SLEEPJ, 2019, 1–10

doi: 10.1093/sleep/zsz179
Advance Access Publication Date: 13 August 2019
Original Article

ORIGINAL ARTICLE

# Sleep duration and post-traumatic stress disorder symptoms: a twin study

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## Abstract

**Study Objectives:** Long and short sleep duration are associated with greater risk of developing post-traumatic stress disorder (PTSD); however, it is unknown how genetic and environmental influences affect this relationship. Thus, we investigated the association between sleep duration and PTSD symptoms using twin models.

**Methods:** Data were obtained from 1865 monozygotic and 758 dizygotic twin pairs enrolled in the community-based Washington State Twin Registry. PTSD symptoms were assessed using the Impact of Events Scale (IES). A classical twin model decomposed the variances of sleep duration and IES score into additive genetic, shared environmental, and unique environmental components. We used correlated factor models to examine the moderation of variance components of sleep duration and IES.

**Results:** Shorter and longer sleep duration were associated with higher IES scores with a quadratic association ($p < 0.001$). The heritability of sleep duration was 36%, and IES 31%. Variance in sleep duration attributable to shared ($b_{1C1} = 2.91$, 95% CI = 1.40 to 4.43; $p < 0.001$) and unique ($b_{1E1} = 0.18$, 95% CI = 0.10 to 0.27; $p < 0.001$) environment was moderated by IES score. Similarly, but to a lesser extent, variance in IES attributable to additive genetics ($b_{1A2} = -0.23$, 95% CI = −0.45 to 0.00; $p = 0.048$) was moderated by sleep duration.

**Conclusions:** Greater PTSD symptom severity was associated with short and long sleep duration. Increasing PTSD symptoms increased variability in sleep duration primarily via shared environmental factors, whereas decreasing sleep duration increased variability in PTSD symptoms primarily via additive genetic factors. This suggests childhood experiences affect variability of sleep duration and genetic factors affect the variability of PTSD symptoms in trauma-exposed individuals.

### Statement of Significance

Post-traumatic stress disorder (PTSD) is a serious pathologic response to trauma with genetic, epigenetic, and environmental components. Sleep problems, including chronic short and long sleep duration, are associated with higher PTSD risk. Our study results add to an important body of literature showing that numerous medical and psychiatric health conditions occur at the extremes of sleep duration. Our results help untangle the complexity of genetic and environmental factors in post-traumatic stress by demonstrating the impact of childhood experiences on adult sleep duration. Sleep duration also moderates genetic influences on PTSD symptom variability, providing a possible mechanism on how poor sleep is associated with increased risk of PTSD symptoms in traumatized individuals with genetic susceptibility.

**Key words:** post-traumatic stress disorder; sleep duration; monozygotic; dizygotic; twins

**Submitted:** 8 January, 2019; **Revised:** 18 June, 2019

## Introduction

Post-traumatic stress disorder (PTSD) is a debilitating psychiatric syndrome characterized by persistent intrusive memories, avoidance of trauma reminders, negative thoughts and feelings, and trauma-related hyperarousal precipitated by a traumatic event. PTSD is diagnosed using the *Diagnostic and Statistical Manual* (*DSM*) criteria, which requires the presence of at least one avoidance symptom, two cognition and/or mood symptoms, and two arousal-related symptoms persisting for at least one month after a traumatic experience [1]. Although trauma exposure is common throughout the world with 50%–90% of people experiencing an event such as a violent personal assault, serious motor vehicle accident, war, or natural disaster at some point in their life [2], only 4% develop PTSD, with higher prevalence reported for combat veterans (12%–30%) and assault victims (20%–30%) [2–7].

PTSD pathophysiology is complex and theorized to encompass genetic, epigenetic, developmental, and psychosocial factors [8–10]. Early family studies on PTSD found the disorder is more common in family members of Holocaust survivors and Cambodian refugees with PTSD than those without [11, 12]. Twin studies later showed genetics contributes to both the trauma exposure and PTSD development [13, 14]. Genetic factors account for an estimated 23.5%–71% of the variance in PTSD development in combat veterans and civilians even after controlling for trauma exposure, with lower heritability estimates found in the US Vietnam Era Twin Registry (23.5%) [14] and a civilian population in Vancouver, Canada [15], and higher heritability estimates found in female civilian participants [16–18].

Sleep problems after trauma exposure consistently predict the onset and persistence of PTSD and other stress-related psychiatric disorders in civilian and veteran populations [19–23]. In particular, very short sleep duration (≤5 hours/night) is associated with higher PTSD risk and psychiatric illness in US Iraq war veterans compared to short (6–7 hours/night) and average sleep duration (≥7 hours/night) [24]. Bivariate correlations between sleep duration and psychiatric comorbidities in US Afghanistan/Iraq era war veterans show soldiers with very short (≤5 hours) and long (≥9 hours) sleep have three times the rate of PTSD and major depressive disorder (MDD) relative to soldiers with average sleep duration (7–8.9 hours) [25]. In community samples, short sleep (≤5 hours/night) is associated with a significantly increased likelihood of PTSD [26]. However, another study found no difference in Post-traumatic Stress Disorder Checklist scores between short sleep (≤6 hours/night) and normal/long sleepers in male, young adult African Americans, although notably normal and long sleepers were grouped together for the analysis [27].

Sleep derangements are not only symptoms of PTSD, but may also contribute to its development [28]. Prospective studies in civilian and veteran populations show poor sleep and short sleep duration occurring prior to traumatic events increase risk of PTSD, MDD, and panic disorder following trauma [29, 30]. Uncertainty exists whether poor sleep is primarily a risk factor for PTSD or a result of the disorder itself.

