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Impact of Alcohol Use, Traumatic Stress, and Cigarette Smoking on Cognitive Functioning in Veterans With Co-occurring Alcohol Use Disorder and Posttraumatic Stress Disorder

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ABSTRACT

Introduction:

Alcohol use disorder (AUD) and PTSD have high rates of co-occurrence in U.S. Military Veterans resulting in incrementally worse functional outcomes relative to having either one of these disorders alone. Cognitive dysfunction can impede one's ability to benefit from standard behavioral AUD and PTSD treatments. Cigarette smoking is also highly prevalent among U.S. Military Veterans, and cognitive dysfunction is associated with chronic cigarette use among individuals with AUD and PTSD independently. However, much less is known about to what extent cigarette smoking further impairs cognitive functioning in individuals with both co-occurring AUD and PTSD.

Materials and Methods:

U.S. Veterans with co-occurring AUD and PTSD ($n = 162$) completed a comprehensive cognitive assessment covering various domains: working memory, processing speed, mental switching, cognitive inhibition, auditory-verbal learning, auditory-verbal memory, and verbal fluency. To examine the impact of alcohol use, traumatic stress, and cigarette smoking on cognitive function, we conducted a three-way interaction examining the moderated effects of smoking status on the association between alcohol use and PTSD symptoms on a composite domain of global cognition.

Results:

Smoking status in Veterans with co-occurring AUD and PTSD moderated the relationship between alcohol use and global cognition ($P = .042$), such that higher levels of alcohol use in the past week were related to worse global cognitive function among Veterans cigarette smokers ($P = .015$) but not among nonsmokers ($P = .833$). On follow-up analyses of individual cognitive domains, greater alcohol use in the past week was associated with lower cognitive inhibition in smokers but not nonsmokers, with traumatic stress symptoms moderating this effect ($P = .039$). Additionally, smoking status moderated the relationship between alcohol use and auditory-verbal learning, such that there was a differential relationship between alcohol use and auditory-verbal learning between smokers and nonsmokers.

Conclusions:

Overall, results provide evidence for the compounding impact of alcohol use, traumatic stress, and cigarette smoking on cognitive functioning. Impaired cognitive performance on a global level as well as on individual domains of cognitive inhibition and auditory-verbal learning were evident. Cognitive dysfunction may impede a Veteran's ability to benefit from therapeutic treatment, and these cognitive domains may represent potential targets for cognitive training efforts. Further, study results support smoking cessation initiatives and smoke-free policies enacted at Veterans Affairs healthcare facilities and medical centers.

INTRODUCTION

Alcohol use disorder (AUD) and PTSD show high rates of co-occurrence in U.S. Military Veterans,[1,2] with the presence of AUD or PTSD increasing the lifetime likelihood of meeting criteria for the other (63% of Veterans with AUD met PTSD criteria; 55-68% of Veterans with PTSD met AUD criteria).[2] Although both AUD and PTSD are each independently associated with poorer functional outcomes,[1,3] co-occurring AUD and PTSD results in an incremental burden (e.g., increased suicidal risk, increased mental health utilization, increased homelessness rates, and decreased functioning) relative to having either one of the disorders.[1,3] Despite accessibility to evidence-based treatments for co-occurring AUD and PTSD and growing evidence supporting the feasibility and effectiveness of these treatments, those with co-occurring AUD and PTSD present with a more complex treatment course and less favorable treatment outcomes relative to those diagnosed with one of these disorders alone.[4] As such, this highlights the importance of identifying transdiagnostic factors present at treatment initiation that could

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impact symptom presentation and lead to improved treatment gains.

