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Relations between cognitive functioning and alcohol use, craving, and posttraumatic stress: An examination among trauma exposed military Veterans with alcohol use disorder

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Abstract

Cognitive dysfunction is commonly observed among individuals with Alcohol Use Disorder (AUD) and trauma exposure and is, in turn, associated with worse clinical outcomes. Accordingly, disruptions in cognitive functioning may be conceptualized as a trans-disease phenomenon representing a potential high-yield target for intervention. Less is known though about how different cognitive functions co-vary with alcohol use, craving, and posttraumatic stress symptom severity among trauma exposed individuals with AUD. Sixty-eight male and female trauma exposed military Veterans with AUD, entering treatment trials to reduce alcohol use, completed measures assessing alcohol use and craving, posttraumatic stress symptom severity, and cognitive functioning. In multivariate models, after controlling for posttraumatic stress symptom severity, poorer learning and memory was associated with higher alcohol consumption and higher risk-taking/impulsivity was associated with stronger pre-occupations with alcohol and compulsions to drink. Alcohol consumption and craving, but not performance on cognitive tests, were positively associated with posttraumatic stress symptom severity. Findings suggest that interventions to strengthen cognitive functioning might be used as a preparatory step to augment treatments for AUD. Clinicians are encouraged to consider a standard assessment of cognitive functioning, in addition to posttraumatic stress symptom severity, in treatment planning and delivery for this vulnerable and high-risk population.

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Keywords

Alcohol; PTSD; Trauma; Cognition; Memory; Executive Functioning; Impulsivity

Problematic alcohol use is common among patients exposed to traumatic events[1], and alcohol use disorder (AUD) is the most prevalent and costly substance use disorder (SUD) among military veterans.[2,3] In civilians, 8-20% of those exposed to trauma will go on to develop Posttraumatic Stress Disorder (PTSD)[4,5,6] and among military veterans, trauma exposure is dramatically elevated with rates as high as 87% in a recent large national study.[7] Importantly, compared to AUD alone, those with co-occurring posttraumatic stress symptoms experience worse occupational, psychosocial and health outcomes, lower reported quality of life, increased interpersonal problems, higher rates of hospitalization and service utilization, and increased risk of suicide and mortality.[8-11] Unfortunately, despite availability of empirically-supported treatments for co-occurring AUD and posttraumatic stress[12,13], rates of relapse and non-response indicate an urgent need to identify risk factors that will better inform interventions for this growing and highly vulnerable population.[14,15]

In order to advance AUD treatment research and clinical practice for trauma-exposed individuals, it is critical to obtain greater knowledge of the common factors that may be associated with symptom presentation upon treatment entry. There is compelling evidence to suggest that neurocognitive dysfunction is one such factor that may represent a high-yield, trans-disease target for intervention. However, at present, there is a dearth of research available to help profile how cognitive functions are associated with symptoms resulting from AUD and trauma exposure. This knowledge gap is unfortunate because higher levels of clinical severity upon treatment entry (i.e., alcohol use, craving, posttraumatic stress symptom severity), significantly increase risk for reduced AUD treatment success.[16-19]

Neurocognitive Functioning, AUD, and Posttraumatic Stress

Trauma exposure has been prospectively associated with changes in neuropsychological functioning[20] and the neuropsychological sequelae of AUD and PTSD psychopathology include deficits in basic attention, processing speed, learning, memory, and executive functioning.[21-27] Executive functions are higher-order cognitive skills that are involved in the planning, initiation, and regulation of goal-directed behavior.[28,29] A wealth of research demonstrates that individuals with either AUD or posttraumatic stress have cognitive impairments compared to healthy controls. For instance, an estimated 50-70% of persons diagnosed with an AUD demonstrate some degree of neurocognitive deficit.[22] In a comprehensive meta-analysis of studies examining cognitive dysfunction among individuals with AUD, the domains of speed of processing, problem solving/executive functions, inhibition/impulsivity, verbal learning, and verbal memory were found to be moderately impaired after 2 to 12 months of abstinence.[27] Reviews and meta-analyses indicate that compared to controls, those with posttraumatic stress tend to demonstrate reduced auditory attention and working memory, selective and sustained attention, inhibitory functions, and cognitive flexibility/rapid attention switching.[21,24,26,30-32] Of particular importance, such cognitive deficits are linked with poorer treatment outcomes and lower retention.[33-41]

