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Elizabeth Riley, Alex Mitko, Anna Stumps, Meghan Robinson, William Milberg, Regina McGlinchey, Michael Esterman, and Joseph DeGutis

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Clinically Significant Cognitive Dysfunction in OEF/OIF/OND Veterans: Prevalence and Clinical Associations

Elizabeth Riley
Cornell University

Alex Mitko, Anna Stumps, and Meghan Robinson
VA Boston Healthcare System, Boston, Massachusetts

William Milberg and Regina McGlinchey
VA Boston Healthcare System, Boston, Massachusetts, and
Harvard Medical School

Michael Esterman
VA Boston Healthcare System, Boston, Massachusetts, and
Boston University School of Medicine

Joseph DeGutis
VA Boston Healthcare System, Boston, Massachusetts, and Harvard Medical School

Objective: Cognitive performance in trauma-exposed populations, such as combat Veterans, has been shown to be worse than in nonexposed peers. However, cognitive performance has typically been within the normal range (within 1 *SD* of normative mean), and the prevalence of *clinically significant* cognitive dysfunction (i.e., performance more than 1 *SD* below the mean on multiple measures in a domain) in younger adults with trauma exposure remains unknown. The objective of our study was to measure this.

Method: We applied *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*) cutoffs for clinically significant cognitive dysfunction (>1 *SD* below the mean in multiple measures within a domain) in the domains of memory, executive function, and attention to a sample of combat-exposed Operation Enduring Freedom/Operation Iraqi Freedom/Operation New Dawn (OEF/OIF/OND; $N = 368$, mean age = 31.7 years, 90% men) Veterans. We then compared psychiatric, physiological, and neural measures, as well as functional outcomes, between those with and without cognitive dysfunction.

Results: Veterans with cognitive dysfunction ($n = 129$, 35.1%) had lower premorbid reading ability and more severe psychological distress, including increased anxiety, depression, posttraumatic stress disorder (PTSD), sleep difficulties, pain, and alcohol consumption. Those with cognitive dysfunction also had worse functional outcomes, with mild but significant disability. In contrast, we found associations between outcome and age, traumatic brain injury, physiological and neural measures to be weak or not significant. **Conclusions:** Together, this suggests that premorbid abilities and trauma-related psychological symptoms contribute significantly to cognitive dysfunction in OEF/OIF/OND Veterans, and that neurological insult and aging may play less of a role. Cognitive dysfunction may be at least partially ameliorated by treating trauma-related symptoms.

General Scientific Summary

In this study, we examined the psychological, physical and neurological health profiles of returning OEF/OIF/OND Veterans who had significant cognitive dysfunction. Though we did not find any evidence of physical or neural decline in those with cognitive dysfunction, we did find that those with

Elizabeth Riley, Department of Human Development, School of Human Ecology, Cornell University; Alex Mitko, Anna Stumps, and Meghan Robinson, Translational Research Center for TBI and Stress Disorders (TRACTS), VA RR&D TBI Center of Excellence, VA Boston Healthcare System, Boston, Massachusetts; William Milberg and Regina McGlinchey, Translational Research Center for TBI and Stress Disorders (TRACTS), VA RR&D TBI Center of Excellence, VA Boston Healthcare System, and Department of Psychiatry, Harvard Medical School; Michael Esterman, Translational Research Center for TBI and Stress Disorders (TRACTS), VA RR&D TBI Center of Excellence, VA Boston Healthcare System, and Department of Psychiatry, Boston University School of Medicine; Joseph DeGutis, Translational Research Center for TBI and Stress Disorders (TRACTS), VA RR&D TBI Center

of Excellence, VA Boston Healthcare System, and Department of Psychiatry, Harvard Medical School.

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Correspondence concerning this article should be addressed to Elizabeth Riley, Department of Human Development, School of Human Ecology, Cornell University, 162 Human Ecology Building, Ithaca, NY 14850. E-mail: er482@cornell.edu

cognitive dysfunction suffered from more intense psychological distress (posttraumatic stress disorder, depression, anxiety, sleep difficulties, pain) than those without. Our results suggest that cognitive dysfunction in younger returning Veterans might be ameliorated with appropriate treatment for psychological distress conditions.

Keywords: cognitive dysfunction, trauma exposure, OEF/OIF/OND Veterans, PTSD, alcohol consumption

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Cognitive performance in trauma-exposed populations such as combat Veterans has been shown to be worse than in those not exposed to trauma, particularly in those suffering from posttraumatic psychological symptoms such as posttraumatic stress disorder (PTSD) and depression (for a review, see Stetz et al., 2007). In particular, studies have associated worse cognitive performance in these populations with trauma sequelae such as PTSD, depression, and traumatic brain injury (TBI; Brandes et al., 2002; Trivedi & Greer, 2014; Vaishnavi, Rao, & Fann, 2009). Studies have also found worsening cognitive performance pre- to postmilitary deployment, even after controlling for these trauma sequelae (e.g., Vasterling et al., 2006). Despite these reports of cognitive compromise, group-level performance in trauma-exposed populations has typically been within normal limits (i.e., within 1 *SD* of the pooled normative mean; Polak, Witteveen, Reitsma, & Olf, 2012), and the prevalence of clinically significant cognitive dysfunction (CD; 1 *SD* or more below the mean on multiple measures within a domain) remains to be characterized. This is important because clinically significant CD may be particularly predictive of poor daily life functioning (Kalechstein, Newton, & van Gorp, 2003; Reppermund et al., 2011). In addition to determining the prevalence in this population, the profile of trauma-exposed individuals with clinically significant CD remains unknown. Comparing the clinical, physiological, and neural measures between trauma-exposed individuals with and without clinically significant CD could provide a better understanding of the current mechanisms of CD in trauma-exposed populations. It could also provide a benchmark for future studies investigating longer-term effects of trauma exposure on CD. To address these issues, in the present study we applied *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*) cutoffs for clinically significant CD to younger combat-exposed Operation Enduring Freedom/Operation Iraqi Freedom/Operation New Dawn (OEF/OIF/OND) Veterans and compared a comprehensive battery of measures between those with and without CD.

