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ORIGINAL ARTICLE

The natural history of insomnia: high sleep reactivity interacts with greater life stress to predict the onset of acute insomnia

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Abstract

Study Objectives: Prior research suggests that some individuals have a predisposition to experience insomnia following acute stressors (i.e. sleep reactivity). The present study was a proof of concept and specifically aimed to provide additional empirical evidence that the link between stressful life events and the onset of acute insomnia is moderated by sleep reactivity.

Methods: About 1,225 adults with a history of good sleep (M_{age} = 53.2 years, 68% female, 83% white) were recruited nationwide for an online study on sleep health. Participants completed surveys to assess sleep reactivity (baseline), sleep patterns (daily sleep diary), and stressful life events (weekly survey). All daily and weekly measures were completed for a one-year period. Sleep diary data were used to identify sleep initiation/maintenance difficulties, including whether they met criteria for acute insomnia at any point during the one-year interval.

Results: Participants with high sleep reactivity compared to low sleep reactivity were at 76% increased odds of developing acute insomnia during the one-year interval. In general, greater weekly stressful life events were associated with greater insomnia during the subsequent week. Those participants with high sleep reactivity demonstrated a stronger relationship between weekly stressful life events and insomnia, such that they reported the greatest levels of insomnia following weeks where they experienced a greater number of stressful life events.

Conclusions: These results further support the sleep reactivity model of insomnia, and specifically, provide evidence that sleep reactivity predicts the incidence of acute insomnia in a sample of participants with no history of insomnia.

Statement of Significance

The current study used high-density data (daily/weekly measures) to track stressful life events and sleep, which allowed for a close examination of whether sleep reactivity predicts acute insomnia and how the association between stressful life events and sleep disturbance varies as a function of sleep reactivity. Due to the high variability of stressful life events and sleep over time, frequent data points were needed to test this etiological model of insomnia, and provide a proof of concept for sleep reactivity. Our findings have clinical utility for establishing sleep reactivity as a potential target for prevention/intervention efforts for insomnia. Future studies could examine the effectiveness of minimizing the impact of stressful life events on sleep in individuals with high sleep reactivity.

Key words: sleep reactivity; acute insomnia; stress

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Introduction

Insomnia is a highly prevalent disorder with significant public health and economic consequences. In fact, nearly 50% of adults in the US experience insomnia at some point each year [1–4]. In addition, insomnia is associated with an increased risk for numerous chronic disease outcomes [5], including cardiovascular disease [6], cancer [7, 8], and metabolic dysregulation [9]. Insomnia's economic consequences are often calculated in terms of direct and indirect costs, with a larger proportion of the economic burden being attributed to indirect costs, particularly absenteeism (i.e. habitual pattern of unplanned absences from work) and presenteeism (i.e. loss of productivity when employees are not fully functioning in the workplace) [10]. Based on data collected from workers in the United States, annual losses in work performance due to insomnia amount to nearly $91.7 billion [11]. These burdensome consequences highlight the need for prevention and intervention efforts that are informed by a better understanding of the factors that predispose and precipitate individuals to developing insomnia.

According to Spielman's three-factor (or 3P) model of insomnia, the most common precipitating factors or triggering events for insomnia are acute stressors. That said, the likelihood or extent to which someone will experience a sleepless night following a stressful life event varies by several factors, such as the nature or severity of the stressor. The impact of stressful life events on sleep also varies based on several individual factors, also known as predisposing factors. Among other things, these factors include a tendency toward cognitive or physiological hyperarousal at night or in response to stress. This phenomenon is also known as sleep reactivity, or a vulnerability to stress-related sleep disturbance in response to real or perceived threat [12]. Individuals with high sleep reactivity, regardless of whether they have a current or past history of insomnia, are more likely to experience difficulties with initiating or maintaining sleep following a real or anticipated acute stressor (e.g. marital conflict or work-related acute stressors), relative to those with low sleep reactivity [13–15]. These sleep reactive tendencies are considered stable over time and across different acute stressors (e.g. job loss, death of a loved one, first night in a laboratory) [12]. Therefore, according to this diathesis-stress model of insomnia, the combination of high sleep reactivity and a sufficiently stressful event can potentially lead to more persistent insomnia symptoms and even Insomnia Disorder [12, 13, 16, 17].