One factor that may represent a high-yield AUD/PTSD transdiagnostic target for intervention is cognition. Some of the most common empirically supported therapies for treating co-occurring AUD and PTSD include Seeking Safety, Concurrent Treatment of PTSD and Substance Use Disorders Using Prolonged Exposure, cognitive processing therapy, and integrated cognitive behavioral therapy.[4,5] Critically, a core treatment component across these protocols is cognitive functioning, with all of these treatments being cognitive behaviorally based.[4–6] In fact, cognitive dysfunction is associated with worse outcomes for both AUD and PTSD treatment.[7,8] Cognitive impairment interferes with successful treatment as it poses challenges in changing well-established maladaptive behavioral and cognitive patterns.[6,9] Conversely, cognitive rehabilitation enhances PTSD and AUD treatment recovery.[7,9]

Despite the critical role of cognition in AUD and PTSD treatment, there is less focus in treatment research on the impact of cognitive impairments with AUD and PTSD symptomatology. Greater alcohol use has been associated with cognitive dysfunction across many domains, including poorer performance on measures of executive functioning and learning and memory.[10,11] Multiple meta-analyses have found PTSD to be consistently associated with cognitive decline,[12,13] especially in domains of verbal learning, processing speed, attention/working memory, and executive functioning.[13] In particular, increased traumatic stress has also been independently associated with cognitive dysfunction. Taken together, cognitive function necessary for therapeutic behavioral change also tends to be impaired initially across both AUD and PTSD.[6]

A marked concern impacting cognition in those with co-occurring AUD and PTSD is the impact of cigarette smoking. Cigarette use is one of the most prevalent types of substance misuse, with especially high prevalence among U.S. Veterans.[14–17] Combined heavy alcohol and cigarette use has been found to have a compounding, deleterious effect on cognitive function,[14,18,19] with the combination predicting poorer cognitive performance in general.[14] In fact, greater smoking severity in AUD individuals has been associated with decreased cognitive proficiency and executive functioning,[14,16,20] whereas nonsmoking AUD individuals performed better on measures of set-shifting and processing speed.[14,21] Additionally, studies found that nonsmoking AUD individuals exhibit significantly greater improvement during the initial weeks to months in early recovery in multiple domains of cognitive functioning as compared to cigarette smoking AUD individuals.[14,22] In contrast with alcohol use, to our knowledge, only one study has examined the impact of co-occurring PTSD and smoking on cognitive function.[23] Erjavec et al.[23] found that cigarette smoking was a significant predictor of cognitive decline in Veterans with PTSD. Although previous research has found cognitive dysfunction to be associated with chronic smoking among individuals with AUD and PTSD independently,[11,14,16,20,24] it is unclear to what degree smoking further impairs cognitive functioning in individuals with both co-occurring conditions.

The objective of the current study was to further examine the effects of alcohol use, traumatic stress, and cigarette smoking on cognitive functioning in Veterans with co-occurring AUD and PTSD. Since smoking can have a compounding, negative effect with alcohol use on cognitive function,[14,16,18–20] we hypothesized that smoking status in Veterans with co-occurring AUD and PTSD would moderate the relationship between alcohol use and cognition, such that Veterans who smoke would show greater cognitive dysfunction relative to nonsmokers at higher levels of alcohol use. We also hypothesized that traumatic stress would moderate this relationship.

METHODS

Participants

Participants were U.S. Military Veterans ($n = 162$) who underwent screening procedures in the Addiction Research Program at the San Francisco Veterans Affairs Health Care System (SFVAHCS). Primary inclusion criteria for this study were as follows: aged 18-69, met diagnostic criteria for Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) AUD in the past month, met standard recommended PTSD cutoff (i.e., 33 or greater) on the PTSD Checklist for DSM-5[25] (PCL-5), and met criteria for "at risk" or "heavy" levels of recent alcohol use (defined by the National Institute on Alcohol Abuse and Alcoholism; as at least 15 standard alcohol drinks per week on average over the past 4 weeks for men or at least eight standard alcohol drinks on average over the past 4 weeks for women). Primary exclusion criteria included unstable psychiatric or medical conditions and experiencing acute alcohol withdrawal (Clinical Institute Withdrawal Assessment score >11 at assessment).

Psychiatric Assessment

Alcohol use

DSM-5 AUD diagnosis was confirmed using the Structured Clinical Interview for DSM-5.[26] Quantity and frequency of past week alcohol use were assessed using the timeline followback (TLFB) method.[27] Interview data were used to calculate the number of alcohol drinks in the past week and number of heavy alcohol use days in the past week.

Traumatic stress

PTSD symptom severity was assessed using the 20-item self-report PCL-5,[25] which includes intrusion, avoidance, negative cognitive mood, and arousal subscales.