Several previous studies have described associations between trauma sequelae and poorer attention, executive functions, and memory performance. Veterans with PTSD and depression have been shown to perform worse on tests of verbal memory (Dolan et al., 2012), attention (e.g., distractor suppression, Esterman et al., 2013), and executive functioning (e.g., inhibitory control, DeGutis et al., 2015; Swick, Honzel, Larsen, Ashley, & Justus, 2012). Increased heavy drinking is common after trauma exposure in both men and women (Ullman, Filipas, Townsend, & Starzynski, 2005) and has also been associated with worse cognitive performance across several cognitive domains (A. Green et al., 2010; Houston

et al., 2014). Mild traumatic brain injury (mTBI) and blast exposure, common co-occurrences with military trauma (O'Neil et al., 2013), may also lead to worse verbal memory performance (e.g., Grande et al., 2018). Some Veterans with mTBI have persistent cognitive complaints (Silver, McAllister, & Arciniegas, 2009), possibly related to accelerated white matter brain-aging trajectories (Trotter, Robinson, Milberg, McGlinchey, & Salat, 2015) or impaired functional connectivity (Robinson, Clark, Milberg, McGlinchey, & Salat, 2017). Furthermore, cardiometabolic dysfunction (Levine, Levine, & Levine, 2014), a constellation of maladaptive cardiovascular, renal, metabolic, prothrombotic, and inflammatory abnormalities, is highly prevalent in those with PTSD (29% of Veterans under 40 years of age with PTSD demonstrated metabolic syndrome; see Wolf, Bovin, et al., 2016). Metabolic dysfunction may also lead to reduced cognitive performance, particularly later in life (Taylor & MacQueen, 2007; Xu, Marseglia, Kalpouzos, Bäckman, & Fratiglioni, 2017), and some studies have even shown associations between compromised metabolic functioning and CD in younger and middle-aged adults (e.g., Yaffe, 2007). Despite each of these trauma sequelae being associated with worse cognitive performance, studies have yet to compare these sequelae between those with and without clinically significant CD. This is important because clinically significant CD is associated with worse functional outcomes, such as the ability to stay employed (Kalechstein et al., 2003) and carry out instrumental activities of daily living (Reppermund et al., 2011).

There are multiple hypotheses regarding the cause of CD in trauma-exposed populations. Some have suggested that trauma-related psychological distress (e.g., symptoms of PTSD, depression, and sleep difficulties) may directly cause CD, such as by increasing attentional/working memory load (Brady & Sinha, 2005; Qureshi et al., 2011) and compromising top-down control (DeGutis et al., 2015). This suggests that CD could improve if psychological distress is reduced. A second related possibility is that cognitive vulnerabilities may be present *before* trauma exposure (e.g., military deployment), which may make an individual more susceptible to trauma-related psychological distress. For example, studies have found that premorbid memory difficulties or poor executive functions result in greater incidence of PTSD (Aupperle, Melrose, Stein, & Paulus, 2012; Brandes et al., 2002; Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001). These pre-trauma cognitive vulnerabilities may be further exacerbated by trauma-related psychological distress, resulting in significant CD (Marx, Doron-Lamarca, Proctor, & Vasterling, 2009).

An alternative, nonmutually exclusive hypothesis is that clinically significant CD in trauma-exposed populations is caused by

aging-related processes, such as physiological decline and neurodegenerative processes (Jeste & Palmer, 2016). A recent study of OEF/OIF Veterans provides evidence that advanced DNA methylation age is associated with reduced neural integrity and poorer working memory performance (e.g., Wolf, Logue, Hayes, et al., 2016). Cardiometabolic dysfunction, common in trauma-exposed populations, has also been related to widespread cortical thinning in temporal, parietal, and frontal regions in OEF/OIF Veterans (Wolf, Sadeh, et al., 2016). Other studies in OEF/OIF Veterans have shown exacerbated age-related decreases in white matter integrity in those exposed to blasts (Trotter et al., 2015). Furthermore, mTBIs may increase the risk of neurodegeneration (McKee & Robinson, 2014), including cortical thinning and Alzheimer's disease (Hayes et al., 2017), and in younger and middle-aged adults, memory difficulties (Croall et al., 2014; Vincent, Roebuck-Spencer, & Cernich, 2014). Cognitive deficits related to mTBI have shown to be worse in those with preexisting cognitive vulnerabilities (e.g., those with reduced cognitive reserve, Oldenburg, Lundin, Edman, Nygren-de Boussard, & Bartfai, 2016). Together, this framework predicts that clinically significant CD in trauma-exposed populations would be associated with physical compromise and aging-related factors: presence of mild TBI and blast injury, increased chronological age (and DNA methylation age), worse cardiometabolic functioning, and neural degeneration.

The psychological distress and aging-related hypotheses for CD have distinct treatment implications. The aging-related framework suggests that addressing clinically significant CD would involve treating metabolic dysfunction and perhaps implementing compensatory cognitive rehabilitation. On the other hand, the psychological distress framework suggests that treating psychological distress symptoms such as through behavioral (e.g., cognitive-behavioral therapy) or pharmacological interventions could improve cognitive functioning. Thus, evaluating the psychological and physical differences between Veterans with and without clinically significant CD cannot only help decide between these models but could also better inform treatment of trauma-exposed populations.

To address these questions, we recruited a relatively large sample of OEF/OIF/OND Veterans ($N = 368$, mean age = 31.7) and defined clinically significant CD (distinct from formal diagnosis of neurocognitive disorder, which we did not pursue) using a battery of well-validated neuropsychological tests. In particular, we incorporated multiple measures from domains of memory, attention, and executive function, domains most often compromised in trauma-exposed populations (see Stricker et al., 2017) and also administered a standalone performance validity test (important for robust results, see Lippa, Lange, French, & Iverson, 2017). To determine whether there were psychological and clinical differences between those with and without CD, we examined assessments of depression, anxiety, PTSD, sleep, pain, and substance abuse. We also tested whether CD in younger Veterans was associated with physical compromise or aging/neurodegeneration (Wolf, Sadeh, et al., 2016) by comparing mTBI and blast history, cardiometabolic measures (blood pressure, glucose, high-density lipoprotein [HDL], triglycerides, waist circumference), neural measures (cortical thickness using structural magnetic resonance imaging [MRI]), as well as age differences between groups. To determine whether premorbid factors affected postdeployment CD, we also examined a measure of premorbid verbal ability. Finally,

to compare how Veterans with and without CD were able to carry out daily life activities, we examined the World Health Organization Disability Assessment Schedule II (WHODAS; Üstün, 2010), a validated self-report functional measure that assesses social participation, communication, mobility, self-care, and activities of daily living.

