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High Dose Alcohol Consumption Predicts Less Reduction in Post-Traumatic Stress Symptoms After a Campus Mass Shooting

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Abstract

Background: Recognizing that alcohol might affect subsequent processing of trauma-related information, this study examined whether high dose alcohol consumption (HDAC) following a campus mass shooting affected the relation between shooting exposure and post-traumatic stress symptoms (PTSS).

Methods: Female participants ($N = 691$) recorded levels of physical exposure to the shooting event, alcohol use, and PTSS 1 month following the shooting event and 8 months later.

Results: No evidence was found to suggest that pre-shooting HDAC moderated the relationship between trauma exposure and PTSS 1 month following the shooting. HDAC in the month following the shooting predicted less resolution of PTSS 8 months later. Specifically, at higher (but not lower) levels of HDAC, shooting exposure was associated with less reduction in PTSS from 1 to 8 months post-trauma. Several alternate explanations were ruled out.

Conclusions: Less reduction in PTSS seems to occur at high levels of both shooting exposure and high dose alcohol consumption. Theoretical implications and directions for future research are discussed.

Keywords

POST-TRAUMATIC STRESS; HIGH DOSE ALCOHOL CONSUMPTION; CAMPUS MASS SHOOTING

Several reasons have been offered to explain the co-occurrence between alcohol use and anxiety symptoms, including post-traumatic stress symptoms (PTSS; Kushner, Abrams, & Borchardt, 2000). Researchers have suggested that PTSS cause individuals to engage in drinking to escape negative affect (e.g., Brown & Wolfe, 1994). Others have suggested that alcohol use increases the vulnerability for developing PTSS because it alters neurobiological systems needed to process trauma in a meaningful fashion (Jacobsen, Southwick, & Kosten,

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Footnotes

¹This definition was different than typical United States standards. Based on the National Institute on Alcohol Abuse and Alcoholism 4 or more drinks in a sitting are considered to represent heavy drinking for women.

2001; Stewart, 1996). It is also possible that alcohol use leads to the maintenance of PTSS by increasing the likelihood of experiencing additional traumatic events (e.g., Parks & Fals-Stewart, 2004) or by reducing social support (Peirce, Frone, Russell, Cooper, & Mudar, 2000). There are few studies that have examined whether alcohol use predicts PTSS in a prospective manner following a traumatic event (e.g., Chilcoat & Breslau, 1998; Kaysen et al., 2006; Kaysen et al., 2011). The present study examined whether high dose alcohol consumption (HDAC) – the type of alcohol use that is most likely to affect underlying neurobiology as well as psychosocial factors important for processing trauma – is associated with subsequent difficulties in resolving PTSS following a campus mass shooting.

High Dose Alcohol Consumption and the Processing of Trauma

PTSS appear to result from difficulties processing trauma-related information as well as integrating such information into the existing memory network (Brewin, 2014; Ehlers & Clark, 2000), although there has recently been debate as to whether autobiographical memory fragmentation is a true result of PTSD (Rubin et al., 2016a, 2016b). Many individuals endorse heightened levels of PTSS immediately following a trauma, but most experience symptom reduction in the following months (e.g., Bonanno & Diminich, 2013; Shalev et al., 1998). A smaller percentage of individuals maintain high levels of PTSS, suggesting a failure to process the traumatic event (Davidson, Stein, Shalev, & Yehuda, 2004). Several models of PTSD highlight the importance of intact neurobiological systems underlying both emotion regulation and information-processing efforts in resolving PTSS (e.g., Buckley, Blanchard, & Neill, 2000). Alterations in such structures, particularly the hippocampus (Rodrigues et al., 2009) and the prefrontal cortex (e.g., Bush, Luu, & Posner, 2000), may account for disruptions in trauma-processing that are linked with the failure to diminish initial post-trauma responses. Neuro-imaging studies have found diminished hippocampal volumes and decreased hippocampal activity during fear-extinction recall paradigms in individuals with PTSD compared to healthy controls (for review, see Shin, Rauch, & Pitman, 2006; Milad et al., 2009). Neuro-imaging studies have also found smaller medial prefrontal cortex and anterior cingulate cortex volumes as well as diminished activity in these areas during fear-inducing paradigms for individuals with PTSD than healthy controls (e.g., Bremner, Staib, & Kaloupek, 1999; Woodward, Kaloupek, & Streeter, 2006).