Multiple studies have now demonstrated, using longitudinal designs, that greater sleep reactivity predicts new onset chronic insomnia or Insomnia Disorder (i.e. insomnia symptoms occurring at least 3 nights per week for 3 or more months). This literature, however, has yet to definitively answer two questions: (1) does sleep reactivity predict *acute* episodes of insomnia (i.e., symptoms present for less than 3 months) and (2) does sleep reactivity moderate the association between stressful life events and insomnia symptoms? The latter question provides a proof-of-concept for the sleep reactivity hypothesis of insomnia. The primary issue is that past studies have not had the temporal resolution to assess whether individuals with greater sleep reactivity are more likely to experience acute bouts of insomnia and whether those insomnia symptoms occur following an acute stressor. To do so, relatively high-density data is needed to track stressful life events and insomnia symptoms over time—e.g. at least weekly/monthly changes in stressful life events and sleep. Although a previous study found that sleep reactivity moderated the effects of stress on insomnia disorder [12], the study measured stressful life events and sleep reactivity annually. The conclusions that can be drawn from these data regarding acute sleep continuity disturbance are limited given that both stressful life events and sleep are highly variable over time [18, 19]. To address this limitation, the current study used a sampling strategy that allowed for a closer examination of whether sleep reactivity predicts a greater incidence of acute insomnia and how the week-to-week association between stressful life events and insomnia varies as a function of sleep reactivity. We hypothesized that individuals with high sleep reactivity will (1) report a greater incidence of acute insomnia during a one-year interval, and (2) be more likely to experience insomnia symptoms following a week with a greater number of self-reported stressful life events.

Methods

Participants and procedure

This study was part of a larger parent study investigating the incidence rates of acute and chronic insomnia in a sample of *initially* good sleepers [20]. Two nationwide platforms (Zogby Analytics and ResearchMatch) were used to recruit participants over three recruitment intervals, separated by approximately 1 year. The final sample included 1,225 participants (M_{age} = 53.2 years, range = 27–89 years). The present sample was primarily female (68%, n = 831) and white (83%, M = 1,019). The study was conducted in two phases, described below. Please also refer to the original paper [20] for additional details regarding the sample.

Phase-1. A preliminary screener survey was administered through Zogby (an international polling agency) and ResearchMatch to identify participants without sleep disorders or a history of insomnia. Specifically, study candidates responded "yes" to the following questions: "Are you a good sleeper? That is, do you reliably (5 or more nights per week) take less than 15 min to fall asleep and are awake during the night for less than 15 min? Has this been true for you for at least the last 6 months?". These screening criteria were conservative to increase the likelihood that only persistently good sleepers were recruited. No other inclusion or exclusion criteria were applied. Potential participants were then referred to the study website where they (1) reviewed HIPPA forms and provided their informed consent, (2) completed an intake survey (profiling sleep, health, and mental health status and history), and (3) completed 2 weeks of online sleep diaries (baseline assessment) to confirm their presentation as good sleepers.

Phase-2. Participants whose baseline sleep diary assessment reflected good sleep (i.e. SL ≤ 15 min and WASO ≤ 15 min, ≥ 5 nights per week) were eligible for Phase-2 and were subsequently assessed for one year via an online study website. This site included questionnaires that assessed: daily morning and evening sleep diaries, weekly and bi-weekly instruments (e.g. medical symptoms checklist, stressful life events), and monthly instruments. Participants were removed from the study for non-compliance if their completion of online sleep diaries dropped below four

completed diaries per week across 2 weeks at any point during the study. Noncompliance was monitored daily by study staff and manually disenrolled from the study if they met criteria for noncompliance.

Subject attrition. A total of 3,287 participants were positively screened for good sleep and entered Phase-1 (consent, baseline questionnaires and two weeks of daily sleep diaries). 85 participants (2.6%) were disenrolled due to noncompliance during the 2-week baseline assessment (daily diaries). Of the remaining 3,202 participants that completed the baseline assessment and entered Phase-2 of the study, 1,954 participants (59.4%) were excluded from our final analyses. Of the 1,954 participants that were excluded in Phase-2, 926 participants were excluded for not meeting the 60% adherence threshold and 1,028 participants were excluded for meeting criteria for AI (i.e. they did not enter the study as good sleepers). A total of 1,246 participants (38.0%) entered and completed Phase-2. Of those, 21 participants did not have sleep reactivity (i.e. FIRST; Ford Insomnia Response to Stress Test) data and were therefore excluded from the current analyses. The final sample consisted of 1,225 participants.