Twin studies allow investigation of the gene-environment interplay between inadequate or excessive sleep and PTSD symptoms by decomposing genetic and environmental sources of variance and distinguishing risk factors from sequelae [31]. This is important when studying complex disorders such as PTSD, which are known to have genetic, epigenetic, and environmental influences on pathophysiology [8, 18, 31]. As noted earlier, short and long sleep duration are linked to PTSD development in veterans, but this has not been studied as extensively in civilian populations [24–26]. Although military populations have been known to experience restricted sleep [24], civilian populations also face an epidemic of chronic sleep deprivation perpetuated by societal pressures and technologies, leading to increasingly later bedtimes and earlier rise times for adults and children. Health surveys conducted by the Centers for Disease Control and Prevention indicate that 35%–40% of US adults sleep less than 7–8 hours per night, with 15% sleeping less than 6 hours [32]. The makers of one consumer sleep technology (Fitbit Inc, San Francisco, CA) report an average nightly sleep duration of 6 hours and 50 minutes for women and 6 hours and 26 minutes for men, both substantially less than the minimum sleep duration of 7 or more hours recommended by the American Academy of Sleep Medicine and Sleep Research Society [33, 34]. Short sleep (under 7 hours/night) and in some studies, long sleep (at least 8–9 hours/night), have been associated with a myriad of chronic health conditions, including cardiovascular conditions [35, 36], diabetes [37, 38], cognitive impairment [39], motor vehicle accidents [40], and psychiatric disorders such as depression and substance dependence [41, 42].

In this study, we examine the relationship between PTSD symptoms and sleep duration in same-sex identical (monozygotic; MZ) and fraternal (dizygotic; DZ) twins enrolled in a large civilian community-based twin registry. We determine whether short and long sleep duration are associated with greater PTSD symptom severity, estimate the contribution of genetic and environmental influences on sleep duration and PTSD symptoms, and analyze moderating relationships between sleep duration and PTSD symptom severity. Analysis of these relationships using a twin study provides a better understanding of sleep problems with PTSD, and the role genetic and environmental influences play in this relationship. If short or long sleep duration is indeed a risk factor for PTSD, early prevention and/or treatments targeting habitual sleep length may improve stress-related outcomes after trauma exposure.

## Methods

### Participants

Data were obtained from a sample of 2623 same sex adult twin pairs (1865 MZ pairs and 758 DZ pairs) enrolled in the community-based Washington State Twin Registry (WSTR) [43]. Twins were aged 18–91 years (mean = 41.57, *SD* = 17.56) at the time of the survey, 69.4% female, and raised together. Most participants (94.2%) self-identified as white. 57.5% reported being married or living with a partner, 30.7% were single, 9.6% were divorced, and 2.2% were widowed. 48.3% of respondents reported having at least a Bachelor's degree level of education, 33.2% had a college or Associate's degree, 15.0% completed high school or had a GED, and 3.2% completed less than a high school level of education.

Details of WSTR construction are described elsewhere [43, 44]. Twins completed surveys including items on sociodemographic data, physician-diagnosed health conditions, habits, environmental exposures, and various abridged screening questionnaires for physical and mental health symptoms. Zygosity is determined through survey items focused on childhood similarity. This method correctly classifies zygosity with at least 95% accuracy compared to biological zygosity indicators [45–47].

## Measures

### *Sleep duration*

Sleep duration was self-reported by the survey item, "On average, how long do you sleep at night?" recorded in hours and minutes.

### *Post-traumatic stress disorder symptoms*

Trauma history was self-reported with the item, "Some people have terrible experiences happen to them. Have you experienced any of the following?," with potential Yes/No answers for combat, fire/explosion, physical assault, or "other." PTSD symptoms were assessed using the Impact of Events Scale (IES) from the WSTR Enrollment Survey. The IES is a questionnaire rating subjective symptoms of distress occurring within the last 7 days when thinking about an upsetting event [48]. IES items correspond to the intrusion and avoidance symptom clusters for PTSD diagnosis in the *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition (*DSM-5*) [1]. The Enrollment Survey includes 11 of the 15 original IES items that correlate most highly with the IES intrusion and avoidance subscales [44, 48, 49]. Respondents rate how often they experience symptoms of intrusive thoughts, sleep disturbance, and avoidance of event reminders, with four response categories (0 = not at all, 1 = rarely, 3 = sometimes, or 5 = often). The total IES score can range from 0 to 55. We grouped scores into terciles of increasing distress levels, with 0–10 indicating low distress, 11–27 indicating moderate distress, and ≥28 indicating high distress [44, 49].

In previous studies, the IES was strongly correlated with a PTSD diagnosis [50, 51]. IES sensitivity as a screening tool for PTSD ranges from 0.94 to 1.00, with a specificity ranging from 0.78 to 0.84 [51]. A prior study using the WSTR modified IES demonstrated high internal reliability and validity between our IES score terciles and self-reported physician diagnosed PTSD [49]. We used IES, rather than lifetime self-reported physician diagnosis of PTSD, because individuals with PTSD are often underdiagnosed and undertreated [52]. Use of the IES also ensured that PTSD symptoms were current (within the last 7 days), as the lifetime physician diagnosis of PTSD may be from a previously experienced episode from which the individual has recovered. Finally, using the IES allowed us to analyze PTSD symptom severity as a continuous rather than binary variable.

### *Other variables*

We additionally analyzed relationships between PTSD symptoms and other factors known to be related to PTSD, including the presence of insomnia, alcohol use, depression, and chronotype. Insomnia was assessed with the enrollment survey item, "How often do you have difficulty falling asleep or staying asleep?" with potential responses of, "Never," "Sometimes," "Often," and "Always." Alcohol use was assessed with the item, "How many drinks of alcohol do you have on a typical day when you are drinking?" with responses of, "1 to 2," "3 to 4," "5 to 6," "7 to 9," "10 or more," or "I don't drink alcohol." Depression symptoms were assessed with the Patient Health Questionnaire-2. Chronotype was assessed with the item, "One hears about 'morning' and 'evening' types of people. Which one of these types do you consider yourself to be?" with responses of "Definitely a morning type," "More a morning than an evening type," "More an evening than a morning type," or "Definitely an evening type."

## Statistical analyses

Descriptive statistics were performed in the statistical program R 3.5.1 (R Core Team) [53]. All latent variable path analyses were conducted using the computer program Mplus v. 8.1 (Muthén & Muthén, 2013). Our initial analysis examined the correlation between sleep duration and IES score. Visual examination of the data revealed a nonlinear association, which we then analyzed using the following quadratic equation: IES = $b_0 + b_1 * sleep + b_2 * sleep^2$, where "IES" is the total IES score, $b_0$ is the intercept, $b_1$ is the linear slope, $b_2$ is the quadratic association, and "sleep" is the sleep duration.