In addition to neurobiological explanations, it is well-known that experiencing certain psychosocial factors following traumatic experiences, such as increased life stress or reduced social support, increases risk for maintaining PTSS (Brewin, Andrews, & Valentine, 2000). Importantly, increased HDAC has been observed to predict subsequent reduction in social support (Peirce, et al., 2000). Additional stress, particularly in the form of experiencing additional traumatic events, has also been observed as an outcome of increased HDAC (Parks & Fals-Stewart, 2004; Testa & Parks, 1996). Taken together, there are several potential mechanisms by which HDAC might interfere with processing trauma, although limited research has examined this possibility in a longitudinal manner.

Because alcohol affects underlying neurobiology (e.g., De Bellis et al., 2005; Nagel et al., 2005) and important psychosocial factors involved in processing trauma (e.g., Peirce et al., 2000), alcohol use might impede resolution of PTSS. We hypothesize that the relationship

between alcohol use and the subsequent course of PTSS is *specific to HDAC* versus overall alcohol use due to the harmful impact of HDAC. These hypotheses are examined in the context of symptom resolution following a campus mass shooting.

The Current Study

We propose that HDAC *prior to* the campus mass shooting would moderate the association between shooting exposure and PTSS severity 1 month following the shooting. That is, higher levels of exposure should be associated with higher levels of PTSS 1 month following the shooting, especially at high levels of pre-shooting HDAC. This effect is proposed because HDAC is expected to interfere with the initial processing of the traumatic event. We refer to this prediction as the *Pre-shooting Interference Hypothesis*.

Recognizing that the HDAC may also interfere with the ongoing processing of the traumatic event, we propose that HDAC *after* the campus mass shooting would moderate the association between shooting exposure and PTSS severity 8 months following the shooting. That is, higher levels of exposure should be associated with higher levels of PTSS 8 months following the shooting, especially at high levels of post-shooting HDAC. We refer to this prediction as the *Post-shooting Interference Hypothesis*. This moderation relationship should also be evident in the change in PTSS from 1 to 8 months. We expect these relationships to be specific to HDAC (versus overall alcohol use).

Methods

Participants and Procedures

Data were collected from female participants who completed three waves of a longitudinal study that was originally intended to examine the longitudinal factors related to sexual revictimization in women. The current study received approval from the Northern Illinois University (NIU) Institutional Review Board. The original sample consisted of 1,045 females enrolled in an introductory psychology course (Time 1 [T1]). The only prerequisites for the original sample were that participants be women 18 years of age or older and fluent in English. Data from the original sample were collected between September 2006 and February 2008. Another description of this data set can be found in Kumpula, Orcutt, Bardeen, & Varkovitzky (2011).

On February 14, 2008, a gunman opened fire on the NIU campus, killing five students, wounding 21 others, and then killing himself. Seventeen days post-shooting, participants from the original sample who consented to follow-up contact and were NIU students at the time of the campus mass shooting ($N = 812$) were sent an email invitation to complete a brief battery of online self-report measures related to their reactions to the campus mass shooting. Six-hundred and ninety-one (85%) of these eligible participants completed the 1-month post-shooting assessment (Time 2 [T2]).

Participants who completed the T2 assessment identified as White (68%), African-American (20%), Hispanic (3%), Asian (3%), or Multiracial/Mixed (6%). The median family income was between \$60,000 and \$69,000. Participants were offered \$40 compensation or partial

course credit. Most participants completed the T2 assessment within 40 days. The average time elapsed between the shooting event and T2 was 27 days ($SD = 12$).

Approximately 8 months later (Time 3 [T3]), participants who completed the T2 assessment were invited to complete another battery of self-report measures. Of the 691 participants who completed the T2 assessment, 588 (85%) completed the T3 assessment. Participants who completed the T3 assessment were offered \$40 compensation. The average time elapsed between the shooting event and the T3 assessment was 35 weeks ($SD = 3.1$).

Measures

Distressing Events Questionnaire (DEQ; Kubany, Leisen, Kaplan, & Kelley, 2000).

The DEQ is a 17-item self-report measure of PTSS. Participants record the extent to which they experienced each symptom over the past month ($1 = \text{almost never}$ to $5 = \text{always}$). The DEQ was administered at the T1, T2, and T3 assessments. At T1, participants recorded responses based on their previous most distressing traumatic experiences. Thus, T1 PTSS was based on idiosyncratic traumatic events; however, participants were asked to reference the campus mass shooting event during both the T2 and T3 assessments. The full scale was used by summing all the items. Higher sum scores reflected higher PTSS. T1 and T3 DEQ scores were transformed using a square-root transformation to reduce non-normality (pre-transformation skew = 1.57 and 1.97, post-transformation skew = 0.36 and 1.15). Internal consistency statistics were excellent at the three time points (α 's = .92, .94, and .94, respectively).

