Cue-Dependent Inhibition in Posttraumatic Stress Disorder and Attention-Deficit/Hyperactivity Disorder

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Highlights

- Response inhibition was compared in 161 adult men with PTSD and/or ADHD and controls.
- PTSD and ADHD+PTSD were linked to impaired response execution and inhibition.
- PTSD and ADHD+PTSD showed slower, more variable reaction time relative to controls.
- No cue dependency differences were observed by PTSD or ADHD diagnosis.

Abstract

Objective: Attention-deficit/hyperactivity disorder (ADHD) and posttraumatic stress disorder (PTSD) are common among military veterans, but the comorbidity of these two psychiatric disorders remains largely unstudied. Evaluating response inhibition and cue-dependent learning as behavioral and neurocognitive mechanisms underlying ADHD/PTSD can inform etiological models and development of tailored interventions.

Method: A cued go/no-go task evaluated response inhibition in 160 adult males. Participants were recruited from the community and a Veterans Administration medical center. Four diagnostic groups were identified: ADHD-only, PTSD-only, ADHD+PTSD, controls.

Results: Group differences were observed across most indices of inhibitory functioning, reaction time, and reaction time variability, whereby PTSD-only and ADHD+PTSD participants demonstrated deficits relative to controls. No cue dependency effects were observed.

Conclusion: Findings complement prior work on neurocognitive mechanisms underlying ADHD, PTSD, and ADHD+PTSD. Lack of expected group differences for the ADHD-only group may be due to limited power. Additional work is needed to better characterize distinctions among clinical groups, as well as to test effects among women and youth.

Keywords: ADHD; PTSD; comorbidity; response inhibition; cued reaction time task; executive functioning

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Cue-Dependent Inhibition in Posttraumatic Stress Disorder and Attention-Deficit/Hyperactivity Disorder

Post-traumatic stress disorder (PTSD) is a commonly occurring and debilitating condition following exposure to life-threatening events (e.g., physical assault, sexual assault, combat exposure). Epidemiological studies suggest that even though the vast majority of US citizens experience a traumatic event in their lifetime (Breslau, 2009), the 12-month prevalence of PTSD in US adults is only 3.5%. However, this rate is substantially higher among veterans. For example, 48.5% of Operation Enduring Freedom (OEF) or Operation Iraqi Freedom (OIF) soldiers screened positive for PTSD symptoms (Khaylis, 2011). Further, 46% of active duty or retired OEF/OIF soldiers met diagnostic criteria for PTSD (Pittman, 2012). In addition to the high rates of PTSD in veterans, lifetime comorbidity of PTSD with any psychiatric disorder (e.g., depression, substance use disorders, anxiety disorders) has been found to be as high as 88%.

Attention-deficit/hyperactivity disorder (ADHD) has also been shown to commonly occur among military personnel with estimates at approximately 10% (Antshel, 2013; Hanson, 2012), whereas estimates of adult ADHD in the general population range from 1.6-4.4% (Kessler, 2006; Simon, 2009). ADHD is estimated to co-occur with PTSD at rates ranging from of 12-28% in veterans (Adler, 2004; Harrington, 2012). Rates of PTSD among adults with ADHD were six times higher than among adults without ADHD (Antshel, 2013). Furthermore, research has demonstrated that ADHD symptom severity significantly predicts PTSD symptom severity (Harrington, 2012).

Though research has begun to uncover the increased risk for PTSD among individuals with ADHD, less is known about the mechanisms by which these two disorders co-occur. Neurocognitive performance represents one potential area that may help to explain ADHD-

PTSD comorbidity. Given that both ADHD and PTSD literatures individually identify deficits in neurocognitive performance (Barkley, 1997; Bremner, 1999; Buckley, 2000), investigating specific facets of neurocognitive functioning signifies a promising line of research. Understanding neurocognitive performance among people with PTSD, ADHD, comorbid ADHD-PTSD, and healthy controls will help in building more sophisticated etiological models of the disorders, and help guide targeted treatments that address underlying deficits.

