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ARTICLE

Traumatic Brain Injury and Incidence Risk of Sleep Disorders in Nearly 200,000 US Veterans

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Abstract

Objective

To test the hypothesis that veterans with traumatic brain injury (TBI) have an increased subsequent risk of sleep disorders, we studied the longitudinal association between TBI and incident sleep disorders in nearly 200,000 veterans.

Methods

We performed a cohort study of all patients diagnosed with a TBI in the Veterans Health Administration system from October 1, 2001, to September 30, 2015, who were age-matched 1:1 to veterans without TBI. Veterans with prevalent sleep disorders at baseline were excluded. Development of sleep disorders was defined as any inpatient or outpatient diagnosis of sleep apnea, hypersomnia, insomnia, or sleep-related movement disorders based on ICD-9 codes after the first TBI diagnosis or the random selection date for those without TBI. We restricted the analysis to those with at least 1 year of follow-up. We used Cox proportional hazards models to examine the association between TBI and subsequent risk of sleep disorders.

Results

The study included 98,709 veterans with TBI and 98,709 age-matched veterans without TBI (age 49 ± 20 years). After an average follow-up of 5 (1–14) years, 23,127 (19.6%) veterans developed sleep disorders. After adjustment for demographics, education, income, and medical and psychiatric conditions, those who had TBI compared to those without TBI were 41% more likely to develop any sleep disorders (hazard ratio 1.41 [95% confidence interval 1.37–1.44]), including sleep apnea (1.28 [1.24–1.32]), insomnia (1.50 [1.45–1.55]), hypersomnia (1.50 [1.39–1.61]), and sleep-related movement disorders (1.33 [1.16–1.52]). The association was stronger for mild TBIs, did not differ appreciably by presence of posttraumatic stress disorder, and remained after a 2-year time lag.

Conclusion

In 197,418 veterans without sleep disorders, those with diagnosed TBI had an increased risk of incident sleep disorders over 14 years. Improved prevention and long-term management strategies for sleep are needed for veterans with TBI.

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Go to Neurology.org/N for full disclosures. Funding information and disclosures deemed relevant by the authors, if any, are provided at the end of the article.

Glossary

CI = confidence interval; **CTBIE** = Comprehensive Traumatic Brain Injury Evaluation; **HR** = hazard ratio; **ICD-9** = *International Classification of Diseases, 9th revision*; **mTBI** = mild TBI; **NPCD** = National Patient Care Databases; **PTSD** = posttraumatic stress disorder; **TBI** = traumatic brain injury; **VHA** = Veterans Health Administration.

One of the most common and immediate consequences of a traumatic brain injury (TBI) is sleep complaints, which significantly impair patients' quality of life and rehabilitation process.[1–3] In a retrospective study of 116 soldiers returning from combat with mild to moderate TBI, almost all (97.4%) reported sleep disturbances such as hypersomnia and sleep fragmentation.[4] Notably, almost all previous studies of TBI and sleep have focused on self-reported sleep complaints immediately or shortly after TBI, and many did not have a control group for comparison. Moreover, few studies have examined the association between TBI and clinically diagnosed sleep disorders such as insomnia, hypersomnia, and sleep apnea. While it has been hypothesized that TBI might lead to permanent damage to sleep-related brain regions and thus long-lasting sleep disorders,[5,6] evidence on the longitudinal association between TBI and the development of sleep disorders years after the injury is limited. It is also unclear whether different severities of TBI might have a different impact on sleep and if the effects of TBI on sleep disorders are more immediate or latent. Sleep disorders affect rehabilitation in patients with TBI and lead to further negative cognitive and functional outcomes.[7,8] Understanding the long-term risk of different sleep disorders is critical to the development of targeted prevention and management strategies.

Military veterans with TBI often have comorbid psychiatric conditions, yet little is known about the association between TBI and risk of incident clinical sleep disorders in this population.[1,9] In particular, comorbid TBI and posttraumatic stress disorder (PTSD) are common and might further exacerbate sleep disturbances.[10–12] However, little is known about how PTSD might influence the association between TBI and subsequent risk of sleep disorders.

