


## Trauma Sequelae are Uniquely Associated with Components of Self-Reported Sleep Dysfunction in OEF/OIF/OND Veterans

Joseph DeGutis, Christopher Chiu, Michelle Thai, Michael Esterman, William Milberg & Regina McGlinchey

To cite this article: Joseph DeGutis, Christopher Chiu, Michelle Thai, Michael Esterman, William Milberg & Regina McGlinchey (2016): Trauma Sequelae are Uniquely Associated with Components of Self-Reported Sleep Dysfunction in OEF/OIF/OND Veterans, Behavioral Sleep Medicine, DOI: [10.1080/15402002.2016.1173550](https://doi.org/10.1080/15402002.2016.1173550)

To link to this article: <http://dx.doi.org/10.1080/15402002.2016.1173550>

 View supplementary material 

 Published online: 16 May 2016.

 Submit your article to this journal 

 View related articles 

 View Crossmark data 

# Trauma Sequelae are Uniquely Associated with Components of Self-Reported Sleep Dysfunction in OEF/OIF/OND Veterans

Joseph DeGutis

*Boston Attention and Learning Laboratory, Boston Division VA Healthcare System, Boston, Massachusetts, USA*  
*Geriatric Research Education and Clinical Center, Boston Division VA Healthcare System, Boston, Massachusetts, USA*  
*Department of Medicine, Harvard Medical School, Boston, Massachusetts, USA*

Christopher Chiu and Michelle Thai

*Boston Attention and Learning Laboratory, Boston Division VA Healthcare System, Boston, Massachusetts, USA*  
*Geriatric Research Education and Clinical Center, Boston Division VA Healthcare System, Boston, Massachusetts, USA*

Michael Esterman

*Boston Attention and Learning Laboratory, Boston Division VA Healthcare System, Boston, Massachusetts, USA*  
*Geriatric Research Education and Clinical Center, Boston Division VA Healthcare System, Boston, Massachusetts, USA*  
*Department of Psychiatry, Boston University School of Medicine, Boston, Massachusetts, USA*

William Milberg and Regina McGlinchey

*Geriatric Research Education and Clinical Center, Boston Division VA Healthcare System, Boston, Massachusetts, USA*  
*Translational Research Center for TBI and Stress Disorders (TRACTS), VA RR&D TBI Center of Excellence, VA Boston Healthcare System, Boston, Massachusetts, USA*

---

This article not subject to US copyright law.

Correspondence should be addressed to Joseph DeGutis, Geriatric Research Education and Clinical Center (GRECC), Boston VA Healthcare System, 150 S. Huntington Ave. (182-JP), Boston, MA 02130. E-mail: [degutis@wjh.harvard.edu](mailto:degutis@wjh.harvard.edu)  
Color versions of one or more figures in the article can be found online at [www.tandfonline.com/hbsm](http://www.tandfonline.com/hbsm).

While the associations between psychological distress (e.g., posttraumatic stress disorder [PTSD], depression) and sleep dysfunction have been demonstrated in trauma-exposed populations, studies have not fully explored the associations between sleep dysfunction and the wide range of common physical and physiological changes that can occur after trauma exposure (e.g., pain, cardiometabolic risk factors). We aimed to clarify the unique associations of psychological and physical trauma sequelae with different aspects of self-reported sleep dysfunction. A comprehensive psychological and physical examination was administered to 283 combat-deployed trauma-exposed Operation Enduring Freedom/Operation Iraqi Freedom/Operation New Dawn (OEF/OIF/OND) veterans. The Pittsburgh Sleep Quality Index (PSQI) and PSQI Addendum for PTSD (PSQI-A) were administered along with measures of PTSD, depression, anxiety, pain, traumatic brain injury, alcohol use, nicotine dependence, and cardiometabolic symptoms. We first performed a confirmatory factor analysis of the PSQI and then conducted regressions with the separate PSQI factors as well as the PSQI-A to identify unique associations between trauma-related measures and the separate aspects of sleep. We found that the PSQI global score was composed of three factors: Sleep Efficiency (sleep efficiency/sleep duration), Perceived Sleep Quality (sleep quality/sleep latency/sleep medication) and Daily Disturbances (sleep disturbances/daytime dysfunction). Linear regressions demonstrated that PTSD symptoms were uniquely associated with the PSQI global score and all three factors, as well as the PSQI-A. For the other psychological distress variables, anxiety was independently associated with PSQI global as well as Sleep Efficiency, Perceived Sleep Quality, and PSQI-A, whereas depression was uniquely associated with Daily Disturbances and PSQI-A. Notably, cardiometabolic symptoms explained independent variance in PSQI global and Sleep Efficiency. These findings help lay the groundwork for further investigations of the mechanisms of sleep dysfunction in trauma-exposed individuals and may help in the development of more effective, individualized treatments.

Studies suggest that as many as 60–90% of individuals have experienced at least one traumatic event in their lifetime, including natural disasters, sexual assault, war, and accidents (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Kilpatrick et al., 2013) and sleep problems are one of the most commonly reported complaints among trauma-exposed individuals (Lavie, 2001). Additionally, of the approximately 10% of those exposed to trauma who develop posttraumatic stress disorder (PTSD; Kilpatrick et al., 2013), 70–87% have trouble initiating or maintaining sleep or experience nightmares, making sleep difficulties the most highly reported PTSD symptom (Maher, Rego, & Asnis, 2006). Sleep difficulties commonly occur immediately after trauma exposure (Wood, Bootzin, Rosenhan, Nolen-Hoeksema, & Jourden, 1992) and may persist long after the trauma (> 45 years later, Goldstein, van Kammen, Shelly, Miller, & van Kammen, 1987; Rosen, Reynolds, Yeager, Houck, & Hurwitz, 1991). Further, sleep difficulties in trauma-exposed populations can arise indirectly from other issues resulting from trauma, such as depression or anxiety (e.g., Sheikh, Woodward, & Leskin, 2003), substance abuse (e.g., Stewart, Pihl, Conrod, & Dongier, 1998), and cardiometabolic dysfunction (e.g., Heppner et al., 2009; Vgontzas, Bixler, & Chrousos, 2005). Sleep problems can significantly impair cognitive performance and general daytime functioning, can increase the risk of accidents (Durmer & Dinges, 2005; Swanson et al., 2011), can increase suicide risk (Krakow, Artar et al., 2000). Understanding sleep in trauma-exposed individuals is important, but what is not understood is what factors resulting from trauma are most related to disrupted sleep. The goal of the current study is to (a) identify the different aspects of sleep dysfunction in individuals exposed to trauma and (b) characterize more fully how specific trauma sequelae (e.g., PTSD, depression, anxiety, substance use, cardiometabolic risk factors, pain) are associated with unique variance in these different aspects of sleep.

For years, researchers and clinicians have appreciated that sleep difficulties and nightmares are core aspects of PTSD (for a review, see Ross, Ball, Sullivan, & Caroff, 1989, and Germain, 2013) and some studies even suggest a reciprocal PTSD/sleep relationship (Maher et al., 2006; though see Zayfert & DeViva, 2004). For example, PTSD is associated with increased arousal, potentially resulting in lighter sleep, as well as nightmares, which can cause frequent awakenings. Furthermore, in order to avoid stressful nightmares, individuals with PTSD may postpone sleeping (Harvey & Bryant, 1998). Conversely, sleep problems prior to and following trauma exposure have also shown to predispose individuals to develop PTSD as well as aggravate and prolong PTSD symptoms (Harvey, Jones, & Schmidt, 2003; Van der Kolk, Hartmann, Burr, & Blitz, 1980). In particular, Harvey and Bryant (1998) found that sleep difficulties measured within one month after trauma exposure increased the likelihood of developing PTSD symptoms 6 months posttrauma. Moreover, several studies have demonstrated that treatments targeting sleep problems (e.g., cognitive behavioral therapy for nightmares, Germain et al., 2012; imagery rehearsal therapy, Krakow et al., 2001; Krakow, Hollifield et al., 2000; pharmacotherapies, Maher et al., 2006; or cognitive behavioral therapy for insomnia, Talbot et al., 2014) can effectively reduce PTSD symptoms. Interestingly, treating PTSD symptoms may not always improve sleep (Zayfert & DeViva, 2004) and unfortunately, many PTSD treatment trials neglect to report formal sleep outcomes. Still, the available data suggest that PTSD symptoms and sleep difficulties may be at least partially dissociable.

Other psychiatric sequelae of trauma beyond PTSD can also independently impact sleep. For example, anxiety-related problems, including panic disorder and generalized anxiety disorder, are more likely to occur after exposure to trauma (Kessler et al., 1995; Krakow, Melendrez et al., 2002; Krakow, Schrader et al., 2002). Anxiety over falling asleep may delay sleep onset and anxiety responses to nightmares may also increase sleep disturbances (Lamarche & De Koninck, 2007). The development of mood disorders, including major depressive disorder and dysthymic disorder, is also prevalent following trauma (Perkonig, Kessler, Storz, & Wittchen, 2000). More severe depression is associated with a higher risk of developing insomnia (Alvaro, Roberts, & Harris, 2013) and studies suggest a bidirectional relationship between depression and insomnia (e.g., Sivertsen et al., 2012).

Beyond psychological distress, more recently researchers have focused on trauma and physical outcomes, which may have unique associations with sleep (Dedert, Calhoun, Watkins, Sherwood, & Beckham, 2010). Cardiometabolic syndrome, a constellation of maladaptive cardiovascular, renal, metabolic, prothrombotic, and inflammatory abnormalities, substantially increases cardiovascular disease (CVD) morbidity and mortality (e.g., Castro, El-Atat, McFarlane, Aneja, & Sowers, 2003). Talbot and colleagues (2015) recently found that PTSD was strongly linked to increased cardiometabolic risk in healthy younger adults ( $M$  age = 30.6 yrs) and that this was related to reduced sleep duration. Further, Dedert and colleagues (2010) report that exposure to trauma is linked with cardiovascular and metabolic morbidity such as obesity. Obesity is the strongest risk factor for sleep-disordered breathing, a contributor to poor sleep and daytime sleepiness (Bennett, Barbour, Langford, Stradling, & Davies, 1999; Redline & Strohl, 1999). Additionally, a growing literature provides evidence that poor sleep can lead to increased cardiometabolic risk factors (e.g., high blood pressure, diabetes, and metabolic dysregulation, Schmid, Hallschmid, & Schultes, 2015), which may explain part of the relationship between trauma and increased cardiometabolic symptoms. It is unclear whether the relationship between cardiometabolic risk factors and PTSD is independent of or mediated by the relationship between sleep and cardiometabolic risk factors. One of the goals of the current study is to better understand the nature of these relationships.

After a traumatic experience, individuals are also more likely to develop substance use problems, such as with alcohol and nicotine (Stewart, 1996). Long-term excessive alcohol use is associated with poorer, more fragmented sleep (Lamarche & De Koninck, 2007). Similarly, nicotine use is associated with an increased difficulty falling asleep, excessive daytime sleepiness, and increased number of awakenings (for a review see Jaehne, Loessl, Bárkai, Reimann, & Hornyak, 2009). Chronic pain is also prevalent among individuals who have experienced trauma (Löwe et al., 2011) and is related to increases in sleep disturbances and nightmares (Gellis, Gehrman, Mavandadi, & Oslin, 2010). Additionally, traumatic brain injuries (TBI) can be associated with trauma exposure, especially in veterans (Klein, Caspi, & Gil, 2003). Up to 80% of individuals who experienced a TBI (including mild TBI) experience sleep difficulties ranging from daytime sleepiness to insomnia (Lippa et al., 2015; Orff, Ayalon, & Drummond, 2009). Thus, it is clear that many variables are associated with poor sleep, and more thoroughly characterizing the independent relationships of each of these trauma sequelae to different aspects of sleep difficulties could better inform models of sleep and trauma and also provide targets for treatments.