Alcohol Use.

At the T2 assessment, a screening item asked participants if they have ever drank alcohol, more than just a sip. Most individuals endorsed drinking behavior at the T2 assessment (87%) and completed additional items about their alcohol consumption. Participants who denied alcohol use were included in the current study by imputing ratings that corresponded with no drinking for each item. Alcohol use was measured for two distinct time periods: 6 months prior to the shooting event (i.e., pre-shooting drinking) and since the shooting event (i.e., post-shooting drinking).

For pre-shooting drinking, participants retrospectively answered pre-shooting drinking items based on their alcohol use during the 6 months prior to the shooting event. Item 1 instructed participants to rate "how often did you drink any kind of alcoholic beverages, including beer, wine, flavored malt beverages (alcopops), wine coolers, or liquor" with responses ranging from 0 (*not at all*) to 8 (*everyday*). Participants rated on average drinking between 2–3 times a month and 1 time a week (Mean = 3.13, $SD = 1.76$, skew = 0.03). Item 2 instructed participants to enter "how many drinks on average would you have on a typical day when you drank." A drink was defined as either a 4 oz. glass of wine, a 12 oz. flavored malt beverage (alcopops) or wine cooler, a 12 oz. beer, or a shot or drink containing 1 oz. of liquor. Participants reported having an average of 3.48 drinks ($SD = 3.10$, skew = 1.79) on a typical day they drank in the last 6 months.

Item 3 instructed participants to rate “how often did you have 5 or more drinks¹ of any kind of alcoholic beverage in a single day” with responses ranging from 0 (*5 times a week or more*) to 8 (*never in the past six months*). Participants reported binge drinking on average between 3–5 times and 6 times in the past 6 months (Mean = 2.69, *SD* = 2.37, skew = 0.36). Item 4 instructed participants to rate “how often did you drink enough to get drunk or very, very high” with responses ranging from 0 (*5 times a week or more*) to 8 (*never in the past six months*). Participants had an average response (Mean = 3.00, *SD* = 2.41, skew = 0.28) to this item that corresponded with getting drunk 6 times in the past 6 months. Items 3 and 4 were reverse-scored and then aggregated to form a pre-shooting HDAC composite ($\alpha = .91$).

For post-shooting drinking, participants answered items based on alcohol use since the shooting event. The same items as the pre-shooting drinking items were administered; however, response options were modified to account for the survey being launched 17 days post-shooting and a 6-month response window would overlap with pre-shooting drinking. Participants were instructed to answer numerically based on number of days or drinks. Participants reported drinking an average of 3.54 days (*SD* = 4.99, skew = 2.92) since the shooting (average 27 days), which is slightly more than during the 6 months prior to the shooting. Participants reported having an average of 3.49 drinks (*SD* = 3.03, skew = 1.42) on a typical day when drinking, about the same amount as during the 6 months prior to the shooting. Participants reported binge drinking an average of 2.03 days (*SD* = 3.70) and reported being very drunk an average of 2.51 days (*SD* = 3.97), somewhat more than during the 6 months prior to the shooting. Post-shooting drinking items 3 and 4 were aggregated and then square-root transformed to reduce non-normality (pre-transformation skew = 3.24, post-transformation skew = 0.95) to form the post-shooting HDAC composite ($\alpha = .86$).

For both pre-shooting drinking and post-shooting drinking, items 1 and 2 were multiplied to create the Quantity Frequency Composite (e.g., Stockwell et al., 2004). After this computation, both composites were square-root transformed to reduce non-normality (pre-transformation skew = 2.33 and 4.93, post-transformation skew = 0.41 and 1.63, respectively).

Physical Exposure to Mass Shooting Questionnaire.

At the T2 assessment, participants completed a 12-item measure of shooting exposure that was modified from another shooting exposure measure (see Littleton, Grils-Taquechel, & Axsom, 2009). The measure contains 12 yes/no questions measuring exposure to different aspects of the shooting. Items were summed to create a total count score ranging from 0 to 12 which reflected the extent of physical exposure to the campus mass shooting event. This variable is hereafter referred to as Trauma Exposure.