Method

Participants

Our initial sample included 433 Veterans of the conflicts in Iraq and Afghanistan, aged 20–62, who enrolled in a study at the Translational Research Center for TBI and Stress Disorders (TRACTS), a VA Rehabilitation Research and Development Traumatic Brain Injury National Network Research Center at VA Boston Healthcare System. Participants were service members of Operation Enduring Freedom/Operation Iraqi Freedom/Operation New Dawn (OEF/OIF/OND) recruited from New England and the Greater Boston Metropolitan area by a full-time recruitment specialist. The recruitment methods and study protocol has been described in detail by McGlinchey et al. (McGlinchey, Milberg, Fonda, & Fortier, 2017). For our analysis, participants were excluded for serious neurological illness ($n = 2$), personality disorders ($n = 2$), cognitive performance such that they could not complete the study tasks ($n = 2$), moderate or severe TBI ($n = 16$), inadequate task effort as determined by a score of less than or equal to 85 on any measure of the Medical Symptom Validity Test (MSVT: $n = 22$, P. Green, 2004), or lack of at least one deployment ($n = 21$). After exclusion of these 65 individuals, the final cohort consisted of 368 participants.

In addition to the MSVT, three of our metrics (Table 1) included embedded performance validity tests. Seven participants failed the Test of Variables of Attention Symptom Exaggeration Index (score > 2), three had a Reliable Digit Span of less than seven on the Wechsler Adult Intelligence Scale (4th ed., WAIS-IV; Wechsler, 2008) indicating poor effort, and eight failed the California Verbal Learning Test Forced Choice Recognition (score < 15). Of these 18 individuals, three were categorized as having CD. Removing these 18 individuals from the sample ($n = 350$) did not change the results of any of our statistical analysis; therefore, they were allowed to remain in the final cohort. Results using this reduced cohort are included in the online supplemental materials.

On the day of testing, after giving consent, participants gave a fasting whole blood sample, underwent a battery of neuropsychological tests, participated in a series of diagnostic clinical interviews, and finally were given an MRI. All procedures and the informed consent process were approved by the Institutional Review Board of Human Studies Research at the VA Boston Healthcare System.

Neurological and Physical Health

The Boston Assessment of TBI-Lifetime (BAT-L) semistructured clinical interview (Fortier et al., 2014) was used to diagnose the number and determine the severity of TBIs and blast exposures during military deployment and throughout the lifetime. MTBIs were those which resulted in less than 30 min of time unconscious,

Table 1

Neuropsychological Tests Used to Diagnose Clinically Significant Cognitive Dysfunction (CD)

Domain	Test	Reference	Cognitive dysfunction	No cognitive dysfunction
Attention	Test of Variables of Attention (TOVA)	(Henry, 2005)	44 (reaction time) 30 (d')	10 (reaction time) 5 (d')
	Digit Span (DSP)	(Wechsler, 2008)	24	9
	Trail Making Test (Trails—number sequencing subtest)	(Delis et al., 2001)	8	1
Executive functioning	Trail Making Test (Trails—number/letter switching subtest)	(Delis et al., 2001)	25	3
	Stroop Test	(Delis et al., 2001)	13	2
	CANTAB Intra-Extra Dimensional Set Shift (IED)	http://www.cantab.com	10	4
	Verbal Fluency (FAS)	(Delis et al., 2001)	17	6
	Auditory Consonant Trigrams (ACT)	(Stuss et al., 1985)	46	19
Memory	California Verbal Learning Test-II (CVLT)	(Woods, Delis, Scott, Kramer, & Holdnack, 2006)	39 (short delay) 45 (long delay) 41 (recognition score)	15 (short delay) 4 (long delay) 1 (recognition score)

Note. The rightmost columns indicate the percent of Veterans who scored 1 *SD* below the mean on each particular test.

less than 24 hr of altered mental status, less than 1 day of post-traumatic amnesia, and a Glasgow Coma Scale score of at least 13. Those with mTBIs were included in the study ($n = 259$, 70% of total sample, average time since mTBI = 8.33 years), while individuals with moderate and severe TBIs were excluded from the current sample. The Deployment Risk and Resiliency Inventory: Combat Experiences and Post Battle Experiences Modules (DRRI) was used to quantify combat experience. Pain levels and sleep quality were assessed by self-report using the McGill short-form pain questionnaire (Melzack, 1975) and Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), respectively. It should be noted that pain and sleep can reflect both physical and psychological health. For this reason, we chose not to include these variables in either the physical or psychological models. Blood pressure was measured according to American Heart Association guidelines; two blood pressure readings were taken in both standing and seated position and then averaged. A blood draw was taken to measure levels of cholesterol (both high-density lipoprotein [HDL] and low-density lipoprotein [LDL]), triglycerides and blood glucose. Waist size was measured by a trained clinician. By using cutoff scores for risk factors including waist circumference, blood pressure, cholesterol, triglycerides and blood glucose, a cardiometabolic syndrome score, defined as 3 or more factors above the cutoff, was calculated by adding up the number of risk factors. Briefly, the cutoff values were waist size above 102 cm for men/88 cm for women, triglycerides above 150, HDL less than 40 for men or less than 50 for women, systolic blood pressure above 130, diastolic blood pressure above 85, and fasting blood glucose above 110 mg/dL.

Psychological Assessments

The Clinical-Administered PTSD Scale (CAPS-IV) structured interview was given to diagnose PTSD and obtain a continuous measure of PTSD symptom severity (Weathers et al., 2018). The Structured Clinical Interview for *DSM-IV* (SCID) was administered to diagnose mood, anxiety and substance abuse disorders (First, Gibbon, Smith Benjamin, & Spitzer, 1999). Self-report subtests from the Depression, Anxiety, and Stress Scale (DASS;

Lovibond & Lovibond, 1995) were used to measure severity of self-reported symptoms of anxiety, depression, and stress. Early life trauma was assessed using the early life trauma questionnaire Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000). Subjects with a mTBI were asked whether they experienced memory difficulties following their injury as part of a medical background questionnaire (McGlinchey et al., 2017). Finally, we obtained continuous measures of substance use/abuse using the Lifetime Drinking History Interview (average drinks on a drinking day [DDD], total lifetime drinks corrected for body weight) as well as self-report from the Short Michigan Alcohol Screening Test (SMAST; Selzer, Vinokur, & van Rooijen, 1975). Only 8 of 368 participants (2.2%) reported current substance abuse other than alcohol; therefore, abuse of other substances was not included in our analysis.