In trauma-exposed populations, both polysomnography (PSG) and self-reports of sleep quality are commonly used to characterize sleep anomalies (Babson & Feldner, 2010). Though PSG studies typically focus more on the effects of PTSD diagnosis than trauma exposure, one notable early PSG study of a variety of traumatic events (Holocaust, combat, and sea disaster) found that trauma survivors had reduced sleep efficiency, increased sleep latency, and reduced rapid eye movement (REM) time compared to controls not exposed to a traumatic event (Hefez, Metz, & Lavie, 1987). However, more recent PSG studies have not found differences between those exposed to trauma (without PTSD) and controls (e.g., Breslau et al., 2004). Focusing on the effects of PTSD diagnosis, a PSG meta-analysis found that individuals with PTSD have slightly more stage 1 (shallower) sleep and less slow-wave sleep as well as *greater* rapid-eye movement density (amount of rapid eye movements per unit of time) compared to controls without PTSD (Kobayashi, Boarts, & Delahanty, 2007). However, these effects in PTSD are small and somewhat variable and several studies have found no differences between those with and without PTSD (e.g., Maher et al., 2006). One explanation of these rather variable PSG results is that individuals suffering from trauma exposure and PTSD may actually sleep better in a “safe” laboratory setting than at home (Davis, 2009).

In contrast to these PSG results, trauma-exposed and PTSD populations more consistently show sleep dysfunction on self-report measures such as the Pittsburgh Sleep Quality Index (PSQI; see Lavie, 2001, for a review). Recent studies of self-reported sleep have found that rather than being a monolithic construct, sleep is made up of several components, such as sleep initiation, maintenance, dreams and nightmares, and daytime functioning (Cole et al., 2006). Understanding how self-reported sleep is broken down into subcomponents could provide both important mechanistic and clinically relevant information. One widely accepted PSQI model is the 3-factor model from Cole et al. (2006), consisting of Sleep Efficiency, Perceived Sleep Quality, and Daily Disturbances factors. Several studies have validated Cole’s 3-factor model in healthy populations of different ethnicities and backgrounds (Babson, Blonigen, Boden, Drescher, & Bonn-Miller, 2012; Koh, Lim, Chia, & Lim, 2015; Magee, Caputi, Iverson, & Huang, 2008). To our knowledge, only two studies have explored the factor structure of the PSQI in trauma-exposed populations (Babson et al., 2012; Casement, Harrington, Miller, & Resick, 2012). Babson and colleagues (2012) studied 226 veterans ( $M$  age = 51.8) and found the

data was best fit by a 2-factor model composed of Perceived Sleep Quality and Sleep Efficiency. They found that depression independently predicted both factors but PTSD only independently predicted Perceived Sleep Quality. In contrast, Casement and colleagues (2012) examined 319 female victims of sexual and physical assault with PTSD and confirmatory factor analyses supported Cole's (2006) 3-factor model composed of Sleep Efficiency, Perceived Sleep Quality, and Daily Disturbances. They further found that severity of depressive symptoms and severity of physical symptoms (e.g., racing heart, headaches) correlated the highest with the Daily Disturbances factor (Casement et al., 2012). In contrast, PTSD symptoms were most strongly associated with Perceived Sleep Quality and Sleep Efficiency. Together, these results demonstrate a particularly strong relationship between PTSD symptoms and sleep quality (e.g., sleep latency and perceived sleep quality) and further suggest that other clinical variables, such as physical symptoms and depression, are related to separable aspects of sleep.

Despite these important insights, studies have not yet fully explored, in a single sample, the range of sleep dysfunction-related variables beyond PTSD, depression, and general physical symptoms in trauma-exposed individuals. Characterizing a wider range of variables is important because clinicians may assume that PTSD symptoms, and possibly depressive symptoms, are the only cause or consequence of patients' sleep difficulties and may ignore other important clinical variables, missing potential opportunities for treatment. In the present study, we aimed to clarify the unique association of a full range of trauma sequelae to different aspects of sleep in a relatively large sample ( $N = 283$ ) of trauma-exposed Operation Enduring Freedom/Operation Iraqi Freedom/Operation New Dawn (henceforth OEF/OIF/OND) veterans. We measured sleep using the PSQI and PSQI Addendum for PTSD (PSQI-A) and several relevant trauma-related variables, including common symptoms of psychological distress (PTSD symptoms, depression, anxiety), pain, cardiometabolic risk factors (waist circumference, triglycerides, HDL cholesterol, blood pressure, and glucose), substance use, and traumatic brain injury. To characterize the underlying factor structure of participants' sleep complaints, we used a confirmatory factor analysis (CFA) approach, comparing previous empirical models of the PSQI, including Cole et al. (2006), Magee et al. (2008), and Babson et al. (2012). Next, to identify variables that are associated with unique variance in global sleep quality as well as subcomponents of sleep, we performed regression analyses using these sleep factors, PSQI global, and PSQI-A as dependent measures. We hypothesized that PTSD symptoms would be strongly associated with all aspects of sleep and that physical symptoms (e.g., cardiometabolic risk factors, pain) would be uniquely associated with components of sleep independent of PTSD symptomology. For observed significant trauma sequelae and sleep relationships, we also sought to perform follow-up analyses, such as mediation, to better determine the nature of these relationships (e.g., whether the cardiometabolic risk factor and sleep association mediates the relationship between PTSD and cardiometabolic risk factors).

## METHODS

### Participants

Participants were drawn from a pool of 331 OEF/OIF/OND veterans recruited between 2009 and 2014 into the TBI Center of Excellence at the Boston VA Healthcare System—the Translational



Research Center for TBI and Stress Disorders (TRACTS). Participants were recruited from the Boston metropolitan area via a full-time recruitment specialist who attended Yellow Ribbon Events, Task Force Meetings, and other events involving U.S. Air Force, Marine, Army, National Guard, and Reserve units. Participants were not specifically recruited from medical or mental health clinics; however, a minority of participants also contacted our recruitment specialist in response to flyers posted in our VA (Veterans Affairs) medical center. Participants took part in TRACTS for research purposes examining the psychological, physical, and neurological health of returning veterans and received compensation for their time. Each participant underwent a comprehensive neuropsychological and psychological evaluation lasting 8–10 hr as well as physical health assessments (e.g., blood pressure, blood draw to determine cholesterol) and were reimbursed \$210 for their time and travel costs.

A total of 48 participants were excluded. In particular, participants were excluded if there was evidence of reduced effort (according to the Medical Symptom Validity Test [Green, 2004],  $n = 19$ ), if their traumatic brain injury (TBI) severity was greater than mild head injury severity according to the Boston Assessment of Traumatic Brain Injury-Lifetime (Fortier et al., 2014; moderate and severe TBIs may be qualitatively different from mild TBIs,  $n = 12$ ), and/or if they were not deployed to combat ( $n = 19$ ). Psychiatric exclusionary criteria included psychotic disorders, bipolar disorder, and suicidal or homicidal ideation requiring intervention as determined by the Structured Clinical Interview for the DSM-IV (American Psychiatric Association, 1994;  $n = 1$ ). The final sample consisted of 283 participants (age:  $M = 31.51$ ,  $SD = 8.16$ ; 253 males). The participant pool completed an average of 13.80 years of education ( $SD = 1.89$ ) and had an average estimated IQ of 102.38 based on the Wechsler Test of Adult Reading ( $SD = 11.56$ ). The average time since deployment was 37.27 months ( $SD = 29.60$ ) and the average length of total deployments was 14.20 months ( $SD = 8.32$ ). The race and ethnic breakdown of our selected clinical sample was: 71.7% Caucasian, 15.5% Hispanic/Latino, 8.5% African American, 1.8% Asian, and .7% Native American. Out of these 283 participants, 167 had a current diagnosis of PTSD according to the Clinician Administered PTSD Scale (CAPS; see below for more details; Weathers, Ruscio, & Keane, 1999). This study was approved by the VA Boston IRB; written consent was obtained from all participants and research was conducted according to the Declaration of Helsinki.

## Sleep Measures

### *Sleep quality*

The Pittsburgh Sleep Quality Index (PSQI) is a 19-item self-report questionnaire that assesses sleep quality over the last month. From these items, seven component scores and a global score are extracted as established by Buysse and colleagues (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The first four items are free responses and include questions about bedtime, wake time, and sleep duration. The questionnaire also includes items that are rated on a 4-point Likert scale (0 = not during the last month, 3 = three or more times a week or 0 = very good, 3 = very bad), covering the quality of sleep and the frequency of common sleep disturbances, sleep medication, as well as daytime fatigue. To determine the frequency of problematic sleep patterns in our sample (see Figure 1), we used established cutoffs for each of the following components

and global score: a total PSQI global score greater than 5 indicates general sleep problems (Buysse et al., 1989); an answer of either “Fairly Bad” or “Very Bad” to the question, “How would you rate your sleep quality over all?” indicates self-reported bad sleep (Buysse et al., 1989); a score of less than 85% (calculated by dividing the number of hours slept by the number of hours in bed) indicates poor sleep efficiency (Perlis et al., 2010). Sleep duration is assessed by the question “How many hours of actual sleep did you get at night?” Carpenter and Andrykowski (1998) reported a Cronbach’s  $\alpha$  of .80 for the global PSQI score and  $\alpha = .70$  to .78 for the sleep disturbance component. The PSQI global score correlates highly with other widely used measures of sleep (e.g., sleep problems: Symptom Experience Report; sleep quality: Sleep, Energy, and Appetite Scale), all  $r$ ’s  $\geq .65$  (Carpenter & Andrykowski, 1998).

### *Disruptive nocturnal behavior*

The PSQI addendum (PSQI-A), a 7-item supplementary assessment to the PSQI, focuses on 7 disruptive nocturnal behaviors (DNB) to assess sleep-related difficulties associated with PTSD. Participants report the frequency of each DNB (e.g., nightmares, feeling nervous) for this measure. A cutoff score of 4 has shown to indicate severe PTSD symptomology based on DNB (Germain, Hall, Krakow, Katherine Shear, & Buysse, 2005). Germain et al. (2005) reported a Cronbach’s  $\alpha$  of .85. PSQI-A significantly correlates with the Clinical Administered PTSD Scale ( $r = .53$ ).

## Psychological Assessments

### *PTSD*

The Clinician Administered PTSD Scale (CAPS) is a semistructured interview that assesses the presence and severity of PTSD based on the 17 symptoms of PTSD as defined by the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; DSM-IV; American Psychiatric Association, 1994); CAPS is considered the gold standard for the diagnosis of PTSD (Blake et al., 1995). PTSD diagnosis was based on the CAPS scoring rule of three, where to receive a PTSD diagnosis, one must have  $\geq 1$  symptom for Criterion A,  $\geq 3$  symptoms for Criterion B, and  $\geq 2$  symptoms for Criterion C and a frequency rating + intensity rating of 3 or higher for each symptom (Blanchard et al., 1995). Weathers, Keane, and Davidson (2001) found Cronbach’s  $\alpha$  ranges from .80 to .90. CAPS also correlates at or above .70 with other well-established self-report measures of PTSD (e.g., the Mississippi Scale for Combat-Related PTSD and the PTSD Checklist; see Weathers et al., 2001 for a review). The variable of interest was current CAPS total score without the 2 sleep symptoms, which is a total of all of PTSD Criterion scores after excluding CAPS B2: distressing dreams and CAPS D1: difficulty falling or staying asleep. We also ran the analyses with CAPS and sleep items left in and the results were extremely similar to the results without these sleep items (see supplementary materials).

### *Traumatic life events*

To complement the CAPS and further assess for trauma exposure, we administered the Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000). The TLEQ is a 22-item measure that includes 21 life experiences that could be experienced as traumatic, including



events such as motor vehicle accidents, physical violence, and natural disasters. Participants indicate whether they experienced each life event, the number of times they encountered each event, and whether they experienced *intense fear*, *helplessness*, or *horror*. This measure has demonstrated acceptable validity and test–retest reliability, an average Cohen’s kappa of .69 after one week with university students, and an average kappa of .52 after two weeks with Vietnam combat veterans. Kubany et al. (2000) found a kappa of .71 comparing disclosure rates between the TLEQ and the Traumatic Life Events Interview.

