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Traumatic Experiences and Reduced Alcohol Self-efficacy in Alcohol Dependent Inpatients: Attentional and Autonomic Mediators

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ABSTRACT:

Traumatic experiences and reduced alcohol self-efficacy in alcohol dependent inpatients: attentional and autonomic mediators

Objective: Self-medication with alcohol has been documented among individuals exposed to chronic trauma who may be unable to resist urges to drink in high-risk situations. Persistent alcohol use can result in attentional bias (AB) toward alcohol cues and psychophysiological reactivity such as changes in cue-elicited heart rate variability (HRV). The present study tested the hypothesis that individual differences in alcohol AB and HRV mediate the association between lifetime exposure to traumatic events and alcohol-related self-efficacy among a sample of alcohol dependent adults in inpatient treatment.

Method: This paper details a secondary data analysis from a sample of alcohol dependent adults (N = 58) who completed standardized psychosocial instruments, an affect-modulated cue reactivity protocol, and a spatial cueing task.

Results: Path analyses indicated statistically significant direct effects of levels of lifetime trauma exposure on Low Frequency/High Frequency-HRV cue-reactivity, alcohol AB, and alcohol self-efficacy. A statistically significant indirect effect was found from lifetime trauma exposure to alcohol-related self-efficacy through alcohol AB.

Conclusions: Results suggest that alcohol AB partially mediates the relationship between greater lifetime trauma exposure and reduced alcohol self-efficacy. Hence, alcohol dependent individuals with more extensive trauma histories may benefit from therapies aimed at increasing self-awareness and self-regulation of attentional and autonomic reactivity to prevent risk of relapse.

Keywords: alcohol dependence, attentional bias, heart rate variability, self-efficacy, traumatic experiences, PTSD

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INTRODUCTION

Individuals exposed to traumatic life experiences are at risk for adverse outcomes, including the development of alcohol-use disorders[1]. Traumatic events are experienced by 40 to 90% of individuals in the general population[2]. Over two-thirds of persons with substance use disorders report experiencing one or more traumatic life events[3], with alcohol use disorders reported as the most commonly co-occurring condition[4]. Individuals exposed to childhood trauma, assaultive violence, and the unexpected death of a loved one have twice the odds of developing concomitant alcohol-use disorders[1]. Among individuals with alcohol dependence, concurrent traumatic stress occurs at rates of 22 to 43%[4].

Substance use is predominantly driven by self-

medication motives among individuals who experience trauma[5]. The self-medication hypothesis postulates that individuals suffering from anxiety and negative mood symptoms use substances to alleviate fear and dysphoria[6]. In that regard, Leeies et al. found that 20% of persons suffering from psychological sequelae of trauma used substances as a means of symptom relief. Moreover, central nervous system (CNS) depressants appear to temporarily reduce trauma-related symptoms[7]; thus, negative reinforcement conditioning may elicit alcohol use when these symptoms recur[4].

Beyond negative reinforcement, persistent use of alcohol can increase the incentive salience of alcohol cues (e.g., smell of alcohol, sight of a bar), thereby strengthening motivation to drink[8]. Heightened incentive salience is indicated by autonomic and attentional reactivity to alcohol cues[9]. Autonomic reactivity to alcohol cues may be indexed by heart rate variability (HRV) – i.e., beat-to-beat changes in heart rate elicited by alcohol-related stimuli[10-13]. High-frequency HRV (HF-HRV), observed in the beat-to-beat modulation of heart rate by the vagus nerve, is thought to be parasympathetically mediated, whereas the ratio of low- to high-frequency HRV (LF/HF HRV) reflects predominately sympathetic nervous system activation[14]. Attentional reactivity to alcohol cues may be indexed by alcohol attentional bias (alcohol AB), evidenced on dot probe tasks by shorter reaction times to probes replacing alcohol photos relative to probes replacing neutral photos[10,17]. Alcohol AB is positively associated with craving[15] and may be primed by stress[16]. Heightened alcohol AB and HF-HRV to stress-primed alcohol cue exposure predicts alcohol relapse vulnerability[10]. As such, traumatic stress may promote alcohol AB and elicit psychophysiological reactivity, which may, in turn, result in self-medication with alcohol as a means of alleviating distress.

