, and avoidance symptoms, all of which predicted greater problem drinking, which then increased risk of sexual assault. Problem drinking may increase victims' risk of being revictimized in several ways. Drinking reduces women's intentions and abilities to resist unwanted sexual advances (Testa, VanZile-Tamsen, Livingston, & Buddie, 2006). Women incapacitated by alcohol may be targeted by perpetrators for their vulnerability (Kilpatrick, Resnick, Ruggiero, Conoscenti, & McCauley, 2007), and engage in greater drinking to cope (unlike other types of assault), and problem drinking appears to affect revictimization in college women (Messman-Moore et al., 2015). While CSA, PTSD, and problem drinking are risk factors for revictimization, it is less clear how new incidents of sexual victimization influence PTSD and problem drinking over time. In this study, CSA and multiple time points of revictimization are examined in a model to evaluate how these experiences affect PTSD and greater problem drinking in ASA survivors. PTSD is also expected to affect revictimization possibly by increasing vulnerability in survivors and decreasing their ability to protect themselves. While PTSD may relate to problem drinking, we believe any link is actually due to effects of these symptoms on survivors' responses (e.g., drinking to cope) and increased revictimization risk. This analysis extends prior work in a different sample of sexual assault to see whether revictimization continues to affect women's symptomatology over time.

2. Method

2.1 Participants and Procedure

Advertisements in local newspapers and fliers distributed throughout the Chicago metropolitan area (e.g., college/universities, women-oriented businesses, mental health and agencies serving victims of violence against women) invited women aged 18 or older with unwanted sexual experiences since age 14 they had told someone about to participate in a confidential mail survey. Interested women were mailed the initial survey (i.e., Time 1 [T1]) with a cover letter, information sheet describing the study, informed consent form, a list of community resources. 1,863 women (85%) completed the T1 survey, 76% completed the T2

survey, and 54% ($N = 1,012$) completed the T3 survey, after removing those who reported never disclosing an assault). Women received \$25 for completing each survey. All 3 surveys were similar in content and format, except we asked about assaults, PTSD, and drinking in the past year at Waves 2 and 3.

The longitudinal sample was quite ethnically diverse (39.7% Caucasian, 43.8% African American, and 16.5% other; with 13.7% Hispanic ethnicity assessed separately), and women were 18 to 70 years old ($M = 36.24$ years, $SD = 12.39$). Thirteen percent of women had graduated from high school and an additional 78.8% had attended at least some college. Forty-six percent of women were employed and 55.6% of women reported household incomes of less than \$20,000 per year. The 1012 eligible participants who completed three waves of data did not differ demographically from those that were non-completers other than being slightly older ($M = 38$ compared to 35) and having more time pass since their most serious assault ($M = 15.86$ and 13.72 in completers versus noncompleters, likely reflecting age differences). We focused on the 768 (76%) of those reporting past year drinking at wave 1. Compared to nondrinkers, past-year drinkers were more likely to be White, employed, more educated, higher income, but no different on Hispanic ethnicity. Women were treated in accordance with the ethical guidelines of the University of Illinois at Chicago.

2.2 Measures

2.2.1 Sexual victimization—Each type of sexual victimization (i.e., CSA, ASA, revictimization) was assessed dichotomously (*experienced, not experienced*) using the Sexual Experiences Survey-Revised (SES-R; Testa et al., 2004).

On the T1 survey, the SES assessed completed rape, attempted rape, sexual coercion, or unwanted sexual contact *at age 14 years or older* (i.e., ASA) and *before age 14 years* (i.e., CSA). All women in this sample had experienced ASA and 64% had experienced CSA. On the T2 survey, the SES revealed that 36.4% had experienced another ASA since the last survey, as did 31.9% on the T3 survey. Dichotomous variables were used for any SES revictimization.

2.2.2. PTSD symptoms—PTSD symptoms were assessed with the Posttraumatic Stress Diagnostic Scale (PDS; Foa, 1995), a standardized 17-item instrument based on DSM-IV criteria. On a scale ranging from 0 (*not at all*) to 3 (*almost always*), women rated how often each symptom (i.e., re-experiencing/intrusion, avoidance/numbing, hyperarousal) bothered them in relation to an assault in the past year. The PDS has good test-retest reliability for a PTSD diagnosis in victims over 2 weeks ($\kappa = .74$; Foa, Cashman, Jaycox, & Perry, 1997) and was reliable using Cronbach's alpha in our sample. Women completed the PDS at T1 ($M = 18.84$, $SD = 12.24$, $\alpha = .93$), T2 ($M = 17.86$, $SD = 11.67$, $\alpha = .94$), T3 ($M = 17.86$, $SD = 11.67$, $\alpha = .94$).

2.2.3. Problem drinking—Problem drinking symptoms were assessed using the Michigan Alcoholism Screening Test (MAST; Selzer, 1971), a standardized 25-item screening instrument for alcohol abuse and dependence. The MAST items formed a reliable measure of problem drinking in our sample. Past-year problem drinking was assessed at T1 ($M =$

3.42, $SD = 4.34$, $\alpha = .64$), and women responded to the measure at T2 and T3 based on their drinking experiences since the last survey (T2: $M = 2.56$, $SD = 4.09$, $\alpha = .77$ T3: $M = 2.48$, $SD = 3.91$, $\alpha = .75$).