With respect to shooting exposure, 76% of the sample was on campus at the time of the shooting, 69% saw emergency responders or police, and 49% were locked-down in a campus building. One-hundred and fifty-two (22%) participants saw individuals who had been wounded or killed, 6% heard gunfire, 4% were in the building where the shooting took place, 2% saw the gunman, 2% saw the gunman firing his weapon, and 0.4% were wounded in the shooting. Two-hundred and thirty-five (34%) participants knew someone who had been wounded in the shooting and 23% knew someone who had died in the shooting.

Results

Preliminary Analyses

In order to detect any potential effects related to attrition, independent sample t-tests were conducted comparing participants who did not complete a measurement to those who did complete a measurement based on their mean values for variables measured at prior time points. Specifically, T1 variables (i.e., trauma exposure and T1 PTSS) were compared between participants completing T2 measures and those who did not complete T2 measures. Additionally, T1 variables and T2 variables (i.e., pre-shooting QFI, pre-shooting HDAC, post-shooting QFI, post-shooting HDAC, T2 PTSS) were compared between participants reporting T3 PTSS and those who did not report T3 PTSS. These 17 analyses were predominantly nonsignificant (p -values ranged from .11 to .99), with the exception of 1 significant finding that participants who did not complete pre-shooting HDAC items, compared to those who did complete, reported lower levels of T1 PTSS. Given that a systematic pattern of attrition biases was not observed, we continued with the following analyses testing the *Pre-shooting Interference Hypothesis* and the *Post-shooting Interference Hypothesis*.

Pre-shooting Interference Hypothesis

Means and standard deviations of and intercorrelations among the study variables are presented in Table 1. The Pre-shooting Interference Hypothesis was tested using a hierarchical regression with an interaction effect (e.g., Aiken & West, 1991). These results are presented in Table 2. The dependent variable in this analysis was T2 PTSS. On Step 1, Interval, Trauma Exposure, Pre-Shooting Alcohol Quantity-Frequency, T1 PTSS, and Pre-Shooting HDAC were mean-centered and entered as predictors. On Step 2, the Pre-Shooting HDAC X Trauma Exposure interaction effect (calculated as the product of the mean-centered variables) was entered as a predictor. Although Trauma Exposure, Interval, and T1 PTSS accounted for significant variance in T2 PTSS, Pre-Shooting Alcohol Quantity-Frequency, Pre-Shooting HDAC and the Pre-Shooting HDAC X Trauma Exposure interaction effect did not. These results failed to support the Pre-shooting Interference Hypothesis.

Post-shooting Interference Hypothesis

A similar analytic approach was used to test the Post-shooting Interference Hypothesis. The dependent variable in this analysis was T3 PTSS. Results from this analysis are presented in Table 2. On Step 1, T1, PTSS, T2 PTSS, Pre-Shooting HDAC, Pre-Shooting Alcohol Quantity-Frequency, Post-Shooting Alcohol Quantity-Frequency, Trauma Exposure, and Post-Shooting HDAC were mean-centered and entered as predictors. On Step 2, the Post-Shooting HDAC X Trauma Exposure interaction effect was entered as a predictor. On the final step, Trauma Exposure, T1 PTSS, T2 PTSS, and the Post-Shooting HDAC X Trauma Exposure accounted for significant variance in T3 PTSS. These results provided support for the Post-shooting Interference Hypothesis.

Simple Slopes Analysis.

Follow-up simple slope analyses revealed that Trauma Exposure was significantly related with T3 PTSS at higher (e.g., +1SD), but not lower levels of Post-Shooting HDAC (e.g., -1SD) while controlling for Pre-Shooting HDAC, Pre-Shooting Alcohol Quantity-Frequency, Post-Shooting Alcohol Quantity-Frequency, T1 PTSS, and T2 PTSS. The observed interaction effect and simple slopes are presented in Figure 1. Regions of significance analyses revealed that Trauma Exposure was only significantly related to T3 PTSS ($p < .05$) when Post-Shooting HDAC levels were above -0.26 standard deviations above the Post-Shooting HDAC mean; and Post-Shooting HDAC was only significantly related to T3 PTSS ($p < .05$) when Trauma Exposure levels were above 3.00 standard deviations above the Trauma Exposure mean.

Consideration of Nonlinear Effects

Given that interaction effects can spuriously reflect quadratic effects (Lubinski & Humphreys, 1990) and alcohol use has demonstrated quadratic relations with PTSS (McFarlane et al., 2009), we explored whether the Post-shooting Interference findings (i.e., significant interaction effect) could be explained by quadratic relationships of trauma exposure and alcohol use with subsequent PTSS. Step 1 remained the same as the previous analysis. The mean-centered Trauma Exposure and Post-Shooting HDAC variables were squared and entered in Step 2 with the Post-Shooting HDAC X Trauma Exposure interaction. The Post-Shooting HDAC X Trauma Exposure interaction effect remained significant ($p < .01$); the quadratic effects were nonsignificant (p 's $> .05$). These findings suggest that the observed significant interaction was not explained by quadratic effects.