### *Combat experiences*

The Combat Experiences module of the Deployment Risk and Resilience Inventory (DRRI) was developed based on contemporary war zones to assess PTSD and trauma exposure independent from other anxiety-related symptomology (King, King, Vogt, Knight, & Samper, 2006). Validity and reliability of the DRRI have been well-established using samples of Gulf War veterans (King et al., 2006) as well as with Operation Iraqi Freedom (OIF) veterans (Vogt, Proctor, King, King, & Vasterling, 2008) with internal consistency reliability ranging from .55 to .90.

### *Depression and anxiety*

To assess mood and anxiety-related symptoms, we also administered the Depression Anxiety and Stress Scale (DASS), which is a 21-item self-report questionnaire composed of 3 different scales to measure the common symptoms related to depression and anxiety in relation to the past week. The questionnaire is scored based on a 4-point Likert scale for both severity and frequency (Lovibond & Lovibond, 1995). Antony, Bieling, Cox, Enns, & Swinson (1998) reported a Cronbach’s  $\alpha$  of .94 for the depression Subscale and an  $\alpha$  of .87 for the anxiety subscale. The DASS depression scale correlates with a well-known measure of depression symptomology, the Beck Depression Inventory,  $r = .79$ , and the DASS anxiety scale correlates with the Beck Anxiety Inventory,  $r = .85$ . Our variables of interest were the total scores for the depression scale and for the anxiety scale.

## Physical, Physiological, and Traumatic Brain Injury Assessment

### *Traumatic brain injury*

The Boston Assessment of Traumatic Brain Injury–Lifetime (BAT-L) is a semistructured interview to diagnose and characterize TBIs throughout a participant’s life span (Fortier et al., 2014). Injuries are assessed before military service, during military service, and after military service. The BAT-L defines a mild TBI as having a loss of consciousness (LOC) between 5 and 30 min or having either an alteration of mental status (AMS) or posttraumatic amnesia (PTA) for greater than 15 min but less than 24 hr. A moderate TBI is defined as having a LOC between 30 min and 24 hr, or AMS/PTA for greater than 24 hr. A severe TBI is defined as having a LOC greater than 24 hr or a PTA greater than 7 days. Fortier et al. (2014) reported strong interrater reliability for the BAT-L, all Cohen  $\kappa$ ’s  $> .80$ . The BAT-L also highly correlates with a well-established measure of TBI, the Ohio State University TBI Identification Method, Cohen  $\kappa = .89$ ; Kendall  $\tau_{-b} = .95$ . Our variable of interest was the total

number of military TBIs because recent work has shown that cumulative mild TBIs explain sleep disturbances in military personnel above and beyond PTSD and depression (Bryan, 2013) and, out of our TBI variables, the number of military TBIs showed the largest average zero-order correlation with the sleep components.

### *Alcohol use*

The Lifetime Drinking History Questionnaire (LDH) is a semistructured interview that examines alcohol use throughout the life span. The LDH identifies drinking phases over the life course to help identify current and lifetime alcohol problems (Skinner & Sheu, 1982). The variable of interest was the average number of standard drinks consumed on a drinking day. Skinner and Sheu (1982) reported a reliability estimate of .68 for the daily average variable. Furthermore, LDH daily average correlates with the Michigan Alcohol Screening test ( $r = .50$ ; Skinner & Sheu, 1982).

### *Pain*

The Short-form McGill Pain Questionnaire (SF-MPQ) is a self-report questionnaire consisting of 3 types of measures: a visual analog scale rating index of average pain in the last month (ranging from “no pain” to “worst possible pain”), 4-point Likert scale ratings (0=none, 3=severe) to different pain adjectives, and a 6-point rating of current overall pain (ranging from “no pain” to “excruciating”). The assessment uses 15 adjectives to evaluate quantitative experiences of subjective pain (Melzack, 1987). Hawker, Mian, Kendzerska, and French (2011) found Cronbach’s  $\alpha$  ranges from .73 to .89. The questionnaire correlates strongly with the original MPQ questionnaire ( $r$ 's  $> .62$ ) (Melzack, 1987) as well as another well-established measure of pain (e.g., the Short Form 36 Health Survey Bodily Pains Scales,  $r = -.36$ ; Hawker et al., 2011). Because the PSQI asks about sleep over the last month, for consistency we chose to use the measure of average pain over the last month rather than the measure of current specific pain.

### *Cardiometabolic syndrome risk factors*

Every participant also underwent a blood screen and a brief physiological exam. Fasting blood was drawn and processed for analysis of serum levels of cholesterol (high density lipoprotein-HDL, low density lipoprotein-LDL, triglycerides, and total cholesterol) as well as fasting glucose. Obesity was quantified using a standard procedure to measure waist circumference.

Systolic and diastolic blood pressure (BP) were recorded in a seated position after 5 min of rest with the arm at rest at the level of the heart. A second measurement was obtained 5 min later and the average of two values was recorded and used as the dependent measure.

Specific criteria for each cardiometabolic risk factor was defined according to the National Cholesterol Education Program (NCEP; Cleeman, Grundy, Becker, & Clark, 2001): waist circumference  $> 102$  cm (male) or 88 cm (female); dyslipidemia (triglycerides  $\geq 150$  mg/dL); BP  $\geq 130/85$  mm HG or current treatment for hypertension; HDL  $< 40$  mg/dL (male) or 50 mg/dL (female); and fasting plasma glucose  $\geq 110$  mg/dL. The dependent variable was the total cardiometabolic risk factor score for each participant, ranging from 0 to 5, representing the total number of elevated/abnormal risk factors. We used the sum of the cardiometabolic risk factors because it represents a unified construct (metabolic syndrome) and has been used in previous

studies (e.g., Kaur et al., 2014). Further, the sum of the cardiometabolic risk factors showed a stronger average correlation with the sleep variables than any individual risk factor (see supplementary Table 1).

### *Nicotine use*

The Fagerstrom Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) is a questionnaire based on the Fagerstrom Tolerance Questionnaire and measures current and chronic cigarette use or use of smokeless tobacco products. The FTND is strongly associated with biochemical indices of cigarette use. Since nicotine dependence has been most consistently associated with disrupted sleep due to both the arousing effects of nicotine and early morning withdrawal, our variable of interest was nicotine dependence, indicated by a total score of 6 or higher (de Leon et al., 2003).

### Data Analysis

We first performed confirmatory factor analyses with R using a maximum likelihood estimator (lavaan R package; Rosseel, 2012), specifying a 3-factor model (Cole et al., 2006), two 2-factor models (Magee et al., 2008; Babson et al., 2012), and a 1-factor model. To compare the four models, we used multiple fit indices: chi-squared ( $X^2$ ), standardized root-mean-square residual (SRMR), root mean square error of approximation (RMSEA), comparative fit index (CFI), and Tucker-Lewis index (TLI). We considered a ratio of  $X^2$  to degrees of freedom  $\leq 3$ , SRMR and RMSEA values  $\leq .06$ , and CFI and TLI values  $> .95$  to be indicators of good model fit (Hu & Bentler, 1999; Schreiber, Nora, Stage, Barlow, & King, 2006).

We next correlated (using Spearman's rho) our measures of interest with PSQI global score and PSQI factors, using factor scores obtained using Thurstone's least squares regression approach (DiStefano, Zhu, & Mindrila, 2009). Normality tests on our clinical variables demonstrated that several of our variables had skewness values greater than positive .50 and also violated the Kolmogorov-Smirnov normality test, all  $p$ 's  $< .001$  (Evans & Olson, 2007). To correct for positive skewness in these variables that violated normality, we transformed them using the method that best corrected for skewness (log, square root, and reciprocal transformation). We used log transformation ( $\log(X+1)$ ) for LDH average, DASS Depression, and DASS Anxiety and square root transformation ( $\sqrt{x}$ ) for SF-MPQ, reducing positive skewness to acceptable values ( $< .50$  for all the variables; Bulmer, 2012). We ran regression models on our PSQI factors and PSQI global score using the transformed measures. Unless otherwise noted, in all regression analyses we entered the independent variables simultaneously. The one exception is that we ran hierarchical regressions when determining whether PTSD is associated with sleep after accounting for combat exposure.

## RESULTS

### Participant Characteristics

Characterization of the 283 participants is shown in Table 1. Although this study technically employed a convenience sample, similar to previous reports from our laboratory (Lippa et al., 2015), there were

TABLE 1  
Average Participant Characteristics

	Mean (SD)	Interpretation
Demographics		
Age	31.51 (8.16)	
Education	13.80 (1.89)	
WTAR (estimated IQ)	102.38 (11.56)	
Deployment Related Measure		
# of deployments	1.41 (.72)	
Cumulative deployment duration	14.20 (8.32)	
Combat exposure total score <sup>c</sup>	16.77 (12.14)	Moderate exposure
Clinical/Physical Measures		
PSQI global	9.34 (4.62)	Severe sleep problems
PTSD total score <sup>a</sup>	49.22 (29.15)	Moderate PTSD symptoms
Depression total score <sup>b</sup>	8.10 (9.31)	Normal range
Anxiety total score <sup>b</sup>	6.31 (7.42)	Normal range
Average pain in last month <sup>c</sup>	29.04 (25.03)	
Alcohol use: avg. # drinks <sup>d</sup>	5.74 (3.88)	
Total cigarette score <sup>f</sup>	2.93 (2.49)	
Total # military TBIs (mild TBIs)	.73 (1.42)	
Cardiometabolic syndrome sum	1.05 (1.07)	Meets around 1/5 components
Weight(lbs.)	194.06 (34.78)	
Body mass index	28.21 (4.80)	Slightly overweight
Waist circumference(cm) <sup>§</sup>	95.62 (12.03)	Normal range
Average systolic blood pressure <sup>§</sup>	116.39 (12.26)	Normal range
Average diastolic blood pressure <sup>§</sup>	75.66 (9.72)	Normal range
HDL cholesterol <sup>§</sup>	47.40 (12.49)	Normal range
Triglycerides <sup>§</sup>	142.48 (139.39)	Normal range
Glucose <sup>§</sup>	89.78 (31.40)	Normal range

<sup>§</sup>Included in the sum of cardiometabolic symptoms variable.

<sup>a</sup>CAPS current total score including sleep symptoms. <sup>b</sup>DASS total score. <sup>c</sup>SF-MPQ average pain in last month. <sup>d</sup>LDH. <sup>e</sup>DRRI. <sup>f</sup>FTND.

no significant differences in age, gender, or branch of service between our sample and the OEF/OIF/OND veterans utilizing VA Health Care (U.S. Department of Veterans Affairs, 2012).

At TRACTS baseline assessment, according to the CAPS, all participants had been exposed to a traumatic event that met Criterion A1. Using the DSM-IV standard scoring rule of 3 for the CAPS (Weathers et al., 1999), we found that 167 of the 283 participants had a PTSD diagnosis. To help guide the CAPS, participants also filled out a self-reported questionnaire about life experiences that could be potentially traumatic (TLEQ). Subjects reported an average of 17.77 of these potentially traumatic experiences ( $SD = 12.64$ ) across their lifetime. Two hundred forty-nine participants reported feeling *intense fear, helplessness, or horror* during at least one of these experiences (suggestive of a traumatic experience), and reported experiencing a psychological or emotional impact for an average of 3.92 ( $SD = 2.73$ ) of the 21 items on the TLEQ.