Our goal was to examine the independent association between TBI and long-term risk of incident sleep disorders in a large national sample of veterans who receive care in the Veterans Health Administration (VHA) health care system. We also explored the association for different severities of TBI and according to different lengths of follow-up, and we examined whether the association differs by the presence of PTSD.

Methods

Participants and Sampling

This is a longitudinal cohort study comprising VHA patients from the Comprehensive Traumatic Brain Injury Evaluation (CTBIE) database and the VHA inpatient and outpatient visits database (National Patient Care Databases [NPCD]) from October 1, 2001, to September 30, 2015. The CTBIE is an accruing national database of Iraq- and Afghanistan-era veterans who have enrolled in VHA health care and have completed a comprehensive TBI exposure survey since the program began in 2007. The CTBIE helps determine whether there was a TBI, its severity, and symptoms associated with TBI. Details about the CTBIE database have been summarized previously.[13,14] In addition, we included cases of TBI diagnosed by a comprehensive list of ICD-9 codes, created by the Defense and Veterans Brain Injury Center and the Armed Forces Health Surveillance Branch for TBI surveillance (2012 criteria).[15] We included veterans with a TBI diagnosis in NPCD from October 1, 2001, to September 30, 2015. Veterans without TBI were identified from a 2% random selection of individuals who have been under VHA health care during the same time frame.

In this study, we defined a study baseline index date for each individual as the first TBI diagnosis date or the random selection date for veterans without a TBI diagnosis. To construct a cohort of veterans with and without TBI, we first excluded all participants with a diagnosis of sleep disorders at or before their baseline index date. We included veterans with at least 1 year of follow-up. Next, we age-matched patients with TBI 1:1 to those without TBI. The final age-matched cohort consisted of 98,709 patients with TBI and 98,709 patients without TBI. Of this sample, 5,992 (3.0%) were from the CTBIE database, 183,029 (92.7%) were from the NPCD, and 8,397 (4.3%) were in both databases. We defined TBI severity as mild or moderate-severe according to the 2010 Department of Defense clinical criteria or the Defense and Veterans Brain Injury Center and Armed Forces Health Surveillance Branch ICD-9 2012 criteria.[15]

Sleep Disorders

We defined newly diagnosed sleep disorders as all inpatient or outpatient diagnoses based on ICD-9 codes (780.5, 307.4, 327, 347) after the baseline index date. Specific sleep disorders of interest included insomnia (327.0, 780.51, 780.52), hypersomnia disorders and narcolepsy (327.1, 780.53, 780.54, 347), sleep-related breathing disorders (327.2, 780.51, 780.53, 780.57), and sleep-related movement disorders (327.5, 780.58).

Other Measures

The NPCD contained information on demographics and medical and psychiatric comorbid conditions. Self-reported demographics included age, sex, and race or ethnicity (non-

Table Baseline Characteristics of 197,418 Veterans With or Without TBI

	No TBI (n = 98,709)	Any TBI (n = 98,709)	*p* Value
Demographics			
Age, mean (SD), y	50.32 (19.05)	48.16 (18.44)	<0.001
Female	14,731 (14.92)	8,424 (8.53)	<0.001
Race			<0.001
Non-Hispanic White	55,040 (55.76)	61,199 (62.00)	
Non-Hispanic Black	12,055 (12.21)	12,689 (12.85)	
Hispanic	1,026 (1.04)	2,286 (2.32)	
American Indian, Asian, or Native Hawaiian	1,524 (1.54)	1,883 (1.91)	
Decline/unknown	29,064 (29.44)	20,652 (20.92)	
>25% college-educated in zip code	49,368 (50.01)	49,046 (49.69)	0.007
Median income tertile in zip code			<0.001
Low (<$25,338)	31,083 (31.49)	31,790 (32.21)	
Middle	31,763 (32.18)	31,171 (31.58)	
High (>$33,347)	31,630 (32.04)	31,244 (31.65)	
Medical			
Diabetes	4,229 (4.28)	3,864 (3.91)	<0.001
Hypertension	11,880 (12.04)	11,811 (11.97)	0.633
Myocardial infarction	1,099 (1.11)	1,616 (1.64)	<0.001
Cerebrovascular disease	1,761 (1.78)	6,820 (6.91)	<0.001
Psychiatric			
Mood disorder	9,184 (9.30)	22,132 (22.42)	<0.001
Anxiety	4,307 (4.36)	10,319 (10.45)	<0.001
PTSD	4,374 (4.43)	19,202 (19.45)	<0.001
Any substance abuse	5,177 (5.24)	11,277 (11.42)	<0.001
Smoking or tobacco use	8,620 (8.73)	13,349 (13.52)	<0.001

Abbreviations: PTSD = posttraumatic stress disorder; TBI = traumatic brain injury.
Values are N (%) unless otherwise specified. The *p* values are based on paired *t* test for age and McNemar test for other variables.