Cigarette use

Quantity and frequency of cigarette use were assessed using the TLFB method.[27] Interview data were used to calculate

the average number of cigarettes smoked in the past 90 days. To examine the effects of smoking on cognitive functioning, we classified participants by smoking status (i.e., nonsmokers or smokers). Nonsmokers ($n = 66$) were those who smoked zero cigarettes within the past 90 days. Smokers ($n = 96$) were those who smoked any cigarettes in the past 90 days, with these participants smoking on average 72.43 cigarettes in the past week and 72.36 cigarettes per week within the past 90 days.

Cognitive Assessment

Cognitive domains

Participants completed a battery of cognitive assessments covering a broad range of cognitive domains. Raw scores on cognitive assessments were converted to standardized *t*-scores using appropriate normative data (adjusted for age, gender, ethnicity, and/or years of education).

Working memory

Working memory is an individual's short-term system for storing and managing information necessary to carry out cognitive tasks. Working memory was calculated by averaging Scaled Scores on the Wechsler Adult Intelligence Scale—Fourth Edition[28] Arithmetic and Digit Span subtests. Scores were normed for participant age.

Processing speed

Processing speed is the pace at which an individual performs a cognitive task. Processing speed was calculated by averaging scores on Trail Making Test A,[29] Stroop Word,[30] and Stroop Color.[30] Scores were normed for age, gender, education, and race/ethnicity using Heaton Compendium Norms.[31]

Mental switching

Trail Making Test B is an index of executive functioning that involves both mental set-shifting and flexibility.[32] Scores were normed for age, gender, education, and race/ethnicity using Heaton Compendium Norms.[31]

Cognitive inhibition

The Stroop Color and Word Test[30] is an index of cognitive inhibition that assesses an individual's ability to inhibit cognitive interference, which occurs when the processing of a stimulus feature impedes the simultaneous processing of a different stimulus feature.[30] Scores were normed on age, gender, education, and race/ethnicity using Heaton Compendium Norms.[31]

Auditory-verbal learning

The Hopkins Verbal Learning Test—Revised[33] (HVLT-R) was used to assess learning. Total sum of learned words across the three learning trials was recorded, and scores were normed for participant age.

Auditory-verbal memory

Auditory-verbal memory was calculated by averaging three other scores from the HVLT-R[33] (HVLT-R Delayed Recall, HVLT-R Retention, and HVLT-R Recognition Discrimination Index). HVLT-R scores were normed for participant age.

Verbal fluency

Verbal fluency is the cognitive ability that facilitates the retrieval of verbal information from memory. Verbal fluency was calculated by averaging scores on the Controlled Oral Word Association Test[34] FAS and Animals. Scores were normed on age, gender, education, and race/ethnicity using Heaton Compendium Norms.[31]

Global cognition

A global cognition composite was calculated by averaging standardized *t*-scores across all the cognitive domains previously listed.

Data Analyses

Moderated moderation models

We utilized a moderated moderation model (Hayes PROCESS Model 3; see Supplementary Fig. 1), where smoking status (categorical moderator W) and traumatic stress (PCL-5 total; continuous moderator Z) are entered as moderators of the primary association between alcohol use (mean-centered past week number of alcohol drinks; independent variable X) on global cognition (dependent variable Y). Age and years of education were included as covariates (C_1 and C_2, respectively) in the model, and these covariates were trimmed from the final model when not predictive of the dependent variable. To further parse interactions, we plotted interactions between cognitive function at different levels of alcohol use and total traumatic stress (low, mid, and high) based on the mean $\pm$ 1 *SD*. Similar models were used in follow-up assessments to examine each of the cognitive domains making up the global cognition composite score.