In addition to such reactivity, individuals' beliefs about their capacity to effectively implement coping techniques – that is, their level of self-efficacy – strongly influence alcohol relapse outcomes[18]. Alcohol dependent individuals with low alcohol-related self-efficacy are less able to resist cravings[19] and tend to exhibit increased difficulty in adapting to environmental stressors and alcohol cues[20]. Therefore, lower alcohol-related self-efficacy among alcohol dependent individuals, coupled with impaired regulation of attentional and autonomic reactions to stress and alcohol cues, may increase relapse risk in stressful and traumatic contexts[20].

Few studies have examined interrelations among attentional and autonomic processes linking traumatic stress to alcohol self-efficacy in individuals with alcohol use disorders. The purpose of this study was to examine alcohol AB and HRV as mediators of the association between lifetime exposure to traumatic events and alcohol-related self-efficacy among alcohol dependent adult patients. Data were derived from an earlier study[21]. This paper makes a unique contribution by reporting previously unexamined mediational relationships between trauma and alcohol self-efficacy. It was hypothesized that: a) extent of lifetime trauma exposure would be negatively associated with alcohol self-efficacy; b) alcohol dependent persons with more extensive trauma histories would exhibit greater alcohol AB and HRV cue-reactivity than individuals with less trauma exposure; and c) alcohol AB and HRV cue-reactivity would mediate the association between lifetime trauma exposure and reduced alcohol-related self-efficacy.

METHODS

Participants

Alcohol dependent adults (n= 58) who resided for an average of 22.2 months in a residential treatment facility for alcohol use disorders located in in the US were recruited for this study. Potential participants met study inclusion criteria if they were ≥18 years old and satisfied lifetime Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) alcohol dependence criteria as assessed with an interview adapted from section I

of the Mini-International Neuropsychiatric Interview (MINI)[22]. Diagnostic interviews were conducted by a licensed psychiatrist and licensed clinical social worker trained in making DSM-IV substance use and other psychiatric disorder diagnoses. Participants met 6.5 (SD=1.0) DSM-IV alcohol dependence criteria (3 were required for a diagnosis), and the sample's average total Alcohol Use Disorders Identification Test score was 32.4 (SD= 5.6). In the year before entering treatment, participants consumed a mean number of 18.9 (SD= 10.8) alcoholic drinks per day. All participants reportedly abstained from use of psychoactive substances during their stay in the treatment facility. These reports were verified through random urinalyses and observations by the program staff. No participants took medications that affected cardiovascular functions.

Potential participants were recruited through flyers, direct referrals from facility staff, and through an informational presentation about the study made at the treatment facility. Participants were paid $25 for their participation. The majority of participants were male (81%) and they primarily identified as African American (55.2%) and Caucasian (39.7%). The average age of the sample was 39.8 (SD= 9.3). In the year prior to entering treatment, 56.9% of participants had earned less than $20,000 and 29.3% had earned between $20,000 and $40,000.

Procedure

Data were collected in a single assessment. Measures were completed in the following order: 1) psychosocial instruments, 2) a computer-based spatial cueing task designed to assess alcohol attentional bias, and 3) an affect-modulated cue-reactivity protocol measuring HRV responses to stress-primed cues[23]. All participants provided written informed consents, and this study was conducted with Institutional Review Board (IRB) approval from the University of North Carolina at Chapel Hill IRB and in accordance with the Helsinki Declaration.

Measures

Trauma history. Lifetime extent of trauma history was assessed by asking participants to indicate whether or not they had experienced each of a series of traumatic events in their lifetime[24]. This variable was calculated by summing the number of affirmative responses to ever having experienced a traumatic event as assessed by the following questions: "witnessed someone severely injured or killed in person"; "having been badly hurt"; "had something very bad or terrifying happen to you"; "hit by someone who was trying to hurt you"; "been mugged by a weapon or by force"; "attacked with a weapon or by someone trying to hurt or kill you"; "witnessed someone be severely beaten"; and "been sexually touched against your will."

Alcohol self-efficacy. The Situational Confidences Questionnaire (SCQ) is a 39-item measure that demonstrates high reliability (α= 0.81-0.97)[25]. Participants are asked to indicate how confident they are on a 6-point likert scale that they could cope with alcohol urges in various high-risk scenarios. Previous studies have shown SCQ scores to predict time to relapse[26].