3. Results

Using a structural equation modeling framework, we conducted an observed variables path analysis using AMOS 23 with maximum likelihood estimation to test a cross-lagged, partially mediated model of PTSD and problem drinking continuous symptom measures in ASA survivors. The model included 768 women who reported a sexual assault at Time 1 and completed all three surveys, exceeding the suggested ratio of 10 cases for each model parameter (Kline, 1998). Missing data were handled by estimating means and intercepts in AMOS 23. Measures of PTSD symptoms were univariate normal with skew less than 3 and kurtosis less than 3 (Kline, 1998), but problem drinking had high kurtosis (T1 = 3.38, T2 = 11.61, T3 = 10.93). From T1 to T3, 24- 38% scored 0 indicating no problem drinking, with fewer problem drinkers over time. However, we used untransformed variables because the measure was not too skewed (T1 = 1.89, T2 = 2.92, T3 = 2.87), results were the same using transformed variables, and effects of violations of normality assumptions regarding kurtosis are minimal in larger samples (Tabachnick & Fidell, 2001).

No first-order bivariate correlations was above .80 (see Table 1), indicating that multicollinearity across variables was not a problem (Kline, 1998). Because of significant correlations, PTSD and problem drinking were correlated in the model within each wave for all three time points.

Several models were tested in arriving at the final two models described below to arrive at the best fitting model to describe the data: a) fully mediated, b) direct effects only, c) direct and indirect effects, and models both with and without cross paths between PTSD and drinking.

We tested a fully mediated model of the relationships, of CSA, PTSD, and drinking with sexual revictimization mediators (dichotomous no/yes measures) across the three time points to determine whether revictimization helped to explain PTSD and drinking over 3 years. This model had adequate fit, $\chi^2 (17, 768) = 77.38$, $p = .000$ (IFI = .97, NFI = .97, and RMSEA = .068). As predicted, CSA was associated with greater T1 PTSD symptoms and problem drinking. Further, the effect of CSA on sexual revictimization between surveys was partially mediated by T1 symptoms of PTSD and problem drinking. After controlling for survivors' symptoms and the correlation between them within each time point, revictimization appeared to partially mediate the path from PTSD to future PTSD, yet did not mediate the path between drinking and future drinking.

We then compared this model to one with added cross-paths of PTSD predicting drinking and drinking predicting PTSD. Contrary to self-medication model predictions, controlling for revictimization, earlier PTSD did not have a direct effect on later problem drinking, and contrary to the hypothesis that greater problem drinking leads to more severe PTSD symptoms, problem drinking did not have a direct effect on later PTSD. This pattern of the

nonsignificance of cross-paths (PTSD-drinking and drinking to PTSD) held across T1-T2 and T2-T3, so the final model did not include these paths (see Figure 1). It is important to note that we did include correlated error terms in our model (not shown in Figure) for PTSD and drinking within each timepoint and those associations were statistically significant at each wave (T1: $b = .29$, T2 $b = .13$, T3 $b = .23$, all significant $p < .001$).

4. Discussion

This is the first 3-year longitudinal study examining the role of revictimization, PTSD, and problem drinking in ASA victims. Although PTSD and problem drinking were significantly correlated at each time point, we found no evidence that PTSD directly influenced problem drinking over the long term. While this does not support the self-medication model, it fits with other research showing null effects of PTSD on prospective heavy or problem drinking (e.g., Testa, Livingston, & Hoffman, 2007; Ullman & Najdowski, 2009).

While PTSD and problem drinking were correlated at the bivariate level, it appears that revictimization is more important in predicting PTSD over time at least in ASA victims. Problem drinking also did not have a direct effect on PTSD symptoms over time. It appeared that PTSD influenced prospective problem drinking in sexually revictimized women in an earlier 2-wave study on a different sample (Ullman & Najdowski, 2009). In addition, the sequelae of CSA and ASA appeared to increase women's risk for experiencing subsequent sexual victimization, which may further exacerbates those same symptoms and also increases women's risk of developing other symptoms (Ullman & Najdowski, 2009). However, perhaps timing of the associations was not fully captured here and PTSD affects drinking over a shorter time period. This may also explain why revictimization did not consistently predict drinking at the same time point when PTSD was in the model.

The effect of CSA on prospective PTSD was mediated by revictimization, but, of interest, the effect of CSA on revictimization was also indirect, mediated by T1 measures of PTSD. Our results corroborate a wealth of other research showing that PTSD (and problem drinking to a lesser extent) are risk factors for revictimization (Ullman & Najdowski, 2009), and suggest that these are important assault outcomes that may help to understand victims' risk for revictimization. Future research should examine what leads to repeated sexual revictimization and whether there are common factors to ASA victims or a subgroup of women at higher risk, such as those with parental CSA and perhaps histories of intimate partner violence as adults.

