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Does Sexual Victimization Predict Subsequent Alcohol Consumption? A Prospective Study among a Community Sample of Women

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Abstract

Although rape and sexual victimization experiences have been hypothesized to contribute to subsequent heavy drinking and alcohol problems among women, little prospective evidence exists. The present prospective study examined whether sexual victimization contributes to subsequent heavy drinking among a community sample of women, 18 – 30 years of age (n = 927). Using three waves of data, 12 months apart, we examined the impact of T1 sexual victimization on T2 heavy drinking, and of T2 sexual victimization on T3 heavy drinking. There were significant bivariate differences between sexually victimized and non-victimized women on heavy drinking both concurrently and prospectively. However, after controlling for prior heavy drinking and demographic variables, most differences disappeared. We also tested the hypothesis that Post-Traumatic Stress Disorder (PTSD) Symptoms would mediate the relationship between T2 sexual victimization and T3 heavy drinking. Although T2 sexual victimization predicted T2 PTSD symptoms, PTSD did not contribute to subsequent heavy drinking. Findings suggest that heavy drinking is relatively stable over time and that sexual victimization does not make a substantial independent contribution to heavy drinking among women in the general population.

Keywords

alcohol abuse; alcohol drinking patterns; posttraumatic stress disorder; rape; victimization

There is a well-documented positive association between women's alcohol consumption and their experiences of sexual victimization (see Abbey, Zawacki, Buck, Clinton, & McAuslan, 2004; Testa & Parks, 1996 for reviews). However, determining the directionality of this relationship is challenging. Drinking alcohol may increase women's vulnerability to sexual assault either directly, by impairing their risk perception and resistance, or indirectly, due to its association with risky settings. Alternatively, women who have experienced sexual victimization may subsequently increase their drinking in response to the trauma (see Stewart, 1996; Stewart, Pihl, Conrod, & Dongier, 1998). Substances such as alcohol, which have arousal- and anxiety-reducing properties, may be used as a means of self-medication for the trauma symptoms that result from sexual victimization (see Conrod & Stewart, 2003). Many of the studies that have considered the association between women's drinking and sexual victimization have been cross-sectional, and therefore unable to clearly distinguish between

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these possibilities. In contrast, the current prospective study was designed to examine whether experiencing adult sexual victimization contributes to subsequent alcohol consumption among a community sample of women.

Many retrospective studies report higher levels of current drinking among women reporting a history of prior sexual victimization in childhood, adolescence, or adulthood (Bensley, Eenwyk, & Simmons, 2000; Champion, Foley, DuRant, Hensberry, Altman, & Wolfson, 2004; Koss & Dinero, 1989; McMullin & White, 2006; Wilsnack, Vogeltanz, Klassen, & Harris, 1997). Although these findings are consistent with the notion that sexual victimization results in increased drinking, other interpretations are possible. For example, some studies find that the relationship between victimization and alcohol consumption disappears when controlling for stressful life events (Horwitz, Widom, McLaughlin, & White, 2001; see Rind, Tromovitch, & Bauserman, 1998 for a review). In addition, few studies control for pre-assault drinking, and thus cannot rule out the possibility that women victimized in adulthood were heavier drinkers before the assault.

To establish that victimization experiences lead to increased heavy drinking or alcohol problems, prospective designs are necessary, ideally, controlling for prior levels of drinking. However, few studies have taken this approach. Testa and Livingston (2000) examined whether sexual victimization reported at Time 1 predicted alcohol use or problems reported at Time 2, 12 months later, after controlling for Time 1 levels of these alcohol variables. They failed to find a relationship of victimization to subsequent alcohol use or alcohol problems; however, the sample size was small and limited to women who initially drank moderately to heavily. Kilpatrick et al's (1997) National Women's Study, based on a nationally representative sample of 3006 women, has been widely cited as supporting the notion that sexual victimization contributes to subsequent alcohol problems among women. After controlling for initial alcohol abuse or dependence, experiencing physical or sexual assault during the 24 month follow-up period was positively associated with past year alcohol abuse/dependence reported at the end of the 24 month follow-up period. Although findings are consistent with the notion that victimization experiences contribute to subsequent drinking problems, because the reporting period for alcohol abuse overlapped with the period in which victimization occurred, it is possible that in some cases drinking problems may have preceded victimization. Moreover, the impact of sexual assault was not examined separately from the impact of physical assault, which was considerably more common. Finally, Kilpatrick et al. did not examine the impact of victimization on alcohol consumption per se, leaving open the possibility that increased alcohol problems among recent victims may reflect a greater willingness of victims to attribute interpersonal problems to alcohol use, and not necessarily increased drinking following victimization.