Alcohol attentional bias. Alcohol AB was measured with a spatial cueing task that was created in E-Prime 2.0 (PST Inc., Pittsburgh, PA) in which participants were shown alcohol-related and neutral stimuli. Alcohol-related stimuli included 13 pictures of alcoholic drinks (i.e., liquor, beer, wine, etc.) in addition to 7 images of persons consuming alcohol. Neutral stimuli consisted of 13 pictures of kitchen items as well as 7 photos of individuals in kitchen scenes. The visual properties of the alcohol and neutral stimuli did not differ significantly vis-a-vis spectral peak (neutral: 0.018, alcohol: 0.017, $t(38)= 0.38$, $p= 0.70$) or spectral width (neutral: 59.20, alcohol: 59.29, $t(38)= -0.03$, $p= 0.98$) characteristics. Each trial was presented on an IBM T60 laptop with a 15 inch screen and began with a fixation cross presented for 500 ms. Then, one alcohol-related image and one neutral image, both in grayscale, were presented side-by-side for 2000 ms. The position of the alcohol-related image (right or left) was randomized and

counterbalanced across 20 practice trials and 160 trials. A two-dot target probe substituted one of the images and a one-dot distractor probe substituted the other image after a 50 ms inter-stimulus interval (ISI). Probes were presented for 100 ms. Utilizing a keypad, participants indicated the location of the target by pressing a left or right button. Alcohol and neutral cues were randomly replaced by target probes at an equal rate. This spatial cueing task has been tested in prior studies for construct validity. Results identified associations between AB scores on this task and alcohol dependence risk factors, including mindlessness, cue-reactivity, and time-to-relapse[9,10,30].

Psychophysiological cue-reactivity. An affect-modulated cue-reactivity protocol was used to measure psychophysiological responses to stress-primed alcohol cues. This protocol first involved attaching electrocardiogram (ECG) electrodes to participants' right and left pectoral muscles. Raw ECG data, sampled at a frequency of 500 Hz, were acquired using a Biopac MP150 data system (Biopac Systems, Goleta, CA). During the 5-minute baseline, participants were instructed to remain quiet and motionless and "not think about anything in particular". Next, participants were shown stress cues including 30 aversive images from the International Affective Picture System presented on a 15 inch monitor for 10 seconds each (duration: 5 min.). Participants fixated on the image stream while remaining motionless. While continuing to remain motionless, next 30 images of alcohol beverages were presented for 10 seconds each (duration: 5 min.). This task elicits stress and craving responses in alcohol dependent adults[21].

Data Analysis

Alcohol AB scores were calculated for each participant by subtracting their mean reaction time to target probes replacing alcohol images from their mean reaction time to target probes replacing neutral images, wherein positive bias scores indicated an attentional bias toward visual alcohol cues. Data were eliminated as outliers (mean 2.5±1.5 trials per participant, 1.5% of all trials) if trials had extreme reaction times, defined as those with reaction times 3 SD above or below the individual mean reaction time (31). Moreover, error trials were also eliminated (mean 6.4±0.6 trials per participant, 4% of all trials). Data are reported as means±SD.

Using Nevrokard aHRV software (Medistar, Stegne, Ljubljana, Slovenia), HRV cue-reactivity was analyzed by detecting R-R intervals in the raw ECG data. Then, the R-wave file was visually inspected to correct misidentified or omitted R-waves. Spectral analysis of HRV was conducted using Kubios 2.0 (Biosignal Analysis and Medical Imaging Group, University of Finland). A fast Fourier transform to extract high-frequency (HF) and low-frequency (LF) HRV in the 0.15–0.40 Hz and 0.04–0.15 Hz frequency bands, respectively, from a de-trended, end-tapered inter-beat interval time series was applied[32]. HF-HRV was used as an index of parasympathetically-mediated HRV. In order to estimate sympathetically-mediated changes in HRV, the LF/HF ratio was selected[14,33]. For the 5-minute baseline and 5-minute alcohol cue exposure periods, mean HRV was computed. To compute HRV reactivity, the difference (Δ) between the 5-minute mean baseline level and the 5-minute mean during alcohol cue-exposure was used, with positive values indicating increased HRV from baseline to cue-exposure[34].