## Univariate twin model

We used the classical twin model to decompose the variances of sleep duration and IES score into three components (Figure 1): additive genetic variance (A), which represents the additive effect of an individual's genes; shared environmental variance (C), which represents shared environmental experiences that make members of the family more similar; and non-shared environmental variance (E), which represents unique environmental experiences [54]. The A variance components correlate at $r = 1.0$ between MZ twins (who share 100% of their genetic sequence) and $r = 0.5$ between DZ twins (who share 50% of their segregating genes, on average). The C variance components correlate at $r = 1.0$ for both MZ and DZ twins. The E variance components do not correlate between twins as they represent experiences unique to the individual and are not shared between members of a twin pair.

## Correlated factor model

We used a correlated factor model to examine the association between sleep duration and IES score [54]. (Figure 2) The correlated factor model is an extension of the univariate twin model, in which the A and E components of sleep duration are correlated with those of IES score ($r_a$ and $r_e$, respectively). As the univariate twin model showed 0% of the variance of IES is attributed to C, the C component for IES was fixed at 0, and no correlation between the C components was estimated.

## Moderated correlated factor model

By extending the correlated factor twin model, we were able to test for the moderation of the ACE variance components of sleep duration by IES score, and AE variance components of IES score by sleep duration. As illustrated in Figure 3, the A, C, and E components of sleep duration ($A_1$, $C_1$, and $E_1$, respectively) can all be modified by IES score, and the A and E components of IES score ($A_2$ and $E_2$, respectively) can be modified by sleep duration. For each of the modified paths, IES score or sleep duration is the moderating variable; the $b_0$ terms are the values of the ACE variances where IES score or sleep duration is zero, and the $b_1$ terms represent the rate of increase or decrease for a given variance component as a function of the moderator (IES score or sleep duration). An exponential function was included to constrain the estimated parameters to be positive. As with the correlated factor twin model above, the C component for IES score was fixed at 0. The $\exp(b_{0A1} + b_{1A1}IES)$

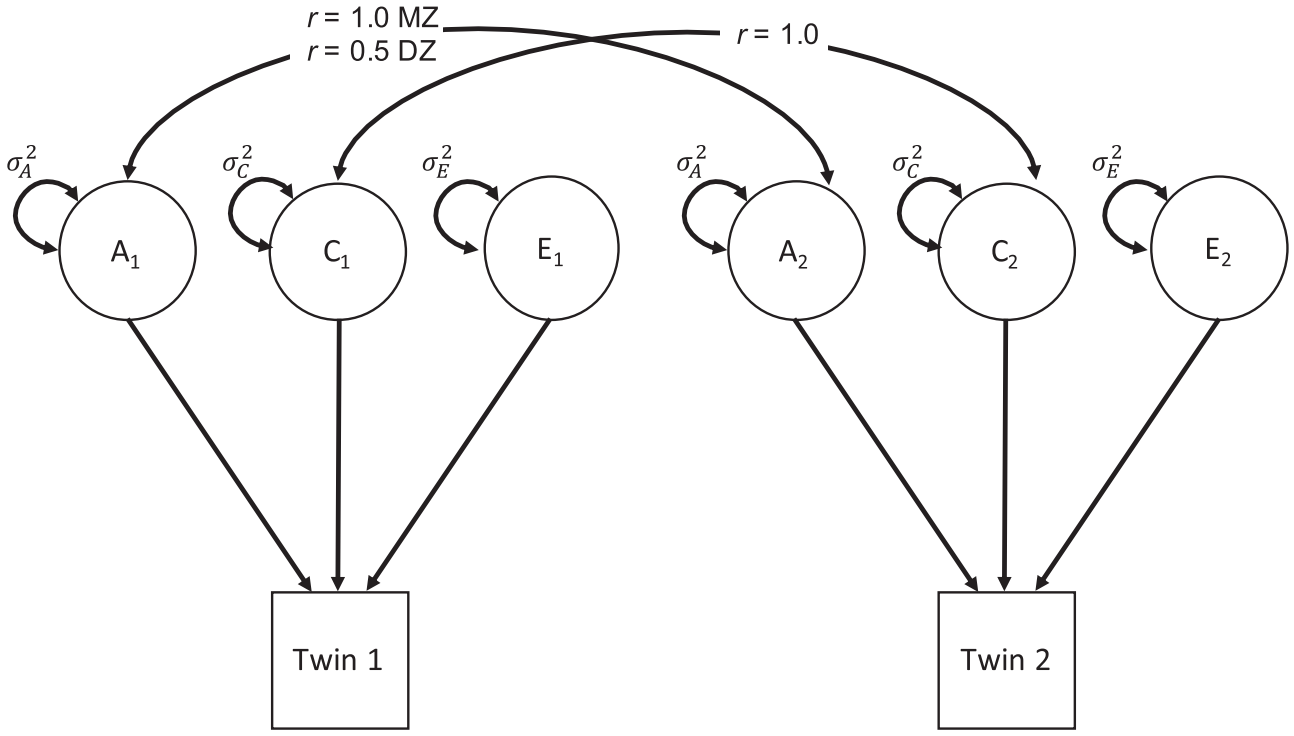


**Figure 1.** Classical univariate twin model for a single variable. The variance of the observed variable is decomposed into three components (shown in circles): additive genetic influences (A), environmental influences that are shared between twins (C), and non-shared environmental influences that are unique to each twin (E). The total variance is the sum of the squares of these three components. MZ = monozygotic; DZ = dizygotic.