According to the BAT-L, 113 participants reported a mild TBI during deployment. Drinking behavior is reported in Table 1 and according to the Structured Clinical Interview for DSM-VI (SCID), 43 had a current diagnosis of substance use disorder, while 189 had a past diagnosis of

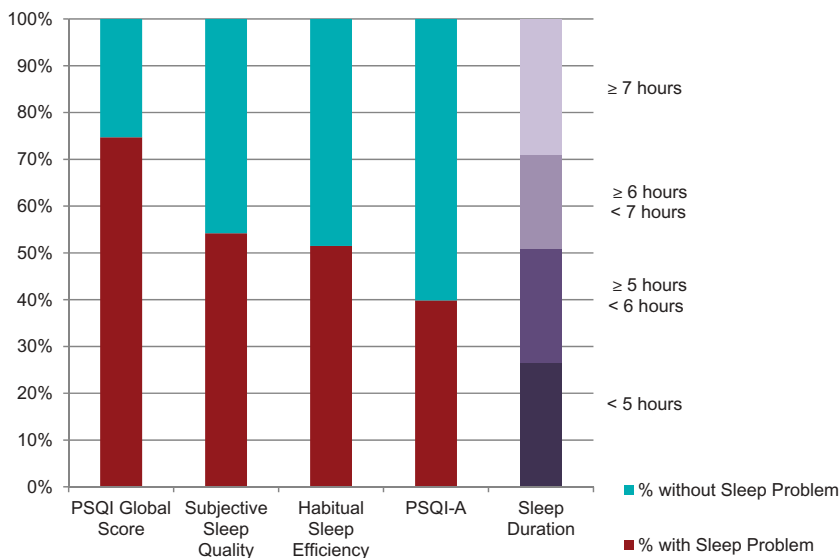
substance abuse. The most prevalent drug used was alcohol, with 20 reporting current dependence and 112 reporting past dependence. Other than alcohol, very few had issues with dependence on other drugs (opioid: Current = 2, Lifetime = 16; cannabis: Current = 4, Lifetime = 22; cocaine: Current = 1, Lifetime = 14; amphetamine: Current = 0, Lifetime = 2; polysubstance: Current = 0, Lifetime = 8; other: Current = 1, Lifetime = 2). Eighty-five participants had a history of nicotine use and participants reported an average total score of 2.93 on the Fagerstrom at the time of assessment. Of this group, only 16 participants endorsed nicotine dependence.

### Prevalence of Sleep Problems

We first sought to characterize the prevalence of self-reported sleep problems in the sample as measured by subcomponents of the PSQI and the PSQI-A (see Figure 1). In our sample, 75% of participants had general sleep difficulties according to the PSQI global score cutoff (Plumb, Peachey, & Zelman, 2014), and 54% reported poor sleep quality. Additionally, 51% of participants reported poor sleep efficiency, and 71% of the population slept for < 7 hr. Lastly, 40% of our sample had PTSD-related disruptive nocturnal behaviors according to the PSQI-A. These results suggest this population has significant sleep problems similar to other trauma-exposed samples (e.g., Gellis et al., 2010).

### Factor Analyses to Determine Components of Sleep Dysfunction

Our next goal was to determine which factor model best fit self-reported sleep in this population. To compare alternative structures of the PSQI components, we employed factor structures based



**FIGURE 1** Percentage of participants meeting various indices of sleep problems based on components of the PSQI and the PSQI-A. Criteria: PSQI Global Score > 5 (Buysse et al., 1989), Subjective Sleep Quality PSQI item #9 (Bad or Very bad; Buysse et al., 1989), sleep efficiency < 85% (Perlis et al., 2010), PSQI-A total score > 4 (Germain et al., 2005).

on the PSQI factor analyses conducted by Cole et al. (2006; 3-factor), Magee et al. (2008; 2-factor), and Babson et al. (2012; 2-factor), and also included a 1-factor model. For the 1-factor model, we loaded all seven components onto one latent variable. For 2 factors, we included two models: (a) for Magee et al.'s (2008) 2-factor model, we loaded sleep duration and sleep efficiency onto the first latent variable and loaded sleep disturbance, sleep quality, sleep latency, daytime dysfunction, and use of sleep medication onto the second latent variable; and (b) for Babson et al.'s (2012) 2-factor model, we loaded sleep duration and sleep efficiency onto the first latent variable and loaded sleep disturbance, sleep quality, sleep latency, and daytime dysfunction onto the second latent variable. For the 3-factor model, we loaded habitual sleep efficiency and sleep duration onto the first latent variable (Sleep Efficiency), loaded sleep quality, sleep latency, and sleep medications onto the second latent variable (Perceived Sleep Quality), and loaded sleep disturbance and daytime dysfunction onto the third latent variable (Daily Disturbances). Fit indices for 1-, 2-, and 3-factor models of the PSQI are shown in Table 2. The 3-factor model was the only one that demonstrated acceptable fit across all indices (5 out of 5), while the other models failed to surpass any of the goodness of fit thresholds. Thus, Cole et al.'s (2006) 3-factor solution of Sleep Efficiency, Perceived Sleep Quality, and Daily Disturbances fit the data the best. Figure 2 shows a path diagram of the 3-factor model with standardized parameter estimates between the factors and PSQI components.

### Associations Between Clinical Variables and Different Aspects of Sleep

Having established that the PSQI is best characterized as three separable factors, we next determined which clinical measures were independently associated with each factor as well as with the PSQI-A and PSQI global score. We first conducted zero order Spearman correlations between all the variables (see Table 3), ensuring that the predictor variables were not too highly correlated (all  $\rho$ 's < .69). For nicotine dependence, our only categorical variable included in this regression, unpaired *t*-tests showed that participants with nicotine dependence scored significantly higher on PSQI global score, all 3 PSQI factors, and PSQI-A (see supplementary Table 1b for statistics). We then conducted five separate regression models with these nine variables predicting PSQI global, PSQI-A, Sleep Efficiency (Factor 1), Perceived Sleep Quality (Factor 2), and Daily Disturbances (Factor 3; see Figure 3 and supplementary Table 2 for additional details

TABLE 2  
Fit Indexes for 1-, 2-, and 3-Factor Models of the PSQI

	<i>1-Factor</i>	<i>Magee et al. 2-Factor (2008)</i>	<i>Babson et al. 2-Factor (2012)</i>	<i>Cole et al. 3-Factor (2006)</i>
$X^2/df$	5.26	3.21	3.88	<b>1.96</b>
RMSEA	.13	.09	.10	<b>.06</b>
SRMR	.07	<b>.05</b>	<b>.05</b>	<b>.03</b>
CFI	.88	.94	.94	<b>.98</b>
TLI	.81	.90	.90	<b>.96</b>

Note.  $X^2/df$ , ratio of chi-squared to degrees of freedom; RMSEA, root mean square error of approximation; SRMR, standardized root-mean-square residual; CFI, comparative fit index; TLI, Tucker-Lewis Index. Bold indicates a fit index was met.



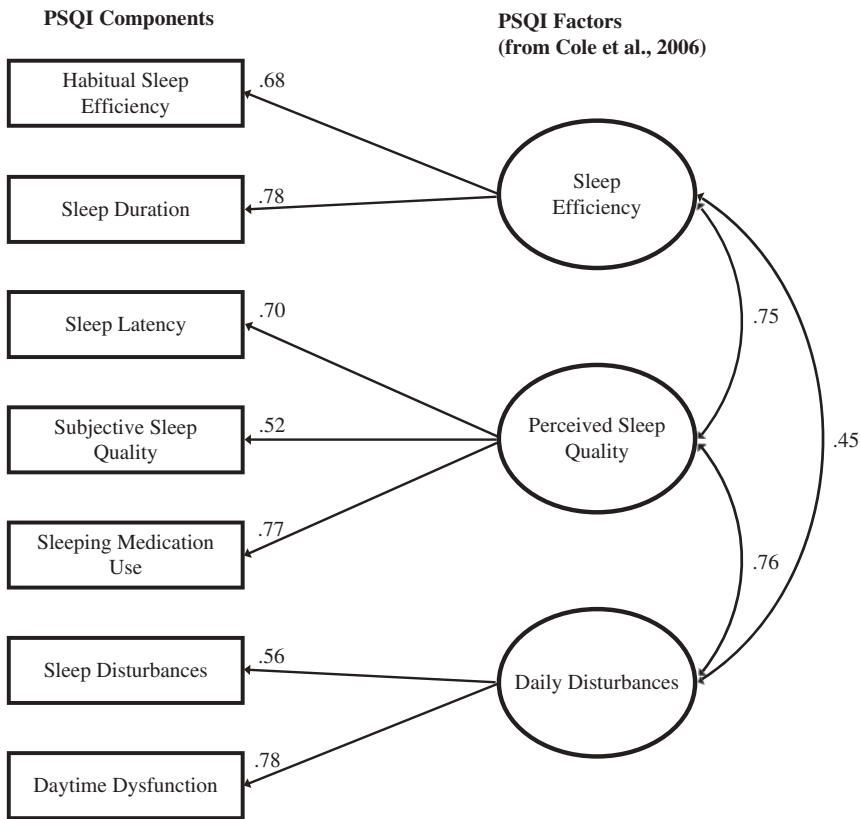


FIGURE 2 Pittsburgh Sleep Quality Index 3-factor model with standardized parameter estimates between the factor solution and the PSQI components.

about these models). We did not include gender because *t*-tests comparing sleep dysfunction in males and females did not show any significant differences in terms of the 3 factors, PSQI Global, or PSQI-A (all *p*'s > .11).

We first examined PSQI global, finding that PTSD symptoms (note that we did not include the sleep items), the sum of cardiometabolic risk factors, and anxiety symptoms significantly accounted for unique variance, explaining 48.6% of the variance in PSQI Global,  $R^2 = .51$ ,  $R^2_{\text{adj}} = .49$ ,  $F(9, 229) = 25.97$ ,  $p < .001$ . The results demonstrate that while PTSD symptomatology shows the strongest association with sleep dysfunction in trauma-exposed individuals, other psychological symptoms (anxiety) and physical symptoms (cardiometabolic symptoms) are also significantly associated with global sleep. This pattern of results between PTSD and PSQI global (as well as between PTSD and the PSQI factors below) was nearly identical when including the CAPS sleep symptoms (see supplementary Table 3).

When examining the Sleep Efficiency factor, we found that PTSD symptoms, anxiety symptoms, depressive symptoms, and cardiometabolic symptoms were all significantly associated with unique variance, accounting for 24.6% of the variance in Sleep Efficiency,  $R^2 = .27$ ,

TABLE 3  
Correlations Among PSQI Factors, Global Score, PSQI-A, and Clinical Variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12
1. Sleep Efficiency	—											
2. Perceived Sleep Quality	<b>.65**</b>	—										
3. Daily Disturbances	<b>.43**</b>	<b>.69**</b>	—									
4. PSQI: Global Score	<b>.81**</b>	<b>.92**</b>	<b>.76**</b>	—								
5. PSQI: Addendum	<b>.46**</b>	<b>.68**</b>	<b>.69**</b>	<b>.69**</b>	—							
6. PTSD <sup>a</sup>	<b>.43**</b>	<b>.59**</b>	<b>.66**</b>	<b>.61**</b>	<b>.66**</b>	—						
7. Anxiety <sup>b</sup>	<b>.40**</b>	<b>.54**</b>	<b>.57**</b>	<b>.56**</b>	<b>.66**</b>	<b>.67**</b>	—					
8. Depression <sup>b</sup>	<b>.21**</b>	<b>.45**</b>	<b>.66**</b>	<b>.47**</b>	<b>.54**</b>	<b>.62**</b>	<b>.60**</b>	—				
9. Pain <sup>c</sup>	<b>.23**</b>	<b>.37**</b>	<b>.49**</b>	<b>.39**</b>	<b>.48**</b>	<b>.41**</b>	<b>.42**</b>	<b>.39**</b>	—			
10. Cardiometabolic Symptoms	<b>.23**</b>	<b>.15*</b>	<b>.17**</b>	<b>.21**</b>	<b>.16**</b>	<b>.12*</b>	<b>.12*</b>	0.05	<b>.16*</b>	—		
11. Age	-.01	<b>-.13*</b>	.03	-.06	-.09	-.02	-.03	-.01	-.03	<b>.20**</b>	—	
12. Military TBI	<b>.20**</b>	<b>.28**</b>	<b>.28**</b>	<b>.28**</b>	<b>.31**</b>	<b>.34**</b>	<b>.32**</b>	<b>.25**</b>	<b>.23**</b>	.05	<b>-.05</b>	—
13. Alcohol Use <sup>d</sup>	<b>.24**</b>	<b>.21**</b>	<b>.16*</b>	<b>.26**</b>	<b>.24**</b>	<b>.21**</b>	<b>.27**</b>	<b>.17**</b>	.09	<b>.16**</b>	<b>-.21**</b>	<b>.14*</b>

N: 245–283.