The current study examined the association between the occurrence of adult sexual victimization and subsequent heavy episodic drinking in a large household sample of young women, addressing some of the limitations of prior research. We focused on young women because compared to older women, young women are at elevated risk of sexual victimization (Bureau of Justice Statistics, 1997) and have higher rates of substance use (Midanik & Clark, 1994). Women who experience sexual victimization are likely to have been heavier drinkers initially, reflecting increased vulnerability to sexual assault associated with heavy drinking (Mohler-Kuo, Dowdall, Koss, & Wechsler, 2004; Testa, VanZile-Tamsen, & Livingston, 2007). Thus, to determine whether victimization results in a change in subsequent heavy drinking, it is necessary to control for pre-assault alcohol consumption. We hypothesized that after controlling for initial levels of heavy episodic drinking, victimization will predict increased drinking over the subsequent year.

Because heavy drinking appears to be higher among younger, Caucasian, and unmarried women (Caetano, Ramisetty-Mikler, Floyd, & McGrath, 2006) and lower among those with children (Testa & Leonard, 2001), we controlled for these demographic factors. In addition, to determine whether effects on drinking are specific to sexual victimization, we controlled for the effects of other traumatic events, including intimate partner violence (IPV). Negative life events have been positively associated with alcohol consumption in adult and adolescent samples (Cooper, Frone, Russell, & Peirce, 1997) and IPV victimization has been associated prospectively with women's substance use (Martino, Collins, & Ellickson, 2005; Testa & Leonard, 2001).

A secondary goal was to explore whether consideration of trauma symptoms improves prediction of women's drinking. Although rates of subsequent substance use disorders are higher among individuals previously exposed to trauma relative to those not so exposed (e.g., Breslau, Davis, Peterson & Schultz, 1997), not everyone who experiences rape or other trauma develops such a disorder. Stewart (1996) suggests that post-traumatic stress disorder (PTSD) symptoms play a key role in the development of subsequent alcohol use disorders and that prediction of these disorders may be improved by considering PTSD symptoms. Supportive of this contention, Chilcoat and Breslau (1998) found that people with a history of PTSD were more likely to subsequently develop drug abuse or dependence relative to those not exposed to a traumatic event; however, exposure to trauma without PTSD did not increase risk of drug abuse/dependence. Numerous studies have established that rape is positively associated with the development of PTSD among women (e.g., Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Saunders, Kilpatrick, Hanson, Resnick, & Walker, 1999; Valentiner, Foa, Riggs, & Gershumy, 1996). Moreover, PTSD precedes alcohol use disorders in most cases and severity of PTSD predicts severity of substance use disorder (see Stewart & Conrod, 2003 for a review). Studies using cross-sectional data suggest that PTSD symptoms mediate the relationship between sexual victimization and alcohol use (Miranda, Meyerson, Long, Marx & Simpson., 2002) or abuse (Epstein, Saunders, Kilpatrick, & Resnick, 1998). As a more rigorous test of mediation, we used prospective data to test the hypothesis that the effect of sexual victimization on subsequent heavy drinking is mediated via increased PTSD symptoms.