Correlations

We also examined the associations between number of alcohol drinks in the past week, number of heavy alcohol use days, traumatic stress (total score and intrusion, avoidance, negative cognitive mood, and arousal subscale scores), cigarette use in the past week, and cognitive functioning (global and individual cognitive domains) via pairwise correlations. Number of alcohol drinks in the past week, number of heavy alcohol use days in the past week, and number of cigarettes smoked in the past week are count outcomes, and correlations between count outcomes and cognitive domains were analyzed via Spearman correlations. Cognitive domains and PTSD symptom cluster outcomes are continuously scaled, and correlations were examined with Pearson correlations. As 66 participants were

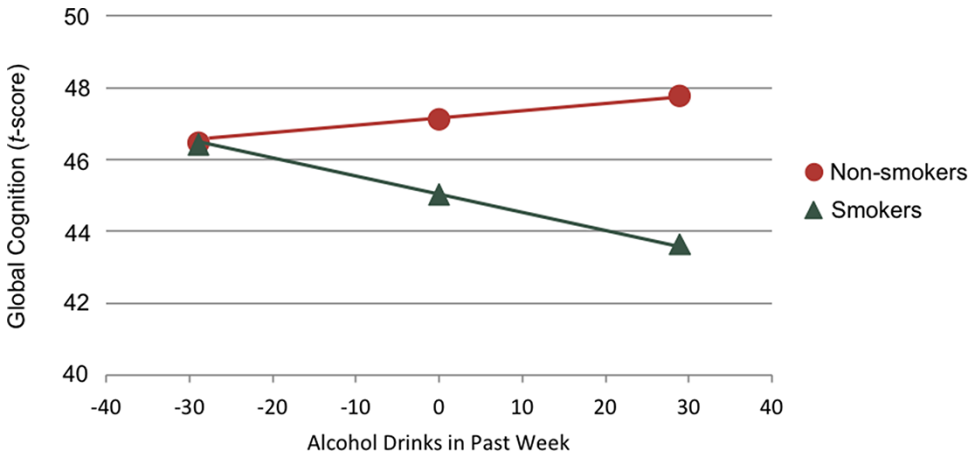

FIGURE 1. Alcohol use × Smoking Status predicted global cognition ($P = .042$), such that greater alcohol use in the past week was associated with lower global cognition in smokers but not nonsmokers.

nonsmokers, we examined correlations separately by smoking status.

Procedure

All participants provided informed consent, and study procedures were approved by the University of California, San Francisco, and the SFVAHCS. The study was conducted in accordance with the latest version of the Declaration of Helsinki. Data and analyses presented in the study were from standard screening assessment sessions prior to beginning any involvement in specific studies conducted by the Addiction Research Program.

RESULTS

Participant Characteristics

Descriptive statistics of participant characteristics and psychiatric assessment variables are presented in Table I. Participants were heavy alcohol drinkers [as defined by NIAAA], drinking on average 33.88 alcohol drinks in the past week. For traumatic stress, PCL-5 scores were indicative of moderate to high posttraumatic stress symptom severity.

TABLE I. Participant Characteristics

Characteristics	Nonsmokers (mean ± *SD*)	Smokers (mean ± *SD*)
Male *n* (%)	56 (84.85)	90 (93.75)
Age	50.78 ± 12.83	50.37 ± 12.13
Education	14.68 ± 2.15	13.38 ± 1.40
Race[a] *n* (%):		
White (Hispanic/Latinx)	33 (50.00)	39 (40.63)
Black/African American	14 (21.21)	33 (34.38)
Asian	3 (4.55)	2 (2.08)
Native American/Alaskan Native	0 (0.00)	1 (1.04)
Native Hawaiian/Pacific Islander	1 (1.52)	1 (1.04)
Mixed race	9 (13.64)	16 (16.67)
Unknown/not reported	6 (9.09)	4 (4.17)
Cigarettes in past week	0 (0.00)	72.43 ± 51.98
Alcohol drinks in past week	32.04 ± 26.79	35.14 ± 30.37
Heavy drinking days in past week	2.77 ± 2.38	2.84 ± 2.51
PCL-5 total	60.21 ± 15.83	63.98 ± 14.03
PCL-5 intrusion	14.68 ± 4.92	15.41 ± 3.97
PCL-5 avoidance	20.92 ± 6.02	22.20 ± 5.61
PCL-5 negative cognitive mood	21.08 ± 6.33	22.43 ± 5.86
PCL-5 arousal	18.47 ± 4.92	19.76 ± 4.54
Military combat, *n* (%)	27 (40.91)	36 (37.50)

PCL-5 = PTSD Checklist for DSM-5.
[a]Racial identity was self-reported by participants. These data were collected to help describe the study sample.