<sup>a</sup>CAPS (current total excluding sleep symptoms). <sup>b</sup>DASS (total)(log). <sup>c</sup>SF-MPQ (Pain Average)(square root). <sup>d</sup>LDH (Daily Average)(log). \* $p < .05$ . \*\* $p < .01$ . Bold indicates significant correlation.

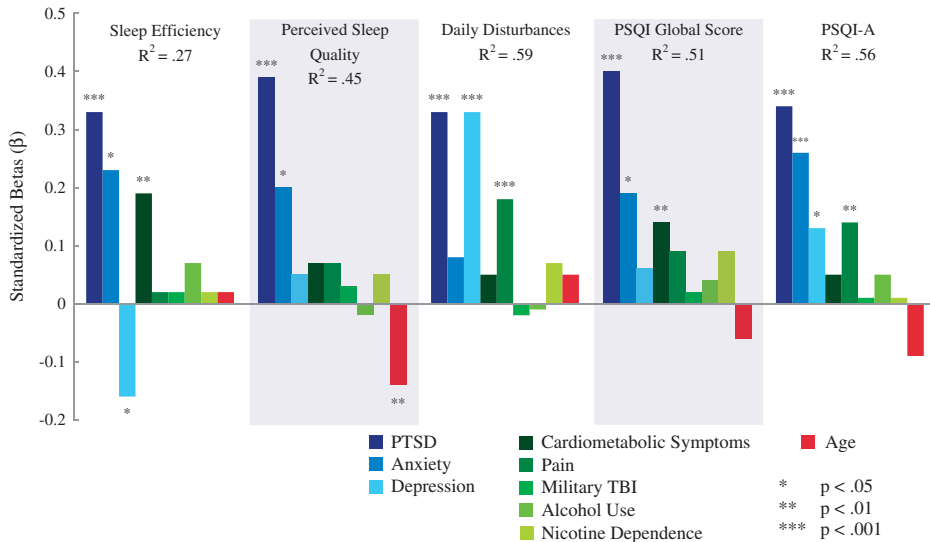


FIGURE 3 A graph of the standardized betas of our multiple regression models predicting sleep dysfunction (PSQI factors, PSQI global score, and PSQI-A) with independent variables entered simultaneously. Psychological variables are plotted in blue and physical variables are plotted in green.

$R^2_{\text{adj}} = .25$ ,  $F(9, 229) = 9.63$ ,  $p < .001$ . It is notable that though depression showed a positive zero-order correlation with Sleep Efficiency (see Table 3), in the regression model predicting Sleep Efficiency it showed a negative standardized beta (i.e., potential suppression effect).

In the model predicting the Perceived Sleep Quality factor, PTSD symptoms, anxiety symptoms, and age all significantly explained unique variance, accounting for 42.3% of the variance,  $R^2 = .45$ ,  $R^2_{\text{adj}} = .42$ ,  $F(9, 229) = 20.42$ ,  $p < .001$ . Interestingly, increasing age was associated with significantly better Perceived Sleep Quality.

For the Daily Disturbances factor, PTSD symptoms, depressive symptoms, and pain were all associated with unique variance, accounting for 57.4% of the variance in Daily Disturbances,  $R^2 = .59$ ,  $R^2_{\text{adj}} = .57$ ,  $F(9, 229) = 36.70$ ,  $p < .001$ . It is not surprising that pain was associated with unique variance in daily disturbances since “have pain” is an item on the sleep disturbances component.

We finally examined the PSQI-A, which contains sleep items specific to PTSD (nightmares, feeling nervous). PTSD symptoms, depression, anxiety, and pain were all significantly associated with unique variance in the PSQI-A, accounting for 54.3% of the variance,  $R^2 = .56$ ,  $R^2_{\text{adj}} = .54$ ,  $F(9, 230) = 32.60$ ,  $p < .001$ . Though the PSQI-A correlated highly with Perceived Sleep Quality, Daily Disturbances, and PSQI global score (all  $p$ 's  $> .68$ ), its regression pattern was distinct from these measures.

### Are the Results Similar When Using Diagnostic Variables From the SCID and CAPS?

We next sought to determine if this pattern of results held when we used dichotomous diagnostic measures of PTSD (CAPS current diagnosis), mood, anxiety, and substance abuse disorders (all from SCID–current diagnosis). The results were similar for PTSD and cardiometabolic

symptoms but less consistent for depression, anxiety, and pain (see supplementary Table 5). In particular, depression was no longer independently associated with unique variance in Sleep Efficiency and anxiety only showed a trend. For Perceived Sleep Quality, depression and pain explained unique variance whereas they did not in the model using continuous measures. For Daily Disturbances, nicotine dependence was associated with unique variance whereas it was not in the model using continuous measures. For PSQI Global, depression, pain, and nicotine dependence explained unique variance, whereas they failed to in the continuous model. In sum, these analyses replicated the continuous models for PTSD and cardiometabolic risk factors, though showed differences for depression, anxiety, pain, and nicotine dependence.

### Does Sleep in Those With PTSD Only Differ From Those With Both PTSD and Common Comorbidities?

We found that, compared to those with a PTSD diagnosis and no comorbid condition, participants with PTSD+mTBI (PTSD without mTBI,  $n = 80$ ; comorbid,  $n = 87$ ), PTSD+anxiety disorder diagnosis (PTSD without anxiety disorder diagnosis,  $n = 128$ ; comorbid,  $n = 39$ ), and PTSD+mood disorder diagnosis (PTSD without mood disorder diagnosis,  $n = 102$ ; comorbid,  $n = 65$ ) reported generally worse sleep across all sleep variables (see supplementary Table 4). However, after controlling for CAPS total score, these significant differences between PTSD only and comorbid conditions were all abolished (all  $p$ 's  $> .09$ ), suggesting that greater PTSD symptoms were driving these results.

### Does Combat Exposure Account for the Relationship Between PTSD and Sleep?

Since combat exposure has shown to be related to PTSD symptoms and sleep dysfunction (Insana, Hall, Buysse, & Germain, 2013), one possible explanation of the current PTSD/sleep association is that the degree of combat trauma exposure (as operationalized by the DRR1 combat experience score) explains the association between PTSD and sleep. To clarify if the relationship between PTSD and sleep can be accounted for by combat exposure, we ran hierarchical regressions predicting PSQI global, the three PSQI factors, and PSQI-A. In the hierarchical regression, we inputted combat exposure first and then inputted the rest of the variables in our original regressions (continuous measures of PTSD, anxiety, depression, pain, cardiometabolic risk factors, age, number of military TBIs, and categorical measure of nicotine dependence). We found that even after accounting for all variance in sleep explained by combat exposure, PTSD continued to be independently associated with all sleep measures, all  $p$ -values for change in  $R^2 < .001$  (see supplementary Table 6 and 6b).

### Does the Relationship Between PTSD and Sleep Mediate the Association Between PTSD and Cardiometabolic Syndrome?

Several studies have suggested that PTSD is associated with greater incidence of cardiometabolic syndrome and we also find a somewhat modest but significant association between PTSD symptoms and the sum of cardiometabolic risk factors in the current study ( $\rho = .13$ ,  $p < .05$ ). The strong associations between sleep/PTSD (e.g., Sleep Efficiency,  $\rho = .43$ ,  $p < .001$ ) as well as associations between sleep and cardiometabolic risk factors (e.g., Sleep Efficiency,  $\rho = .23$ ,  $p < .001$ ) leave open the possibility that the PTSD–cardiometabolic risk factor relationship may be mediated by sleep. To

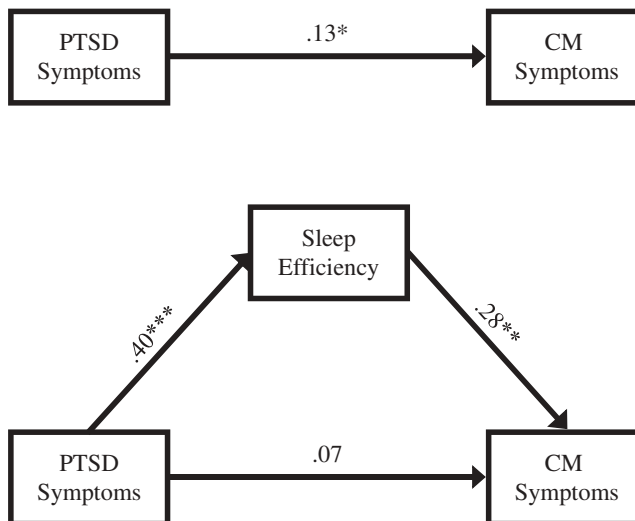


FIGURE 4 Standardized regression coefficients for the relationship between PTSD symptoms and cardiometabolic symptoms as mediated by Sleep Efficiency. \* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

test this, we performed mediation analyses using a bootstrap procedure (Hayes, 2013). As can be seen in the path diagram in Figure 4, we found that there was a significant indirect effect of PTSD symptomology on cardiometabolic symptoms through Sleep Efficiency,  $b = .0035$ , biased-corrected and accelerated confidence interval (BCa CI; Efron, 1987; [.0012, .0063]), representing a small effect,  $\kappa^2 = .0774$ , 95% BCa CI [.0250, .1371]. Similarly, there was a significant indirect effect of PTSD symptomology on cardiometabolic symptoms through PSQI global,  $b = .0053$ , BCa CI [.0012, .0097], representing a small effect,  $\kappa^2 = .1022$ , 95% BCa CI [.0247, .1786]. In contrast, there was no significant indirect effect of PSQI global,  $b = .0033$ , BCa CI [-.0186, .0265], or Sleep Efficiency,  $b = .0294$ , BCa CI [-.0231, .0989], on cardiometabolic symptoms through PTSD symptomology. Thus, our findings suggest that either poor global sleep or reduced sleep efficiency can significantly account for the relationship between PTSD symptoms and cardiometabolic syndrome.

## DISCUSSION

The goal of this study was to clarify the unique contribution of trauma sequelae to self-reported sleep dysfunction. Factor analyses of the PSQI confirmed three separable components of self-reported sleep: Sleep Efficiency, Perceived Sleep Quality, and Daily Disturbances. Multiple regressions demonstrated that PTSD symptoms were robustly associated with unique variance in the PSQI-A, PSQI global, as well as all three sleep factors, suggesting the PTSD symptoms are related to all aspects of self-reported sleep in trauma-exposed individuals. In addition to PTSD, anxiety independently explained variance in PSQI global, PSQI-A, as well as Sleep Efficiency and Perceived Sleep Quality, whereas depression showed less consistent results, only explaining independent variance in Daily Disturbances and PSQI-A. Besides symptoms of psychological

distress, physical symptoms were also associated with unique variance in self-reported sleep, with cardiometabolic symptoms explaining independent variance in PSQI global and Sleep Efficiency. Together, these results emphasize the importance of both psychological and physical–physiological symptoms toward understanding self-reported sleep dysfunction in those exposed to trauma.