One aspect of neurocognition that has been routinely explored in individuals with ADHD is response inhibition or the inhibition of prepotent or ongoing responses (Barkley, 1997; Oosterlan, 1998). This deficit in response inhibition and interference control has been suggested as a basis for the core features of ADHD (Barkley, 1997). Response inhibition has been routinely measured using the "Go/No Go" Task in which there are two stimuli, a "go" and a "no-go." Participants are then instructed to press a button, as quickly as possible, when a "go" stimulus is presented and to inhibit this response when a "no-go" stimulus is presented (Simmonds, 2008). Research suggests adults with ADHD show lower accuracy rates, increased commission errors, increased response interference, and a decreased likelihood to consciously detect these errors (Morein-Zamir, 2014; O'Connell, 2009; Sebastian, 2012; Woltering, 2013).

Response inhibition also has been investigated, albeit to a lesser degree, within the PTSD literature. According to the DSM-5, PTSD is comprised of four clusters of symptoms: re-experiencing symptoms, avoidance, negative cognitions/mood, and arousal (American Psychiatric Association, 2013). Kertzman and colleagues (2014) pointed to re-experiencing symptoms as well as sensory stimuli leading to cognitive deficits such as response inhibition in patients with PTSD. Self-reported behavioral inhibition is significantly related to PTSD symptoms, especially PTSD symptoms in the avoidance cluster among veterans (Myers, 2012).

Research using the Go/No-Go task has demonstrated that veterans with PTSD show a decreased

inhibitory response and greater false-alarm rate (Tillman, 2010) as well as a significantly more

variable reaction times (Swick, 2013) when compared to veterans without PTSD. Several studies

have investigated behavioral and neural processing deficits in both inhibitory functioning and

evaluation of contextual cues in veterans and civilians with PTSD, in line with evidence that

problems in inhibiting a fear response and interpreting environmental safety vs. danger

information may underlie symptoms of the disorder (Falconer, 2012; Jovanovic, 2012;

Jovanovic, 2013; van Rooij, 2015). Results from those studies suggest that behavioral and

neural deficits in inhibition and contextual cue processing may extend beyond fear responses in

PTSD and may be more generalized, core deficits of the disorder (Jovanovic 2012; Jovanovic

2013; van Rooij, 2014; van Rooij, 2015). Although some of the studies evaluated the potential

effects of comorbid psychiatric conditions (e.g., mood disorders), none directly reported on the

potential influence of ADHD on observed group differences.

Little is known about the inhibitory functioning among individuals with ADHD and

comorbid PTSD. To our knowledge there have been no studies that have looked at response

inhibition in groups of veterans and civilians with ADHD, PTSD, and comorbid ADHD-PTSD

compared to veterans and civilians without a history of psychopathology. It is unknown whether

people with comorbid ADHD-PTSD have worse inhibitory functioning than people with either

disorder alone.

When evaluating inhibitory responses, it is important to consider the potential influence

of environmental factors, such as cues that may guide response preparation. Given the symptoms

seen in PTSD, especially those related to arousal and hypervigilance (American Psychiatric

Association, 2013), veterans with PTSD may be especially attuned to environmental cues to

inform behavioral control (e.g., vigilance for danger cues). Some evidence suggests that valid

cues can aid in the anticipation of an appropriate response against inhibitory impairment. For

example, in alcohol use literature, antecedent cues during the Go/No-Go task can help dampen

inhibitory deficits when individuals are under the influence of alcohol (Marczinski, 2005).

However, the use of antecedent cues during the Go/No-Go task has not been shown to

significantly improve response inhibition deficits in adults with ADHD (Roberts, 2016). It is

unknown whether having a diagnosis of PTSD would influence cue dependency among veterans

and community adults with and without comorbid ADHD.

The current study seeks to investigate how response inhibition is influenced by diagnoses

of ADHD, PTSD, or comorbid ADHD-PTSD in veterans and civilians compared to healthy

controls. Furthermore, the study will examine how antecedent cues effect response inhibition in

these groups. We predicted that participants with ADHD – with and without PTSD – would

demonstrate inhibitory deficits relative to controls. We also hypothesized that participants with

PTSD would demonstrate greater cue dependency than participants without PTSD.

Method

Participants

Participants in this study were 160 men (mean age = 30.8 years, SD = 7.6; 75.2%

white/Caucasian; 14.9% black/African-American; 3.0% biracial; 3.1% Hispanic). Participants

were either recruited from the local community or through a large Veterans Administration (VA)

Medical Center in the southeastern U.S. Participants enrolled from the VA were combat

veterans of Operation Iraqi Freedom/Operation Enduring Freedom (OIF/OEF) missions. Of

enrolled participants, 17 met criteria for ADHD only (47% veterans), 25 met criteria for PTSD

only (100% veterans), 26 met criteria for ADHD+PTSD (100% veterans), and 93 participants

met criteria for neither ADHD nor PTSD (23% veterans). Basic demographic characteristics for each group are summarized in Table 1.