Cognitive Descriptive Statistics

On average participants scored within the normal range on global cognition and the individual cognitive domains (*t*-scores = 43.57-50.52), with evidence of low average performance for the auditory-verbal learning domain (*t*-score = 37.47; Table II). Of note, a meaningful proportion of our co-occurring AUD and PTSD participants scored within the low average ($35 < t$-score ≤ 42) and borderline/impaired range (t-score ≤ 35) on multiple cognitive domains. For global cognition, 22.84% of participants scored within the low average range and an additional 2.47% of participants scored within the borderline/impaired range. Across the individual cognitive domains, a large proportion of the co-occurring AUD and PTSD participants scored in the low average to borderline/impaired range (16.05-66.67%), with the lowest performances for auditory-verbal learning (low average = 19.75%; borderline/impaired = 46.91%), auditory-verbal memory (low average = 18.52%; borderline/impaired = 30.25%), and cognitive inhibition (low average = 29.01%; borderline/impaired = 16.67%) domains.

TABLE II. Global Cognition and Cognitive Domains *t*-scores and Proportion of Participants Scoring in the Low Average and Borderline/Impaired Range

	t-score (mean ± *SD*)	Low average (%) (35 < *t*-score ≤ 42)	Borderline/ impaired (%) (*t*-score ≤ 35)
Global cognition	45.85 ± 5.55	22.84	2.47
Cognitive domains			
Working memory	47.51 ± 7.88	23.46	6.17
Processing speed	43.61 ± 5.83	25.93	9.26
Mental switching	49.76 ± 9.97	18.52	6.79
Cognitive inhibition	43.57 ± 9.35	29.01	16.67
Auditory-verbal learning	37.47 ± 11.38	19.75	46.91
Auditory-verbal memory	40.61 ± 10.64	18.52	30.25
Verbal fluency	50.52 ± 8.57	11.73	4.32

SD = standard deviation.

Global Cognition Moderated Moderation

For global cognition (see Supplementary Table I for full final model results), the three-way interaction of Alcohol Use × Smoking Status × Traumatic Stress did not significantly predict global cognition scores ($P = .363$). However, the two-way interaction of Alcohol Use × Smoking Status significantly predicted global cognition ($P = .042$), such that greater alcohol use in the past week was associated with lower global cognition in smokers but not nonsmokers (Fig. 1).

Cognition Domains Moderated Moderation

In follow-up analyses, we examined moderated moderation models for each of the cognitive domains (see Supplementary Table II for full final model results). The three-way interaction of Alcohol Use × Smoking Status × Traumatic Stress significantly predicted cognitive inhibition scores ($P = .039$), when accounting for the positive association with greater years of education ($P = .017$). Parsing this three-way interaction, greater alcohol use in the past week was associated with lower cognition inhibition in smokers ($\beta = -.04$, $P = .015$) but not nonsmokers ($\beta = .01$, $P = .832$), with total traumatic stress moderating this moderation at low ($\beta = -.27$, $P = .002$) and average ($\beta = -.17$, $P = .003$), but not high ($\beta = -.07$, $P = .228$), levels of traumatic stress (Fig. 2A).

Although the three-way interaction of Alcohol Use × Smoking Status × Traumatic Stress did not significantly predict auditory-verbal learning scores ($P = .698$), the two-way interaction of Alcohol Use × Smoking Status significantly predicted auditory-verbal learning ($P = .036$), such that there was a differential relationship between alcohol use and auditory-verbal learning between smoking and nonsmoking groups (Fig. 2B). The within group (smoking and nonsmoking groups) association between alcohol use and learning was not significant upon post hoc regression analyses.

There were no other significant main effects or interactions ($ps > 0.123$) for other cognitive domains (working memory, mental switching, auditory-verbal memory, and verbal fluency). There was a statistical trend for nonsmokers to have greater processing speed relative to smokers ($P = .059$).

Correlations Among Alcohol Use, Traumatic Stress, Cigarette Use, and Cognitive Functioning

Among those who smoked cigarettes, number of alcohol drinks in the past week was positively correlated with PCL-5 total ($r_s = 0.22$, $P = .031$) and PCL-5 avoidance ($r_s = 0.31$, $P = .002$). Average number of heavy alcohol use days per week in the past week was positively correlated with total PCL-5 arousal ($r_s = 0.25$, $P = .015$). Additionally, number of cigarettes smoked in the past week was positively correlated with PCL-5 avoidance ($r_s = 0.25$, $P = .016$). Global cognition and other cognitive domains were not significantly correlated with alcohol use, traumatic stress, or cigarette use.