Neuropsychological Assessment and Defining CD

To assess neuropsychological function, participants were administered a battery of validated neuropsychological tests in the domains of attention, memory and executive function (Table 1). Performance was converted to scaled, age-corrected z -scores, and CD was operationalized as a score more than 1 *SD* below the mean on at least two measures within at least one domain, as defined by the *Diagnostic and Statistical Manual* (5th ed; *DSM-5*, American Psychiatric Association, 2013) diagnosis for neurocognitive disorder. The importance of requiring more than one score below cutoff has been clearly demonstrated in the literature (Holdnack et al., 2017). Although neurocognitive disorders cannot be diagnosed without a documented decline in function, the cutoffs are nevertheless useful in determining what constitutes poor functioning (American Psychiatric Association, 2013, p. 607). When categorizing participants as having CD, we chose to require a below-cutoff score on at least two measures within a domain because this greatly reduces the likelihood of miscategorizing an individual as having CD based on a single, erroneously low score. We used a cutoff of *below* 1 *SD* (not including 1 *SD*) from the mean because age adjustment of the scaled scores may have included small rounding errors and we wanted to take a more conservative ap-

proach to labeling CD. This approach, deemed the “comprehensive approach” by Jak et al. (2009), in their article on defining mild cognitive impairment (Jak et al., 2009), has been used in several other recent studies, for example, Stricker et al. (2016).

MRI Acquisition and Processing

Of our 368 participants, 260 received MRIs. There were no significant differences in demographics, neuropsychological performance, or any other study variable between those who received an MRI and those who did not (all $p > .1$). For each participant, two Magnetization Prepared Rapid Gradient Echo (MPRAGE) T1-weighted anatomical sequences were acquired (Repetition Time/Echo Time [TR/TE] = 2530/3.32 ms, 1 mm × 1 mm × 1 mm, flip angle 7 degrees) on a 3T Siemens TIM Trio scanner. Cortical and subcortical surface and volume reconstructions were computed from these images using the parcellations provided by FreeSurfer (Fischl, 2012). The automated reconstruction for each participant was inspected by trained observers. Areas where automatic identification of the pial or white matter boundaries were incorrect were adjusted by manually identifying voxels as being white matter or nonbrain as necessary. Then, the image was reanalyzed, per the published recommendations of the FreeSurfer development group. Following manual editing and reanalysis, cortical reconstructions were inspected again, and additional rounds of manual edits and reanalysis were completed until the reconstruction matched the anatomical boundaries that were apparent by visual inspection. Cortical thickness was defined as the shortest distance between the white matter and CSF (the boundaries of the gray matter) for every point on the tessellated surface. These values were smoothed with a 20 mm² kernel along the surface. For statistical analyses between participants, general linear models were used to compare the contributions of the CD group, while controlling for age, and for volume comparisons, total intracranial volume. Corrections for multiple comparisons were completed using FreeSurfer's `mri_glmfit-sim`, which utilizes a Monte-Carlo simulation to determine the likelihood of a cluster of a given size occurring by chance. Clusters were defined by a vertex-wise threshold of $p < .05$, and were considered significant if the corrected p value of the cluster was less than 0.05.

Functional Disability Testing

Functional disability was assessed by self-report with the WHODAS II (Üstün, 2010), including the following subscales: Understanding and Communicating, Participation [in society], Getting Along, Life Activity, Self-Care, Work and School, and Mobility.

Clinically Significant CD Associations With Psychological Distress Versus Aging-Related Factors

To test the hypotheses that clinically significant CD is associated with psychological distress versus aging-related factors, we first ran separate overall logistic regression models predicting the presence or absence of CD. For the psychological distress model, we included continuous measures of depression and anxiety (DASS depression and anxiety) and PTSD (CAPS), as well as alcohol consumption (DDD from the Lifetime Drinking History [LDH]) as independent variables. We chose number of drinks on a drinking day rather than other alcohol consumption variables because it has been shown to be

reliable and predictive in our previous studies with this sample (DeGutis et al., 2018). For the aging-related factors model, we included age, individual cardiometabolic measures (blood pressure, triglycerides, cholesterol, waist circumference and glucose levels), as well as the presence of a military TBI. We included the presence of a TBI that occurred during deployment (“military TBI”) in this model because it is specific to deployment and combat/trauma exposure and has been linked to neurodegeneration (McKee & Robinson, 2014). In addition to these models predicting the presence or absence of clinically significant CD, we also examined analogous psychological distress and aging-related models predicting continuous cognitive score. We calculated a global continuous cognitive score for each participant by first z -scoring each test score against published norms (see Table 1), then computing an average for each cognitive domain (memory, attention, and executive function) and finally, averaging the three domain scores for each participant.

Significant models were followed up with between group t tests (presence or absence of clinically significant CD) or Pearson correlations (for continuous global cognitive score) for each variable, using the false discovery rate (FDR) correction (Benjamini & Hochberg, 1995) to correct for multiple comparisons.

Post Hoc Analyses

To examine whether premorbid verbal ability (Wechsler Test of Adult Reading [WTAR Score]) explained postdeployment group differences, we ran a series of analyses of covariance (ANCOVAs) testing whether between-groups differences remained when controlling for WTAR score. For each neuropsychological variable that differed significantly between groups, we ran an ANCOVA using SPSS in which that variable was the dependent variable, cognitive status was the fixed factor, and WTAR was the covariate.

To determine which factors uniquely predicted clinically significant CD, we performed a logistic regression using MATLAB including the six variables that showed significant differences between the CD and control groups after FDR correction: CAPS, DASS anxiety, DASS depression, PSQI, pain, DDD, and WTAR (WHODAS II variables were not included because they are considered outcome variables). We also performed a linear regression to determine which of the aforementioned six variables significantly predicted each participant's continuous cognitive score. Last, we performed hierarchical regression analysis to determine which variables, in addition to WTAR, would uniquely predict CD status or continuous cognitive score. In these analyses, WTAR was entered first as a predictor of CD status/cognitive score. Second, a set of variables that differ significantly between CD and control groups was entered to determine which had additional significant predictive value.

Results

Demographics, Premorbid Ability, and Military Service

Our analysis included 368 Veterans with an average age of 31.7 years ($SD = 8.18$). 90% were male. A total of 74% were White, 15% Hispanic or Latino, 8% Black or African American, 1% Asian, and less than 1% American Indian. Participants had an average of 13.83 ($SD = 1.88$) years of education and an average

estimated premorbid IQ of 103.06 ($SD = 11.40$). Participants had an average of 1.41 deployments ($SD = 0.69$), an average duration of deployment of 14.32 months ($SD = 8.47$), and an average DRRI score (combat intensity) of 17.30 ($SD = 11.93$), indicating on average a moderate amount of combat exposure. The Veterans' average time since deployment was 3.38 years ($SD = 2.65$).