2. Method

2.1 Participants

Random digit dialing of households in the Buffalo, NY area, between May 2000 and April 2002, was used to identify women 18 – 30 years of age. The sampling frame contained all telephone exchanges in the city of Buffalo and most from surrounding Erie County, excluding areas most geographically distant from downtown Buffalo where the initial interview was conducted. In-person interviews were completed with 1,014 women, or 61% of eligible women identified, a completion rate similar to that of surveys conducted solely by telephone (e.g., Simon et al., 2001; Welte, Barnes, Wieczorek, Tidwell, & Parker, 2001). The sample appeared to be a good representation of the local population from which it was drawn. For example, the sample was 75.3% Caucasian and 16.9% African American, with small percentages of Hispanic (3.2%), Asian (1.6%), Native American (.7%), and women of mixed or other background (2.4%). The expected percentages of Caucasians and African-Americans for the area, based on 2000 census data, are 78.1% and 16.5%, respectively. Median household income for the sample was about $35,000, compared to $36,498 for the area. Most participants had graduated from high school (95%), compared to 89% of young women for the area as a whole. Although college housing was not included in our sampling frame, 39.8 % were enrolled in higher education (32.6% in college and 7.2% in graduate school), compared to 35.9% of similar aged women in the county. At Time 1 (T1), most were unmarried (76% never married, 3% divorced or legally separated), with an average age of 23.76 (*SD* = 3.71) years.

Of the 1,014 women who completed T1, 927 (91.4%) completed all 3 waves and are included in subsequent analyses. Non-completers did not differ from completers on age, income, marital status, childhood sexual abuse, adult sexual victimization, alcohol or drug abuse, or sexual risk behavior. However, non-Caucasians were more likely to be non-completers (16.5%) than Caucasians (5.7%), χ^2 (1) = 28.63, $p < .001$.

2.2 Procedure

The study was conducted in compliance with the University Institutional Review Board. Eligible women were asked to participate in a longitudinal study of women's social experiences, consisting of three waves of data collection, 12 months apart. Initial participation involved a two-hour session conducted at the Research Institute on Addictions (RIA), for which participants were paid $50. Upon arrival at RIA, study procedures were explained and written informed consent was obtained. T1 data were collected using both a computer-assisted self interview (CASI) and a confidential personal interview with a trained female interviewer. At T2 and T3, questionnaire booklets were mailed to participants' homes and returned in a postage-paid envelope. Measures were similar to those used at T1 but focused on the past 12 months. On average, T2 booklets were completed 366.40 (*SD* = 14.38) days after the T1 interview and T3 booklets were completed 371.02 (*SD* = 28.28) days after completion of T2. Women were sent a $50 check upon receipt of the completed questionnaires.

2.3 Measures

2.3.1 Adult Sexual Victimization—Sexual victimization was assessed at each Wave using a modified version of the Sexual Experiences Survey (SES, Koss, Gidycz, & Wisniewski, 1987), with demonstrated external validity (Testa, VanZile-Tamsen, Livingston, & Koss, 2004). The SES consists of 11 behaviorally specific items that assess unwanted sexual contact, verbally coerced intercourse, attempted rape, and rape resulting from force or incapacitation (e.g., from alcohol or drugs). For purposes of analyses, women who responded positively to one or more items were coded as experiencing sexual victimization. Women who reported an experience of completed or attempted sexual intercourse due to force or incapacitation were classified as experiencing rape.

At T1, women were asked about sexual victimization experiences occurring since age 14. Women who responded positively to any item subsequently participated in a face-to-face interview regarding the most recent incident of sexual assault. As part of this interview, women were asked the date of this incident, allowing us to determine whether sexual victimization had occurred within the past 12 months. At T2 and T3, we used the same 11 SES items to assess the occurrence of sexual victimization over the past 12 months.

2.3.2 Childhood Sexual Abuse—At T1, women were asked a series of 8 items, adapted from Whitmire, Harlow, Quina and Morokoff (1999), regarding unwanted sexual touching and intercourse experiences occurring before age 14. Women who reported at least one unwanted sexual experience before age 14 were considered to have experienced childhood sexual abuse (CSA).

2.3.4 Intimate Partner Violence—At each assessment, women's experiences of intimate partner violence (IPV) were assessed using the Revised Conflict Tactics Scales (CTS-2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996). Women responded yes or no to a series of items assessing minor and severe physical violence perpetrated by their intimate partners. At T1, we assessed IPV within the past year, as well as over the lifetime. At T2 and T3 we assessed IPV experiences over the past 12 months.