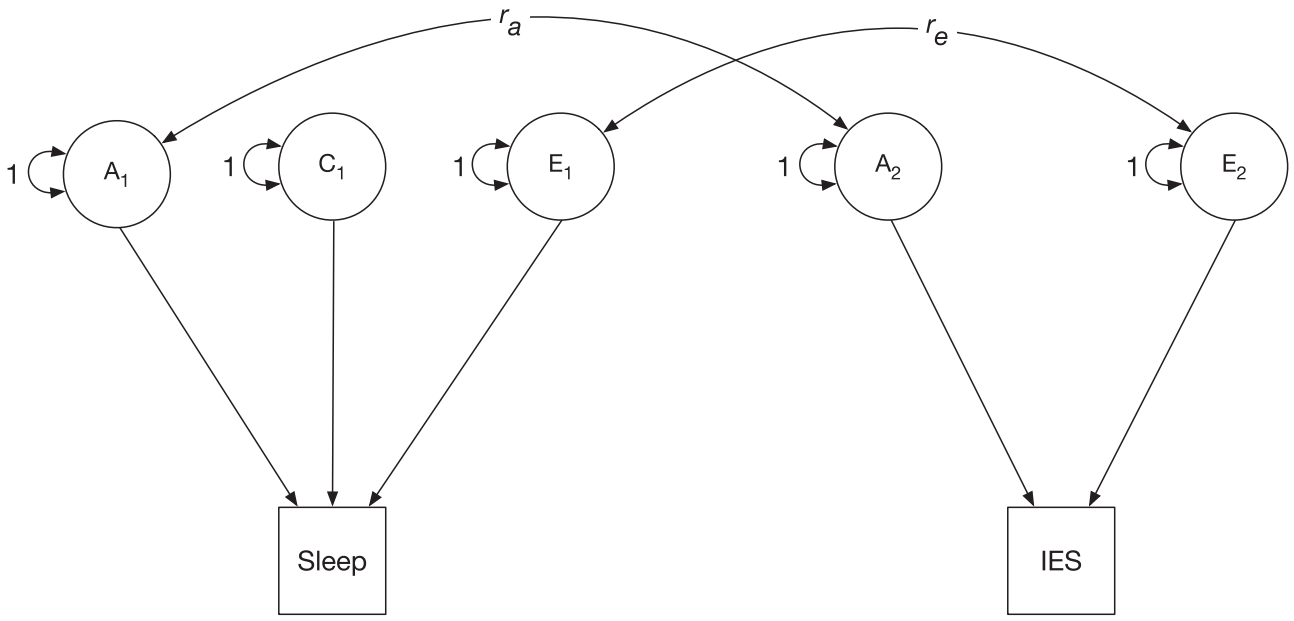


**Figure 2.** Correlated factor model between sleep duration and IES score. Only one twin is shown here for simplicity. The variance of sleep duration is decomposed into three components: additive genetic influences ($A_1$), environmental influences that are shared between twins ($C_1$), and non-shared environmental influences that are unique to each twin ($E_1$). The variance of IES is decomposed into additive genetic influences ($A_2$), and non-shared environmental influences ($E_2$). $C_2$ is not shown because 0% of the IES variance was attributable to shared environment. $r_a$ refers to the correlation between the two A components, and $r_e$ refers to the association between the two E components.

expression estimates the extent to which the A variance component of sleep ($A_1$) is moderated by IES score. A positive $b_{1A1}$ parameter reflects that the amount of sleep duration variance attributable to additive genetic variance (i.e., the A variance) increases as IES score increases, whereas a negative $b_{1A1}$ parameter reflects that the amount of sleep duration variance attributable to additive genetic variance decreases as IES score increases.

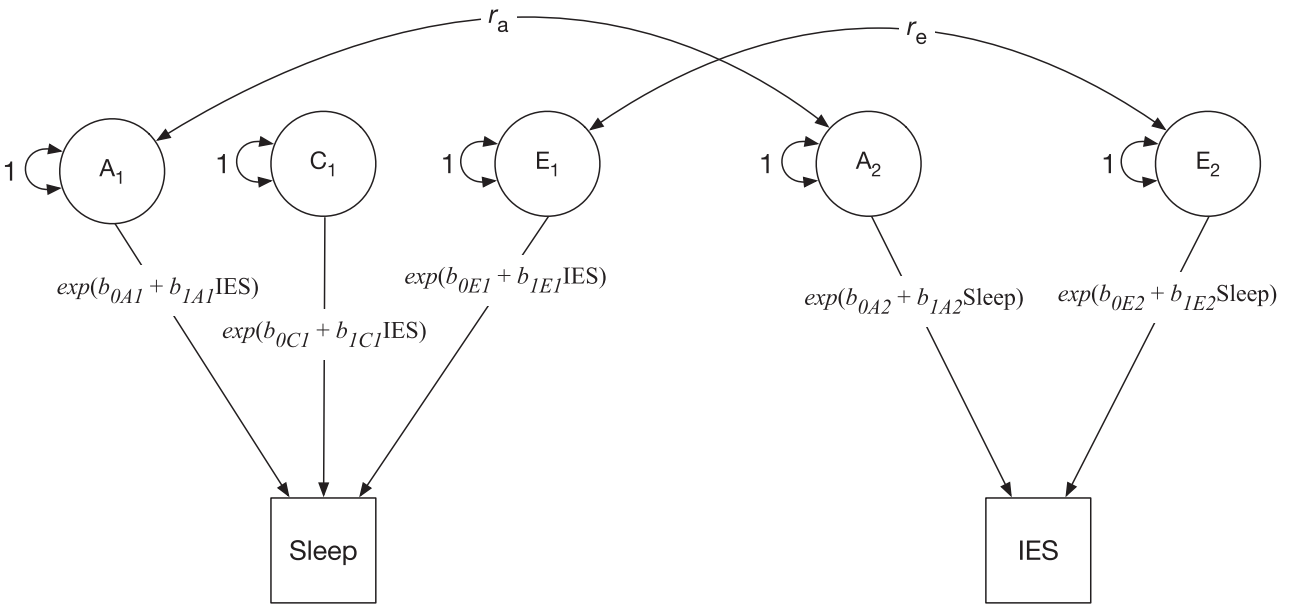


**Figure 3.** Moderated correlated factor model between sleep duration and IES score. Only one twin is shown here for simplicity. As with the basic correlated factor model, the variance of sleep duration and IES score are decomposed into ACE components. $r_a$ refers to the correlation between the two A components, and $r_e$ refers to the correlation between the two E components. Each of the ACE components of sleep duration is a linear function of IES score, and each of the AE components of IES is a linear function of sleep duration. The exponential function is used to ensure a positive value for the estimated A, C, and E variance components.