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Impulsive and risky behavior, a clinical profile of suboptimal decision making commonly observed in both AUD and trauma exposed populations[42,43], is considered a manifestation of poor executive control.[44,45] This is because deficits in "supervisory" executive control make it difficult to combat the automatic habit responses unleashed by the reward-seeking system (e.g., by employing positive coping strategies).[46] Indeed, individuals with AUD are more inclined to respond automatically and struggle to problem-solve, learn from reward prediction errors, and consider the long-term consequences of an action[47-51] and similar patterns are observed among individuals with posttraumatic stress.[52-54]

Although cognitive dysfunction has been well-documented in uni-morbid AUD and trauma-exposed populations[21,22,26], little research has described and examined how these functions are associated with indices of clinical severity among individuals with both AUD and trauma exposure. Given the established associations between cognitive dysfunction and poor treatment outcomes coupled with the cognitive demands made of patients during the treatment process, it is critical to better characterize relations between cognitive functioning and clinical severity outcomes upon treatment entry. The objective of the current study is to examine associations among key measures of cognitive functioning (processing speed, executive functioning, risk-taking/impulsivity, verbal learning and memory) and indices of clinical severity (posttraumatic stress symptom, quantity and frequency of alcohol use, and craving) among a sample of trauma-exposed military veterans with AUD entering pharmacotherapy treatment trials for AUD.[55-58] Several hypotheses were advanced to address study objectives. First, after accounting for posttraumatic stress symptom severity, we expect that lower processing speed, executive functioning, and verbal learning and memory, and higher risk-taking/impulsivity will be positively associated with quantity and frequency of drinking and craving for alcohol. Second, after accounting for alcohol consumption and craving, we expect a similar pattern of relations to emerge between cognitive domains and posttraumatic stress symptom severity.

METHODS

Participants

Participants were 68 U.S. military veterans (mean age = 49.74, SD = 12.93; 90% male; 57% Caucasian, 24% African American, 12% mixed race, 3% Asian, 4% other; 21% identified ethnicity as Hispanic/Latino) who were drawn from three different randomized controlled trials of topiramate treatment for AUD. Participants were included in these studies if they met Diagnostic and Statistical Manual of Mental Disorders IV-TR (DSM-IV) criteria[59] for current AUD as assessed by the Structured Clinical Interview for DSM-IV (SCID I/P).[60] All participants also reported "at-risk" or "heavy" drinking in accordance with NIH/NIAAA criteria (at least 15 standard drinks per week on average over the 4 weeks prior to study entry for men and at least 8 standard drinks per week on average for women)[61] and all expressed a desire to reduce alcohol consumption with the possible long-term goal of abstinence. For inclusion in the current study, participants must have also endorsed exposure to trauma as assessed by the Life Events Checklist,[62] which is strongly associated with PTSD symptoms in combat veterans.[63] Participants were excluded if they were known to have any clinically significant unstable psychiatric or medical conditions that would

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interfere with study participation, or had a suicide attempt or suicidal ideation in the six months prior to enrollment.

Procedure

Participants were recruited, and all procedures took place at the San Francisco Veterans Affairs Medical Center (SFVAMC). All participants provided written informed consent and underwent procedures approved by the University of California, San Francisco, the SFVAMC, and the Department of Defense. Each participant was assessed in 2-3 visits extending over approximately one week during which they completed the measures and tasks described below. Assessments for the current report were completed prior to randomization to each trials study group. No participants were assigned to receive Topiramate at the time of the assessments.

Psychiatric Assessment

Alcohol Use—The Time-Line Follow-Back (TLFB) interview[64,65] was conducted with participants to assess quantity and frequency of alcohol consumption prior to entering treatment. Data from the TLFB interview were used to calculate average number of drinks consumed per week and average number of drinking days per week in the 90 days prior to treatment. TLFB is considered the standard for alcohol use outcome measurement in clinical trials.[66]

Alcohol Craving—Craving for alcohol was assessed with the Obsessive Compulsive Drinking Scale (OCDS), which is widely used in clinical AUD populations and possesses strong psychometric properties.[67,68] The OCDS is designed to measure obsessive thoughts and behavioral compulsions and urges associated with alcohol craving among heavy drinkers and is comprised of two subscales; drinking obsessions (obsessive thoughts related to drinking) and compulsions (compulsive drinking urges and behaviors). Participants respond to items using a 5-6 point Likert-type scale and items are summed to yield a total score that ranges from 0 to 56.