   Inclusion and exclusion criteria. To be eligible for this study, participating veterans were required to have a history of combat exposure, as evidenced by formal release or discharge paperwork (i.e., DD Form 214), a report of combat exposure during the interview with a psychiatrist [masked], and a minimum score of 10 on the Combat Exposure Scale (CES) (Lund, 1984). Participants were not required to meet diagnostic criteria for PTSD or ADHD to enroll, and participants with major depression, and anxiety disorders were included. Subjects with other Axis I psychiatric disorders were excluded from this study; this included current or lifetime DSM-IV schizophrenia, other psychotic disorders, bipolar disorder, and active substance abuse or dependence in the past six months. Individuals with a past history of substance abuse and dependence were included if the last use of the substance was over 6 months prior to the enrollment. There were no inclusion or exclusion criteria based on sociodemographic characteristics.

Procedure

   The study protocol was approved by the Institutional Review Board (IRB) of the academic institution where this research was conducted. A brief description of the study, including explanation of the voluntary nature of participation, was given to potential participants by a trained research assistant. People who expressed interest in participation were screened to determine eligibility for study involvement using the inclusion and exclusion criteria described below. Institutionally approved informed consent was obtained from all participants before the protocol began.

After collecting demographic and deployment information, participants were assessed by a trained research assistant for the presence of psychiatric disorders with the *Mini-International Neuropsychiatric Interview* (MINI) (Lecrubier, 1997). PTSD symptoms were assessed with the Clinician Administered PTSD Scale (CAPS) (Blake, 1995), and ADHD symptoms were assessed with the Conners Adult ADHD Diagnostic Interview for *DSM-IV* (CAADID) (Conners, 1999). A board-certified psychiatrist (author initials) interviewed participants for combat exposure history and to confirm PTSD status per *DSM-IV* criteria, as well as other major psychiatric illnesses that would be exclusionary. Participants who met eligibility criteria were allowed to proceed with further assessments, including the Conners' Adult ADHD Rating Scales (CAARS) and a psychiatric clinical interview to determine ADHD diagnostic status. All measures were completed during a single visit to the clinical laboratory.

Materials

ADHD Symptoms: Conners' Adult ADHD Rating Scales–Self-Report: Short Version (CAARS-S:S). The CAARS–S:S (Conners, 1999) contains 26 items measuring symptoms of ADHD. Raw scale scores in the subscales (Inattention/Memory Problems; Hyperactivity/Restlessness; Impulsivity/Emotional Lability; Problems with Self Concept) are transformed into a standard T-score with a mean of 50 and a standard deviation of 10, normed by gender and age. T-scores above 65 indicate clinically significant symptoms.

PTSD Symptoms: Clinician Administered PTSD Scale (CAPS). The CAPS (Blake, 1995) is a gold standard diagnostic interview for current and lifetime PTSD and has excellent psychometric properties. Across samples, the CAPS demonstrates high inter-rater reliability (i.e., above .86) and internal consistency on each of the three PTSD symptom clusters (range .63 to .89), and correlates strongly (i.e., above .61) with other measures of PTSD. When tested in

conjunction with the SCID, the CAPS provided a PTSD diagnosis with specificity ranging from

94% to 95%, and sensitivity ranging from 84% to 90% (Radnitz, 1998; Hyer, 1996). Developed

by researchers at the National Center of PTSD, this structured interview assesses all 17

symptoms of PTSD for frequency (scored on a 0 [never] to 4 [daily or almost every day]) and

intensity (0 [none] to 4 [extreme, incapacitating distress]). These 17 symptoms can be totaled for

a dichotomous diagnostic index or a continuous measure of posttraumatic stress severity, as well

as DSM-related symptom cluster subscales (Reexperiencing, CAPS-B; Avoidance, CAPS-C;

Hyperarousal, CAPS-D).