Among nonsmokers, there were no significant correlations between alcohol use, traumatic stress symptoms, and cognitive functioning.

DISCUSSION

The current study examined the effects of alcohol use, traumatic stress, and cigarette smoking on cognitive functioning in Veterans with co-occurring AUD and PTSD. We examined the moderating effect of smoking status and traumatic stress on the association between alcohol use on cognition. Smoking status moderated the relationship between alcohol use and global cognition, such that smokers showed greater global cognitive dysfunction relative to nonsmokers at higher levels of alcohol use. We then examined follow-up analyses of each cognitive domain. For cognitive inhibition, greater alcohol use in the past week was associated with lower cognitive inhibition in smokers but not nonsmokers, with traumatic stress symptoms moderating this effect at low and average levels of traumatic stress. For auditory-verbal learning, smoking status moderated the relationship between alcohol use and cognition. There was a differential relationship between alcohol use and auditory-verbal learning between smoking and nonsmoking group. There was also a statistical trend for smoking status (which did not interact with alcohol use or traumatic stress), such that nonsmokers tended to have greater processing speed relative to smokers.

Consistent with research showing impaired cognitive performance in individuals with AUD[10,11] and PTSD,[12,13] over 25% of our Veterans with co-occurring AUD and PTSD participants scored within the low average and borderline/impaired range on global cognition and across the individual cognitive domains. Across the different cognitive domains, Veterans exhibited the lowest performance for auditory-verbal learning, auditory-verbal memory, and cognitive inhibition with between 46% and 67% scoring in the low average to borderline/impaired range. Impairment of these cognitive domains in this Veteran population is consistent with previous research[10–13] and highlights the importance of assessing