2.3.5 Traumatic Events—Following procedures used by Resnick, Kilpatrick, Dansky, Saunders, and Best (1993), at T1, women were asked whether at any time in their lives they had experienced any of seven traumatic events ("Criterion A" events) including natural disaster, serious accident, physical assault, and witnessing of someone seriously injured or killed. For each event they had experienced, women were asked their age at the time, allowing us to determine whether any events had occurred in the past year. At T2 and T3, women were asked whether any of these events had occurred in the past 12 months. Women were assigned a score based on how many traumatic events they had experienced, ranging from 0 to 7. This measure allowed us to control for the potential effects of traumatic events other than sexual assault on subsequent alcohol consumption.

2.3.6 Post-Traumatic Stress Disorder Symptoms (PTSD)—At T2, PTSD symptoms were assessed using The National Women's Study (NWS) PTSD Module (Resnick et al., 1993). The respondent was asked to indicate whether there has been a period of one month or more in which she experienced each of a series of 21 symptoms of PTSD corresponding to DSM-IIIR criteria. An advantage of this approach is that respondents are not required to link symptoms with events, facilitating assessment of symptoms among individuals who may have experienced multiple traumatic events. Items assess symptoms of avoidance ("You deliberately tried very hard not to think about something that happened to you"), hyperarousal ("unexpected noises startled you more than usual"), and re-experiencing ("you felt a lot worse in a situation that reminded you of something that happened in the past"). Cronbach alpha was .91 and subscales were intercorrelated from .76 – .82.

2.3.7 Alcohol consumption—At each assessment, women were asked a series of questions about their alcohol use, allowing us to characterize it in terms of quantity, frequency, and maximum consumption. We focused on two primary outcome measures that we believe are most likely to reflect heavy drinking in response to a stressor. First, we assessed frequency of heavy episodic drinking (HED), using two questions: frequency of consuming five or more drinks on a single occasion in the past 12 months, and frequency of drinking until intoxicated in the past 12 months. Both items were assessed on a six-point scale ranging from 0 (*never*) to 5 (*5 or more days per week*). Because these items were highly correlated at each time period (r's = .69 – .73), they were averaged to form a single measure of HED. Women who reported no drinking during the past 12 months were assigned a score of 0 on this item. We also asked women to indicate the maximum number of drinks per occasion that they might consume, ranging from 1 (*2 or fewer drinks*) to 10 (*22 or more drinks)*. Women who abstained completely were coded 0 and the three highest categories (16–18, 19–21, and 22 or more) were combined due to low frequencies [1].

2.3.8 Alcohol Abuse and Dependence—Lifetime alcohol abuse and dependence were assessed at T1 using a computer administered version of the DIS, based on DSM-IV criteria (Robins, Cottler, Bucholz, & Compton, 1997).

2.3.9 Demographic Variables—Age and ethnic background were assessed at T1, with ethnicity classified as either Caucasian (1) or non-Caucasian (0). We assessed marital status and parenting status at each time period. For analysis purposes, women were classified as

[1]Due to an error in skip patterns at T2, women who reported drinking less than monthly for at least 6 months in a row (n = 199) were skipped out of the remaining alcohol consumption questions. Although these women tended to be light and infrequent drinkers relative to the rest of the sample, the majority of them (83.4%) reported some drinking at T1, thus, it did not seem appropriate to assume that these women were abstaining at T2. Because alcohol consumption measures were highly correlated across Waves for the remainder of the sample (r's from .65 – .70 for drinking frequency, average quantity, maximum quantity, and frequency of heavy episodic drinking from T1 to T2), we substituted T1 heavy drinking data for the missing T2 data for these women. To ensure that this substitution was not influencing results, we repeated all analyses omitting these women. No findings were altered.

married (1) or not married (0). Women who had given birth and were living with the child were classified as parents (1), all others as not parents (0).

3. Results

3.1 Bivariate Associations between Sexual Victimization and Heavy Drinking

As shown in Table 1, as expected, women who reported rape at T1 reported higher levels of heavy episodic drinking and maximum drinks per occasion both concurrently (at T1) and over the following 12 months (at T2). Similarly, women who reported rape at T2 reported heavier drinking at both T2 and T3. The pattern of means was similar for women who experienced any sexual victimization versus no victimization. Although suggestive of a relationship between sexual victimization and heavy drinking, multivariate and prospective analyses are necessary to examine the independent effect of victimization after controlling for the effects of prior drinking and other potential confounding variables.