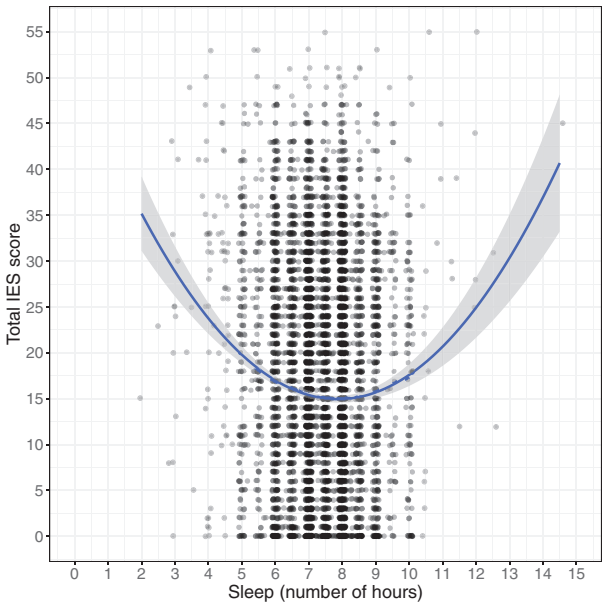


**Figure 4.** Nonlinear relationship between sleep duration and total IES score. The association between sleep duration and IES score was nonlinear: IES = 51–9.2 sleep + 0.6 sleep$^2$, using a quadratic equation. Shaded areas denote ±1 standard error.

## Results

The mean sleep duration for all participants was 7.35 hours (*SD* = 1.10). For all twins, the mean IES score was 15.87 (*SD* = 13.01), which corresponds to a moderate level of distress. As shown in Figure 4, the association between sleep duration and IES score was nonlinear, with higher IES scores associated with shorter and longer sleep durations. To ensure this pattern is not related to outlier values, the association was estimated separately for twins sleeping <7 hours, 7–8.9 hours, and ≥9 hours. For twins with sleep duration <7 hours, there was a significant negative association between sleep duration and IES score ($b_1 = -2.26$, $p > 0.001$), whereas for twins with sleep duration ≥9 hours there was a significant positive association between sleep duration and IES score ($b_1 = 3.52$, $p < 0.001$). There was no significant association between sleep duration and IES score among participants with sleep duration of 7–8.9 hours ($b_1 = -0.60$, $p = 0.117$). The results of these analyses support the finding of the U-shaped curve demonstrated by the quadratic analysis. Table 1 demonstrates the estimated coefficients for the quadratic association between sleep duration and IES score.

When analyzing associations between IES score and other factors known to be related to PTSD, we found a weak correlation between IES score and insomnia ($r = 0.23$, $p < 0.001$). There was essentially no correlation between IES score and alcohol use ($r = -0.04$, $p < 0.001$), with statistical significance felt not to be meaningful due to the sample size. There was a modest association between IES score and depression ($r = 0.37$, $p < 0.001$). The association between IES score and chronotype (morningness versus eveningness) was analyzed using analysis of variance with post hoc pair-wise comparisons showing consistently higher IES scores for "evening" types compared to "morning" types. (Table 2)

### Univariate twin model

Twin correlations and the standardized biometric variance components for sleep duration and IES are displayed in Table 3. For both sleep duration and IES score, most of the variance was attributable to non-shared environmental influences (E = 63% and 69% for sleep duration and IES score, respectively), with a smaller proportion attributable to additive genetics (A = 36% for sleep duration and 31% for IES score, representing the

**Table 1.** Components of quadratic association between sleep duration categories and total IES score

| Component | | Estimate | SE | 95% CI | t | P |
|---|---|---|---|---|---|---|
| Short sleep (<7 hours) | $b_0$ | 31.08 | 2.95 | 25.29 to 36.86 | 10.53 | <0.001 |
| | $b_1$ | −2.26 | 0.49 | −3.22 to −1.29 | −4.57 | <0.001 |
| Average sleep (7–8.9 hours) | $b_0$ | 19.54 | 2.90 | 13.85 to 25.24 | 6.73 | <0.001 |
| | $b_1$ | −0.60 | 0.38 | −1.34 to 0.15 | −1.57 | 0.117 |
| Long sleep (≥9 hours) | $b_0$ | −16.20 | 8.49 | −32.84 to 0.44 | −1.91 | 0.057 |
| | $b_1$ | 3.52 | 0.90 | 1.75 to 5.29 | 3.90 | <0.001 |

Estimated coefficients for the quadratic association between sleep duration and total IES score, where $b_0$ is the intercept and $b_1$ is the linear slope. SE = standard error; CI = confidence interval.

**Table 2.** Post hoc analysis of IES score differences by chronotype

| Type | | Mean difference | 95% CI | Adjusted P |
|---|---|---|---|---|
| More evening than morning | Evening | −0.71 | −2.02 to 0.59 | 0.496 |
| More morning than evening | Evening | −1.76 | −3.07 to −0.45 | 0.003 |
| Morning | Evening | −2.44 | −3.82 to −1.07 | < 0.001 |
| More morning than evening | More evening than morning | −1.05 | −2.11 to 0.01 | 0.055 |
| Morning | More evening than morning | −1.73 | −2.87 to −0.59 | < 0.001 |
| Morning | More morning than evening | −0.68 | −1.83 to 0.46 | 0.419 |

Chronotype was assessed with a questionnaire item asking, "One hears about 'morning' and 'evening' types of people. Which one of these types do you consider yourself to be?" Potential answers were on a spectrum from "morningness" to "eveningness." The table shows post hoc comparisons of IES scores for each chronotype response. IES scores were higher for the evening chronotypes, reaching statistical significance between chronotype responses that were the furthest apart on the morningness to eveningness spectrum. CI = confidence interval.