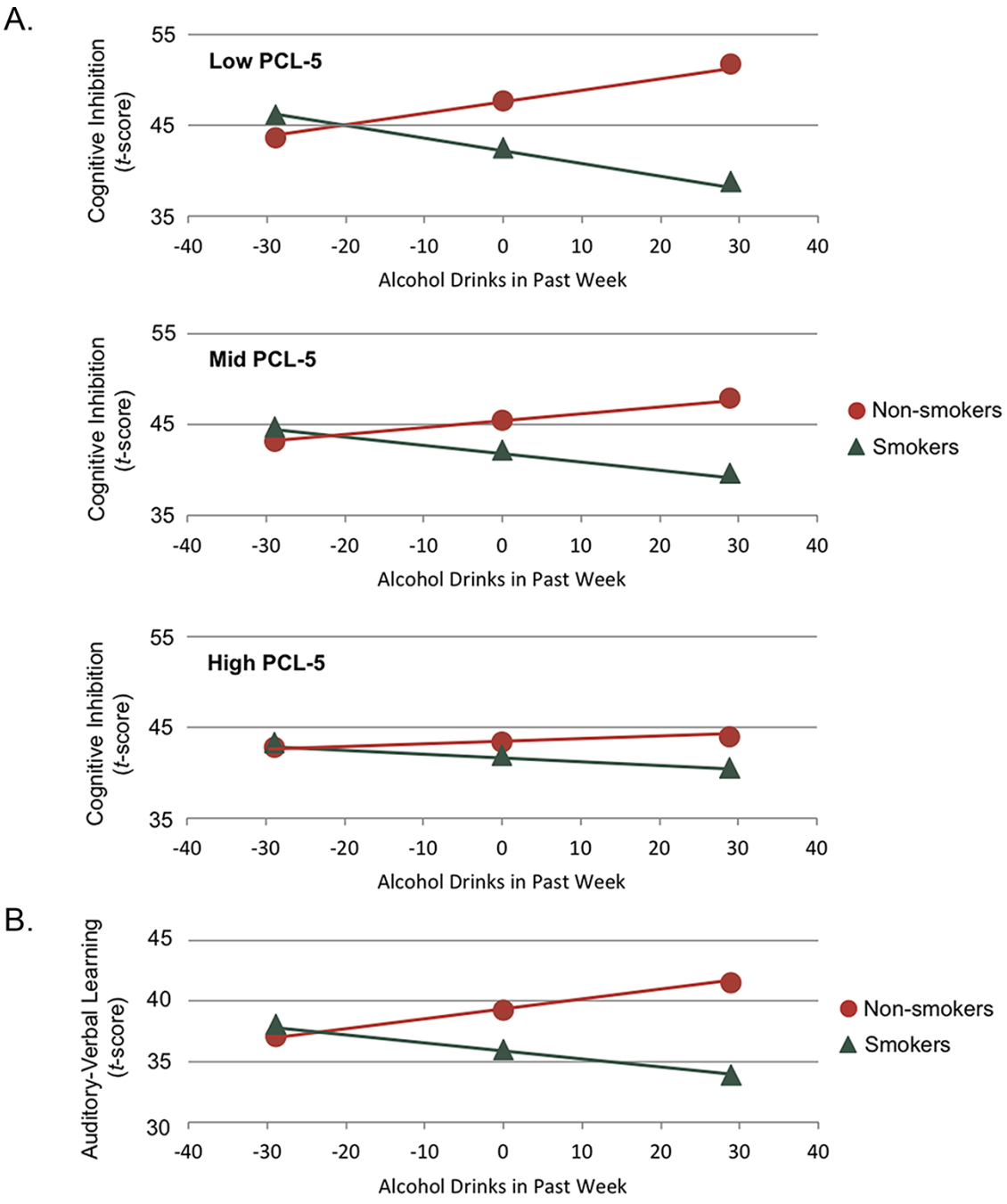

FIGURE 2. (A) Alcohol Use × Smoking Status × Traumatic Stress predicted cognitive inhibition scores ($P = .039$). Greater alcohol use in the past week was associated with lower global cognition in smokers but not nonsmokers, with traumatic stress moderating this moderation at low ($P = .002$) and average ($P = .003$) levels of traumatic stress. (B) Alcohol Use × Smoking Status predicted auditory-verbal learning ($P = .036$), with different relationships between alcohol use and auditory-verbal learning between smoking and nonsmoking groups.

cognitive function and targeting cognitive deficits in order to enhance treatment benefits.

Results from the current study provide further evidence of cognitive dysfunction in Veterans with co-occurring AUD and PTSD and establish the additional detrimental impact cigarette smoking has on cognition. Previous work has shown that nonsmoking AUD individuals exhibit significantly greater cognitive performance and improvement on auditory-verbal learning and processing speed compared to smoking AUD individuals during early recovery.[22,24] Results from the current study are consistent with evidence that co-occurring cigarette smoking and alcohol use in individuals with AUD has a worse negative effect on cognition than just alcohol alone.[14,35]

In regard to our hypothesis on traumatic stress, we found a significant three-way interaction, such that greater alcohol

use in the past week was associated with lower cognitive inhibition in smokers but not nonsmokers, with total traumatic stress moderating this association at low and average levels of traumatic stress, but not high levels of traumatic stress. As traumatic stress scores increased in smokers, the strength of the relationship between recent alcohol use and cognitive inhibition decreased. PTSD appears to have a synergistic effect with alcohol use and smoking on cognitive inhibition. It may be that the impact of alcohol use and cognitive inhibition may not be readily seen among smokers with high levels of PTSD, as PTSD symptoms may have a greater impact on cognitive inhibition and negate the individual effects of alcohol and smoking. In fact, Veteran participants with high PTSD symptoms had lower overall cognitive inhibition scores, regardless of alcohol use or smoking status. This finding is consistent with research finding that greater smoking severity in AUD individuals is associated with decreased cognitive proficiency and executive functioning.[14,16,20] More research is needed to replicate and further examine this result. Additionally, it could be that increased traumatic stress triggers AUD[36] as well as cigarette smoking and not solely that individuals with co-occurring AUD and PTSD self-medicate with cigarettes to cope with increased traumatic stress. Future studies should further examine the temporal relationships among AUD, traumatic stress, and cigarette use.