Multivariate path analysis was employed to test a model in which alcohol AB, HF-HRV reactivity, and LF/HF-HRV reactivity could mediate the relationship between lifetime traumatic events and alcohol-related self-efficacy controlling for differences in age and gender (see Figure 1). Path analysis was conducted AMOS 17.0, which uses Full Information Maximum Likelihood (FIML) methods to estimate missing data[35] – an approach that can produce unbiased estimates of regression coefficients for small samples (N~60) with up to 20% of missing data[36]. The χ^2/df ratio was examined to assess the overall model fit along with the Comparative Fit Index (CFI)[37] and the Root Mean Squared Error of Approximation (RMSEA) Index[38].

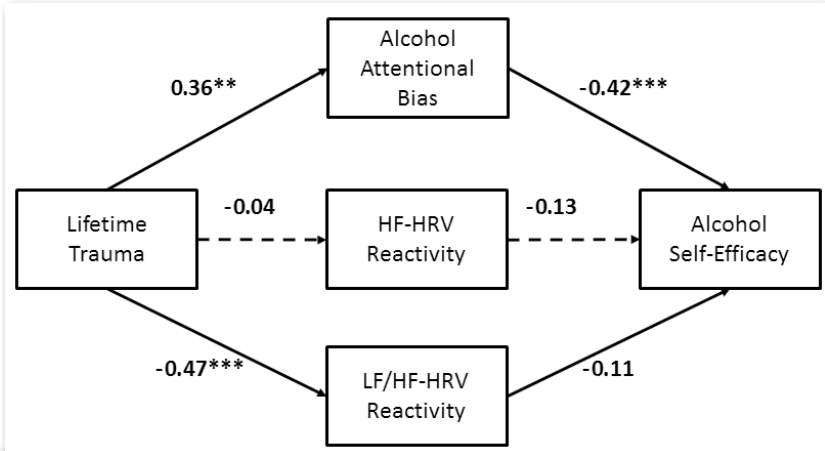

Figure 1: A multivariate path model testing attentional and autonomic mediators of the association between number of lifetime traumatic experiences and alcohol-related self-efficacy (controlling for age and gender) in an inpatient sample of alcohol dependent adults (n= 58) is presented. The indirect effect from lifetime trauma to alcohol-related self-efficacy through alcohol AB was statistically significant (Sobel z = 1.99, p= 0.046). Model R^2= 0.22. **p<0.01 ***p<0.001. Lifetime trauma = number of categories of traumatic lifetime incidences experienced by participants. Alcohol attentional bias = higher scores indicate greater attentional fixation on alcohol cues. HF-HRV reactivity = change in high frequency heart rate variability from resting baseline through alcohol cue-exposure. LF/HF reactivity = change in the low frequency/ high frequency heart rate variability ratio from resting baseline through alcohol cue-exposure. Alcohol self-efficacy = perceived ability to resist temptation to drink in high-risk situations.

RESULTS

Extent of Lifetime Trauma in the Sample

Participants had experienced a wide range of traumatic events. Participants reported having: been hit by someone trying to hurt them (92.5%), been in danger of getting badly injured or killed (80%), experiencing a terrifying event (80%), been attacked by someone who had attempted to seriously injure or kill them (77.5%), witnessed someone else being severely injured or killed (72.5%), been mugged or attacked with a weapon (67.5%), and having been raped (22%). On average, participants had experienced 5.7 (SD= 1.9) types of traumatic events.

Zero-Order Correlation Between Lifetime Trauma and Alcohol Self-efficacy

Lifetime trauma was significantly negatively correlated with alcohol self-efficacy (r= -0.36, p= 0.02).

Alcohol AB and HRV Cue-Reactivity

The mean reaction time to target probes replacing alcohol photos presented for 500 ms was 363.0±123.0 ms, whereas mean reaction time for neutral photos was 359.7±124.5 ms. Paired t-tests revealed nonsignificant differences between reaction times to alcohol and neutral photos presented for 500 ms (p>0.10). However, there was substantial heterogeneity in individual attentional bias scores, such that nearly one-half (44%) of the individuals in the sample had an attentional bias towards alcohol cues, whereas the other half (56%) had an attentional bias away from alcohol cues; hence, an individual difference analysis was conducted to model the heterogeneity in attentional bias responses.