**Table 3.** Twin correlations and standardized variance components for sleep duration and total IES score

| | rMZ | | rDZ | | A | | C | | E | |
|---|---|---|---|---|---|---|---|---|---|---|
| Variable | Est (SE) | 95% CI | Est (SE) | 95% CI | Est (SE) | 95% CI | Est (SE) | 95% CI | Est (SE) | 95% CI |
| Sleep duration (hour) | 0.37 (0.02) | 0.33 to 0.41 | 0.19 (0.03) | 0.13 to 0.25 | 0.36 (0.08) | 0.20 to 0.52 | 0.01 (0.07) | 0 to 0.15 | 0.63 (0.02) | 0.59 to 0.67 |
| Total IES score | 0.31 (0.02) | 0.27 to 0.35 | 0.16 (0.01) | 0.14 to 0.18 | 0.31 (0.02) | 0.27 to 0.35 | 0 (0.001) | 0 to 0.002 | 0.69 (0.02) | 0.65 to 0.73 |

rMZ = twin correlation between MZ twins; rDZ = twin correlation between DZ twins; A = additive genetic component; C = shared environmental component; E = non-shared environmental component; Est = estimate; SE = standard error; CI = confidence interval.

heritability of each phenotype), and only 1% and 0% attributable to shared environmental influences (C) for sleep duration and IES score, respectively.

## Correlated factor model

The correlated factor model showed no statistically significant correlation between the A variance components for sleep duration and IES score ($r_a$ = −0.05, 95% CI = −0.15 to 0.05; $p$ = 0.339). There was a weak, negative association between the E variance components for sleep and IES ($r_e$ = −0.08, 95% CI = −0.12 to −0.04; $p$ = 0.001), indicating that the greater the variance of the unique environmental component for sleep duration, the smaller the variance for the unique environmental component of the IES score.

## Moderated correlated factor model

We next fitted a moderated correlated factor model to analyze the ACE variance components of sleep duration as a function of IES score, and the AE variance components of IES score by sleep duration. (Figure 3) Table 4 shows that the C and E variance components of sleep duration were moderated by IES score ($b_{1c1}$ = 2.91 and $b_{1E1}$ = 0.18; both $p$ < 0.001). This means the amount of sleep variance attributable to shared environment (C) increased 2.91 *SD* units for each additional unit of IES (as IES score increased),

**Table 4.** Parameter estimates for the moderated correlated factor model of sleep duration and IES score

| Parameter | Estimate | SE | 95% CI | P |
|---|---|---|---|---|
| Variance of sleep duration moderated by IES | | | | |
| $b_{0A1}$ | −1.05 | 0.07 | −1.19 to −0.92 | <0.001 |
| $b_{1A1}$ | 0.00 | 0.09 | −0.17 to 0.17 | 0.97 |
| $b_{0C1}$ | −7.20 | 1.99 | −11.10 to −3.30 | <0.001 |
| $b_{1C1}$ | 2.91 | 0.78 | 1.40 to 4.43 | <0.001 |
| $b_{0E1}$ | −0.49 | 0.03 | −0.56 to −0.43 | <0.001 |
| $b_{1E1}$ | 0.18 | 0.04 | 0.10 to 0.27 | <0.001 |
| Variance of IES moderated by sleep duration | | | | |
| $b_{0A2}$ | −1.20 | 0.08 | −1.36 to −1.04 | <0.001 |
| $b_{1A2}$ | −0.23 | 0.12 | −0.45 to 0.00 | 0.048 |
| $b_{0E2}$ | −0.37 | 0.03 | −0.43 to −0.31 | <0.001 |
| $b_{1E2}$ | 0.04 | 0.04 | −0.04 to 0.13 | 0.32 |

The $b_0$ terms are the values of the ACE variances where IES score or sleep duration is zero, and the $b_1$ terms represent the rate of increase or decrease for a given variance component (A, C, or E) as a function of the moderator (IES score or sleep duration). SE = standard error; CI = confidence interval.

and the amount of sleep variance attributable to non-shared environment (E) increased 0.18 *SD* units for each additional unit of IES (as IES score increased). The A variance component of sleep duration was not moderated by IES scores ($b_{1A1}$ = 0, $p$ = 0.97). These results are illustrated in the stacked variance plots in Figure 5,