These findings show that proximal assaults (i.e., ASA) rather than distal assaults (i.e., CSA) are more important for understanding women's vulnerability for future sexual victimization (e.g., Gidycz, Coble, Latham, & Layman, 1993; Himelein, 1995) and psychological outcomes (Nishith, Mechanic, & Resick, 1990; Ullman & Najdowski, 2009). Effects of earlier traumas like CSA may be transmitted via revictimization. All of the women in our sample experienced ASA, so it was not possible to examine the relative contributions of CSA and ASA to subsequent revictimization or prospective PTSD or problem drinking. We did observe that revictimization only predicted the subsequent wave sequelae, but not that in

later waves. This suggests that interrupting the revictimization cycle and associated PTSD, is critical to reducing the cumulative consequences of sexual assault.

This study was limited by having 3 waves of data collected from survivors who varied at baseline in time since they had been sexually assaulted. This large sample was diverse in terms of ethnicity and socioeconomic status and included survivors who are hard to reach with traditional random sampling methods (e.g., women without telephones, in shelters). In addition, many survivors experienced new ASAs over the 3-year study period. This is a sample of survivors who disclosed to someone and therefore may differ as those with more stereotypical assaults, more distress, less self-blame, and higher likelihood of acknowledging their experiences as assault or rape are more likely to disclose (Kilpatrick et al., 2007). However, this should not bias the sample very much as most survivors tell someone at some point following sexual assault. Despite variation among women in this nonrepresentative community sample, the model predicting prospective symptomatology fit the data well. It is possible that PTSD would show a link with problem drinking over a shorter time frame than one year in sexual assault victims, so this should be evaluated in future studies. Theories explaining comorbid PTSD and problem drinking in ASA survivors should account for repeated sexual victimization.

Using the MAST to measure problem drinking in our nonclinical sample is a limitation, although Shields, Howell, Potter, and Weiss (2007) found that the MAST is generally an acceptable measure in most research situations. Problems associated with this measure may result from its reliance on responses to face valid items: Participants may not answer honestly regarding the extent of their problem drinking. Also, women's self-reports on this measure may have reflected distress rather than actual problem drinking, which, as suggested by Stewart et al. (1998), may explain the link between PTSD and substance use.

This study suggests intervening to reduce sexual revictimization is most important as it is those new experiences that best predict continued PTSD and to a lesser extent problem drinking in ASA victims. Assertiveness and self-defense training appear to be possible solutions for helping women to avoid future assaults (see Gidycz & Dardis, 2014 for a review). Future longitudinal research should examine how specific coping strategies (e.g., drinking to cope, Kaysen et al., 2006; Messman-Moore et al., 2015; Ullman & Najdowski, 2009) and other modifiable psychosocial factors relate to PTSD and problem drinking to reduce these outcomes in women survivors. Specifically, other factors such as PTSD chronicity, illicit drugs (overlapping problem drinking, but much less prevalent in our sample), intimate partner violence, and other psychiatric disorders should be examined in future research. It is important to note that we measured all revictimization, not just substance-involved revictimization which may have influenced the results and lessened the likelihood of finding a link of between revictimization and problem drinking, as shorter time intervals show such associations in college women (Messman-Moore et al., 2015). Research should also seek to understand exactly how these outcomes influence women's likelihood of experiencing further assault (e.g., impaired risk judgments) and how interventions can be developed to decrease women's vulnerability for experiencing any sexual assault. We are currently doing analyses to examine prospective predictors of revictimization in this sample

(Relyea & Ullman, under review), but other studies are also needed using representatively sampled victims.

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Highlights

Child sexual abuse was related to greater PTSD and problem drinking

Revictimization predicted PTSD and problem drinking over time

PTSD did not directly affect problem drinking over time

Revictimization mediated the effect of child sexual abuse on PTSD

Child sexual abuse mediated the effect of revictimization on PTSD

Revictimization predicted PTSD over time but not problem drinking

Note. ** $p < .005$, *** $p < .001$. Significant correlated error terms between PTSD and Problem drinking at each wave not shown

Figure 1.
Final Model of the Relations among CSA, Sexual Revictimization, PTSD Symptoms, and Problem Drinking

Table 1

Bivariate correlations between Sexual Victimization, PTSD, and Problem Drinking Symptoms ($Ns = 645-728$)

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. CSA	—	.29***	.18***	.19***	.26***	.13***	.12**	.22***	.14***
2. T1 PTSD symptoms		—	.31***	.20***	.66***	.22***	.20***	.57***	.20***
3. T1 Problem drinking			—	.18***	.22***	.66***	.16***	.26***	.56***
4. T2 Revictimization				—	.27***	.20***	.46***	.28***	.19***
5. T2 PTSD symptoms					—	.23***	.28***	.74***	.22***
6. T2 Problem drinking						—	.19***	.25***	.64***
7. T3 Revictimization							—	.32***	.22***
8. T3 PTSD symptoms								—	.32***
9. T3 Problem drinking									—

Note:

**
 $p < .01$

 $p < .001$