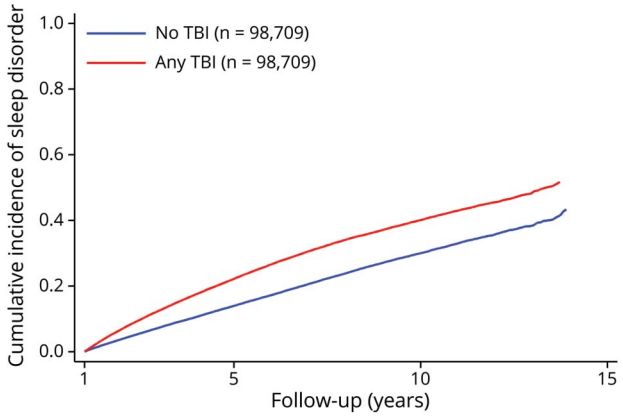
Hispanic Black, non-Hispanic White, Hispanic or other/unknown). Veterans were classified as living in broad educational and income groups based on the 2000 US Census data and zip code at the index date. We defined education as the proportion of the adult population who have completed college education or more in zip code tabulation areas, dichotomized into ≤25% and >25%.[13,14] Income was grouped as tertile of median zip code tabulation area income for adults <75 or ≥75 years of age.

We defined medical and psychiatric comorbid conditions as diagnoses by ICD-9 codes in any inpatient or outpatient visit at baseline or during the 2 years before baseline. For this study, medical conditions included hypertension, diabetes, cerebrovascular disease, and myocardial infarction; psychiatric disorders included anxiety, mood disorder (dysthymia, depression, bipolar disorder), PTSD, tobacco use, and substance abuse.

Statistical Analysis

We first compared the baseline characteristics by TBI status using paired *t* tests and McNemar χ^2 tests for continuous and categorical variables, respectively. Multivariable Cox proportional hazards models were adopted to assess time to sleep disorder diagnosis with years of follow-up used as the time scale. Individuals were censored at the last medical encounter or death. To examine the independent effects of TBI on subsequent sleep disorders, we progressively controlled for demographics (age, sex, race, education, and income), medical conditions (hypertension, diabetes,

Figure 1 Cumulative Incidence of Any Sleep Disorder Among Veterans With and Without TBI



Unadjusted cumulative incidence of all sleep disorders is shown as a function of the presence of traumatic brain injury (TBI). After adjustment for sex, race, education, income, and medical and psychiatric conditions, those who had TBI were 41% more likely to develop any sleep disorders (hazard ratio 1.41 [95% confidence interval 1.37–1.44]).

cerebrovascular disease, and myocardial infarction), and psychiatric disorders (anxiety, mood disorder, PTSD, substance use disorder, and tobacco use) in separate models. Cumulative incidence of specific sleep disorders by TBI status was also assessed graphically. We performed a number of sensitivity analyses, including (1) study of the severity of TBI; (2) adjusting further for the number of follow-up visits; (3) stratifying according to length of follow-up (<3 vs ≥3 years) to explore the impact of follow-up length on the association; and (4) adding random effect to paired identification to account for the intrapair correlation. To determine whether the relationship between TBI and incident sleep disorders differed by comorbid PTSD, we further stratified the analysis by PTSD and examined the interaction between TBI and PTSD using a likelihood ratio test. To ensure that sleep disorders occurred after TBI diagnosis, we also performed a sensitivity analysis by imposing a lag time or washout period of 2 years from baseline to sleep disorder diagnosis date, such that participants with a diagnosis of sleep disorders within 2 years after baseline were excluded. We tested the Cox proportional hazards model assumptions and found no evidence against these assumptions. Two-sided p values were used, and we defined $p < 0.05$ as statistical significance. We performed all analyses with SAS version 9.4 (SAS Institute Inc, Cary, NC).