The current PSQI results were best fit by a 3-factor model; neither the 2- nor the 1- factor solutions consistently exceeded goodness-of-fit indices. Interestingly, rather than finding a unique structure of the PSQI in trauma-exposed veterans, we replicated the 3-factor model of the PSQI found in aging populations (Cole et al., 2006), participants with chronic fatigue syndrome (Mariman et al., 2012), and women with PTSD (Casement et al., 2012). This suggests that the 3-factor model may represent a general structure of self-reported sleep across populations. We further demonstrated that a 3-factor approach provides additional information compared to only using the PSQI global score. For example, increasing age was independently related to improvements on the Perceived Sleep Quality factor but this was not true for the PSQI global score (and if anything this relationship went in the other direction for PSQI global). Furthermore, the 3-factor model demonstrated that the significant association of cardiometabolic symptoms with PSQI global was driven by the association of cardiometabolic symptoms with the Sleep Efficiency factor. These results further validate the 3-factor solution to the PSQI and suggest that dividing the PSQI into three factors provides a better characterization of sleep difficulties in trauma-exposed individuals than treating self-reported sleep as a unitary construct.

In addition to further validating a 3-factor model of the PSQI, the results demonstrate that of all the independent variables examined, PTSD symptoms by far explain the most independent variance in self-reported sleep, including the PSQI-A, PSQI global, as well as all three PSQI factors. The strong relationship between PTSD symptoms and sleep, independent of depression and anxiety, indicates some degree of specificity in the association between PTSD and sleep. We further found that this PTSD–sleep relationship was independent of DRR1 combat experience demonstrating that PTSD symptoms, even after controlling for degree of trauma exposure, are particularly associated with poor sleep. This robust self-reported sleep association with PTSD contrasts a previous study by Babson and colleagues showing that in those with a diagnosis of PTSD, greater PTSD symptoms are related to a reduction in the Perceived Sleep Quality PSQI factor only (Babson et al., 2012). The stronger PTSD–sleep associations in the current study could be due to the wider range of PTSD symptoms included in our trauma-exposed sample (i.e., the inclusion of those with and without a diagnosis of PTSD).

Besides PTSD, psychological symptoms of anxiety and depression were also significantly independently associated with self-reported sleep problems. Similar to previous studies (e.g., see Foa, Stein, & McFarlane, 2006), we found that anxiety and depression had a high degree of overlap with PTSD symptoms and one another (PTSD–Anxiety  $\rho = .67$ , PTSD–Depression  $\rho = .62$ , Anxiety–Depression  $\rho = .60$ ). Further, anxiety and depression were highly correlated with all sleep variables to a similar extent as PTSD, suggesting that PTSD, depression, and anxiety symptoms all measure aspects of a similar psychological distress construct related to poor sleep. Despite this interrelatedness, studies have also shown that depression and anxiety have unique aspects from PTSD that are associated with sleep issues in trauma-exposed populations (Babson & Feldner, 2010; Gellis et al., 2010). In line with this, we found that both depression and anxiety symptoms explained independent variance in sleep variables apart from PTSD symptoms and also that the diagnosis of mood and anxiety disorders are related to sleep independent of PTSD diagnosis (see supplementary materials). Depression's independent association with the Sleep Disturbances factor may exist because rumination at night has been linked to sleep disturbances (Thomsen,



Mehlsen, Christensen, & Zachariae, 2003), or because depression may further cause daytime fatigue (Carney, Moss, Lachowski, & Atwood, 2014). Additionally, anxiety's independent association with Sleep Efficiency and Perceived Sleep Quality could reflect that it covers additional physical symptoms independent from PTSD symptoms (e.g., trembling in the hands, difficulty breathing). These additional physical symptoms could be related to trauma-related disruption of the hypothalamic-pituitary-adrenal axis (Vgontzas et al., 2001). Further, worry is a part of anxiety, which has been shown to delay sleep onset (Gross & Borkovec, 1982) and could further reduce sleep efficiency. Together, the results suggest that the assessment of PTSD, depression, and anxiety can help to yield a thorough conceptualization of sleep difficulties in trauma-exposed individuals, which will likely enhance treatment.

In addition to symptoms of psychological distress, the current results suggest that symptoms of cardiometabolic syndrome are associated with sleep independent of PTSD and other relevant clinical measures. In particular, we found that the sum of cardiometabolic symptoms was independently associated with the global PSQI score and the Sleep Efficiency factor specifically, which includes sleep efficiency and duration. Though waist circumference was the cardiometabolic risk factor most highly associated with Sleep Efficiency, the overall sum of the risk factors performed better in the regressions and mediation analyses (see supplementary materials), suggesting that it may be more accurate to consider the constellation of cardiometabolic factors when examining associations with self-reported sleep. These findings are in line with studies examining middle-aged healthy adults that have found a strong relationship between diagnosis of cardiometabolic syndrome and both PSQI global (Jennings, Muldoon, Hall, Buysse, & Manuck, 2007) and self-reported sleep duration (Hall et al., 2008). The current study extends these findings to trauma-exposed individuals. In terms of the causal relationship, several recent studies suggest that reduced sleep duration leads to increased metabolic risk factors including obesity, type 2 diabetes, dyslipidaemia, and hypertension (for a review see Schmid et al., 2015), though metabolic risk factors may also lead to sleep apnea and poor sleep (Vgontzas et al., 2005). Further evidence that sleep can lead to increases in metabolic risk factors comes from studies finding that experimentally restricting sleep impairs glucose tolerance (Leproult & Van Cauter, 2010) and increases calorie consumption (Brondel, Romer, Nougues, Touyarou, & Davenne, 2010). The specificity of the metabolic risk factor–sleep duration–efficiency relationship is intriguing; additional studies using PSG could help understand whether specific components affected by sleep restriction (e.g., slow-wave sleep duration) are related to metabolic risk factors.

Particularly relevant to the current population, several studies have linked PTSD to metabolic syndrome (e.g., Heppner et al., 2009) and recent studies have suggested that stress-related changes in neuropeptide Y and glucocorticoid systems may be important physiological mechanisms underlying this relationship (Rasmusson, Schnurr, Zukowska, Scioli, & Forman, 2010). The current results also demonstrate a significant PTSD symptom–metabolic risk factor relationship, and additionally show that this relationship is significantly mediated by sleep (both for PSQI global and the Sleep Efficiency factor). This suggests that sleep may have an important role in the pathogenesis of metabolic syndrome in trauma-exposed individuals. Future longitudinal studies would be useful to understand the timing and interaction between sleep reduction and stress-related changes in neuropeptide Y and glucocorticoid systems in the manifestation of metabolic syndrome in trauma-exposed individuals.

The current findings have potential treatment implications. First, they demonstrate that PTSD symptoms are robustly related to all aspects of self-reported sleep in trauma-exposed individuals.

One possible implication of this is that addressing sleep dysfunction in those with greater PTSD symptoms may require a multipronged approach such as using differential behavioral sleep interventions to boost sleep efficiency (Espie et al., 2014), treat nightmares (Krakow et al., 2001), and ameliorate other symptoms of insomnia (Margolies, Rybarczyk, Vrana, Leszczyszyn, & Lynch, 2013). Additionally, the results suggest that in trauma-exposed individuals with particularly poor sleep efficiency or duration and cardiometabolic risk factors, sleep treatments that promote greater sleep duration and efficiency (e.g., Espie et al., 2014) could possibly improve cardiometabolic outcomes. Conversely, decreasing one's cardiometabolic risk factors may provide a method to improve aspects of sleep efficiency and/or duration.

Although the results of the current study have several strengths, they also have limitations. First, we included only combat-exposed, non-treatment-seeking, mostly male OEF/OIF/OND veterans, many of whom had mTBIs, and these veterans may not be representative of all trauma-exposed individuals or even treatment-seeking veterans. Replicating these results in treatment-seeking veterans, a trauma-exposed civilian population, as well as in a greater sample of females, would test the generality of the current findings. Relatedly, we did not include a control group not exposed to trauma, so we do not know whether the pattern of results is specific to trauma exposure or would show a similar pattern in any population with sleep difficulties. Another limitation is the lack of objective sleep measures, which could have provided insight on how trauma sequelae influence physiological aspects of sleep (e.g., REM or deep sleep). Moreover, there may have been reporting issues with alcohol and nicotine use, such as underreporting due to social desirability bias. Also, because we did not have a specific measure of sleep apnea and because many people do not know they have sleep apnea, we likely included many participants (based on national estimates ~15%) with sleep apnea. Inclusion of these individuals likely worsened overall sleep (particularly the sleep disturbances factor) and likely added unexplained variance to the models. Further, we did not collect information about concurrent medication intake and mental health treatment, which could have explained additional variance. A final important limitation is the cross-sectional nature of the current study and our focus on the independence rather than interdependence of various trauma sequelae relating to sleep. Notably, nearly all the variables we examined in this study were highly correlated and causality between any two of these variables (e.g., PTSD symptoms and PSQI global) is at a minimum bidirectional or significantly more complex. We chose to focus on predicting sleep in our regression models but it would have been just as valid to reverse the direction of the regressions and predict symptoms of PTSD or depression. Longitudinal studies that experimentally manipulate sleep, clinical symptoms, or physical-physiological measures would be useful to better understand the highly complex causal relationships between these trauma sequelae.

The current study examined associations between clinical variables and self-reported sleep in trauma-exposed individuals and found that PTSD, anxiety, depression, and cardiometabolic symptoms all had unique associations with different aspects of sleep. In terms of symptoms of psychological distress, these findings demonstrate that not only is PTSD symptomology very strongly associated with all aspects of sleep dysfunction, but that depression and anxiety also explain unique variance in aspects of sleep beyond PTSD. Additionally, the results show that cardiometabolic symptoms are uniquely associated with sleep, especially sleep efficiency and duration. These findings help lay the groundwork for further investigations of the mechanisms of sleep dysfunction in trauma-exposed individuals and may help in the development of more effective, individualized treatments.

## ACKNOWLEDGMENTS

This research was supported by the Translational Research Center for TBI and Stress Disorders (TRACTS), a VA Rehabilitation Research and Development Traumatic Brain Injury Center of Excellence (B9254-C); by a Veterans Affairs Career Development Award II given to JD; and a VA Clinical Science R&D Career Development Award to ME (1IK2CX000706-01A2).

## SUPPLEMENTAL MATERIAL

Supplemental data for this article can be accessed on the [publisher's website](#).

## REFERENCES

- Alvaro, P. K., Roberts, R. M., & Harris, J. K. (2013). A systematic review assessing bidirectionality between sleep disturbances, anxiety, and depression. *Sleep, 36*(7), 1059–1068. doi:10.5665/sleep.2810
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- Antony, M. M., Bieling, P. J., Cox, B. J., Enns, M. W., & Swinson, R. P. (1998). Psychometric properties of the 42-item and 21-item versions of the Depression Anxiety Stress Scales in clinical groups and a community sample. *Psychological Assessment, 10*(2), 176.
- Babson, K. A., Blonigen, D. M., Boden, M. T., Drescher, K. D., & Bonn-Miller, M. O. (2012). Sleep quality among U.S. military veterans with PTSD: A factor analysis and structural model of symptoms. *Journal of Traumatic Stress, 25*(6), 665–674. doi:10.1002/jts.21757
- Babson, K. A., & Feldner, M. T. (2010). Temporal relations between sleep problems and both traumatic event exposure and PTSD: A critical review of the empirical literature. *Journal of Anxiety Disorders, 24*(1), 1–15. doi:S0887-6185(09)00158-3[pii]10.1016/j.janxdis.2009.08.002
- Bennett, L. S., Barbour, C., Langford, B., Stradling, J. R., & Davies, R. J. (1999). Health status in obstructive sleep apnea: Relationship with sleep fragmentation and daytime sleepiness, and effects of continuous positive airway pressure treatment. *American Journal of Respiratory and Critical Care Medicine, 159*(6), 1884–1890. doi:10.1164/ajrccm.159.6.9808107
- Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Gusman, F. D., Charney, D. S., & Keane, T. M. (1995). The development of a clinician administered PTSD scale. *Journal of Traumatic Stress, 8*(1), 75–90.
- Blanchard, E. B., Hickling, E. J., Taylor, A. E., Forneris, C. A., Loos, W., & Jaccard, J. (1995). Effects of varying scoring rules of the Clinician-Administered PTSD Scale (CAPS) for the diagnosis of post-traumatic stress disorder in motor vehicle accident victims. *Behaviour, Research, and Therapy, 33*(4), 471–475. doi:0005-7967(94)00064-Q [pii]
- Breslau, N., Roth, T., Burduvali, E., Kapke, A., Schultz, L., & Roehrs, T. (2004). Sleep in lifetime posttraumatic stress disorder: A community-based polysomnographic study. *Archives of General Psychiatry, 61*(5), 508–516. doi:10.1001/archpsyc.61.5.50861/5/508 [pii]
- Brondel, L., Romer, M. A., Nougues, P. M., Touyarou, P., & Davenne, D. (2010). Acute partial sleep deprivation increases food intake in healthy men. *American Journal of Clinical Nutrition, 91*(6), 1550–1559. doi:ajcn.2009.28523 [pii]10.3945/ajcn.2009.28523
- Bryan, C. J. (2013). Repetitive traumatic brain injury (or concussion) increases severity of sleep disturbance among deployed military personnel. *Sleep, 36*(6), 941.
- Bulmer, M. G. (2012). *Principles of statistics*. North Chelmsford, MA: Courier Corporation.
- Buysse, D. J., Reynolds, C. F., III, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*(2), 193–213.
- Carney, C. E., Moss, T. G., Lachowski, A. M., & Atwood, M. E. (2014). Understanding mental and physical fatigue complaints in those with depression and insomnia. *Behavioral Sleep Medicine, 12*(4), 272–289. doi:10.1080/15402002.2013.801345