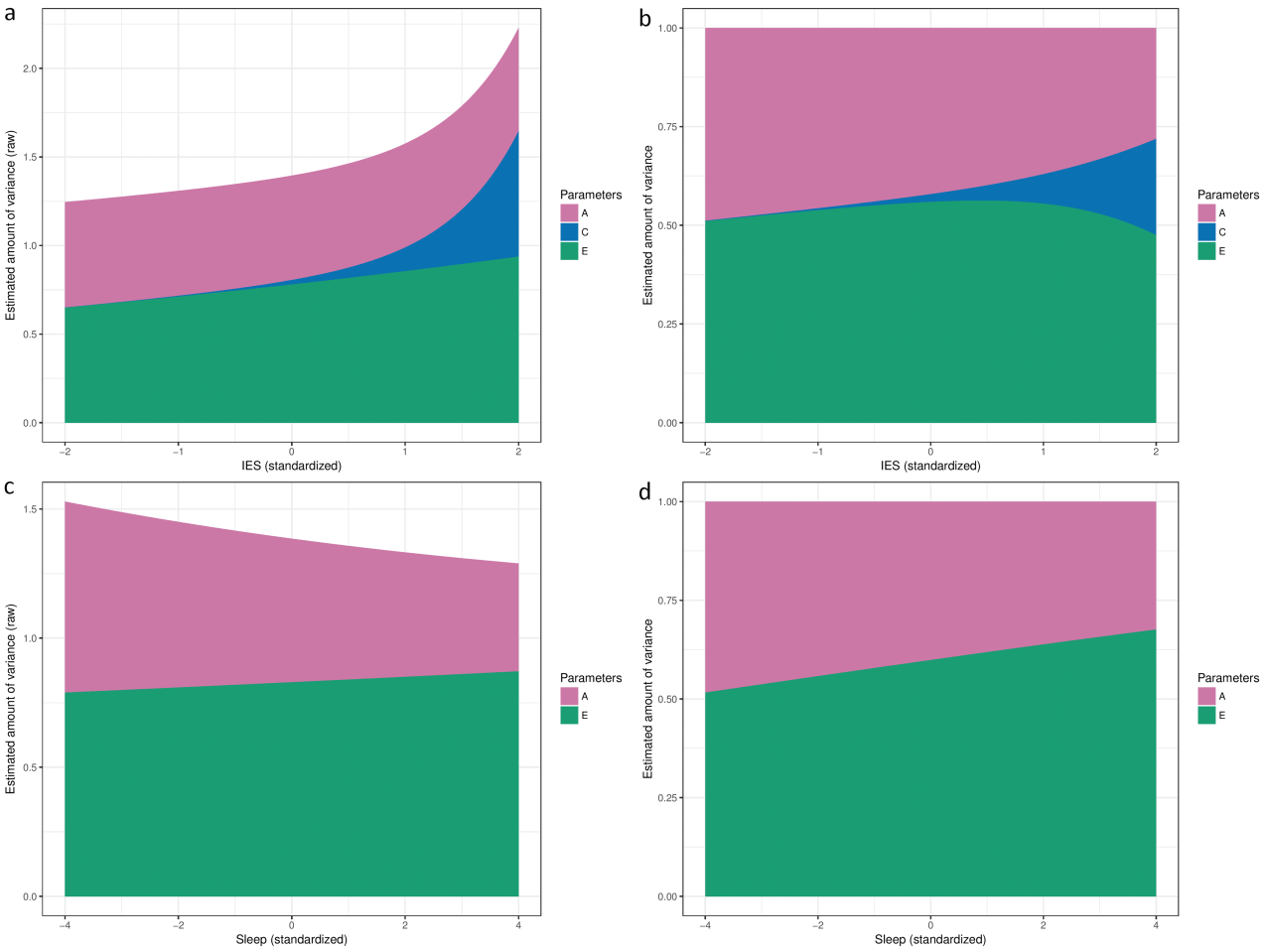


**Figure 5.** Estimated variance of sleep duration and IES score. (**a**) Estimated amount of variance (raw) in sleep duration that is attributable to additive genetic (A), shared environmental (C), and non-shared environmental factors (E) as a function of IES score. The total amount of variance in sleep increases as the total IES score increases. At high IES scores (at least one *SD* above the mean), the amount of variance in sleep attributable to C increases dramatically. (**b**) Estimated amount of variance in sleep duration, displayed as proportions of the total variance. (**c**) Estimated amount of raw variance in IES score that is attributable to A and E components as a function of sleep duration. The amount of IES variance attributable to the A component was significantly moderated by sleep duration, a finding not replicated with the E component. Thus, the total amount of variance in IES increases as sleep duration decreases. The C component was fixed to 0, as 0% of the IES variance was attributable to shared environmental influences. (**d**) Estimated amount of variance in IES score, displayed as proportions of the total variance.

a and b, which shows the shared (C) and non-shared environmental (E) variance of sleep increased as a function of increasing IES score. It is also notable that the total amount of variance in sleep duration increased as a function of IES score.

The A variance component of IES was moderated by sleep duration with marginal significance ($b_{1A2} = -0.23$, $p = 0.048$). The E variance component of IES was not moderated by sleep duration ($b_{1E2} = 0.04$, $p = 0.32$). As illustrated in the stacked variance plots (Figure 5, c and d), there was a slight decrease in the additive genetic (A) variance of IES as sleep duration increased, whereas there was minimal change in the non-shared environment (E) variance of IES score across sleep duration.

We further illustrate the moderating effect of IES score on the ACE variance components of sleep duration by estimating the standardized variance components of sleep duration in pairs in which both twins had lower IES scores (≤27, indicating low or moderate distress), when one twin had an IES score in the highest tercile (>27, indicating high levels of distress) but the other did not, and when both twins had high IES scores. Table 5 shows that when both twins had low IES scores, 60% of the variance in sleep duration is attributed to E, and 40% is attributed to A. Similarly, when both twins had high IES scores, 56% of the variance is attributed to E and 44% is attributed to A. When IES scores were discordant between twins, 73% of the variance in sleep duration is attributed to E, 25% is attributed to C, and 2% is attributed to A, indicating increased variance in sleep duration is predominantly attributable to both shared and unique environmental influences.

## Discussion

Our results demonstrate a complex relationship between genetic and environmental influences on sleep duration and PTSD symptoms in twins. We found shorter and longer habitual sleep duration is associated with significantly higher PTSD symptom scores, illustrating a nonlinear relationship between sleep duration and PTSD symptoms. Numerous studies demonstrate greater occurrence of adverse health conditions with chronic short sleep duration (typically under 6 hours), and to a lesser extent, chronic long sleep duration (more than 9 hours) [25, 34–37, 39, 40, 55, 56]. In particular, our finding is consistent

**Table 5.** Twin correlations and standardized variance components for sleep duration by IES score cutoff

| | | rMZ | | rDZ | | A | | C | | E | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| IES scores | N | Est (SE) | 95% CI | Est (SE) | 95% CI | Est (SE) | 95% CI | Est (SE) | 95% CI | Est (SE) | 95% CI |
| Both twin IES ≤ 27 | 1540 | 0.40 (.03) | 0.34 to 0.46 | 0.20 (.01) | 0.18 to 0.22 | 0.40 (.03) | 0.34 to 0.46 | 0 | — | 0.60 (.03) | 0.54 to 0.66 |
| One twin IES ≤ 27, the other twin IES > 27 | 1430 | 0.27 (.04) | 0.19 to 0.35 | 0.26 (.06) | 0.14 to 0.38 | 0.02 (.14) | –0.25 to 0.29 | 0.25 (.12) | 0.015 to 0.49 | 0.73 (.04) | 0.65 to 0.81 |
| Both twins IES >27 | 435 | 0.44 (.06) | 0.32 to 0.56 | 0.22 (.03) | 0.17 to 0.28 | 0.44 (.06) | 0.32 to 0.56 | 0 | — | 0.56 (.06) | 0.44 to 0.68 |

rMZ = twin correlation between MZ twins; rDZ = twin correlation between DZ twins; A = additive genetic component; C = shared environmental component; E = non-shared environmental component; N = number of twin pairs; Est = estimate; SE = standard error; CI = confidence interval.

with prior research in veterans showing short and long sleep duration are associated with significantly higher risk for PTSD symptoms when compared to veterans with average sleep duration (7–9 hours) [24, 25, 30]. This helps elucidate the increased risk association between chronic short sleep, long sleep, and post-traumatic stress and demonstrates healthy sleep duration is linked with fewer PTSD symptoms following trauma exposure.