Standard Protocol Approvals, Registrations, and Patient Consents

The study protocol was approved by the institutional review boards at the University of California, San Francisco; San Francisco VA Medical Center; and US Army Medical Research and Materiel Command Office of Research Protections and Human Research Protection Office. The need for informed consent was waived given the use of deidentified administrative data.

Data Availability

Data for this study were obtained from VHA electronic health records and contain protected health information, including age, scrambled social security number, date of birth, date of death, and dates of medical diagnoses. Therefore, we are unable to share our dataset with other investigators or place it in a repository. Investigators wishing to replicate these analyses may contact the authors to discuss the process of obtaining access to VHA data.

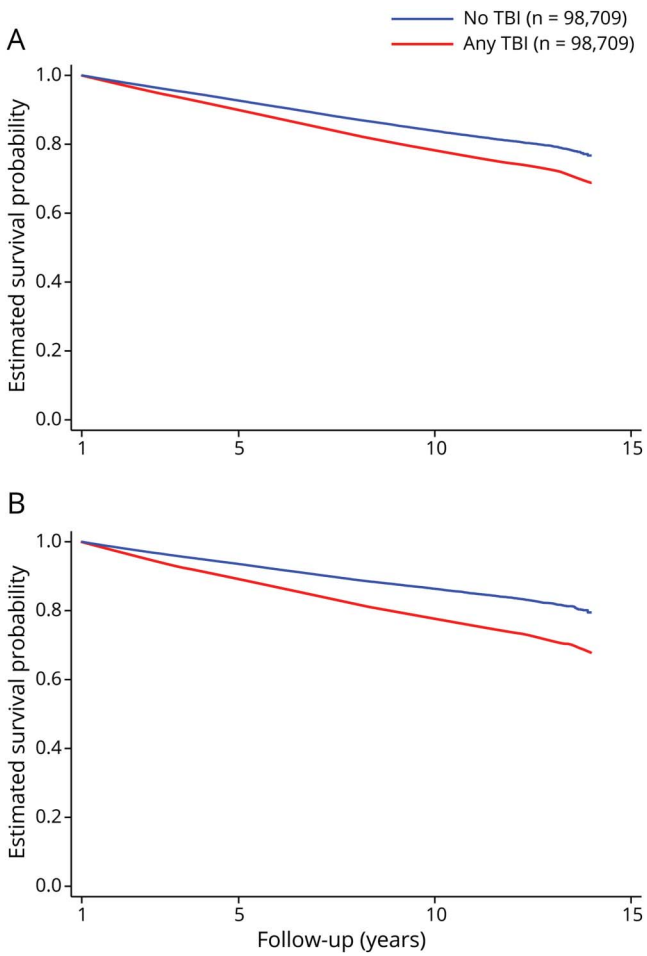
Results

The table shows the baseline characteristics of the nearly 200,000 veterans with and without TBI. In general, veterans with and without TBI were well matched for age. Participants had a mean age of 49 ± 20 years at baseline, and 11.7% were women. Of the patients with TBI, 49,006 (49.6%) were mild cases. Veterans with TBI were less likely to be women and more likely to have comorbid conditions. In particular, those with TBI were much more likely than those without TBI to have psychiatric conditions, including mood disorders (22.4% vs 9.3%), anxiety (10.5% vs 4.4%), PTSD (19.5% vs 4.4%), substance abuse (11.4% vs 5.2%), and smoking or tobacco use (13.5% vs 8.7%). Compared to those with mild TBI (mTBI), the cases of more severe TBI were older, were less likely to be women, and had lower socioeconomic status, more medical comorbid conditions, and fewer psychiatric conditions.

The veterans had follow-up medical record information for an average of 5.0 (1–14.0) years: 4.8 years for those with TBI and 5.1 years for those without TBI. Over the follow-up, 23,127 (23.4%) of veterans with TBI developed sleep disorders, while 15,583 (15.8%) of those without TBI developed sleep disorders. Figure 1 shows the cumulative incidence of any newly diagnosed sleep disorder by the presence of TBI. After adjustment for age, sex, race, education, and income, those who had TBI were 50% more likely to develop any sleep disorders compared to those without TBI (hazard ratio [HR] 1.50 [95% confidence interval (CI) 1.47–1.53]). After further adjustment for medical (diabetes, hypertension, myocardial infarction, and cerebrovascular disease) and psychiatric (mood disorder, anxiety, PTSD, substance use disorder, and tobacco use) disorders, the adjusted HR for developing sleep disorders was 1.41 (95% CI 1.37–1.44).