Discussion

This study tested two hypotheses regarding the role of HDAC during the course of PTSS. As expected, trauma exposure was associated with PTSS 1 month after the trauma. There was no evidence that HDAC prior to the campus mass shooting altered the relationship between trauma exposure and PTSS one month after the trauma (i.e., *Pre-shooting Interference Hypothesis*). Mean levels of PTSS decreased between 1 and 8 months after the trauma. This apparent symptom resolution appeared to be impeded by HDAC in the 1 month following the trauma; HDAC within 1 month following the campus mass shooting moderated the relation between trauma exposure and PTSS 8 months after the trauma. Specifically, trauma exposure was related to 8-month PTSS at high (but not low) levels of post-shooting HDAC. Further, this study was able to rule out several alternate explanations for this finding.

Regarding the Pre-shooting Interference Hypothesis, no support was found. Prior research has shown that HDAC is likely to affect the hippocampus (Hedges & Woon, 2010), and prefrontal cortical structures, which are involved in initial processing of trauma (Rodrigues, LeDoux, & Sapolsky, 2009). In addition, the way in which trauma is initially encoded is likely to affect PTSS (Halligan, Clark, & Ehlers, 2002). This study, however, found no evidence that 1 month PTSS are substantially affected by recent HDAC, at least not within the range observed in this study.

HDAC moderated the relation between trauma exposure and PTSS 8 months following a campus mass shooting event (*Post-shooting Interference Hypothesis*). Typically, the relationship between trauma exposure and PTSS dissolves over time as the trauma is processed (e.g., Bonanno & Diminich, 2013). The current study found that HDAC early in the course of symptom resolution appears to strengthen the relationship between trauma exposure and subsequent PTSS.

The stronger relationship between trauma exposure and subsequent PTSS at high levels of HDAC is likely due to interference in processing the traumatic event. Additional analyses ruled out obvious alternate explanations. First, the interference effect was specific to HDAC and could not be explained by overall alcohol use. HDAC may be particularly involved given its specific influence on the hippocampus (e.g., Hedges & Woon, 2010; Nagel et al., 2005) and prefrontal cortex (e.g., De Bellis et al., 2005). In addition, evidence suggests that HDAC seems more likely than overall alcohol consumption to lead to subsequent traumas (e.g., victimization; Parks & Fals-Stewart, 2004) as well as reduce available helpful social support (Peirce et al., 2000). These variables may be possible mechanisms of the observed interaction effect that could be explored in future research. Second, the interference effect was not explained by quadratic relationships between either trauma exposure and 8-month PTSS or post-shooting HDAC and 8-month PTSS.

The observed role of HDAC on symptom resolution could not be explained by overall alcohol use or the effects of PTSS at 1-month post-shooting. Analyses provided evidence for the specificity of the relationship, but it is possible that the key finding from this non-experimental study may reflect the effects of an unknown third variable. A third variable explanation, however, would need to posit an effect on 1 month HDAC (and not overall alcohol use) and a maturational effect on PTSS that is not evident at 1-month post-shooting but is evident at 8-months post-shooting. As such, it is possible that HDAC may be a marker for maladaptive coping strategies that are associated with the failure to resolve symptoms (Valentiner, Foa, Riggs, & Gershuny, 1996).

It is also important to note that the timing of HDAC relative to experiencing PTSS following the campus mass shooting could not be determined in the current study. This limits the ability to determine whether HDAC occurred at the same time as PTSS and interfered with concurrent trauma processing or occurred relatively outside of the experience of PTSS. Additionally, since our study was limited to self-report measures of PTSS and alcohol use, direct measurement of the functioning, activity, or integrity of potential underlying neurobiological alterations (i.e., hippocampus, prefrontal cortical structures) is needed to determine the mechanism by which HDAC is associated with subsequent problems in symptom resolution. These studies might include direct measures (e.g., neuro-imaging) or functional measures (e.g., working memory, inhibitory control). Since the current study did not include such measures, explanations in terms of neurobiological alterations should be considered speculative. Future research should also measure psychosocial factors, including availability of social support and incidence of additional traumatic experiences, to determine if these variables serve as mechanisms that explain the relation between HDAC and subsequent reduction in PTSS.