Insomnia status. As reported previously [20], the one-year incidence of new-onset acute insomnia (AI) in this sample was 27.0% (n = 337). Each participant's sleep diary data were used to identify instances of sleep initiation and/or maintenance difficulties. Acute insomnia was defined as two consecutive weeks with a frequency of ≥ 3 nights per week of sleep initiation and/or maintenance complaints (sleep latency [SL] ≥ 30 min and/or wake after sleep onset [WASO] ≥ 30 min and/or early morning awakenings [EMA] ≥ 30 min) [21–23]. Of those that met criteria for AI, 72.4% (n = 244) of participants recovered good sleep (AI-REC), and 6.8% (n = 23) developed chronic insomnia (CI). The definition for recovery were at least 7 out of 12 weeks of "good" sleep after an AI episode where the final 4 weeks in that period were designated as "good" sleep (i.e. did not meet criteria for AI). The definition for CI was 10 or more weeks in a 12-week period with the same frequency and severity criteria as AI. Notably, 19.3% (n = 65) neither recovered nor went on to develop CI. This group exhibited what might be best referred to as persistent poor sleep (PPS; problems with sleep initiation or maintenance [SL or WASO or EMA > 30 min] that did not meet or exceed frequency (3 or more days per week) or chronicity criteria (3 or more months in duration).

Measures

Sleep reactivity. The Ford Insomnia Response to Stress Test (FIRST [24]) is a 9-item questionnaire used to assess vulnerability to stress-related sleep disturbance (i.e. sleep reactivity). Participants completed the FIRST once during the baseline assessment (i.e. start of Phase 2). The FIRST measures the probability that a person would be to have difficulty sleeping following (or in anticipation of) a stressful situation. It asks how likely (1 = "not likely"; 2 = "somewhat likely"; 3 = "moderately likely"; 4 = "very likely") the participant would have difficulty sleeping under nine stressful situations (e.g. before an important meeting the next day, after getting bad news during the day, after an argument, and before having to speak in public). Participants were asked to rate the likelihood even if they had not experienced the situation recently. The FIRST has been widely used in previous sleep research [12, 15, 25, 26], and has demonstrated good psychometric properties. In the present study, the measure demonstrated good internal consistency (Cronbach's alpha = 0.88). The total score was equal to the sum of the nine items, with higher scores suggesting greater sleep reactivity. Clinical cut-offs for the FIRST were also recently proposed [27]. Based on these recommendations, the present study used a cut-off score of 16 or greater to define "high" sleep reactivity.

Daily sleep patterns. Sleep patterns were assessed via an online daily sleep diary. Items included in the online sleep diary were based on the Consensus Sleep Diary [28]. The diary was used to measure daily fluctuations in sleep latency (SL), wake after sleep onset (WASO), early morning awakenings (EMA), nocturnal awakenings (NWAK), time in bed (TIB), and total sleep time (TST). SL measured time, in minutes and hours, to initial sleep onset (i.e. "How long did it take you to fall asleep?"). WASO measured how much time participants were "awake during the night". EMA values were reported in the sleep diaries as the number of hours or minutes that the participant spent awake in bed following their final awakening. Specifically, they were asked, "What time was your final awakening?", followed by "How long were you continuously awake before getting out of bed?". TIB is estimated as the difference between "What time did you get out of bed for the day?" and "What time did you try and go to sleep?". TST was assessed two ways: (1) subjective responses to "How much sleep did you get last night?" and (2) the difference between SL, WASO, and EMA from the reported TIB (compute TST = TIB – [SL + WASO + EMA]). Average weekly Total Wake Time (TWT) was also estimated using the sum of sleep latency, wake after sleep onset, and early morning awakenings (TWT = SL + WASO + EMA). Participants completed their diaries using a study-dedicated online portal. Participants received daily email reminders to complete their sleep diaries and email notification if they missed a diary entry. If a participant missed a diary, it would be recorded as a missed entry (i.e. there was no option to retrospectively complete diary entries).