Veterans with TBI were more likely than those without TBI to develop incident sleep apnea (11.4% vs 8.3%), insomnia (12.0% vs 7.2%), hypersomnia (2.1% vs 1.3%), and sleep-related movement disorders (0.6% vs 0.4%). Of the cases with sleep apnea, the majority (92.9%) had obstructive sleep apnea. Figure 2 shows the Kaplan-Meier survival estimates for sleep apnea and insomnia, the 2 most common sleep disorders in

Figure 2 Kaplan-Meier Survival Estimates of Sleep Apnea and Insomnia by TBI



Unadjusted estimated survival probability for (A) sleep apnea and (B) insomnia is shown as a function of the presence of traumatic brain injury (TBI). After adjustment for sex, race, education, income, and medical and psychiatric conditions, the hazard ratios (95% confidence intervals) were 1.28 (1.24–1.32) for sleep apnea, 1.50 (1.45–1.55) for insomnia, 1.50 (1.39–1.61) for hypersomnia, and 1.33 (1.16–1.52) for sleep-related movement disorders. Survival plots for hypersomnia and sleep-related movement disorders are not shown due to small number of cases.

veterans. After adjustment for demographics and medical and psychiatric conditions, the HRs were 1.28 (95% CI 1.24–1.32) for sleep apnea, 1.50 (95% CI 1.45–1.55) for insomnia, 1.50 (95% CI 1.39–1.61) for hypersomnia, and 1.33 (95% CI 1.16–1.52) for sleep-related movement disorders.

Overall, the association was stronger for mTBI compared to moderate to severe TBI: after adjustment for demographics and medical and psychiatric disorders, the HRs of developing any sleep disorder were 1.49 (95% CI 1.45–1.53) for mTBIs and 1.33 (95% CI 1.30–1.37) for moderate to severe TBIs. After additional adjustment for the number of follow-up visits, veterans with TBI were 30% more likely to develop all incident sleep disorders (HR 1.30 [95% CI 1.27–1.33]). The association remained among those with at least 3 years of follow-up: the HR was 1.30 (95% CI 1.26–1.33). The results were similar after random effect for the paired sample was added (HR 1.42 [95% CI 1.33–1.53]).

The association between TBI and sleep disorders was similar for veterans with and without PTSD: with PTSD, the HR was 1.32 (95% CI 1.21–1.45), and without PTSD, the HR was 1.50 (95% CI 1.45–1.55) (p for interaction = 0.11). The association was attenuated but remained statistically significant after the introduction of a 2-year time lag between TBI and the development of sleep disorders. The adjusted HRs were 1.35 (95% CI 1.31–1.38) for developing any sleep disorders, 1.23 (95% CI 1.19–1.27) for sleep apnea, 1.45 (95% CI 1.39–1.50) for insomnia, 1.42 (95% CI 1.30–1.55) for hypersomnia, and 1.28 (95% CI 1.09–1.49) for sleep-related movement disorders.

Discussion

In this cohort of 197,418 veterans without prevalent sleep disorders, those with TBI had an increased risk of developing all major clinical sleep disorders, including sleep apnea, insomnia, hypersomnia, and sleep-related movement disorder, independently of demographics, medical conditions, and psychiatric disorders, over the following 14 years. The association for mTBI was stronger and remained constant throughout the follow-up; the association for moderate to severe TBI was more robust among those with <3 years of follow-up and was attenuated over time. Overall, the association between TBI and sleep disorders was reduced somewhat but still moderate after the introduction of a 2-year washout period. Moreover, the association was similar among those with and those without PTSD.