The use of a female college sample raises questions about the generality of the findings, particularly considering that gender differences are observed with respect to PTSS (e.g., Stein, Walker, & Forde, 2000) and alcohol use (e.g., Nolen-Hoeksema, 2004). Also, the toxic effect of HDAC on underlying neurobiology seems more harmful during the adolescent period (e.g., Fleming et al., 2013), raising the possibility that the observed effect might not generalize to other ages. In addition, the context of a campus shooting does not represent the variety of traumas that lead to PTSS. Similarly, although preceding levels of PTSS were included as covariates in our analyses, T1 PTSS was reported based on idiosyncratic traumatic events which could have unique outcomes with respect to PTSS trajectories. Finally, we did not examine potential effects of polysubstance use (e.g., alcohol, nicotine, and cannabis use) which may alter the observed findings. Considering these limitations, conclusions based on the results of the current study should be tempered until these results can be replicated with other samples and other traumas. The relatively small effect size found in the current study suggests that HDAC is of minor significance for understanding PTSS within an exposed population. The nature of the interaction effect suggests that HDAC is of increasing significance at higher levels of HDAC and at higher levels of trauma exposure. The current findings should inform future work with those populations.

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Figure 1.
The Interaction between Post-Shooting HDAC and Trauma Exposure in Predicting 8-Month PTSS
Note: HDAC = High Dose Alcohol Consumption. PTSS = Post-traumatic Stress Symptoms.

Table 1

Means and Standard Deviations of and Intercorrelations Among Study Variables

	1	2	3	4	5	6	7	8
1. Trauma Exposure								
2. T1 PTSS [^]	.11 **							
3. T2 PTSS	.33 **	.36 **						
4. T3 PTSS [^]	.32 **	.33 **	.56 **					
5. Pre-Shooting HDAC	.15 **	.08	.12 *	.12 *				
6. Pre-Shooting QF [^]	.11 **	.11 **	.14 **	.13 **	.78 **			
7. Post-Shooting HDAC [^]	.11 *	.15 **	.19 **	.15 **	.66 **	.67 **		
8. Post-Shooting QF [^]	.09 *	.12 **	.14 **	.12 **	.61 **	.76 **	.79 **	
<i>Mean</i>	2.87	2.64 ¹	20.42	2.20 ²	5.69	2.86 ³	2.09 ⁴	2.89 ⁵
<i>SD</i>	1.73	1.78	14.37	1.65	4.58	2.20	2.02	3.05
<i>Skew</i>	0.60	0.36	0.53	0.49	0.32	0.41	1.05	1.63
<i>Minimum</i>	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
<i>Maximum</i>	12.00	7.42	68.00	7.68	16.00	12.25	10.00	20.00
<i>N</i>	691	651	682	585	545	668	567	671

Note: PTSS = Post-traumatic Stress Symptoms, HDAC = High Dose Alcohol Consumption, QF = Quantity-Frequency.

[^] = variable transformed to reduce non-normality.**
 $p < .01$ *
 $p < .05$.¹Pre-transformed mean=10.15²Pre-transformed mean=14.37³Pre-transformed mean=13.00⁴Pre-transformed mean=4.55⁵Pre-transformed mean=17.62

Table 2

Regression Analyses Testing the Pre-shooting Interference Hypothesis and the Post-Shooting Interference Hypothesis

Predictors	Standardized Betas		
	R ²	Step 1	Step 2
<i>Pre-shooting Interference Hypothesis</i>			
Dependent Variable = T2 PTSS			
Step 1	.21 **		
Interval		-.17 **	-.17 **
T1 PTSS		.33 **	.33 **
Trauma Exposure		.27 **	.27 **
Pre-shooting QFI		-.02	-.02
Pre-shooting HDAC		.05	.05
Step 2	.00		
Trauma Exposure X Pre-shooting HDAC		-	.01
<i>Post-shooting Interference Hypothesis</i>			
Dependent Variable = T3 PTSS			
Step 1	.39 **		
T1 PTSS		.14 **	.13 **
T2 PTSS		.51 **	.51 **
Trauma Exposure		.16 **	.14 **
Pre-shooting QFI		.02	.03
Pre-shooting HDAC		.00	.00
Post-shooting QFI		-.01	-.02
Post-shooting HDAC		.00	.01
Step 2	.01 *		
Trauma Exposure X Post-shooting HDAC		-	.12 *

*
p < .05

**
p < .01.