3.2 Does Lifetime Sexual Victimization Predict T1 Heavy Drinking?

First, we examined whether a history of prior rape, reported at T1, is associated with current HED and maximum drinks per occasion at T1, after controlling for demographic variables. Correlations among T1 predictors and T2 outcome variables are presented in Table 2[2]. Demographic variables and childhood sexual abuse (CSA) were entered on the first step. As expected, being Caucasian, single, unmarried, and childless were associated with higher levels of both measures of heavy drinking at T1, as was CSA (see Table 3). Lifetime rape experiences, entered on the second step, contributed significantly to prediction of frequency of HED, F (1, 903) = 5.02, $p < .03$. However, with the addition of lifetime physical violence and number of traumatic events on the third step, rape no longer had an independent effect on HED. Lifetime rape improved prediction of maximum drinks per occasion, F (1, 904) = 17.00, $p < .001$ and remained a significant predictor in the final equation. The pattern of results was identical when any sexual victimization was used as the predictor in place of rape.

3.3 Does T1 Sexual Victimization Predict T2 Heavy Drinking?

Although the first set of regression analyses reveal an association between prior adult sexual victimization and current heavy drinking after controlling for demographic variables, these analyses involve cross-sectional data and do not control for pre-victimization drinking. As a more rigorous test of the impact of victimization on subsequent heavy drinking, we examined the impact of T1 victimization on T2 drinking, controlling for T1 drinking (see Table 4). T1 drinking and the demographic variables were entered on the first step; T1 rape was entered on the second step; and T1 IPV and traumatic events were entered on the final step to determine whether any impact of sexual victimization remained after accounting for these other experiences. After controlling for strong effects of T1 HED, β = .70, T1 rape made a modest though significant contribution to prediction of T2 HED, F (1, 914) = 4.41, $p < .05$. However, when IPV and other traumatic events were entered on the third step, the effect of rape became non-significant. After controlling for T1 maximum drinks, β =.72, T1 rape did not improve prediction of T2 maximum drinks, F (1, 915) = .00. Results were similar when T1 any sexual victimization was substituted for T1 rape.

The weak effects of sexual victimization on later drinking may result from the fact that T1 victimization occurred, on average, 4.42 (SD = 3.63) years prior to T1. Thus, sexual victimization may have resulted in increased drinking immediately after the event, but its effects may have dissipated over time. To address this concern, we repeated the above analysis

[2]The pattern of correlations among T2 predictors and T3 outcomes (not shown) was similar.

substituting measures of T1 past year rape, T1 past year IPV, and T1 past year traumatic events in place of the lifetime measures. After accounting for the strong effects of T1 heavy drinking, neither T1 past year rape nor T1 sexual victimization contributed to T2 HED or maximum drinks per occasion. Likewise, past year IPV and past year traumatic events did not contribute to prediction of T2 HED or maximum drinks in any equation.

3.4 Does T2 Sexual Victimization Predict T3 Heavy Drinking

We used a similar strategy to examine whether past year sexual victimization reported at T2 predicted heavy drinking at T3 (see Table 5). T2 demographics and T2 drinking were entered on the first step; followed by T2 rape on the second step, and other T2 traumatic experiences on the third step. After controlling for the significant effects of T2 HED, T2 rape did not predict T3 HED, F (1, 915) = 2.11, p = .15; nor did the other trauma variables entered on the third step. However, T2 rape improved prediction of T3 maximum drinks per occasion, F (1, 916) = 4.06, $p < .05$ and remained significant after entering T2 traumatic events and T2 IPV on the third step. Analyses were repeated using any T2 sexual victimization as the predictor; it was not significant in either equation.

As a simple, graphic way of illustrating the extent to which T2 sexual victimization results in increased heavy drinking at T3, we classified women according to whether their maximum drinks per occasion increased from T2 to T3, decreased from T2 to T3, or stayed the same. Of the 29 women who experienced rape at T2, 9 (31.0%) increased their maximum drinks per occasion, 11 (37.9%) decreased, and 9 (31.0%) remained the same. The proportion of victimized women in these three categories did not differ from the proportion of non-victimized women in these categories, χ^2 (2) < 1. Thus, despite a significant effect of T2 rape on T3 maximum drinks per occasion, only a minority of victimized women reported consuming more drinks per drinking occasion at T3 compared to T2.