PTSD Symptom Severity—PTSD symptom severity was assessed with the 17-item PTSD Checklist for Civilians (PCL-C)[69,70] and directly corresponds to the DSM-IV[59] symptoms of PTSD and subscales (B: Re-experiencing, C: Avoidance/Numbing, and D; Hyperarousal). Respondents indicate the extent to which they have been bothered by each symptom, in response to a stressful life situation, within the past month, using a 5-point Likert-type scale (1=not at all bothered; 5=extremely bothered). Responses are summed to yield a total score, ranging from 17-85, which is reflective of global PTSD symptom severity. The currently recommended cut-off score of 50[70,71] indicates that present symptoms are suggestive of PTSD.

Neurocognitive Assessment

Processing Speed—Trail Making Test Part A requires the respondent to connect a series of 25 numbered circles on a worksheet, as quickly as possible, and is often used an index of processing speed.[72]

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Executive functioning—In Trail Making Test Part B, the respondent connects a series of circles on a worksheet, alternating between numbers and letters, with instructions to work as quickly as possible.[72] Trail Making Test Part B is commonly used to assess executive functioning because it requires mental flexibility and speeded set-shifting.[73] Performance on Part B is correlated with other well-established measures of mental flexibility (Wisconsin Card Sorting Test perseverative errors)[74] and domains of executive function including working memory (WAIS-III digits backwards).[75] Time to complete Trail Making Test Part A and Part B was recorded and the revised comprehensive norms (corrected for age, education, gender, and ethnicity) for the expanded Halstead-Reitan Battery[76] were used for scoring.

Risk-Taking/Impulsivity—The Balloon Analogue Risk Task (BART) is a behavioral measure of impulsivity and risk-taking.[77,78] The BART displays a computer-generated balloon, programmed to explode randomly, and the participant uses the click of a mouse to gradually inflate the balloon, earning 5 cents per click. After each click, the participant has two options, (1) to continue to inflate the balloon at the risk of bursting it and losing all of the money from that balloon trial, or (2) stop clicking and save the accumulated money to a permanent bank. The primary outcome, adjusted average pumps (i.e., the average number of pumps on trials in which the balloon does not explode), has been shown to relate to self-reports of substance use and other health- risk behaviors.[77,78]

Verbal Learning and Memory—The Revised Hopkins Verbal Learning Test (HVLT-R)[79,80] was used to assess verbal learning and memory. The HVLT-R measures recall for a 12-word list across three learning trials, and after a delay (free recall after 20 minutes). Scoring is normed for participant age. A composite score, used as an index of verbal learning and memory was calculated by taking the average of the T-scores for total recall across the three trials and delayed recall.

Data analyses

Descriptive statistics and alpha reliability coefficients were calculated for study measures. The average drinks consumed per week variable was positively skewed and thus, was log transformed prior to statistical analysis. Zero-order correlations were conducted to assess relations between alcohol use, craving, posttraumatic stress symptom severity, and cognitive variables (processing speed [Trail Making Test Part A], executive functioning [Trail Making Test Part B], risk-taking/impulsivity [BART], verbal learning and memory [HVLT-R]). Correlation analyses were also conducted between clinical severity outcomes and demographic variables (gender, age, race, education) to determine whether demographic variables should be included as covariates in regression models. Four hierarchical multiple regression (HMR) models were tested to determine the extent to which cognitive variables explained variance in alcohol use (average drinks per week, average drinking days per week), craving, and posttraumatic stress symptom severity. In the first three HMRs, posttraumatic stress symptom severity was entered on Step 1 as it was robustly correlated with alcohol outcomes. Cognitive variables were entered on Step 2. In the 4th HMR, posttraumatic stress symptom severity was entered as the dependent variable. Quantity of alcohol consumption and craving was entered on Step 1 and cognitive variables were entered on Step 2. Gender, race, age, and education were trimmed from the HRMs because they

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were not associated with outcomes. All continuous variables were standardized prior to entry.[81]

RESULTS

Descriptive statistics for all study variables and estimates of internal consistency for measures are presented in Table 1. The sample was comprised of heavy drinkers, consuming an average of 12.62 drinks (SD = 8.44; Range 2.24 – 49.29) per drinking day in the 90 days prior to treatment trial enrollment. Participants demonstrated average T-scores on Trail Making Test Part A (processing speed) and B (executive functioning) though T-scores for verbal learning and memory were approximately 1 standard deviation below the mean of 50 indicating somewhat worse performance in this domain relative to the general population. The sample had a mean total score of 56.72 (SD = 13.73) on the PCL indicating moderate to high posttraumatic stress symptom severity and a range generally in line with diagnosis cut off levels standard in PTSD research.[70,71]