As described in an earlier report[21], there were no significant mean effects of the cue-reactivity paradigm on HF-HRV or LF/HF ratio (p's >0.10). As with the alcohol AB scores described above, inspection of the raw data revealed heterogeneity in HRV responses to stress-primed alcohol cues during this task, such that some respondents evidenced increases in HRV whereas others evidenced decreases in HRV from baseline through alcohol cue-exposure. In light of this variability in HRV cue-reactivity, an individual difference analysis was performed.

Path Analytic Results

Results of the path analysis are presented in Figure 1. This model exhibited excellent fit to the data: χ^2/df= 0.99, p= 0.44, CFI= 1.00, RMSEA= 0.00 (0.00, 0.16). Results of the multivariate path analysis, controlling for age and gender, revealed significant direct effects of levels of lifetime trauma exposure on alcohol AB (β= 0.36, p= 0.01) and LF/HF-HRV reactivity (β= -0.47, p<0.001), but not on HF-HRV reactivity (β= -0.08, p= 0.61). In turn, there was a direct effect of alcohol AB on alcohol self-efficacy (β= -0.42, p<0.001), but no significant direct effects from LF/HF-HRV (β= -0.11, p= 0.42) or HF-HRV reactivity (β= -0.13, p= 0.36) on self-efficacy. Using the Sobel test[39], the indirect effect from lifetime trauma to alcohol-related self-efficacy through alcohol AB was statistically significant (Sobel z= 1.99, p= 0.046), indicating that alcohol AB partially mediated the relationship between lifetime traumatic incidents and alcohol-related self-efficacy. However, neither HF-HRV nor LF/HF-HRV reactivity mediated the relationship between lifetime trauma exposure and alcohol self-efficacy (Sobel z ps>0.10).

DISCUSSION

The current study explored pathways between individual differences in lifetime trauma exposure, attentional and autonomic reactivity to stress-primed alcohol uses, and alcohol self-efficacy among a treatment-seeking sample of alcohol dependent adults. Results of multivariate path analyses indicated that alcohol dependent individuals with more extensive trauma histories exhibited heightened alcohol AB, and thereby felt less able to resist the urge to drink alcohol in high-risk situations (i.e., low alcohol-related self-efficacy). Further, alcohol dependent individuals with significant trauma histories experienced attenuated LF/HF-HRV reactivity from baseline through stress-primed alcohol cue-exposure, though LF/HF-HRV reactivity to stress-primed alcohol cues was not associated with alcohol self-efficacy. Finally, HF-HRV reactivity was not significantly associated with either lifetime trauma or alcohol self-efficacy. On the whole, these findings are congruent with prior research indicating that alcohol cue-reactivity in stressful and traumatic contexts may influence sustained alcohol dependence[40].

Alcohol-dependent individuals with more extensive trauma histories exhibited greater alcohol AB and lower alcohol self-efficacy than those with less trauma exposure. Further, the association between lifetime trauma and alcohol-related self-efficacy was partially mediated by attentional fixation on alcohol cues. It may be speculated that individuals with more extensive trauma histories have greater difficulty shifting their attention away from alcohol cues, and thus, feel less able to resist the urge to drink in high-risk situations. Indeed, theoretical models specifying feedback circuits between stress, implicit cognition, self-regulation attempts, and the drive to consume alcohol[41] suggest that alcohol misuse in the context of stress and negative affect may establish automatic alcohol-use action schemas - memory structures that coordinate and compel behaviors involved in alcohol consumption. Activation of alcohol-use action schemas by alcohol cues or stressors might weaken conscious cognitive control mechanisms to regulate alcohol urges[42] and thereby undermine the sense of self-efficacy over the compulsion to drink.