In terms of demographic and service-related differences between those with and without CD, we found that the groups, described below, did not differ in terms of age, gender, education, number or duration of deployments, or time since deployment (all $p > .10$; Table 2). The groups did significantly differ in their estimated premorbid IQ, with those with CD showing a lower estimated IQ ($M = 99.70$, $SD = 11.69$) than those without CD ($M = 104.90$, $SD = 10.83$), $p < .001$. When we correlated demographic, premorbid, and service-related variables with continuous global cognitive score, we found a significant association with education, $r = .20$, $p < .001$, as well as a weaker association with time since last deployment, $r = -0.10$, $p = .047$. Because it is common for Veterans to pursue additional education opportunities postdeployment (especially those without clinically significant CD), we did not regard education as a premorbid variable to be controlled for in subsequent analyses. Finally, perhaps unsurprisingly, we found a highly significant positive correlation between premorbid estimated IQ and higher global cognitive scores, $r = .37$, $p < .001$.

Prevalence of CD in the Current Sample

We first determined the number of participants in our sample with clinically significant CD using *DSM-5* cutoffs for mild neurocognitive disorder (mNCD; $<1 SD$ below the age-adjusted mean on at least two measures within at least one domain of memory, attention and, executive function; Table 1). We found that 35.1% of the sample ($n = 129$) met the criteria for CD. Of those individuals, 28.7% had a deficit in the memory domain only ($n = 37$), 24.0% had a deficit in attention only ($n = 31$), and 24.0% had a deficit in executive function only ($n = 31$). The remaining participants had a deficit in more than one domain: 7 with attention and executive deficits, 13 with executive and memory deficits, 7 with attention and memory deficits, and 3 with deficits in all three domains. We also analyzed our data set using a cutoff of 1.5 SD s

below the mean. For those results, see the online supplemental material.

Comparing Psychological Distress Versus Aging-Related Models of CD

Next, we compared two models of clinically significant CD, the psychological distress model (i.e., dysfunction is caused by psychological factors) versus the aging-related model (i.e., dysfunction is caused by processes similar to aging). We first ran separate overall logistic regression models predicting the presence or absence of CD as well as models using the continuous cognitive score. The logistic regression run with psychological distress factors (DASS depression and anxiety subscales, current CAPS score, and DDD) predicting cognitive status (whether or not the participant had CD) was significant, $\chi^2(4,334) = 27.22$, $p < .001$ (also see Table 3). The linear regression with psychological distress factors predicting continuous global cognitive score was also significant, $F(4,334) = 5.57$, $p < .001$, $R^2 = 0.06$. DDD was able to uniquely predict CD status ($\beta = 0.10$, $p = .004$), as well as predict continuous global cognitive score ($\beta = -0.02$, $p = .013$). The anxiety subscale of the DASS was also significant in uniquely predicting those with CD ($\beta = 0.06$, $p = .007$).

Considering these findings, we next sought to determine whether psychological distress predicted CD after accounting for estimated premorbid IQ (as measured by WTAR, the reading ability test). We performed a hierarchical regression, including WTAR in the first step and psychological distress variables in the second step. We first performed analyses with the presence or absence of CD as the dependent variable. In Model 1, WTAR as a predictor of CD was significant ($\chi^2(1, 329) = 19.91$, $p < .001$). Model 2 added in CAPS, depression, anxiety, and DDD to WTAR. Model 2 ($\chi^2(4, 329) = 43.84$, $p < .001$) showed a significant log-likelihood change from Model 1, $\chi^2(4, 329) = 23.93$, $p < .001$. In Model 2, of the five predictors, only WTAR ($\beta = -0.04$, $p < .001$) and DDD ($\beta = 0.11$, $p = .003$), uniquely predicted CD. We applied identical models predicting continuous global cognitive score and showed very similar results. In particular, Model 1 (WTAR only) had an R^2 of 0.16. For Model 2 (WTAR plus CAPS, depression, anxiety, and DDD) the R^2 was 0.21 with a significant R^2 change of 0.04, $F(4, 329) = 4.50$, $p = .001$. Of the five predictors, only WTAR ($\beta = 0.38$, $p < .001$) and DDD

Table 2
Demographic, Premorbid Ability, and Military Background

Measure	Cognitive dysfunction	No cognitive dysfunction	p value for t test	Cohen's d	r	p value for r
Age	31.83 \pm 8.14	31.60 \pm 8.22	.764	.033	-.01	.834
Years of education	13.64 \pm 1.82	13.93 \pm 1.90	.155	-.154	.21	<.001*
Number of lifetime mTBIs	1.71 \pm 2.07	1.59 \pm 2.20	.618	.054	-.08	.154
Number of military mTBIs	0.93 \pm 1.35	0.77 \pm 1.51	.311	.107	-.09	.085
Noticed worsening in memory and attention after mTBI	.62 \pm .49	.47 \pm .50	.011*	.284	-.22	<.001*
DRRI—Combat intensity	18.76 \pm 12.66	16.63 \pm 11.60	.138	.171	-.07	.227
Time since deployment (months)	44.37 \pm 33.15	38.58 \pm 32.13	.107	.178	-.07	.158
WTAR—estimated premorbid verbal ability	99.70 \pm 11.69	104.90 \pm 10.83	<.001*	-.467	.37	<.001*

Note. mTBI = mild traumatic brain injury; DRRI = Deployment Risk and Resiliency Inventory; WTAR = Wechsler Test of Adult Reading.

* Significance after false discovery rate (FDR) correction. Correlation coefficient r is between the continuous cognitive score and each variable.

Table 3
Psychological Health Information

Measure	Cognitive dysfunction	No cognitive dysfunction	<i>p</i> value for <i>t</i> test	Cohen's <i>d</i>	<i>r</i>	<i>p</i> value for <i>r</i>
DASS—Depression	10.11 ± 9.77	8.05 ± 9.39	.057	.216	-.13	.013*
Major depression prevalence	.30 ± .46	.24 ± .43	.252	.128	-.07	.167
DASS—Anxiety	9.28 ± 8.32	5.70 ± 6.97	<.001*	.479	-.19	<.001*
Anxiety prevalence	.21 ± .41	.21 ± .41	.998	.000	-.06	.254
CAPS score	56.81 ± 29.07	47.33 ± 28.01	.003	.334	-.20	<.001*
PTSD prevalence	.66 ± .47	.59 ± .49	.185	.144	-.09	.074
Interpersonal early life trauma	.36 ± .48	.27 ± .44	.063	.214	-.06	.237
LDH—Average drinks on a drinking day (DDD)	7.20 ± 4.38	5.73 ± 3.14	.001*	.405	-.18	<.001*
LDH—Total lifetime drinks (in grams of alcohol per kg bodyweight)	2,960 ± 6,030	1,710 ± 2,100	.026	.316	-.13	.013*
SMAST—Presence of alcohol abuse throughout lifetime	3.23 ± 3.68	2.48 ± 3.12	.057	.227	-.11	.053
SMAST—Presence of alcohol abuse in the last 12 months	1.74 ± 2.55	1.44 ± 2.68	.304	.114	-.06	.271
Current substance abuse diagnosis	.18 ± .38	.14 ± .35	.322	.112	-.11	.043

Note. DASS = Depression, Anxiety, and Stress Scale; CAPS = Clinical-Administered PTSD Scale; PTSD = posttraumatic stress disorder; LDH = Lifetime Drinking History; DDD = average drinks on a drinking day; SMAST = Short Michigan Alcoholism Screening Test.