Most prior studies have focused on the immediate effects of TBI on subjective sleep needs or the prevalence of sleep complaints in the acute phase of TBI.[16–19] One review of 24 studies of military service members suggested that all studies have focused on the prevalence rather than incidence of sleep disturbances in patients with TBI.[9] The current study builds on prior cross-sectional evidence and reports the long-term risk of newly diagnosed sleep disorders after TBI among military veterans. This is consistent with findings from nonveteran populations, including another recent study of a large national sample of older adults that suggested that newly diagnosed sleep disorders were common both before and after TBI.[20] A few small case-control studies have used objective measures to assess short-term sleep changes in patients with TBI and generated inconsistent findings, with some suggesting impaired polysomnography-measured sleep quality and altered sleep staging in patients with mTBI[21–23] and others indicating no change in sleep quality.[24,25] Meanwhile, it was unclear whether sleep changes persist over time after TBI. While several studies found persistence or new onset of sleep-wake disturbances, including excessive daytime sleepiness, hypersomnia, and insomnia, at between 6 months and 3 years after injury,[5,26,27] the majority of these studies did not have a

control group and included only small clinical samples of up to 65 patients with TBI. The Armed Forces Health Surveillance Center compared the prevalence of 14 common symptoms in military service members with TBI and found sleep disorders to have the second largest proportion increase from 3 to 12 months after injury.[28] We found that the association between TBI and risk of incident sleep disorders, although slightly reduced, remained moderate after imposing a 2-year washout period or among those with at least 3 years of follow-up. This indicated a possibility that some of the undiagnosed sleep disorders might be residual symptoms of TBI and may not have persisted past 2 years after TBI, while most other cases were developed after TBI and the latent effects of TBI on sleep could not be neglected.

Notably, the association with incident sleep disorders was generally stronger for mTBIs than for moderate to severe TBIs, which might be due to the different brain injury mechanism. Specifically, mTBIs often involve repetitive concussive or subconcussive injuries such as sports injuries or blast injury among active duty military or acceleration/deceleration injury. This type of damage is more likely to cause diffuse axonal injury and inflammation,[29] while moderate or severe TBI is often due to a direct blow with more focal but severe damage. Therefore, the symptoms and sequelae of mTBIs and more severe TBIs may differ. In this study, veterans with mTBIs were also much more likely to have psychiatric comorbid conditions, including mood disorders and PTSD, which might partly mediate the effects of TBI on sleep disorders, although the effects of mTBIs on sleep disorders remained robust after adjustment for psychiatric comorbid conditions.

In examining specific diagnoses of sleep disorders, we found that patients with TBI had increased risk of developing all major sleep disorders in the future. This is in line with the literature,[30] including findings from a meta-analysis that found among patients with recent TBI a prevalence of 20% to 30% for insomnia, hypersomnia, obstructive sleep apnea, and periodic limb movement during sleep.[17] It is unclear why we found among patients with TBI a potential higher subsequent risk of developing insomnia or hypersomnia. Findings from polysomnography and EEG studies in patients with TBI suggest that sleep activity changes related or similar to the symptoms of insomnia, including frequent and long night awakenings, increased N1 and N2 stages of non-REM sleep and decreased REM sleep and multiple abnormalities in frequency bands in both non-REM and REM sleep.[31–34] Some animal models of TBI[35–37] have shown a hypersomnolence phenotype that resembles reports of daytime sleepiness and nighttime sleep fragmentation.

The mechanism by which TBI leads to the development of sleep disorders is poorly understood, although an increasing number of studies have begun to identify TBI-induced neuropathologic events or endocrine dysregulation that might contribute to both short- and long-term changes in sleep-wake activities after TBI.[29] Some have proposed that structural brain injuries, including damage to the suprachiasmatic nucleus, optic chiasm, hypothalamus, amygdala, and brainstem, lead to direct damage to arousal-promoting neurons and might trigger secondary sleep-wake disturbances after TBI, especially in more severe TBI.[38–40] For instance, hypothalamic injury could contribute to sleep-wake disorders through loss of wake-promoting histaminergic or orexin/hypocretin neurons.[41,42] Risk of sleep apnea might be increased as a result of mechanical changes of craniofacial anatomy, although the exact pathways linking TBI and different forms of sleep apnea remain to be elucidated. Loss of brainstem neurons has also been observed immediately after TBI, including 17% fewer serotonergic dorsal raphe nuclei neurons and 29% fewer noradrenergic locus coeruleus neurons, although this was less severe than previously reported loss of hypothalamic wake-promoting neurons.[43] While it is plausible that an aggregate of these lesions contributes to sleep-wake changes, previous studies have examined brain structural damage and neuron loss only in the acute phase of TBI or at up to 6 months after injury. Similarly, several studies have found reduced secretion of melatonin at up to 6 months after TBI,[44,45] which could also account for the presence of sleep-wake disturbances after TBI. Longitudinal evidence is needed to determine whether TBI induces permanent brain structural damage, whether the effects of TBI on wake-promoting neurons and melatonin secretion persist over time, and whether these mechanisms play a role in the development of sleep disorders in the long term.