Psychiatric Diagnoses: The MINI International Neuropsychiatric Interview and

Chart Review. The MINI (Lecrubier, 1997) is a brief, valid, and reliable structured diagnostic

interview for *DSM-IV* and ICD-10 psychiatric disorders. Participants were administered the

MINI to screen for other Axis I disorders. When participants screened positive for a given

diagnosis, study clinicians used follow up questions and record review to validate diagnoses and

ensure eligibility criteria were met. Many participants met criteria for at least one psychiatric

condition besides ADHD or PTSD. Co-occurring psychiatric conditions were most common in

the ADHD+PTSD (73.1%) and PTSD only (60.0%) groups, followed by the ADHD only

(29.4%) group, and least common in controls (6.5%). As shown in Table 1, depressive disorders

were most common across groups; there was a significant difference in the proportion of

participants with depression in each group ($X^2(3) = 48.7$, $p < .001$), with depression being more

common in the PTSD only and ADHD+PTSD groups than the ADHD only and control group,

and more common in the ADHD only group than in the control group.

Combat Exposure: Combat Exposure Scale (CES) (Lund, 1984) is a 7-item self-report

measure, used to obtain information regarding exposure to wartime stressor events. The measure

yields total scores ranging from 1 to 41, where higher scores indicate greater severity of combat

exposure.

Inhibitory Functioning. Inhibitory control (response inhibition and execution) was

measured using a cued go/no-go (GNG) reaction time task used in other research to measure

inhibitory control in response to alcohol administration (Marczinski, 2005; Fillmore, 2009) and

in adults and youth with ADHD (Weafer, 2009; Derefinko, 2008).

The task was operated using Inquisit software using a pre-programmed script for the

Cued GNG task (Millisecond Software, Seattle, WA) and was performed on a PC. As described

in the task user manual, each trial involved the following sequence. First, a fixation point (+) was

presented for 800 ms followed by a blank white screen for 500 ms. Next, a visual cue was

displayed for one of five stimulus onset asynchronies (SOAs = 100, 200, 300, 400, or 500 ms).

SOAs were distributed evenly across trials and varied to minimize anticipation effects. Following

the cue, a go or no-go target stimulus was displayed until either a correct response occurred or

1000 ms had elapsed. Trials were separated by a 700 ms inter-trial interval.

Cues were rectangles framed in black and filled with white on a white background

(appeared "empty"). Rectangles were either horizontal or vertical in orientation. Targets were

presented as a solid fill to the interior of the rectangular cues. Go targets were green, and no-go

targets were blue. Participants were instructed to respond on the keyboard spacebar with their

right index finger as soon as a green (go) target appeared and to suppress a response when a blue

(no-go) target was presented. The orientation of the cues signaled the probability of a subsequent

go or no-go target. The orientation of the empty rectangles was a visual cue regarding the

probability of a go vs. no-go response requirement. The vertical rectangle had an 80%

probability of being a green go trial and the horizontal rectangle had an 80% probability of being

a blue no-go trial. Cues with a high probability of correctly signaling a target were considered

"valid cues" (vertical-green/go and horizontal-blue/no-go) and cues with a low probability of

correctly signaling a target were considered "invalid cues" (vertical-blue/no-go and horizontal-

green/go).

Participants completed a total of 250 trials with equal distribution of vertical (125; 100

green/go, 25 blue/no-go) and horizontal cues (125; 100 blue/no-go, 25 green/go). For each trial,

the computer recorded whether a response occurred, and if so, the reaction time (RT) in

milliseconds from the onset of target to key press. Feedback (the words "correct" or "incorrect"

along with RT) was presented during the inter-trial interval to promote quick and accurate

responding.

Three primary measures were derived from the cued go/no-go task for this study.

Inhibitory failures were measured as the proportion of no-go targets for which a participant

failed to inhibit a response (i.e., responded to a no-go target). Scores were calculated for each

cue condition (go and no-go). Group effects on proportion of inhibitory failures in the go and no-

go cue conditions were analyzed by separate ANOVAs. *Response execution* was measured by

RT to go targets. Shorter RTs indicated greater facilitation of response execution (Marczinski,

2005; Fillmore, 2009; Weafer, 2009; Derefinko, 2008). Mean RT scores were calculated for each

participant for each cue type. Group effects on RT scores in the go and no-go cue conditions

were analyzed by two ANOVAs. *Omission errors* were measured as non-responses to go targets.

Omission errors were generally infrequent (typically <2 per session), which is consistent with

previously reported findings in adults with ADHD (Weafer, 2009) and children with ADHD

(Derefinko, 2008). Because no significant differences were observed between veteran and

civilian participants on task performance variables, data were pooled for veterans and civilians

within diagnostic group. Post-hoc group contrasts were conducted with Bonferroni or Tamhane's

correction to account for multiple comparisons depending on whether there was homogeneity of

variance across dependent variables by group.