In the present study, alcohol dependent patients with more lifetime trauma exhibited smaller increases in LF/HF-HRV to stress-primed alcohol cues than patients with fewer traumatic experiences. The observed inverse association between lifetime trauma and LF/HF-HRV cue-reactivity is in accordance with prior literature demonstrating blunted stress responses among persons with PTSD. In previous research, PTSD patients have been found to exhibit attenuated HRV and sympathetically-mediated cardiovascular reactivity to recall of traumatic memories and trauma imagery[43-45], as well as trauma and affective visual cues[46], acoustic startle probes[47], and cognitive challenge[48], among other laboratory stress provocations. Moreover, PTSD patients with

the most extensive trauma histories exhibit the greatest degree of blunted cardiovascular reactions to trauma imagery[49]. Thus, findings of the present study add to a growing body of literature demonstrating attenuated cardiovascular responses to emotional provocations in individuals with trauma histories[50]. Moreover, emerging theoretical models suggest that blunted psychophysiological responses to stress and reward (such as the blunted reactivity to stress-primed alcohol cues exhibited by individuals with more extensive trauma histories in the present study) might be indicative of a generalized biological disengagement from life challenges that is associated with adverse behavioral outcomes, including substance addiction[51]. No such relations between lifetime trauma exposure and HF-HRV reactivity were identified in the current study, in contrast to prior studies which have demonstrated an association between posttraumatic stress and lower HF-HRV[52]. Inconsistency between our HF-HRV results and the prior literature may be due to the fact that resting state HRV and cue-elicited HRV (i.e., HRV reactivity) are distinct phenomena that may have different biopsychosocial correlates.

Contrary to our hypothesis, the relationship between lifetime trauma and alcohol self-efficacy could not be accounted for by changes in HRV in the current study. This null finding may stem from the effects of trauma and addiction on self-awareness. Importantly, many neuropsychiatric conditions, including trauma and alcohol dependence, impair the ability to recognize and describe one's own emotional states and behavior[53]. Indeed, abnormalities in self-awareness and concomitant deficits in behavioral control have been attributed to the neural dysfunctions underlying addiction, which may explain the commonly observed dissociation between self-report and behavior in patients with substance use disorders[54]. Such individuals may lack insight into their addiction and thereby exhibit discordance between their physiological and subjective responses to substance-related stimuli, a phenomenon already substantiated by meta-analyses[55]. Thus, individuals who lack self-awareness vis-a-vis their addictive responses may be unable to accurately monitor and interpret interoceptive information from their bodies[54], such as autonomic perturbations elicited by stress and alcohol cues. Consequently, they may make inaccurate predictions regarding their susceptibility to relapse in high-risk situations, and therefore have an inflated sense of alcohol-related self-efficacy.

Certain limitations need to be considered when interpreting the results. First, the cross-sectional design precludes causal inferences. Though path analysis revealed that alcohol AB statistically mediated the relationship between lifetime traumatic experiences and alcohol self-efficacy, without experimental induction of AB toward or away from alcohol cues[56], we cannot ascertain whether alcohol AB is the cause, correlate, or consequence of alcohol-related self-efficacy. Second, we did not assess for posttraumatic stress symptomatology, and so it is unknown to what extent PTSD symptoms might be linked with alcohol AB, HRV reactivity, and self-efficacy. In addition, we did not control for respiration rate in our analyses, which could confound our HRV results[57]; yet, there is substantial debate in the literature regarding the importance of such corrections[58]. Finally, the presentation order of stress and alcohol cues was not counterbalanced, which prevented us from determining the differential contribution of each type of cue to HRV responses. Future studies should replicate these findings in a larger sample of alcohol dependent persons completing a counterbalanced cue-reactivity assessment.

Despite these limitations, study findings have implications for the treatment of alcohol dependence in individuals with a lifetime history of trauma. Results indicate that traumatic experiences among alcohol dependent individuals may lead to heightened attentional and autonomic reactivity to alcohol cues, while reducing the perceived ability to abstain from alcohol use in high-risk situations. Therapeutic techniques, such as the practice of mindfulness, may show promise among individuals with histories of trauma and

attentional biases toward addictive cues[59-61]. Such techniques might aim to attenuate stress reactivity and target implicit, subcortical appetitive processes by way of reducing attentional biases to alcohol cues, which may be accomplished by training individuals to shift and disengage attention from alcohol-related triggers to neutral objects[62]. Moreover, mindfulness-based interventions are designed to increase self-awareness and self-regulation of attentional and autonomic reactivity[62], and thus, may be especially useful in addressing co-occurring trauma and alcohol dependence by strengthening self-efficacy in stressful socioenvironmental contexts abundant with conditioned stimuli associated with past drinking episodes.

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