* Significance after false discovery rate (FDR) correction. Correlation coefficient *r* is between the continuous cognitive score and each variable.

($\beta = -0.14$, $p = .007$) significantly predicted continuous cognitive score. As an aside, it is interesting to note that whether or not a participant was formally diagnosed with various forms of psychological distress (PTSD, anxiety, depression) was far less predictive than the severity of the condition. Using a 1.5 *SD* cutoff for defining CD demonstrated very similar results to 1 *SD* cutoff (see the online supplemental Table 1b). A version of this model in which education was controlled for is also presented in online supplemental Table 1c; this analysis showed that the effect of psychological variables was still present in the between-groups analysis but no longer significant in the continuous cognitive score analysis. This could represent the strong link between education attainment and cognitive functioning and/or that those with less psychological distress are more likely to pursue further education.

In contrast to these significant findings with psychological distress, we found that most of the hypothesized physical health factors (age, military TBI, blood glucose, HDL levels, and triglycerides) did not predict the presence or absence of CD (Table 4). Two of the hypothesized physical health factors were significantly associated with cognitive status: waist size and systolic blood pressure. A larger waist size was associated with an increased risk

of CD, but *higher* systolic blood pressure was associated with a *lower* risk of CD. Because this effect is contrary to an extensive literature on the relationship between blood pressure and cognition (for a review see Birns, Morris, Donaldson, & Kalra, 2006), we removed it from the physical health factors model. Without blood pressure, the physical health factors model was not significant ($R^2 = 0.03$, $p = .642$). Furthermore, the physical health factors predicting the continuous cognitive score also failed to reach significance ($R^2 = 0.01$, $p = .806$). Together, these findings clearly support the psychological distress model of CD more than the physical health-related model. For full models with both 1 *SD* and 1.5 *SD* cutoffs for defining CD, see the online supplementary Tables 2–3.

Are Cortical Thickness and Volume Related to CD?

To determine whether CD was related to thinner cortex in particular brain regions in this population, we compared cortical thickness and volume across the whole brain in a subset of study participants for whom MRI data was available ($n = 260$). Differences in cortical thickness, particularly in aging-associated regions

Table 4
Physical Health Information

Measure	Cognitive dysfunction	No cognitive dysfunction	<i>p</i> value for <i>t</i> test	Cohen's <i>d</i>	<i>r</i>	<i>p</i> value for <i>r</i>
McGill short form pain total score	35.29 ± 27.67	27.13 ± 24.12	.008*	.321	-.18	.001*
PSQI—Overall score	10.76 ± 4.39	9.50 ± 4.90	.014*	.266	-.18	<.001*
Waist (cm)	97.64 ± 11.82	94.16 ± 11.91	.009*	.293	-.04	.416
Average systolic blood pressure	116.90 ± 15.45	119.21 ± 12.37	.082	-.211	.04	.467
Blood glucose level	91.15 ± 30.62	89.55 ± 26.68	.625	.057	-.05	.372
High density lipoprotein (HDL)	46.52 ± 13.90	48.57 ± 12.91	.177	-.154	.01	.897
Triglycerides	136.34 ± 98.28	142.81 ± 142.90	.614	-.050	-.01	.789
Cardiometabolic Syndrome score	1.11 ± 1.10	.95 ± 1.03	.164	.156	-.06	.291

Note. PSQI = Pittsburgh Sleep Quality Index.

* Significance after false discovery rate (FDR) correction. Correlation coefficient *r* is between the continuous cognitive score and each variable.

such as the prefrontal cortex, could help provide support for the aging-related model. After cluster correction, we found that there were no significant correlations between continuous cognitive score and cortical thickness (see online supplemental Figure 1). There were also no between-groups differences in either cortical thickness or volume in any areas of the brain at either 1 *SD* or 1.5 *SD* cutoff levels, including subcortical regions. There were small subthreshold correlations between cortical thickness and continuous cognitive score in superior and inferior temporal cortices that were driven by cognitive scores in the attention domain (see online supplemental Figure 1). These data suggest that at this point in time in the participants' lives, structural differences and global cognitive associations between those with and without CD were minimal. As a positive control, we examined effects of aging on cortical thickness in our study population and, despite the somewhat limited age range in the current sample, found very robust effects (see online supplemental Figure 2).

Measures of Functional Disability

Participants with CD had significantly greater disability according to the WHODAS II global score ($M = 21.47$, $SD = 17.42$, indicating mild disability) compared with those without ($M = 15.08$, $SD = 14.23$, no disability to mild disability, $p < .001$; Table 5). This was driven by the fact that those with CD showed significantly greater disability than those without on the WHODAS II subscales of Understanding and Communicating (which includes several questions about cognition), Mobility, and Community Participation. The two groups did not differ on measures of Self Care, Getting Along, Work and School, or Life Activity. Similarly, correlation analyses revealed significant associations between continuous global cognitive scores and overall WHODAS II, $r = -0.16$, $p = .003$, which were similarly driven by the Understanding and Communicating, Mobility, and Participation subscale scores (Table 3). This suggests that CD is associated with worse self-reported daily functioning, particularly with respect to cognitive and social engagement and mobility.

Exploratory Differences/Associations With CD

We next sought to expand our analyses to explore potential differences and associations with other relevant measures of trauma-related sequelae. MTBIs during deployment were highly

prevalent in our sample. On average, our participants received 0.83 TBIs during military service and 1.63 throughout their lives and 52% of them reported experiencing "memory problems and lapses" following a TBI. Both *t* test and correlation analysis showed that although the CD group did not have more military TBIs or lifetime TBIs, they reported an increased rate of subjective "memory problems and lapses" following TBI (61.5%; Table 2). Note that the response to this question could reflect either their TBI or other co-occurring trauma-related sequelae.