Results

Failure of response inhibition following invalid (go) cues. Inhibitory failures were

generally uncommon across all diagnostic groups (Table 2). Participants with co-occurring

PTSD and ADHD demonstrated approximately twice as many inhibitory failures following

invalid (go) cues as participants in the control group (2.3% vs. 1.2%), though the group effect

was not significant, $F(3, 156) = .74$, $p = .52$, $\eta_p^2 = .014$.

Failure of response inhibition following valid (no-go) cues. Participants in the control

group demonstrated significantly fewer inhibitory failures following valid (no-go) cues relative

to all three clinical groups, $F(3, 156) = 3.02$, $p = .03$, $\eta_p^2 = .055$. There were no significant

differences among the three clinical groups (p-values all $> .05$).

Cue dependency. Cue dependency effects were most prominent in the control and

PTSD+ADHD groups. Participants in the ADHD only and PTSD only groups demonstrated

similar proportions of inhibitory failures to no-go trials regardless of cue condition.

Response execution and RT variability following valid (go) cues. There was a significant

group effect on mean reaction time ($F(3, 156) = 8.62$, $p < .001$, $\eta_p^2 = .142$) and mean reaction

time SD ($F(3, 156) = 12.52$, $p < .001$, $\eta_p^2 = .194$) when go cues were presented prior to the go

target. Post hoc contrasts indicated that the ADHD+PTSD group responded significantly more

slowly on those trials than the control group, $p = .03$ (Table 2). Additionally, the PTSD only ($p =$

.01) and ADHD+PTSD groups ($p = .001$) had more variable response times than the control

group.

Response execution and RT variability following invalid (no-go) cues. There was a significant group effect on mean reaction time ($F(3, 156) = 7.78$, $p < .001$, $\eta_p^2 = .130$) and mean reaction time SD ($F(3, 156) = 7.52$, $p < .001$, $\eta_p^2 = .126$) when no-go cues were presented prior to the go target. Post hoc contrasts indicated that the PTSD only group ($p = .06$) and the ADHD+PTSD group ($p = .06$) responded marginally more slowly on those trials than the control group. Additionally, the PTSD only ($p = .04$) and ADHD+PTSD groups ($p = .009$) had significantly more variable response times than the control group.

Omission errors. Although overall rates of omission errors were very low, significant group effects were observed for both go cue ($F(3, 156) = 5.19$, $p = .002$, $\eta_p^2 = .091$) and no-go cue ($F(3, 156) = 5.58$, $p = .001$, $\eta_p^2 = .097$) trials.   Specifically, although no pairwise comparisons revealed significant differences, post-hoc contrasts indicated that the ADHD and ADHD+PTSD groups demonstrated significantly more omission errors than the control group following both go cues ($p = .010$) and no-go cues ($p = .012$).

Discussion

The purpose of this study was to evaluate behavioral indices of neurocognitive functioning – specifically response execution and inhibition – among veterans with and without ADHD and PTSD. Results indicated that participants with either PTSD or co-occurring ADHD+PTSD demonstrated significantly longer and more variable reaction times than controls. These effects generally held regardless of whether cues preceding the target stimuli were valid or invalid. Considered together, findings suggest that veterans with co-occurring ADHD+PTSD demonstrate slower response execution and impaired response inhibition similar to patterns reported in other samples of veterans and other groups with ADHD. Notably, co-occurring ADHD+PTSD was not associated with amplified deficits in these domains. There is some

evidence that anxiety may attenuate or buffer impulsive responding in commonly observed in

ADHD (Schatz, 2006). It is possible that anxiety or hypervigilance symptoms of PTSD – or

comorbid depression, which was most common among participants with PTSD or ADHD+PTSD

in this study – may have played a similar role, lengthening response time and lowering the risk of

inhibitory failures among participants with ADHD+PTSD.

Participants with only ADHD did not differ significantly from participants with only

PTSD or PTSD+ADHD on any outcome variable. However, they also did not differ from

comparison participants upon post hoc analysis. This trend runs counter to prior findings in the

literature where patients with ADHD performed worse (i.e., more inhibitory failures, more

variable responding, etc.) than healthy controls. Even though a significant group difference was

not observed between participants with ADHD and controls, the mean scores of the ADHD

group were comparable to the ADHD+PTSD and PTSD groups. One reason for the lack of

significant group effects between participants with ADHD only and controls was the overall

smaller sample size and greater variability in the ADHD group. Some trend-level differences

between the ADHD and control group were observed, but after correcting for multiple

comparisons and non-homogeneity of variance between groups, these effects were not

significant. It is possible this absence of group differences is the result of limited power, and

significant differences might emerge in a study that enrolled more participants with ADHD only.