Zero-order associations between alcohol use, craving, posttraumatic stress symptoms and cognitive variables

Total posttraumatic stress symptom severity and symptom clusters were positively associated with craving and quantity of alcohol consumption but not frequency. Higher craving was associated with higher quantity and frequency of alcohol consumption. Riskier performance on the BART was associated with higher total posttraumatic stress symptom severity and avoidance and numbing symptoms (Cluster C) as well stronger alcohol obsessions and cravings. Verbal learning and memory, processing speed, and executive functioning were not associated with posttraumatic stress symptom severity. Poorer learning and memory performance on the HVLT was associated with higher quantity and frequency of drinking in the 90 days prior to treatment. Table 2 provides a complete correlation matrix.

Hierarchical Multiple Regression (HMR)

Four independent HMR analyses were conducted to address the primary study objectives. In the first and third HMR, posttraumatic stress symptom severity was positively associated with both average drinks per week and alcohol craving. After controlling for posttraumatic stress symptom severity, lower verbal learning and memory performance was associated with higher average number of drinks per week and average drinking days per week in the first and second HMR and higher risk-taking/impulsivity (BART) was associated with greater alcohol craving in the third HMR. In the fourth HMR, average drinks per week and craving were positively associated with posttraumatic stress symptom severity; cognitive variables demonstrated no relation with posttraumatic stress symptom severity after controlling for alcohol consumption and craving. See Table 3 for details.

DISCUSSION

The aim of the present study was to examine the extent to which key domains of cognitive functioning were associated with measures of clinical severity among trauma-exposed military veterans seeking treatment for AUD. Consistent with patterns commonly reported in

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the literature[82], posttraumatic stress symptom severity was positively associated with quantity of alcohol (but not frequency) consumed in the 90 days prior to treatment and craving for alcohol. After controlling for posttraumatic stress symptom severity, lower verbal learning and memory was associated with higher quantity and frequency of drinking; no relations emerged between processing speed, risk-taking/impulsivity or executive functioning and alcohol use. Higher risk-taking/impulsivity, but not processing speed, verbal learning and memory or executive functioning, was associated with stronger obsessions and cravings for alcohol. After controlling for alcohol consumption and craving, no relations emerged between cognitive functions and posttraumatic stress symptom severity, which suggests cognitive functioning may hold more relevance for alcohol use and craving than for severity of posttraumatic stress symptoms. Overall, this profile of relations highlights that examination of different aspects of cognitive functioning in relation to markers of clinical severity can yield unique information to inform case conceptualization and treatment planning and delivery.

Counter to hypotheses, executive functioning as indexed by Trail Making Test B was not related to any outcomes. This task represents just one component of executive functioning, mental (cognitive) flexibility, and thus may not be sensitive to all executive functions relevant to drinking behavior and craving. Additional tests of mental flexibility and other measures of executive functioning are necessary to definitively assess these complex relationships. In addition, risk-taking/impulsivity as indexed by the BART was unrelated to alcohol use but was positively associated with obsessions and craving for alcohol. Craving represents a form of negative urgency, a facet of impulsivity characterized by a tendency towards rash and impulsive action in the face of negative affect[83], and is highlighted in predominant models of addiction whereby users shift from engaging in reward-seeking behavior (e.g., drinking) to avoiding negative, aversive states.[84,85] Accordingly, risk-taking/impulsivity appeared to hold more relevance for craving and obsessions with alcohol rather than drinking behavior.

Of note, risk-taking/impulsivity was positively associated with posttraumatic stress symptom severity and cluster C symptoms in particular, which include persistent avoidance of trauma-related thoughts, feelings and situational reminders, social disconnection, numbing and restricted range of affect, anhedonia, and poor memory. Future research should examine the extent to which emotion dysregulation, posttraumatic stress symptom severity, and aspects of impulsivity interact to increase the risk for substance abuse and related problems.[86-90] Finally, posttraumatic stress symptom severity was positively associated with risk-taking/impulsivity but not processing speed, executive functioning, or verbal learning and memory. This is consistent with mixed results in the literature concerning the relation between PTSD and cognitive dysfunction among different samples.[21,24,26]