Participants with CD also had significantly higher rates of sleep disturbance than those without (Table 4), although both groups had scores which indicated moderate to severe sleep problems (Buysse et al., 1989). Those with CD also had more pain than those without, with an average pain score between moderate and severe, compared with an average score between mild and moderate in the nondysfunction group (Table 4). Correlational analyses with continuous cognitive scores similarly showed that greater pain and worse sleep was associated with significantly worse cognitive performance (correlations of 0.18 and 0.16, respectively).

Discussion

The current study demonstrated that one out of every three OEF/OIF/OND Veterans exhibited clinically significant CD as measured by validated, reliable neuropsychological measures of attention, executive function, and memory. When comparing those with and without CD, we found that Veterans with CD showed significantly greater psychological distress (PTSD/anxiety/depressive symptoms/alcohol consumption) and lower estimated premorbid verbal ability. Notably, we found that psychological distress remained a significant predictor of CD even when controlling for premorbid cognitive ability. Examining correlations with a continuous measure of global cognitive performance revealed a nearly identical pattern of results. We also found that CD is associated with worse self-reported daily functioning, particularly with respect to cognitive and social engagement and mobility. We found no group differences or significant associations with physical health factors including age, physiological (cardiometabolic) or neural (cortical thickness) measures, suggesting that Veterans' current CD is not significantly associated with physical or neural decline. Together, this suggests that at this point in time ($M = 3.93$ years postdeployment), CD in trauma-exposed OEF/OIF/OND Veterans is primarily related to premorbid abilities and psycho-

Table 5
Functional Outcomes

Measure	Cognitive dysfunction	No cognitive dysfunction	<i>p</i> value for <i>t</i> test	Cohen's <i>d</i>	<i>r</i>	<i>p</i> value for <i>r</i>
WHODAS—Understanding and communicating	26.72 ± 21.57	19.66 ± 17.37	.002*	.373	-.22	<.001*
WHODAS—Participation	24.74 ± 23.03	17.42 ± 19.77	.003*	.349	-.12	.023
WHODAS—Getting along	23.51 ± 23.46	18.01 ± 19.56	.029	.262	-.11	.036
WHODAS—Life activity	22.83 ± 24.11	17.16 ± 20.11	.029	.263	-.10	.056
WHODAS—Self-care	7.08 ± 12.13	4.46 ± 9.85	.043	.244	-.08	.166
WHODAS—Work and School	23.08 ± 23.04	21.36 ± 22.96	.560	.075	.04	.477
WHODAS—Mobility	18.60 ± 21.54	9.33 ± 14.40	<.001*	.538	-.21	<.001*
WHODAS—Overall score	21.47 ± 17.42	15.08 ± 14.23	<.001*	.414	-.18	.001*

Note. WHODAS = the World Health Organization Disability Assessment Schedule II.

* Significance after false discovery rate (FDR) correction. Correlation coefficient *r* is between the continuous cognitive score and each variable.

logical distress symptoms. This leaves open the possibility that their CD may be at least partially ameliorated by treating trauma-related psychological symptoms.

We found that the prevalence of CD in our sample of OEF/OIF/OND Veterans was 35.1%. We are not aware of any reports measuring the prevalence of clinically significant CD in a younger population without a common serious disease, so it is difficult to determine whether our reported rate of 35.1% represents an unexpectedly high rate of dysfunction. The prevalence of clinically significant CD is necessarily related to the definition of CD, which is broader in the *DSM-5* than in previous versions of the *DSM*. In addition, recent studies have demonstrated that assessment of cognitive status in Veterans with PTSD is complicated by the interrelationship between memory difficulties and anxiety disorders (Lippa et al., 2017), and furthermore, our choice of performance validity score cutoff (the MSVT score below which we did not analyze data any further) may have affected our outcomes (Lippa et al., 2017). However, regardless of whether the prevalence in our sample is unexpected, 35.1% of returning OEF/OIF/OND Veterans constitutes greater than 650,000 individuals in likely need of care (U.S. Department of Veterans Affairs, 2017). Of Veterans with CD, we found that 28.7% had a deficit in the memory domain, 24.0% had executive function deficits, 24.0% had attention deficits, and the remaining Veterans had deficits in more than one domain. It is important that the current results also demonstrate that Veterans with CD reported significantly worse daily life functioning on the WHODAS II global score, with particularly worse scores in the domains of understanding and communicating (which includes several self-report cognitive questions), participating in community/society, and getting around. Overall, Veterans with CD reported mild but significant disability in these categories whereas those without CD had no significant disability. These categories of the WHODAS II are particularly relevant to social functioning and suggest that the objective CD we observed is likely accompanied by real-world self-reported difficulties.

Our findings support the hypothesis that this higher rate of CD and associated disability may be caused, at least in part, by deployment-related psychological distress and factors related to it. We found that those with CD, compared with those without, exhibited greater symptoms of anxiety, depression, and PTSD, as well as increased drinking, showing small to medium effect sizes. Exploratory analyses also revealed that those with CD also had worse sleep and increased pain, which are associated with trauma-related psychological distress in this population (e.g., Lippa et al., 2017). It is important that all of these between-groups differences remained significant after controlling for premorbid verbal ability (WTAR score), suggesting these differences are not fully explained by premorbid factors. These findings build on numerous studies of associations observed between trauma sequelae (e.g., PTSD, depression) and decreased memory, executive, and attention performance (Amick et al., 2013; DeGutis et al., 2015; Esterman et al., 2013; Nietlisbach & Maercker, 2009; Qureshi et al., 2011) by demonstrating that these cognitive impairments can extend into the clinically significant range. It is notable that the categorical diagnoses of PTSD, depression, anxiety, and substance abuse did not do nearly as good of a job of differentiating those with and without clinically significant CD as the continuous measures of these variables. This suggests that those with the severest

levels of PTSD, depression, anxiety, and substance abuse are those experiencing clinically significant CD.

The current results demonstrate that drinking behavior was uniquely predictive of clinically significant CD, while PTSD and depression did not uniquely predict CD and anxiety did not predict CD after controlling for premorbid verbal ability. The failure of PTSD, anxiety, and depression to predict unique variance could be because they measure aspects of the same underlying pathology, whereas alcohol consumption may represent a distinct mechanism. The high levels of alcohol consumption in the current population and its unique association with CD is not surprising given the well-known associations between trauma-related psychological distress and alcohol use (Brady & Sonne, 1999), and between chronic alcohol use and CD (Stavro, Pelletier, & Potvin, 2013). Even when a diagnosable substance use disorder is not present, heavy drinking has shown to be associated with deficits in executive function, memory and visuospatial skills (Green et al., 2010; Houston et al., 2014). However, research also suggests that abstinence/detoxification results in significant improvements to cognition, with little to no remaining effects after 1 year (for a review, see Stavro et al., 2013). If the CD in our sample is caused in part by excessive alcohol consumption, it may be treatable with therapies to reduce alcohol use and improve coping skills, such as cognitive-behavioral therapy (Sannibale et al., 2013).