Stressful life events. Stressful life events were measured using an updated version of the Social Readjustment Rating Scale [29]. Participants completed the measure on a weekly basis by identifying stressful life events that they had experienced within the previous week. Each of the 43 life events listed are coded with a value proportionate to the level of stress typically resulting from the event (e.g. "divorce" has a value of 73, whereas "taking a loan" has a value of 17). These values were estimated using the average distress score (0–100) from a convenience sample and validated in a representative sample [30]. Total scores represent the sum of the values for all events experienced by an individual within that week.

Statistical analyses

In the present study, we operationalized acute insomnia in two ways. For the first set of analyses, we were interested in the link between sleep reactivity and the incidence of acute insomnia. In this case, we operationalized acute insomnia using the "diagnostic" definition of the condition (i.e. greater than 30 min for at least 3 nights per week on any of the insomnia subtypes – SL,

WASO, or EMA). Specifically, we used moving 14-day windows (first window consisted of days 1–14, second window consisted of days 2–15, third window consisted of days 3–16, etc.) to determine whether a participant met criteria for acute insomnia. Logistic regression analyses were used to quantify whether sleep reactivity predicted incidence of acute insomnia (yes/no for any episode across the 1-year study period). In the second set of analyses (mixed effects models), we examined whether sleep reactivity was related to relative differences in week-to-week levels of insomnia. To maximize variability in weekly "sleeplessness" and considering that patients with insomnia may experience any combination of SL/WASO/EMA, we operationalized insomnia symptoms as TWT (or the sum of SL/WASO/EMA). TWT has been previously used as a method to estimate insomnia or sleep continuity disturbance [31–33]. A series of mixed-effects models (via SPSS MIXED 26.0) were used to assess whether stressful life events were associated with average TWT, and whether this association varied as a function of sleep reactivity scores. Due to a positively skewed distribution, weekly stress scores were log transformed and person mean-centered. Sleep reactivity scores were grand mean-centered to aide interpretation of the output. For the mixed-effects models, average weekly TWT was entered as the dependent variable. Weekly stress scores, sleep reactivity scores, and the two-way interaction between stressful life events and sleep reactivity were entered as fixed effects. The intercept was entered as a random effect to control for clustering in the data (i.e. within subject variability in TWT). Clinical cut-offs for sleep reactivity were recently proposed, with sleep reactivity scores ≥ 16 categorized as "high sleep reactivity." [34] To draw more clinically meaningful conclusions, the sleep reactivity scores were also analyzed as a categorical measure with FIRST scores greater than or equal to 16 coded as high sleep reactivity and scores below 16 coded as low first reactivity.

Results

Descriptive statistics

The mean FIRST score for participants in the sample was 16.7 (*SD* = 5.7), with 53% of the sample reporting "high sleep reactivity" (a score ≥ 16). High FIRST scores were greater among women (mean = 17.5) relative to men (mean = 14.9, r = 0.22, p < .001). Of the female participants in the sample, 59% of them reported a high FIRST score (compared to 39% among men). The means and standard deviations for all study variables are reported in Table 1. When comparing participants who scored high versus low on the sleep reactivity measure, participants with high FIRST scores reported greater TWT (high FIRST, median = 18.0 min, IQR = 19.8; low FIRST, median = 14.6, IQR = 17.7; p < .001).

Does sleep reactivity increase risk/incidence of acute insomnia?

When FIRST scores were treated as a continuous variable, results from the logistic regression supported that greater sleep

Table 1. Measures of central tendency, variance, frequency, and percentages for all study variables sorted by: total sample, participants with high sleep reactivity (as assessed by FIRST), and participants with low sleep reactivity; effect size (n^2) and corresponding statistical significance were also included.