In this heavy-drinking sample, participants tended to perform [on average] within normal limits on normed neuropsychological measures. Yet, even with normal performance, associations with clinical symptom severity emerged. Therefore, although not necessarily disrupted, strengthening of neurocognitive functions critical for achieving emotional and behavioral control may be fruitful in promoting treatment success.[21,22,37,91] Difficulties with learning, memory, cognitive flexibility, inhibition and planning can represent a

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significantobstacle for patients across many aspects of the recovery process (e.g., navigation of a healthcare system, medication management, absorption of clinical materials and implementation of new skills, anticipation of and planning for triggering situations). Further, given that clinicians are often poor at identifying cognitive struggles among substance abusing patients[92], and empirically supported treatments have been slow to recognize and address it[91,93], lack of treatment engagement and progress (e.g., inattention, failure to do homework; denial and minimization of problem severity) may be inappropriately interpreted as treatment-resistance or lack of motivation.[94] Clinicians are thus encouraged to consider a standard assessment of cognitive functioning, in addition to assessing for trauma exposure and posttraumatic stress symptom severity, when treating individuals with AUD.

Limitations and Future Directions

Despite several strengths of this study, including a clinically and theoretically-informed multivariate model and examination of research questions within a treatment-seeking sample, limitations should be noted. First, the current study was cross-sectional thus longitudinal examination is required to confirm directionality of relations. For instance, heavy drinking may cause deficits in verbal learning and memory, which subside as abstinence continues.[27] Second, the current study did not investigate cognitive functioning in relation to treatment outcomes (e.g., relapse, adherence, drop-out) and research is sorely needed to examine such questions within this population. For instance, addressing deficits in learning and memory and elevations in risk-taking and impulsivity may potentially help optimize recovery outcomes among patients with greater clinical severity at treatment entry. Third, in addition to examining clinical severity outcomes, future studies should also consider functional outcomes that capture domains such as occupational and interpersonal functioning and self-care. Fourth, the current sample size was relatively small, and these findings should be replicated in a larger sample to improve generalizability. Fifth, when examining these relations, future studies should control for the potential effects of traumatic brain injury and use of other psychotropic medications (e.g., benzodiazepines) and substances that are known to negatively impact cognition.[95,96] Finally, tests of moderation and mediation may help to elucidate the extent to which neurocognitive dysfunction serves to functionally connect posttraumatic stress symptoms with alcohol use and craving. Specifically, poor cognitive functioning may limit the ability to retrieve and employ adaptive coping skills to avoid or reduce alcohol use when posttraumatic stress symptoms are elevated.

In summary, the current study offers a novel contribution to the literature via a multivariate examination of four key cognitive domains in relation to alcohol use, craving and posttraumatic stress symptom severity among a sample of trauma exposed veterans with AUD. Examination of these research questions within a treatment-seeking sample with a range of posttraumatic stress symptom severity has direct implications for clinicians and researchers to better address the role of cognitive dysfunction in the recovery process. Specifically, risk-taking/impulsivity and verbal learning and memory may offer a malleable target to help reduce risk factors that contribute to poor AUD treatment outcomes, especially among those exposed to traumatic events. One possible clinical practice interpretation is that memory compensatory strategies (e.g., external cuing and reminders, repetition, increased

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monitoring) may be beneficial in helping patients who initially present with higher levels of alcohol consumption. Interventions that address aspects of impulsivity (e.g., contingency management) may be well suited to individuals experiencing high levels of craving upon treatment entry. In addition, neuroscience-informed approaches to remediating disrupted cognitive processes may improve clinical and functional outcomes and reduce public health burdens associated with these recalcitrant and highly comorbid conditions. For instance, interventions to reduce impulsivity and improve cognitive functions (e.g., inhibition, planning, memory) such as computerized cognitive training[97-99] may be used as a preparatory step to precede as well as augment existing empirically supported treatments for this vulnerable and high-risk population.