Another hypothesized pathway is that psychiatric comorbid conditions, including comorbid PTSD, in patients with TBI might drive a short-term increase in subjective sleep-wake disturbances[46,47] and possibly more long-term increases. In the present study, the association between TBI and the development of sleep disorders remained after controlling for medical and psychiatric comorbid conditions and did not differ by the presence of PTSD.

This study has several important strengths. We report for the first time a longitudinal association between TBI and risk of developing sleep disorders several years after injury in a large national sample of military veterans. TBI cases were identified through both the CTBIE and NPCD, and the severity of TBI was also assessed; the use of clinical diagnoses of sleep disorders has added clinical implications and allowed us to examine and compare different types of sleep disorders. Furthermore, we were able to control for a wide range of potential confounders and to perform several sensitivity analyses that support a robust finding even after introducing a 2-year time lag and stratifying by follow-up length and the presence of PTSD. There are also a few limitations. We used ICD-9 codes to define TBI and sleep disorders, which may be less sensitive than standardized assessment. Sleep disorders may have been underdiagnosed in medical records, and we were more likely to have captured severe sleep problems. Our sample comprised a veteran population, and we cannot be

certain whether these findings are generalizable to civilian populations. However, given that most TBIs incurred over many years among veterans are not military related,[48] it is likely that the association between TBI and sleep disorders is applicable to nonveteran populations. All veterans were identified within the VHA health care system, and thus, the results might not be generalizable to those receiving care outside the VHA system. As with other studies using electronic health records, cases are identified only when the patient visits the clinic and receives a diagnosis by the physician. To help address this issue, we further adjusted for the number of follow-up visits in sensitivity analysis. Furthermore, most veterans were men, which limits the generalizability of these findings to women.

We show that veterans with TBI and without prevalent sleep disorders at baseline had an increased subsequent risk of developing sleep disorders, including insomnia, hypersomnia, sleep apnea, and sleep-related movement disorders, over 14 years, independently of medical and psychiatric comorbid conditions. This association was stronger for mTBIs, did not differ by PTSD, and remained after the introduction of a 2-year time lag and among those with at least 3 years of follow-up. Increased clinical attention should be paid to both short-term and long-term risk of sleep disorders in patients with TBI, both the mild and more severe cases. Additional research is needed to determine the underlying mechanisms for a longitudinal link between different severity and types of TBI and development of sleep disorders. Ultimately, early identification and prevention strategies for sleep disorders after TBI need to be developed and would be critical for improving quality of life and other long-term outcomes in patients with TBI.

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Disclosure

All other authors declare no conflict of interest. Go to Neurology.org/N for full disclosures.

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Appendix Authors

Name	Location	Contribution
Yue Leng, PhD	University of California, San Francisco	Design and conceptualized study; drafted the manuscript for intellectual content
Amy Byers, PhD	San Francisco Veterans Affairs Health Care System, CA	Acquired and interpreted the data; revised the manuscript for intellectual content
Deborah E. Barnes, PhD	San Francisco Veterans Affairs Health Care System, CA	Interpreted the data; revised the manuscript for intellectual content
Carrie B. Peltz, PhD	San Francisco Veterans Affairs Health Care System, CA	Interpreted the data; revised the manuscript for intellectual content
Yixia Li, MPH	San Francisco Veterans Affairs Health Care System, CA	Analyzed the data; performed statistical analysis; revised the manuscript for intellectual content
Kristine Yaffe, MD	San Francisco Veterans Affairs Health Care System, CA	Major role in the acquisition of data; designed and conceptualized study; revised the manuscript for intellectual content

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