	Total sample	High FIRST	Low FIRST		
	(*n* = 1,225)	(*n* = 643)	(*n* = 582)	*P*-value	n^2
Stressful life events, *Median (IQR)*	0 (13.0)	0.0 (20.0)	0.0 (0.0)	<.001	<0.001
TWT, *Median (IQR)*	16.4 (18.7)	18.2 (19.7)	14.7 (17.7)	<.001	0.012
AGE, *Mean (SD)*	53.2 (11.0)	52.8 (11.0)	53.8 (11.0)	.03	0.001
BMI, *Mean (SD)*	28.9 (7.4)	28.7 (7.5)	29.0 (7.3)	.50	<0.001
GENDER, *n* (%)				<.001	0.034
Female, *n* (%)	831 (67.8)	489 (76.2)	342 (58.9)		
Male, *n* (%)	392 (32.0)	153 (23.8)	239 (41.1)		
RACE, *n* (%)				.01	<0.001
American Indian/Alaskan Native	11 (0.9)	7 (1.1)	4 (0.7)		
Asian American	28 (2.3)	20 (3.1)	8 (1.4)		
Native Hawaiian/Pacific Islander	2 (0.2)	0.0 (0)	2 (0.3)		
Black	86 (7.0)	38 (5.9)	48 (8.2)		
White	1,019 (83.2)	538 (83.7)	481 (82.6)		
Multi-Racial	29 (2.4)	9 (1.4)	20 (3.4)		
Unknown	50 (4.1)	31 (4.8)	19 (3.4)		
HISPANIC, *n* (%)				.19	0.001
Non-Hispanic	1,171 (95.6)	610 (94.9)	561 (96.4)		
Hispanic	54 (4.4)	33 (5.1)	21 (3.6)		
Education, *n* (%)				.62	<0.001
HS or less	147 (12.0)	80 (12.5)	67 (11.5)		
More than HS	1076 (87.8)	562 (87.5)	514 (88.5)		
Annual income, *n* (%)				.76	<0.001
Less than $30K	269 (22.0)	139 (21.7)	130 (22.4)		
Greater than or equal to $30K	954 (77.9)	503 (78.3)	451 (77.6)		

Note. *IQR* and *SD* are used to represent interquartile ranges and standard deviation, respectively. Stressful Life Events = LES scores, TWT = total wake time, BMI = body mass index.

reactivity was associated with a greater likelihood of developing acute insomnia during the one-year interval (OR = 1.05, 95% CI = 1.02–1.07, $p < .001$). This effect remained significant even while controlling for gender (OR = 1.04, 95% CI = 1.02–1.06, $p = .001$). These data indicate that for every unit increase in FIRST, there was approximately 4% increased odds of developing acute insomnia. When FIRST scores were treated as a categorical variable and gender was entered as a covariate, results supported that the high sleep reactivity group, compared to the low sleep reactivity group, was at 76% greater odds of developing acute insomnia during the study period (OR = 1.76, 95% CI = 1.35–2.29, $p < .001$). In this sample, 20.6% of low FIRST participants developed acute insomnia and 32.8% of high FIRST participants developed acute insomnia. Another way to interpret this is that 63.7% of participants with acute insomnia had high FIRST scores (see Figure 1).

Does sleep reactivity moderate the link between weekly stressful life events and insomnia symptoms?

As expected, there was a significant main effect of weekly stressful life events on average weekly total wake time (TWT, $b = 1.49$, 95% CI = 1.30–1.69, $t = 14.8$, $p < .001$) in the full sample. Such that, greater stressful life events were related to greater overall insomnia symptoms. Next, we assessed whether the association between weekly stressful life events and TWT varied as a function of sleep reactivity. Results supported that FIRST scores significantly moderated the association between stressful life events and TWT, two-way interaction, $b = 0.08$, 95% CI = 0.05–0.12, $t = 4.50$, $p < .001$. Specifically, the impact of weekly stressful life events on TWT was greater at higher levels of FIRST (see Figure 2). Because stressful life events were person mean-centered, these data represent the effect of experiencing more stressful life events relative to each participant's mean level of stress (i.e. controls for within-subject differences in stress). When analyzing the FIRST data categorically, results were similar, the association between weekly stressful life events and TWT were significantly greater among participants who reported high sleep reactivity, two-way interaction, $b = 0.42$, 95% CI = 0.18–0.81, $t = 2.05$, $p = .04$. Specifically, among participants with high sleep reactivity the association between weekly LES and TWT was greater, $b = 1.72$, 95% CI = 1.44–2.00, $t = 11.99$, $p < .001$, relative to participants with low sleep reactivity, $b = 1.29$, 95% CI = 1.03–1.56, $t = 9.56$, $p < .001$. We also re-analyzed the data after person mean-centering the outcome variable (TWT). This output provides more information related to within-subject effects by assessing whether relative increases in stressful life events (from each participant's average levels of stress) are related to relative increases in TWT (from each participant's average levels of TWT). These data further confirmed that relative elevations in stressful life events were related to corresponding elevations in TWT and that this was particularly true for participants with greater sleep reactivity (continuous variable), two-way interaction, $b = 0.08$, 95% CI = 0.05–0.12, $t = 4.50$, $p < .001$ (Figure 3). Please see Table 2 for a summary of all the model estimates.