We did not observe significant differences in cue dependency across groups despite our

prediction that participants with PTSD may be more sensitive to cues than participants without

the disorder. Although some research has pointed the importance of cue-dependent learning in

co-occurring PTSD and substance use disorders (Coffey, 2010; Saladin, 2003), it may be that

effects are specific to particular types of cues or triggers, such as threat cues in the case of PTSD

or cues that trigger craving in substance use. In the current study, the cues presented in the task were not linked to a particular rewarding or aversive outcome but rather to the likelihood of a particular type of target stimulus being presented in a given trial. It is possible that a cue dependency effect may be observed among participants with PTSD or ADHD+PTSD relative to participants without ADHD when cues not only predict likelihood of response expectation (press vs. not press) but if successful execution of those behaviors were linked to more salient consequences, such as aversive or rewarding stimuli contingent on performance.

The current study was the first to compare response execution and inhibition, as well cue dependency, in a sample of veterans and community adults with and without both ADHD and PTSD. Whereas other patterns of clinical comorbidity – such as PTSD and mood disorders, PTSD and substance use disorders, and ADHD and substance use disorders – have been studied more extensively, there is relatively little published research on ADHD+PTSD. Given that ADHD and PTSD are both common among military personnel and veterans, as well as in the general population though less frequently, additional research is needed to better understand the mechanisms underlying their comorbidity as well as the effectiveness of existing interventions designed for each disorder among people with both disorders.

Despite the contributions of the current study, a number of limitations should be noted. First, the study enrolled only males, due to relatively high rates of ADHD in males relative to females, higher rates of males served by the VA medical center, and the eligibility criteria of the parent study which involved genetic analyses. Future work should enroll female participants to evaluate for possible sex and gender effects. Second, rates of inhibitory failures and omission errors were low in this study. Low rates of errors have been reported on go/no-go tasks previously, especially among healthy adult control participants, but error rates in this study were

lower than anticipated overall – particularly among the clinical groups. Although the cued

go/no-go reaction time task we used here has been used in other studies (Weafer, 2009;

Derefinko, 2008), it is possible that the task was not sensitive to differences in inhibitory

functioning in the current sample, particularly outside the context of alcohol challenge as has

been investigated previously. Additional studies in other groups, including larger samples with

co-occurring ADHD, mood, and anxiety disorders—as well as in younger samples of children

and adolescents – may help clarify the impact of comorbidity on impulsive responding in

ADHD.   Future studies should include multiple indices of inhibitory functioning, including

versions of go/no-go or stop tasks with more stringent response requirements or that include

more salient consequences to motivate faster responding across trials. The current study was also

limited in its capacity to account for other factors that may have influenced task performance,

such as personality, history of head injury, or substance use disorder.

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Table 1. Demographic and clinical information by group

Variable	ADHD only (*n* = 17)	PTSD only (*n* = 25)	ADHD + PTSD (*n* = 26)	Control (*n* = 92)	*F*	Chi-square
Age, years M(*SD*)	32.4 (7.5)	32.3 (7.0)	33.8 (8.0)	29.3 (.76)	3.21*	--
Race (% white)	79.6	66.7	82.3	65.4	--	29.1
Combat Exposure Scale	20.3 (13.5)	18.5 (7.6)	23.6 (8.3)	17.0 (8.9)	2.43	--
CAPS-Total	35.8 (12.2) [a] (n=8)	65.1 (9.2) [b]	68.7 (10.9) [b]	30.2 (15.1) [a]	54.9** *	