3.5 Trauma Symptoms as a Mediator

We hypothesized that the relationship between sexual victimization and subsequent heavy drinking would be mediated by PTSD symptoms. T2 rape was not associated with T3 HED, hence we could not test for mediation. However, we tested whether the relationship between T2 rape and T3 maximum drinks was mediated via T2 trauma symptoms. First, we regressed T2 current PTSD symptoms on T2 rape, controlling for all of the demographic variables and other trauma variables that were entered in the equation predicting T3 maximum drinks. As expected, T2 rape predicted T2 trauma symptoms β = .13, $p < .001$. However, when T2 PTSD symptoms were entered on the final step of the regression equation predicting T3 maximum drinks (see Table 5), we found no significant effect of PTSD symptoms, β =−.002, p = .94, nor any corresponding reduction in the direct effect of rape on T3 maximum drinks. Thus, the modest effect of T2 rape on T3 maximum drinks per occasion was not mediated via PTSD symptoms.

3.6 Are Effects of Trauma Limited to Women with Alcohol Use Disorders?

Traumatic events may trigger substance use primarily among women who have substance use disorders and use alcohol as a means of coping with trauma and stress (e.g., Hien, Cohen & Campbell, 2005; Saladin et al, 2003). To explore this hypothesis we repeated the analyses examining the effects of T1 victimization on T2 heavy drinking, and examining T2 victimization on T3 heavy drinking, restricting the sample to women who met DSM-IV criteria for lifetime alcohol abuse or dependence at T1 (n = 213). After accounting for the strong effects of prior heavy drinking (β range .58 – .67), we found no evidence that experiences of rape or sexual victimization predicted subsequent HED or maximum drinks among this subsample at either T2 or T3.

4. Discussion

The current study provides a rare, prospective examination of the independent contribution of adult sexual victimization to subsequent heavy drinking among a community sample of young women. Consistent with many prior studies, baseline (T1) heavy drinking was positively associated with baseline reports of childhood sexual abuse, sexual victimization since age 14, and lifetime history of intimate partner violence, after controlling for demographic factors known to influence drinking. We also found cross-sectional associations between sexual victimization and heavy drinking at T2 and T3. Although these findings are consistent with the hypothesis that victimization contributes to heavy drinking, it cannot be determined based on these cross-sectional data whether T1 heavy drinking occurs in response to prior victimization or whether heavy drinking preceded or perhaps contributed to the victimization. Consistent with the latter possibility, heavy drinking is known to be a risk factor for subsequent IPV (Martino, Collins & Ellickson, 2004; Testa et al., 2003) and for some types of sexual victimization (Combs-Lane & Smith, 2002; Testa et al., 2007).

As a more rigorous test of the hypotheses that sexual victimization contributes to subsequent heavy drinking we used the conservative approach of controlling for prior drinking level. Using this approach, we found little evidence supporting the independent role of sexual victimization in prospective prediction of heavy drinking, over two time periods. Despite the relatively young age of the sample, heavy drinking was quite stable over time, even among women with alcohol abuse or dependence (see Kerr, Fillmore, & Bostrom, 2002). Consequently, prior levels of heavy drinking explained nearly all of the variance in heavy drinking at the next time point, with rape and other traumatic experiences having little or no detectable impact.

Although we failed to find a strong impact of sexual victimization on subsequent drinking, this does not mean that these experiences had no psychological impact. As expected, rape was positively associated with higher levels of PTSD symptoms, suggesting that sexual victimization is indeed traumatic. Yet, higher levels of symptomatology were not associated with subsequent heavy drinking, suggesting that for the most part, women were not self-medicating by increasing their alcohol consumption. Because participants were recruited from households in the community, they were generally high functioning. Victimized women may have benefited from social support and from a repertoire of active coping strategies that kept them from resorting to heavy drinking to deal with their trauma. We acknowledge, however, that because of the fairly long intervals between assessments (12 months), short term periods of increased drinking immediately following victimization are likely to have gone undetected. Moreover, measures of drinking may have been insufficiently sensitive to detect change. Finally, although effects of victimization on subsequent heavy drinking were not readily apparent among the sample as a whole, it is possible that sexual assault served as a trigger for increased heavy drinking for a limited number of vulnerable women. For example, sexual assault survivors who subsequently develop drinking problems are more likely to view drinking as a means of coping and to have received negative social reactions from friends (Ullman, Filipas, Townsend, & Starzynski, 2006). Unfortunately, we did not assess either of these potentially important moderating variables. We did, however, examine whether the effects of rape on subsequent heavy drinking were limited to women who met diagnostic criteria for alcohol abuse or dependence and failed to find support.