Though the current results suggest that psychological distress-related trauma sequelae lead to clinically significant CD, they are also consistent with predeployment cognitive vulnerabilities both producing CD and making OEF/OIF/OND Veterans more susceptible to experiencing psychological distress. There is an extensive literature to support the idea that the trait of proneness to psychological distress is a predictor of future cognitive health (Wilson et al., 2003, 2007; Wilson et al., 2005). In addition, those with premorbid cognitive weaknesses such as poor verbal memory and sustained attention are more likely to experience PTSD following a traumatic event (Breslau, Chen, & Luo, 2013; Brewin, Andrews, & Valentine, 2000; Vasterling et al., 2002). Although we had only administered a single measure of premorbid ability, we did find a lower estimate of premorbid verbal ability (WTAR) in the CD group, and WTAR score was found to uniquely predict CD.

Our results suggest that neuropsychological performance could potentially be at least partially remediated via successful treatment of psychological distress. There is a substantial literature supporting the effectiveness of interventions for PTSD (Sutherland & Bryant, 2007), depression (Levkovitz, Caftori, Avital, & Richter-Levin, 2002; Richardson et al., 1994), sleep (Altena, Van Der Werf, Strijers, & Van Someren, 2008; Mander et al., 2010), and substance use disorders to improve cognitive functioning. For example, Sutherland and Bryant (2007) reported improved autobiographical memory performance in those with PTSD after cognitive-behavioral therapy. In studies conducted with younger depressed subjects, pharmacological treatment (selective serotonin reuptake inhibitor) yielded cognitive improvements in declarative memory, psychomotor speed, and attention (Levkovitz et al., 2002). In addition, abstinence from alcohol use has shown to improve cognition, particularly attentional abilities in those with substance use disorders (Schulte et al., 2014).

Education had a moderating effect on the current results and warrants further investigation. When education was included in the model, no psychological distress variables uniquely predicted the

continuous global cognitive score. However, there were still significant associations between CD status and psychological variables even when controlling for education. This result could mean that strengthening cognition, either indirectly (e.g., through diet and exercise, see Gomez-Pinilla, 2011, or continuing education) or possibly directly (through cognitive enhancers, e.g., Ressler et al., 2004; Sofuoglu, Devito, Waters, & Carroll, 2013) might result in decreased symptoms of anxiety, depression and substance abuse. However, it could also mean that Veterans with severe psychological distress receive less education than their less-affected peers as a result of their psychological status. PTSD and other traumatic consequences of military service have previously been shown to be a barrier to continuing education (see Borsari et al., 2017). In our sample, the average time since deployment was almost 3.5 years, and our data set does not differentiate between pre- and postdeployment education. Therefore, the results of similar studies in OEF/OIF/OND Veterans would be helpful in determining the malleability of CD in this population as well as the relative importance of trauma-related versus premorbid factors in postdeployment CD.

In contrast to the psychiatric variables, our data provide little evidence that there are differences in physical health and cortical thinning between those with and without clinically significant CD. Those with and without clinically significant CD did not vary in age, cortical thickness, or cardiometabolic risk factors. Similarly, continuous measures of global cognition did not reveal significant associations with these measures (all $ps > 0.26$). Together, this suggests that aging-related factors were not significant contributors to clinically significant CD in the current population. That said, with increasing age, it is likely that these physical health factors may begin to play more of a role. For example, cardiometabolic indicators may extend more into the pathological range in the future, similar to other Veteran populations (Taylor & MacQueen, 2007; Xu et al., 2017). It has also been shown that CD may lag behind cardiometabolic dysfunction by several years (Taylor & MacQueen, 2007). Thus, differences in age-related factors such as cardiometabolic and neural health may emerge in those with CD in the future. Another possible explanation for why we found no physical health-related effects are that those changes are caused by specific conditions rather than being a general mechanism affecting all Veterans in our sample. Several studies have suggested that PTSD could lead to premature senescence in a variety of physical markers, including telomeres, inflammatory markers and DNA methylation (Lohr et al., 2015; Wolf, Logue, et al., 2016). MTBI, another condition with high prevalence in our population, has also been associated with aging-related, neural compromise (Lindemer, Salat, Leritz, McGlinchey, & Milberg, 2013). Examining CD in a larger population of Veterans with PTSD or mTBI would be a natural next step in determining which mechanism(s) lead to CD in these subpopulations.

Though the current study provides important insights into clinically significant CD in this population, it has limitations. Most important, because this study was cross-sectional rather than longitudinal, our ability to determine the cause(s) of CD was limited, and we were unable to determine whether a decline in function had taken place. Furthermore, we had very few measures of premorbid health and cognitive ability, making it difficult to fully assess the degree to which premorbid differences might have influenced outcome. This issue is of particular importance in the Veteran population because estimates of premorbid cognitive ability that

apply to the general population likely do not apply to Veterans. Admission to the U.S. Armed Forces is allowed only if individuals pass certain aptitude tests, including cognitive tests; therefore, the spectrum of cognitive ability in the Veteran population is likely not as broad as in the general population.

In investigating the relationship between cortical thickness and volume and CD, we found no significant relationship. However, it is possible that the structural MRI scans used in this study were not ideal for detecting the changes in brain structure or anatomy related to CD. Diffusion imaging or functional MRI may be more sensitive to differences in this population (Poole et al., 2016; Spielberg et al., 2017). Future investigations using a more comprehensive battery of neuroimaging measures would be useful to determine whether there are neural differences between groups. In addition, our measure of self-reported daily life functioning, WHODAS II, may have been affected by self-report biases (Rodriguez, Holowka, & Marx, 2012). Including collateral reports from family members and third parties would be useful to better measure the degree of functional disability in those with and without CD. Finally, our exclusion of 22 individuals who showed evidence of reduced effort likely led us to underestimate the incidence of clinically significant CD in this particular population, because OEF/OIF/OND Veterans who failed this measure have shown to exhibit significantly worse cognitive performance and increased psychological distress symptoms compared with those who passed (Clark, Amick, Fortier, Milberg, & McGlinchey, 2014).

In sum, our results suggest that psychological distress is a likely contributor to CD in younger OEF/OIF/OND Veterans, either as a direct result of their traumatic experiences or as a result of underlying cognitive vulnerabilities exacerbated by their experiences. In either case, treatment for depression, anxiety and alcohol use problems would likely improve cognitive function and increase the quality of life of these individuals.

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