				(n=21)	
CAPS-B	6.1 (5.1) [a]	15.6 (6.1) [b]	15.8 (5.6) [b]	5.4 (4.2) [a]	21.3***
	(n=8)			(n=21)	
CAPS-C	12.4 (8.7) [a]	26.3 (7.6) [b]	28.2 (6.0) [b]	11.7 (8.2) [a]	26.6***
	(n=8)			(n=21)	
CAPS-D	17.3 (4.6) [a]	23.2 (4.3) [b]	24.8 (4.1) [b]	13.1 (7.0) [a]	24.4***
	(n=8)			(n=21)	
CAARS-A	61.2 (12.7) [a]	48.6 (8.5) [b]	66.8 (8.9) [a]	44.7 (7.4) [b]	55.9***
CAARS-B	62.2 (8.3) [a]	52.9 (6.7) [a]	60.7 (6.9) [a]	46.2 (9.0) [b]	32.5***
CAARS-C	55.8 (11.5) [ab]	51.5 (9.7) [b]	60.7 (9.1) [a]	42.9 (5.8) [c]	43.0***
CAARS-D	52.5 (12.6) [a]	53.4 (11.0) [a]	64.7 (9.3) [b]	42.9 (7.3) [c]	43.5***
CAARS-E	60.6 (10.1) [a]	51.5 (7.6) [b]	67.5 (5.9) [c]	42.8 (7.1) [d]	90.3***
Disorder (%)					
Adjustment	0.0	20.0	7.7	1.1	16.1**
Antisocial					8.5*
Personality	5.9	0.0	0.0	0.0	
Anxiety	5.9	24.0	11.5	3.3	11.9**
Bipolar	0.0	4.0	7.7	0.0	7.5

Borderline Personality	0.0	0.0	3.8	1.1	2.0
Depression	29.4	48.0	57.7	4.3	48.7***
Insomnia	0.0	4.0	0.0	0.0	4.3
Intermittent Explosive	0.0	0.0	3.8	0.0	5.2
Panic	0.0	8.0	7.7	0.0	8.7*
Social anxiety	0.0	0.0	7.7	1.1	5.9

Note: ADHD: Attention-deficit/hyperactivity disorder. PTSD: Posttraumatic stress disorder. CAPS: Clinician Administered PTSD Scale. CAPS-B: Reexperiencing scale. CAPS-C: Avoidance scale. CAPS-D: Hyperarousal scale. CAARS: Connors Adults ADHD Rating Scale. CAARS-A: Inattention/Memory Problems. CAARS-B: Hyperactivity/Restlessness. CAARS-C: Impulsivity/Emotional Lability. CAARS-D: Problems with Self-Concept. CAARS-E: ADHD Index. $* p < .05$. $** p < .01$. $*** p < .001$. Co-occurring psychiatric diagnoses were determined by a combination of scores on the MINI screening interview and follow-up question and record review by study clinicians. Values with different superscript letters were significantly different per post hoc contrast analysis, $p < .05$, following correction for multiple comparisons (Bonferroni or Tamhane, depending on whether equal variances observed). Values that share a superscript did not differ significantly from each other. Values without superscripts were not significantly different from any other values.

Table 2. Cued Go/No-Go Task performance and reaction time data across groups.

Variable	ADHD only ($n = 17$)	PTSD only ($n = 25$)	ADHD+PTSD ($n = 26$)	Control ($n = 92$)	F
Proportion IF Go Cue	1.4%	1.4%	2.3%	1.2%	.74
Proportion IF No-Go Cue	1.5%	1.2%	1.2%	0.5%	3.02*
Cue dependency	-0.1	0.2	1.1	0.7	
Reaction time Go Cue	353.4 (102.9)	343.1 (63.7)	382.4 (122.7)[a]	307.6 (38.2)[b]	8.62***
Reaction time No-Go Cue	358.2 (103.6)	354.1 (67.0)[a]	385.7 (125.7)[a]	315.3 (34.9)[b]	7.78***
Reaction time SD Go-Cue	80.8 (45.6)	78.6 (35.3)[a]	90.0 (42.2)[a]	52.9 (23.3)[b]	12.52***
Reaction time SD No-Go Cue	75.0 (53.3)	74.1 (40.7)[a]	75.7 (36.3)[a]	49.4 (24.9)[b]	7.52***
Omission errors Go Cue	2.6 (4.7)[a]	1.3 (2.4)	3.5 (7.9)[a]	.44 (1.5)[b]	5.19**

| Omission | .65 (1.2)[a] | .20 (.41) | 1.1 (2.6)[a] | .07 (0.3)[b] | 5.58** |

errors No-Go

Cue

Note. IF = inhibitory failures. SD = standard deviation. Values with different superscript letters were significantly different per post hoc contrast analysis with Tamhane's correction applied, $p <$ .05. * $p < .05$. ** $p < .01$. *** $p < .001$. Values that share a superscript did not differ significantly from each other. Values without superscripts were not significantly different from any other values.