- Carpenter, J. S., & Andrykowski, M. A. (1998). Psychometric evaluation of the Pittsburgh Sleep Quality Index. *Journal of Psychosomatic Research, 45*(1), 5–13.
- Casement, M. D., Harrington, K. M., Miller, M. W., & Resick, P. A. (2012). Associations between Pittsburgh Sleep Quality Index factors and health outcomes in women with posttraumatic stress disorder. *Sleep Medicine, 13*(6), 752–758. doi:S1389-9457(12)00147-5[pii]10.1016/j.sleep.2012.02.014
- Castro, J. P., El-Atat, F. A., McFarlane, S. I., Aneja, A., & Sowers, J. R. (2003). Cardiometabolic syndrome: Pathophysiology and treatment. *Current Hypertension Reports, 5*(5), 393–401.
- Cleeman, J., Grundy, S., Becker, D., & Clark, L. (2001). Expert panel on detection, evaluation and treatment of high blood cholesterol in adults. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP III). *Journal of the American Medical Association, 285*(19), 2486–2497.
- Cole, J. C., Motivala, S. J., Buysse, D. J., Oxman, M. N., Levin, M. J., & Irwin, M. R. (2006). Validation of a 3-factor scoring model for the Pittsburgh Sleep Quality Index in older adults. *Sleep, 29*(1), 112–116.
- Davis, J. L. (2009). *Treating post-trauma nightmares: A cognitive behavioral approach*. New York, NY: Springer.
- Dedert, E. A., Calhoun, P. S., Watkins, L. L., Sherwood, A., & Beckham, J. C. (2010). Posttraumatic stress disorder, cardiovascular, and metabolic disease: A review of the evidence. *Annals of Behavioral Medicine, 39*(1), 61–78. doi:10.1007/s12160-010-9165-9
- de Leon, J., Diaz, F. J., Becoña, E., Gurpegui, M., Jurado, D., & Gonzalez-Pinto, A. (2003). Exploring brief measures of nicotine dependence for epidemiological surveys. *Addictive Behaviors, 28*(8), 1481–1486.
- DiStefano, C., Zhu, M., & Mindrila, D. (2009). Understanding and using factor scores: Considerations for the applied researcher. *Practical Assessment, Research & Evaluation, 14*(20), 1–11.
- Durmer, J. S., & Dinges, D. F. (2005). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology, 25*(1), 117–129. doi:10.1055/s-2005-867080
- Efron, B. (1987). Better bootstrap confidence intervals. *Journal of the American Statistical Association, 82*(397), 171–185.
- Espie, C. A., Kyle, S. D., Miller, C. B., Ong, J., Hames, P., & Fleming, L. (2014). Attribution, cognition and psychopathology in persistent insomnia disorder: Outcome and mediation analysis from a randomized placebo-controlled trial of online cognitive behavioural therapy. *Sleep Medicine, 15*(8), 913–917. doi: S1389-9457(14) 00093-8[pii]10.1016/j.sleep.2014.03.001
- Evans, J. R., & Olson, D. L. (2007). *Statistics, data analysis, and decision modeling*. Upper Saddle River, NJ: Pearson/Prentice Hall.
- Foa, E. B., Stein, D. J., & McFarlane, A. C. (2006). Symptomatology and psychopathology of mental health problems after disaster. *Journal of Clinical Psychiatry, 67*(Suppl 2), 15–25.
- Fortier, C. B., Amick, M. M., Grande, L., McGlynn, S., Kenna, A., Morra, L., ...McGlinchey, R. E. (2014). The Boston Assessment of Traumatic Brain Injury-Lifetime (BAT-L) Semistructured Interview: Evidence of research utility and validity. *Journal of Head Trauma Rehabilitation, 29*(1), 89–98.
- Gellis, L. A., Gehrman, P. R., Mavandadi, S., & Oslin, D. W. (2010). Predictors of sleep disturbances in Operation Iraqi Freedom/Operation Enduring Freedom veterans reporting a trauma. *Military Medicine, 175*(8), 567–573.
- Germain, A. (2013). Sleep disturbances as the hallmark of PTSD: Where are we now? *American Journal of Psychiatry, 170*(4), 372–382.
- Germain, A., Hall, M., Krakow, B., Katherine Shear, M., & Buysse, D. J. (2005). A brief sleep scale for posttraumatic stress disorder: Pittsburgh Sleep Quality Index Addendum for PTSD. *Journal of Anxiety Disorders, 19*(2), 233–244.
- Germain, A., Richardson, R., Moul, D. E., Mammen, O., Haas, G., Forman, S. D., ...Nofzinger, E.A. (2012). Placebo-controlled comparison of prazosin and cognitive-behavioral treatments for sleep disturbances in US military veterans. *Journal of Psychosomatic Research, 72*(2), 89–96. doi:S0022-3999(11)00283-2[pii]10.1016/j.jpsychores.2011.11.010
- Goldstein, G., van Kammen, W., Shelly, C., Miller, D. J., & van Kammen, D. P. (1987). Survivors of imprisonment in the Pacific theater during World War II. *American Journal of Psychiatry, 144*(9), 1210–1213.
- Green, P. (2004). *Green's Medical Symptom Validity Test*. Edmonton, Canada: Green's Publishing.
- Gross, R. T., & Borkovec, T. (1982). Effects of a cognitive intrusion manipulation on the sleep-onset latency of good sleepers. *Behavior Therapy, 13*(1), 112–116.
- Hall, M. H., Muldoon, M. F., Jennings, J. R., Buysse, D. J., Flory, J. D., & Manuck, S. B. (2008). Self-reported sleep duration is associated with the metabolic syndrome in midlife adults. *Sleep, 31*(5), 635–643.
- Harvey, A. G., & Bryant, R. A. (1998). The relationship between acute stress disorder and posttraumatic stress disorder: A prospective evaluation of motor vehicle accident survivors. *Journal of Consulting and Clinical Psychology, 66*(3), 507–512.

- Harvey, A. G., Jones, C., & Schmidt, D. A. (2003). Sleep and posttraumatic stress disorder: A review. *Clinical Psychology Review, 23*(3), 377–407. doi:S0272735803000321[pii]
- Hawker, G. A., Mian, S., Kendzerska, T., & French, M. (2011). Measures of adult pain: Visual Analog Scale for Pain (VAS Pain), Numeric Rating Scale for Pain (NRS Pain), McGill Pain Questionnaire (MPQ), Short-Form McGill Pain Questionnaire (SF-MPQ), Chronic Pain Grade Scale (CPGS), Short Form-36 Bodily Pain Scale (SF-36 BPS), and Measure of Intermittent and Constant Osteoarthritis Pain (ICOAP). *Arthritis Care & Research, 63*(S11), S24–0-S252.
- Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. New York, NY: Guilford Press.
- Heatherton, T. F., Kozlowski, L. T., Frecker, R. C., & Fagerstrom, K. O. (1991). The Fagerstrom Test for Nicotine Dependence: A revision of the Fagerstrom Tolerance Questionnaire. *British Journal of Addiction, 86*(9), 1119–1127.
- Hefez, A., Metz, L., & Lavie, P. (1987). Long-term effects of extreme situational stress on sleep and dreaming. *American Journal of Psychiatry, 144*(3), 344–347.
- Heppner, P. S., Crawford, E. F., Haji, U. A., Afari, N., Hauger, R. L., Dashevsky, B. A., ... Baker, D. G. (2009). The association of posttraumatic stress disorder and metabolic syndrome: A study of increased health risk in veterans. *BMC Medicine, 7*, 1. doi:1741-7015-7-1 [pii]10.1186/1741-7015-7-1
- Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal, 6*(1), 1–55.
- Insana, S. P., Hall, M., Buysse, D. J., & Germain, A. (2013). Validation of the Pittsburgh Sleep Quality Index Addendum for posttraumatic stress disorder (PSQI-A) in U.S. male military veterans. *Journal of Traumatic Stress, 26*(2), 192–200. doi:10.1002/jts.21793
- Jaehne, A., Loessel, B., Bárkai, Z., Riemann, D., & Hornyak, M. (2009). Effects of nicotine on sleep during consumption, withdrawal and replacement therapy. *Sleep Medicine Reviews, 13*(5), 363–377.
- Jennings, J. R., Muldoon, M. F., Hall, M., Buysse, D. J., & Manuck, S. B. (2007). Self-reported sleep quality is associated with the metabolic syndrome. *Sleep, 30*(2), 219–223.
- Kaur, S. S., Gonzales, M. M., Eagan, D. E., Goudarzi, K., Tanaka, H., & Haley, A. P. (2014). Inflammation as a mediator of the relationship between cortical thickness and metabolic syndrome. *Brain Imaging and Behavior, 1*–7.
- Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry, 52*(12), 1048–1060.
- Kilpatrick, D. G., Resnick, H. S., Milanak, M. E., Miller, M. W., Keyes, K. M., & Friedman, M. J. (2013). National estimates of exposure to traumatic events and PTSD prevalence using DSM-IV and DSM-5 criteria. *Journal of Traumatic Stress, 26*(5), 537–547. doi:10.1002/jts.21848
- King, L. A., King, D. W., Vogt, D. S., Knight, J., & Samper, R. E. (2006). Deployment Risk and Resilience Inventory: A collection of measures for studying deployment-related experiences of military personnel and veterans. *Military Psychology, 18*(2), 89–120.
- Klein, E., Caspi, Y., & Gil, S. (2003). The relation between memory of the traumatic event and PTSD: Evidence from studies of traumatic brain injury. *Canadian Journal of Psychiatry, 48*(1), 28–33.
- Kobayashi, I., Boarts, J. M., & Delahanty, D. L. (2007). Polysomnographically measured sleep abnormalities in PTSD: A meta-analytic review. *Psychophysiology, 44*(4), 660–669. doi:PSYP537[pii]10.1111/j.1469-8986.2007.537.x
- Koh, H. W., Lim, R. B., Chia, K. S., & Lim, W. Y. (2015). The Pittsburgh Sleep Quality Index in a multi-ethnic Asian population contains a three-factor structure. *Sleep and Breathing, 19*(1), 113–119. doi:10.1007/s11325-015-1130-1
- Krakov, B., Artar, A., Warner, T. D., Melendrez, D., Johnston, L., Hollifield, M., ...Koss, M. (2000). Sleep disorder, depression, and suicidality in female sexual assault survivors. *Crisis, 21* (4),163–170.
- Krakov, B., Hollifield, M., Johnston, L., Koss, M., Schrader, R., Warner, T. D., & Cheng, D. (2001). Imagery rehearsal therapy for chronic nightmares in sexual assault survivors with posttraumatic stress disorder: A randomized controlled trial. *Journal of the American Medical Association, 286*(5), 537–545. doi:joc10245[pii]
- Krakov, B., Hollifield, M., Schrader, R., Koss, M., Tandberg, D., Lauriello, J., ...Kellner, R. (2000). A controlled study of imagery rehearsal for chronic nightmares in sexual assault survivors with PTSD: A preliminary report. *Journal of Traumatic Stress, 13*(4), 589–609. doi: 10.1023/A:1007854015481
- Krakov, B., Melendrez, D., Johnston, L., Warner, T. D., Clark, J. O., Pacheco, M., ...Schrader, R. (2002). Sleep-disordered breathing, psychiatric distress, and quality of life impairment in sexual assault survivors. *Journal of Nervous and Mental Disease, 190*(7), 442–452. doi:10.1097/01.NMD.0000022444.23912.D2
- Krakov, B., Schrader, R., Tandberg, D., Hollifield, M., Koss, M. P., Yau, C. L., ...Cheng, D. T. (2002). Nightmare frequency in sexual assault survivors with PTSD. *Journal of Anxiety Disorders, 16*(2), 175–190.