Because of the absence of other prospective studies of the impact of sexual victimization on later heavy drinking, it is difficult to know whether these findings are an aberration or whether indeed, there is little relationship between sexual victimization and subsequent heavy drinking within general population samples of women. Among clinical samples, the substantial comorbidity of PTSD and substance abuse, and extremely high rates of prior sexual assault among women in substance abuse treatment (e.g. Dansky, Saladin, Brady, & Kilpatrick,

1995; Miller, Downs, & Testa, 1993), have led to research aimed to understand the underlying processes (e.g., Saladin et al., 2003) and to the development of integrated treatments (e.g., Brady, Back & Coffey, 2004). Our findings do not negate the importance of these trauma processes in alcohol or drug dependent women; however, they point out the danger in extrapolating from clinical to household samples.

More research is needed to determine whether sexual victimization influences heavy drinking among the general population of women. The use of even larger samples will permit researchers to examine whether there are moderating variables that determine who is vulnerable to increased drinking in response to trauma. Shorter assessment intervals, ideally, daily data collection, will also provide a more sensitive test of the hypothesized relationship. The conclusion from this study, however, is that despite the substantial prevalence of sexual victimization among young women in the general population and the traumatic impact of these experiences, the prospective impact of these experiences on heavy drinking appears limited.

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Table 1

Comparison of Mean Heavy Drinking Levels of Women with and without Attempted or Completed Rape at Waves 1 and 2

	Any Lifetime Adult Attempted or Completed Rape at Wave 1				
	Yes (*n* = 202)		**No (*n* = 723)**		
Variable	***M***	***SD***	***M***	***SD***	***t***
T1 Heavy Episodic Drinking	1.14	.98	.92	.81	2.84**
T1 Maximum Number of Drinks	3.46	2.07	2.78	1.76	4.23***
T2 Heavy Episodic Drinking	1.21	1.05	.94	.84	3.38**
T2 Maximum Number of Drinks	3.25	1.99	2.77	1.79	3.12**
	Any Past Year Attempted or Completed Rape at Wave 2				
	Yes (*n* = 29)		No (*n* = 896)		
Variable	*M*	*SD*	*M*	*SD*	*t*
T2 Heavy Episodic Drinking	1.88	.88	.97	.89	5.45***
T2 Maximum Number of Drinks	4.90	2.13	2.81	1.80	5.22***
T3 Heavy Episodic Drinking	1.76	1.07	.95	.87	4.00***
T3 Maximum Number of Drinks	4.55	2.08	2.74	1.68	4.65***
T2 PTSD Symptoms	11.52	6.03	5.15	5.30	5.62***

** $p < .01$.

*** $p < .001$.

Table 2

Correlations among T1 Predictor and T2 Outcome Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Age	-											
2. Race	.07*	-										
3. T1 marital status	.47***	.10**	-									
4. T1 parent status	.40***	−.19***	.39***	-								
5. CSA	.02	−.21***	−.03	.11**	-							
6. T1 HED	−.09**	.16***	−.21***	−.22***	.05	-						
7. T1 max drinks	−.08*	.25***	−.16***	−.20***	.02	.71***	-					
8. T1 sexual vict.	.06	−.00	.03	.03	.21***	.06	.13***	-				
9. T1 rape	.04	.02	−.04	.02	.17***	.10**	.15***	.68***	-			
10. T1 IPV	.14***	−.04	.02	.18***	.18***	.10**	.12***	.25***	.26***	-		
11. T1 events	.07*	−.02	−.01	.04	.12***	.04	.10**	.24***	.25***	.19***	-	
12. T2 HED	−.12***	.17***	−.21***	−.19***	.03	.72***	.59***	.06	.13***	.10**	.06	-
13. T2 max drinks	−.13***	.26***	−.15***	−.22***	−.01	.63***	.75***	.08*	.11**	.07*	.06	.71***

* $p < .05$.