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Table 1

Descriptive statistics for alcohol use, craving, posttraumatic stress symptoms and measures of cognitive functioning

	M (SD)	Alpha (α)
Average Drinks Per Week Past 90 Days	63.53 (47.77)	
Average Drinking Days Per Week Past 90 Days	5.30 (1.71)	
Obsessive Compulsive Drinking Scale	24.10 (10.11)	.91
Obsessions	8.28 (4.90)	.89
Compulsions	15.82 (6.14)	.84
PTSD Checklist	56.72 (13.73)	.92
B – Re-experiencing	16.06 (5.01)	.88
C – Avoidance/Numbing	22.96 (6.20)	.84
D – Hyperarousal	17.71 (4.33)	.81
Trail Making Test Part A* – Processing Speed	46 (11.22)	
Trail Making Test Part B* – Executive Functioning	50 (13.12)	
Balloon Analogue Risk Task+ – Risk-taking/Impulsivity	37.72 (15.65)	
Hopkins Verbal Learning Test – Verbal Learning*	39.62 (11.32)	
Hopkins Verbal Learning Test – Delayed Recall*	41.66 (11.01)	
Hopkins Verbal Learning Test – Learning Memory Composite*	40.64 (10.50)	

*Standardized T-scores

+n = 64, average adjusted pumps

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Table 2

Correlations of alcohol use, craving, posttraumatic stress symptom severity, and measures of cognitive functioning

		1	2	3	4	5	6	7	8	9	10	11	12
1.	Drinks Per Week	-											
2.	Dnking Days Per Wk	.37**	-										
3.	OCDS	.31*	.26*	-									
4.	Obsessions	.33**	.17	.89**	-								
5.	Compulsions	.25*	.30*	.93**	.68**	-							
6.	PCL	.34**	.24	.41**	.42**	.34**	-						
7.	Re-experiencing	.31*	.16	.40**	.42**	.33**	.88**	-					
8.	Avoidance/numbing	.25*	.24	.35**	.35**	.30*	.90**	.67**	-				
9.	Hyperarousal	.37**	.23	.33**	.34**	.28*	.86**	.68**	.66**	-			
10.	Trail Making Test A	−.16	−.11	−.16	−.12	−.17	−.22	−.17	−.24	−.18	-		
11.	Trail Making Test B	−.02	−.16	−.17	−.14	−.18	−.22	−.20	−.19	−.18	.66**	-	
12.	BART	.22	.09	.39**	.38**	.34**	.28*	.20	.30*	.24	−.03	.07	-
13.	HVLT Composite	−.35**	−.25*	−.17	−.22	−.10	−.04	−.06	−.01	−.08	.13	.04	.04

Note. N = 64-68.

BART = Balloon Analogue Risk Task, HVLT = Hopkins Verbal Learning Test; OCDS = Obsessive Compulsive Drinking Scale; PCL = PTSD Checklist.

* $p < .05$

** $p < .01$.

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Table 3

Results from hierarchical multiple regression analyses

	β	R^2	Δ R^2
Regression 1 DV: Average Drinks per Week Past 90 Days			
Step 1		.12	
PCL - PTSD Symptom Severity	.35**		
Step 2		.25	.11
PCL - PTSD Symptom Severity	.30*		
Trail Making Test Part A	−.13		
Trail Making Test Part B	.13		
BART - Risk Taking	.14		
HVLT - Verbal Learning and Memory	−.31*		
Regression 2 DV: Average Drinking Days per Week Past 90 Days			
Step 1		.06	
PCL - PTSD Symptom Severity	.25*		
Step 2		.14	.08
PCL - PTSD Symptom Severity	.21		
Trail Making Test Part A	.07		
Trail Making Test Part B	−.15		
BART - Risk Taking	.06		
HVLT - Verbal Learning and Memory	−.26*		
Regression 3 DV: Alcohol Craving – Obsessive Compulsive Drinking Scale			
Step 1		.17	
PCL - PTSD Symptom Severity	.41**		
Step 2		.29	.13*
PCL - PTSD Symptom Severity	.29*		
Trail Making Test Part A	.00		
Trail Making Test Part B	−.12		
BART - Risk Taking	.33**		
HVLT - Verbal Learning and Memory	−.17		
Regression 4 DV: Posttraumatic Stress Symptom Severity			
Step 1		.22	
Average Drinks Per Week	.24*		
Alcohol Craving - OCDS	.34**		
Step 2		.27	.05
Average Drinks Per Week	.26*		
Alcohol Craving - OCDS	.28*		
Trail Making Test Part A	−.08		

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	β	R^2	ΔR^2
Trail Making Test Part B	−.12		
BART - Risk Taking	.10		
HVLT - Verbal Learning and Memory	.11		

Note.

n = 63-64. DV = Dependent Variable. Please see Table 1 for a list of abbreviations.

** $p < .01$

* $p < .05$.