- Kubany, E. S., Leisen, M. B., Kaplan, A. S., Watson, S. B., Haynes, S. N., Owens, J. A., & Burns, K. (2000). Development and preliminary validation of a brief broad-spectrum measure of trauma exposure: The Traumatic Life Events Questionnaire. *Psychological Assessment, 12*(2), 210–224.
- Lamarche, L. J., & De Koninck, J. (2007). Sleep disturbance in adults with posttraumatic stress disorder: A review. *The Journal of Clinical Psychiatry, 68*(8), 1257–1270.
- Lavie, P. (2001). Sleep disturbances in the wake of traumatic events. *New England Journal of Medicine, 345*(25), 1825–1832. doi:10.1056/NEJMra012893345/25/1825[pii]
- Leproult, R., & Van Cauter, E. (2010). Role of sleep and sleep loss in hormonal release and metabolism. *Endocrine Development, 17*, 11–21. doi:000262524[pii]10.1159/000262524
- Lippa, S. M., Fonda, J. R., Fortier, C. B., Amick, M. A., Kenna, A., Milberg, W. P., & McGlinchey, R. E. (2015). Deployment-related psychiatric and behavioral conditions and their association with functional disability in OEF/OIF/OND veterans. *Journal of Traumatic Stress, 28*(1), 25–33.
- Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour, Research, and Therapy, 33*(3), 335–343. doi:0005-7967(94)00075-U[pii]
- Löwe, B., Kroenke, K., Spitzer, R. L., Williams, J. B., Mussell, M., Rose, M., ...Spitzer, C. (2011). Trauma exposure and posttraumatic stress disorder in primary care patients: Cross-sectional criterion standard study. *The Journal of Clinical Psychiatry, 72*(3), 304–312. doi:10.4088/JCP.09m05290blu
- Magee, C. A., Caputi, P., Iverson, D. C., & Huang, X. F. (2008). An investigation of the dimensionality of the Pittsburgh Sleep Quality Index in Australian adults. *Sleep and Biological Rhythms, 6*(4), 222–227.
- Maher, M. J., Rego, S. A., & Asnis, G. M. (2006). Sleep disturbances in patients with post-traumatic stress disorder: Epidemiology, impact and approaches to management. *CNS Drugs, 20*(7), 567–590. doi:2073[pii]
- Margolies, S. O., Rybarczyk, B., Vrana, S. R., Leszczyszyn, D. J., & Lynch, J. (2013). Efficacy of a cognitive-behavioral treatment for insomnia and nightmares in Afghanistan and Iraq veterans with PTSD. *Journal of Clinical Psychology, 69*(10), 1026–1042.
- Mariman, A., Vogelaers, D., Hanouille, I., Delesie, L., Tobback, E., & Pevernagie, D. (2012). Validation of the three-factor model of the PSQI in a large sample of chronic fatigue syndrome (CFS) patients. *Journal of Psychosomatic Research, 72*(2), 111–113. doi:S0022-3999(11)00277-7[pii]10.1016/j.jpsychores.2011.11.004
- Melzack, R. (1987). The short-form McGill pain questionnaire. *Pain, 30*(2), 191–197.
- Orff, H. J., Ayalon, L., & Drummond, S. P. (2009). Traumatic brain injury and sleep disturbance: A review of current research. *Journal of Head Trauma Rehabilitation, 24*(3), 155–165. doi:10.1097/HTR.0b013e3181a0b28100001199-200905000-00002 [pii]
- Perkonig, A., Kessler, R. C., Storz, S., & Wittchen, H. U. (2000). Traumatic events and post-traumatic stress disorder in the community: Prevalence, risk factors and comorbidity. *Acta Psychiatrica Scandinavica, 101*(1), 46–59.
- Perlis, M. L., Swinkels, C. M., Gehrman, P. R., Pigeon, W. R., Matteson-Rusby, S. E., & Jungquist, C. R. (2010). The incidence and temporal patterning of insomnia: A pilot study. *Journal of Sleep Research, 19*(1 Pt 1), 31–35. doi: JSR768 [pii]10.1111/j.1365-2869.2009.00768.x
- Plumb, T. R., Peachey, J. T., & Zelman, D. C. (2014). Sleep disturbance is common among servicemembers and veterans of Operations Enduring Freedom and Iraqi Freedom. *Psychological Services, 11*(2), 209–219. doi: 2013-41142-001 [pii]10.1037/a0034958
- Rasmusson, A. M., Schnurr, P. P., Zukowska, Z., Scioli, E., & Forman, D. E. (2010). Adaptation to extreme stress: Post-traumatic stress disorder, neuropeptide Y and metabolic syndrome. *Experimental Biology and Medicine (Maywood), 235*(10), 1150–1162. doi: 235/10/1150[pii]10.1258/ebm.2010.009334
- Redline, S., & Strohl, K. P. (1998). Recognition and consequences of obstructive sleep apnea hypopnea syndrome. *Clinics in Chest Medicine, 19*(1), 1–19.
- Rosen, J., Reynolds, C. F., III, Yeager, A. L., Houck, P. R., & Hurwitz, L. F. (1991). Sleep disturbances in survivors of the Nazi Holocaust. *American Journal of Psychiatry, 148*(1), 62–66.
- Ross, R. J., Ball, W. A., Sullivan, K. A., & Caroff, S. N. (1989). Sleep disturbance as the hallmark of posttraumatic stress disorder. *American Journal of Psychiatry, 146*(6), 697–707.
- Rosseel, Y. (2012). lavaan: An R package for structural equation modeling. *Journal of Statistical Software, 48*(2), 1–36.
- Schmid, S. M., Hallschmid, M., & Schultes, B. (2015). The metabolic burden of sleep loss. *The Lancet Diabetes & Endocrinology, 3*(1), 52–62. doi:S2213-8587(14)70012-9[pii]10.1016/S2213-8587(14)70012-9
- Schreiber, J. B., Nora, A., Stage, F. K., Barlow, E. A., & King, J. (2006). Reporting structural equation modeling and confirmatory factor analysis results: A review. *Journal of Educational Research, 99*(6), 323–338.



- Sheikh, J. I., Woodward, S. H., & Leskin, G. A. (2003). Sleep in post-traumatic stress disorder and panic: Convergence and divergence. *Depression and Anxiety, 18*(4), 187–197.
- Sivertsen, B., Salo, P., Mykletun, A., Hysing, M., Pallesen, S., Krokstad, S., ...Øverland, S. (2012). The bidirectional association between depression and insomnia: The HUNT study. *Psychosomatic Medicine, 74*(7), 758–765. doi: PSY.0b013e3182648619 [pii]10.1097/PSY.0b013e3182648619
- Skinner, H. A., & Sheu, W. J. (1982). Reliability of alcohol use indices. The Lifetime Drinking History and the MAST. *Journal of Studies on Alcohol, 43*(11), 1157–1170.
- Stewart, S. H. (1996). Alcohol abuse in individuals exposed to trauma: A critical review. *Psychological Bulletin, 120*(1), 83–112.
- Stewart, S. H., Pihl, R. O., Conrod, P. J., & Dongier, M. (1998). Functional associations among trauma, PTSD, and substance-related disorders. *Addictive Behaviors, 23*(6), 797–812.
- Swanson, L. M., Arnedt, J. T., Rosekind, M. R., Belenky, G., Balkin, T. J., & Drake, C. (2011). Sleep disorders and work performance: Findings from the 2008 National Sleep Foundation Sleep in America poll. *Journal of Sleep Research, 20*(3), 487–494. doi:10.1111/j.1365-2869.2010.00890.x
- Talbot, L. S., Maguen, S., Metzler, T. J., Schmitz, M., McCaslin, S. E., Richards, A., Neylan, T. C. (2014). Cognitive behavioral therapy for insomnia in posttraumatic stress disorder: A randomized controlled trial. *Sleep, 37*(2), 327–341.
- Talbot, L. S., Rao, M. N., Cohen, B. E., Richards, A., Inslicht, S. S., O'Donovan, A., ... Neylan, T. C. (2015). Metabolic risk factors and posttraumatic stress disorder: The role of sleep in young, healthy adults. *Psychosomatic Medicine, 77*(4), 383–391.
- Thomsen, D. K., Mehlsen, M. Y., Christensen, S., & Zachariae, R. (2003). Rumination: Relationship with negative mood and sleep quality. *Personality and Individual Differences, 34*(7), 1293–1301.
- U.S. Department of Veterans Affairs. (2012). Analysis of VA health care utilization among Operation Enduring Freedom, Operation Iraqi Freedom, and Operation New Dawn veterans, from 1st qtr FY 2002 through 1st qtr FY 2012. Washington, DC: Author.
- Van der Kolk, B., Hartmann, E., Burr, A., & Blitz, R. (1980). A survey of nightmare frequencies in a veterans outpatient clinic. *Sleep Research, 9*, 229.
- Vgontzas, A. N., Bixler, E. O., & Chrousos, G. P. (2005). Sleep apnea is a manifestation of the metabolic syndrome. *Sleep Medicine Reviews, 9*(3), 211–224. doi:S1087-0792(05)00006-7[pii]10.1016/j.smrv.2005.01.006
- Vgontzas, A. N., Bixler, E. O., Lin, H. M., Prolo, P., Mastorakos, G., Vela-Bueno, A. . (2001). Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: clinical implications. *Journal of Clinical Endocrinology & Metabolism, 86*(8), 3787–3794. doi:10.1210/jcem.86.8.7778
- Vogt, D. S., Proctor, S. P., King, D. W., King, L. A., & Vasterling, J. J. (2008). Validation of scales from the Deployment Risk and Resilience Inventory in a sample of Operation Iraqi Freedom veterans. *Assessment, 15*(4), 391–403. doi:1073191108316030 [pii]10.1177/1073191108316030
- Weathers, F. W., Keane, T. M., & Davidson, J. R. (2001). Clinician Administered PTSD Scale: A review of the first ten years of research. *Depression and Anxiety, 13*(3), 132–156.
- Weathers, F. W., Ruscio, A. M., & Keane, T. M. (1999). Psychometric properties of nine scoring rules for the Clinician-Administered Posttraumatic Stress Disorder Scale. *Psychological Assessment, 11*(2), 124.
- Wood, J. M., Bootzin, R. R., Rosenhan, D., Nolen-Hoeksema, S., & Jourden, F. (1992). Effects of the 1989 San Francisco earthquake on frequency and content of nightmares. *Journal of Abnormal Psychology, 101*(2), 219–224.
- Zayfert, C., & DeViva, J. C. (2004). Residual insomnia following cognitive behavioral therapy for PTSD. *Journal of Traumatic Stress, 17*(1), 69–73. doi:10.1023/B:JOTS.0000014679.31799.e7