** $p < .01$.

*** $p < .001$

Table 3

Hierarchical Multiple Regression Predicting T1 Heavy Drinking from any Lifetime Adult Attempted or Completed Rape at T1

Predictor Variables	Freq. of Heavy Episodic Drinking at T1 (N=910)			Maximum Number of Drinks at T1 (N=911)		
	ΔR^2	Initial β	Final β	ΔR^2	Initial β	Final β
Step 1	.094***			.104***		
Age at T1		.04	.02		.01	−.01
Race[a]		.17***	.16***		.26***	.25***
T1 Marital status[b]		−.19***	−.17***		−.14***	−.13***
T1 Parent status[c]		−.14***	−.15***		−.10**	−.12**
Childhood sexual abuse		.09**	.07*		.08*	.04
Step 2	.005*			.017***		
T1 Lifetime rape		.07*	.04		.13***	.09**
Step 3	.011**			.014**		
T1 Lifetime traumatic events		.00	.00		.05	.05
T1 Lifetime IPV		.11**	.11**		.11**	.11**
Total R^2	.111***			.135***		

[a] Race is coded 1 for Caucasian, 0 for non-Caucasian.

[b] Marital status is coded 1 for married, 0 for not married.

[c] Parent status is coded 1 for parent, 0 for not a parent.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 4

Hierarchical Multiple Regression Predicting T2 Heavy Drinking from any Lifetime Adult Attempted or Completed Rape at T1 and T1 Heavy Drinking

Predictor Variables	ΔR^2	Freq. of Heavy Episodic Drinking at T2 (N=921)			Maximum Number of Drinks at T2 (N=922)	
		Initial β	Final β	ΔR^2	Initial β	Final β
Step 1	.531***			.575***		
Age at T1		−.03	−.04		−.06*	−.05*
Race[a]		.07**	.07**		.08**	.08**
T1 Marital status[b]		−.06*	−.06*		−.01	−.01
T1 Parent status[b]		.01	.01		−.03	−.03
T1 Heavy drinking measure		.70***	.70***		.72***	.72***
Step 2	.002*			.000		
T1 Lifetime rape		.05*	.04		−.00	.00
Step 3	.001			.000		
T1 Lifetime traumatic events		.02	.02		−.01	−.01
T1 Lifetime IPV		.03	.03		.00	.00
Total R^2	.534***			.575***		

[a] Race is coded 1 for Caucasian, 0 for non-Caucasian.

[b] Marital status is coded 1 for married, 0 for not married and living with husband. [b] Parent status coded 1 for parent, 0 for not a parent.

* $p<.05$.

** $p<.01$.

*** $p<.001$.

Table 5

Hierarchical Multiple Regression Predicting T3 Heavy Drinking from Past Year Attempted or Completed Rape at T2 and T2 Heavy Drinking

	Freq. of Heavy Episodic Drinking at T3 (N=922)			Maximum Number of Drinks at T3 (N=923)		
Predictor Variables	**ΔR^2**	**Initial β**	**Final β**	**ΔR^2**	**Initial β**	**Final β**
Step 1	.466***			.505***		
Age		−.05	−.06*		−.07*	−.07*
Race[a]		.01	.00		.08**	.09**
T2 Marital status[b]		−.09**	−.09**		−.08**	−.08**
T2 Parent status[b]		−.01	−.01		.00	.00
T2 heavy drinking measure		.64***	.64***		.66***	.65***
Step 2	.001			.002*		
T2 Past year rape		.04	.04		.05*	.05
Step 3	.002			.000		
T2 Past year traumatic events		−.01	−.01		.00	.00
T2 Past year IPV		−.04	−.04		.01	.01
Total R^2	.470***			.507***		

[a] Race is coded 1 for Caucasian, 0 for non-Caucasian.

[b] Marital status is coded 1 for married, 0 for not married. [b] Parent status is coded 1 for parent, 0 for not a parent.

* $p < .05$.

** $p < .01$.

*** $p < .001$.