Analyses of other variables associated with PTSD showed positive correlations between IES score and insomnia and depression, as would be expected [1]. Contrary to the high known prevalence of alcohol and substance use associated with PTSD, there was no association between IES score and alcohol use in this sample. We found that subjects with a greater self-reported "evening" chronotype had higher IES scores than those with self-reported "morning" chronotype. An eveningness or "night owl" chronotype is associated with mood disorders, anxiety disorders, and substance abuse [57–59], as well as greater lifetime PTSD symptoms, more sleep disturbance, and more frequent and severe nightmares [58, 60–62]. Chronodisruption has been implicated to play a role in stress vulnerability and PTSD, possibly linked to circadian rhythm abnormalities such as the blunting of cortisol, melatonin, and core body temperature cycles [63–65].

Our twin analyses showed intrapair twin correlations for PTSD symptoms and sleep duration similar to prior studies in both veteran and civilian populations [14, 15, 66]. We found a moderate genetic influence (e.g., heritability) for sleep duration (36%) and IES score (31%), and a larger non-shared environmental influence on sleep duration (63%) and IES score (69%). Our findings on heritability of sleep duration are consistent with prior studies in this sample [66, 67]. The results of our univariate analysis of PTSD symptoms are most consistent with other major twin studies using majority female (65%–77%) community samples in Canada and Australia, showing moderate additive genetic and larger nonshared environmental influences on PTSD symptoms [15, 16]. The heritability estimate from our study was higher than in twin studies using the male US Vietnam Era Twin Registry (23.5%) [14] and lower than that of a community sample of female twins (71%) [17], supporting the hypothesis that heritability of PTSD symptoms may be higher in women [18].

Our moderated correlated factor model found the shared and non-shared environmental components of sleep duration were moderated by the IES score, but the genetic component was not. Furthermore, the amount of variance in sleep duration attributable to the shared environmental component increased dramatically as the reported PSTD symptom levels increased. This suggests at high levels of reported PTSD symptoms, the extent to which one twin's sleep duration can be predicted by their co-twin's sleep duration increases, reflecting an increase in the predictability of sleep in both MZ and DZ twins. Shared environmental influences include all exposures that are common to both twins, and may include in utero exposures, birth history, childhood living conditions, location, and diet. A greater contribution of shared environmental exposures to the predictability of sleep duration in the setting of more severe PTSD symptoms suggests childhood experiences affect variability in sleep duration later in life in trauma-exposed individuals. A large body of literature has demonstrated the influence of early childhood experiences on the functional expression of genes modulating stress response. Epigenetic changes occurring from trauma exposure during early life have been shown to cause dysregulation of the hippocampal–pituitary–adrenal axis [8]. An aberrant stress response may produce long-term alterations in typical sleep duration. Increased variance in sleep duration indicates more individuals sleeping outside the normal range, with possible associated adverse health consequences.

Finally, there is debate regarding whether sleep problems in PTSD occur as a result of PTSD symptoms, or contribute to its development. Prior twin studies suggest some associated features of PTSD, such as reduced hippocampal volume and cognitive impairments, are pre-existing risk factors rather than consequences of PTSD. Other associated features, such as psychophysiological responses, pain levels, and brain metabolism, appear to be altered only after trauma exposure [31]. Although sleep derangements occurring both before and after trauma exposure are known to be associated with the development of PTSD, it is not well understood whether sleep problems are primarily a risk factor or consequence of PTSD, or both. Our analyses found the amount of PTSD symptom variance attributable to genetic factors, but not unique environmental factors, was moderated by sleep duration. This suggests that genetic factors predisposing for pathologic response to stress are affected by sleep duration. Overall, there was greater moderating effect of PTSD symptoms on sleep duration variance than vice versa, with early life factors playing a larger role than genetic or unique environmental influences. Yet ongoing sleep problems triggered by the development of a trauma response may further exacerbate the development or worsening of PTSD symptoms in genetically susceptible individuals. Furthermore, numerous studies have implicated fragmentation of sleep, particularly REM sleep, in the pathophysiology of PTSD [28, 68]. It is feasible that qualitative problems with sleep quality or fragmentation occurring before or during PTSD development lead to subsequent changes in sleep duration. Our study results suggest that a genetic predisposition to pathologic response to stress could be reduced by extending sleep in individuals with chronic short sleep duration.

Limitations of this study include the self-report nature of the sleep and PTSD symptom data. Twins may not have accurately reported sleep duration or included time in bed or naps as part

of their total sleep duration. Self-reported sleep duration approximates objective measures of sleep length [69, 70], although some studies suggest it may be biased by overestimation [71]. In addition, the time frame for IES symptoms is specified to be the past 7 days whereas the sleep duration does not specify a time frame, thus it may only be assumed that the two measures occur over roughly the same time frame. Other limitations include a lack of longitudinal data regarding when trauma occurred and changes in sleep duration before or after trauma. It is unknown to what extent other sleep issues, such as sleep disorders or poor sleep quality, contribute to the relationship between sleep and PTSD symptoms. In addition, this study was constrained by the necessity of using abridged instruments in the Enrollment Survey, including the IES. Research on traumatic stress reactions has frequently used the IES for a variety of populations and types of trauma; however, only a smaller number of studies have used this abridged version, although validation analyses were completed showing reliability and internal validity [44, 49, 50]. The IES notably omits hyperarousal symptoms listed as criteria in the *DSM*, which may be linked to sleep disturbances. As with all twin studies, assumptions are made regarding the correlations of shared and unique environmental variance components occurring with twins raised together. Finally, our sample was predominantly younger adult Caucasian women, and thus caution should be used when applying these results to the general population.

In conclusion, we have shown that there is a nonlinear association between sleep duration and PTSD symptoms, such that greater PTSD symptoms are associated with shorter and longer sleep duration. Twin models additionally reveal that higher severity of PTSD symptoms appears to influence variability in sleep duration primarily through shared and nonshared environmental influences, and shorter sleep duration influences variability in IES through additive genetic influences. The mechanism by which this occurs may involve pathology occurring as a result of childhood exposures that leads to adverse adult sleep habits, and possibly increased risk of pathologic responses to stressful experiences. Further, short sleep may activate genetic pathways predisposing to PTSD symptoms in trauma-exposed individuals. Additional work assessing longitudinal changes of these influences may yield further insight into the complex relationship between sleep and post-traumatic stress.

*Conflict of interest statement*. None declared.

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