Cognitive impairment can result in patient difficulties in making treatment gains.[9] Cognitive inhibition and auditory-verbal learning are particularly important for maximizing benefits of many cognitive behavioral treatments for co-occurring AUD and PTSD.[7,8] Cognitive inhibition allows the mind to exclude stimuli that are irrelevant to the task at hand and is a key executive function necessary to stop automatic responses to cues and cues that result in continued alcohol and cigarette use.[37] Cognitive inhibition is also important in emotion regulation after trauma, and impairments in cognitive inhibition could limit the effectiveness of AUD/PTSD treatments that rely on autobiographical memory retrieval, prolonged exposure, and cognitive reappraisal.[38] Further, auditory-verbal learning is key to the ability to actively learn from what one hears and is necessary in psychotherapy treatment, as patients need to be able to learn new information and recall information that is being taught.

Cognitive rehabilitation has been found to augment successful treatment recovery in AUD and PTSD populations.[39] As such, the dysfunctional cognitive domains observed in the current study represent potential cognitive training targets for future intervention studies that attempt to utilize cognitive training to augment and maximize current therapies. Performance on specific cognitive tests can also be used to inform clinical treatment approaches as well as for developing an individual's risk for relapse profile. Despite promising research results and multiple technological approaches to treatment—that make for more convenient and flexible treatment interventions—implementation of cognitive rehabilitation into clinical practice is mostly absent.[39] Research that delves into the impact that cognitive function has on treatment outcome as well as the application of cognitive training within populations with substance use disorder and common co-occurring conditions is warranted. Similarly, policies that target smoking cessation during early recovery in Veterans with co-occurring AUD and PTSD are likely beneficial in increasing cognitive functioning, with research finding that smoking cessation is associated with rapid recovery in cognitive functioning.[40] In turn, this increase in cognitive functioning could lead to improved treatment outcomes.

Despite several strengths of this current study, including an assessment of a broad array of cognitive domains in a co-occurring AUD and PTSD treatment-seeking sample, there are limitations worth noting. The first limitation is that we only assessed for cigarette smoking and did not account for other forms of nicotine use, such as chewing or vaping. Future studies should assess for all forms of nicotine use. Another limitation is the cross-sectional nature of the research design, which does not allow for directional interpretations of results. Hence, it is unclear if smoking-related cognitive dysfunction is a predisposing factor or a consequence of cigarette use. Longitudinal studies are needed to clarify directionality of smoking-related dysfunction, assess whether negative smoking effects on cognition are permanent, and assess whether cognition will improve with reduced alcohol use or improved PTSD symptoms. Although our cognitive assessment battery was robust, another limitation was that there were some important areas of cognition that were not assessed (e.g., judgment, planning, decision-making, and motor inhibition) and could be important to examine in contexts of treatment-seeking individuals with comorbid substance use. Lastly, we posit that cognitive training and smoking cessation policies could be beneficial in improving cognition and treatment outcomes; however, we did not test this hypothesis in the current study. As such, future studies should examine additional domains of cognition and the effects of cognitive rehabilitation and smoking cessation on treatment outcomes in Veterans with co-occurring AUD and PTSD.

CONCLUSIONS

Overall, results from the current study can help to inform both measurement-based care and policy. Due to the deleterious effect of smoking on cognitive functioning, treatment providers should assess for cigarette smoking and related cognitive dysfunction, as these may delay and obstruct AUD and PTSD treatment progress and improvement. Additionally, as cigarette smoking is a prevalent behavior,[14–17] especially among Veteran populations, and cigarette use, alcohol use, and traumatic stress are highly comorbid,[17] treatment providers for AUD and PTSD should consider a treatment design that concurrently targets smoking cessation in this population subgroup since addressing cigarette smoking-related cognitive dysfunction could improve treatment outcomes. These results also have important implications for policy. Recently, VA healthcare facilities as well as other medical

centers nationwide have been instituting cigarette smoking reduction initiatives as well as cigarette smoking cessation initiatives. These results lend support for the importance of these initiatives, and future research should examine the impact of these policies.

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SUPPLEMENTARY MATERIAL

Supplementary material is available at *Military Medicine* online.

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CONFLICT OF INTEREST STATEMENT

None declared.

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