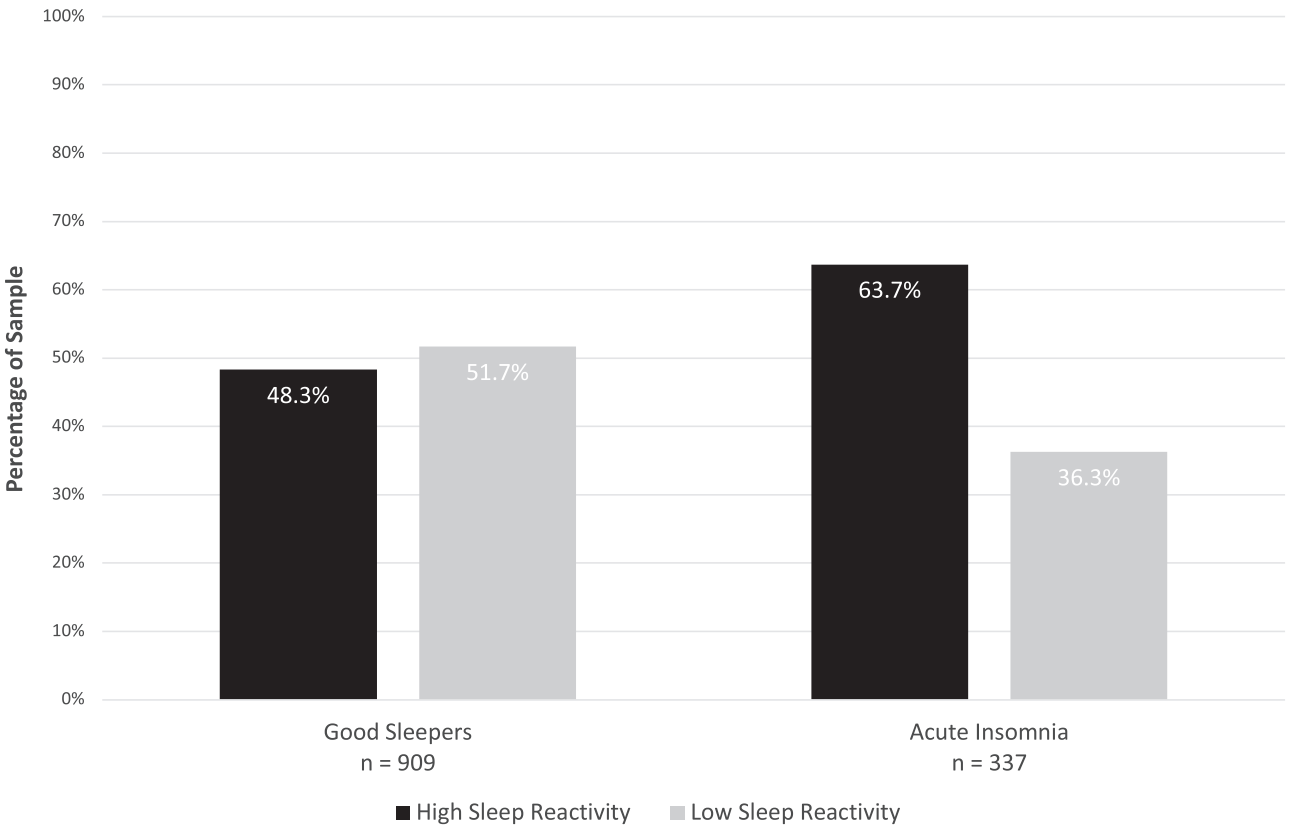

Figure 1. Differences between high and low sleep reactivity in participants who remained good sleepers throughout the one year of data collection versus participants who developed new onset acute insomnia during that time interval. High Sleep Reactivity represents FIRST scores ≥ 16, Low Sleep Reactivity represents FIRST scores < 16.

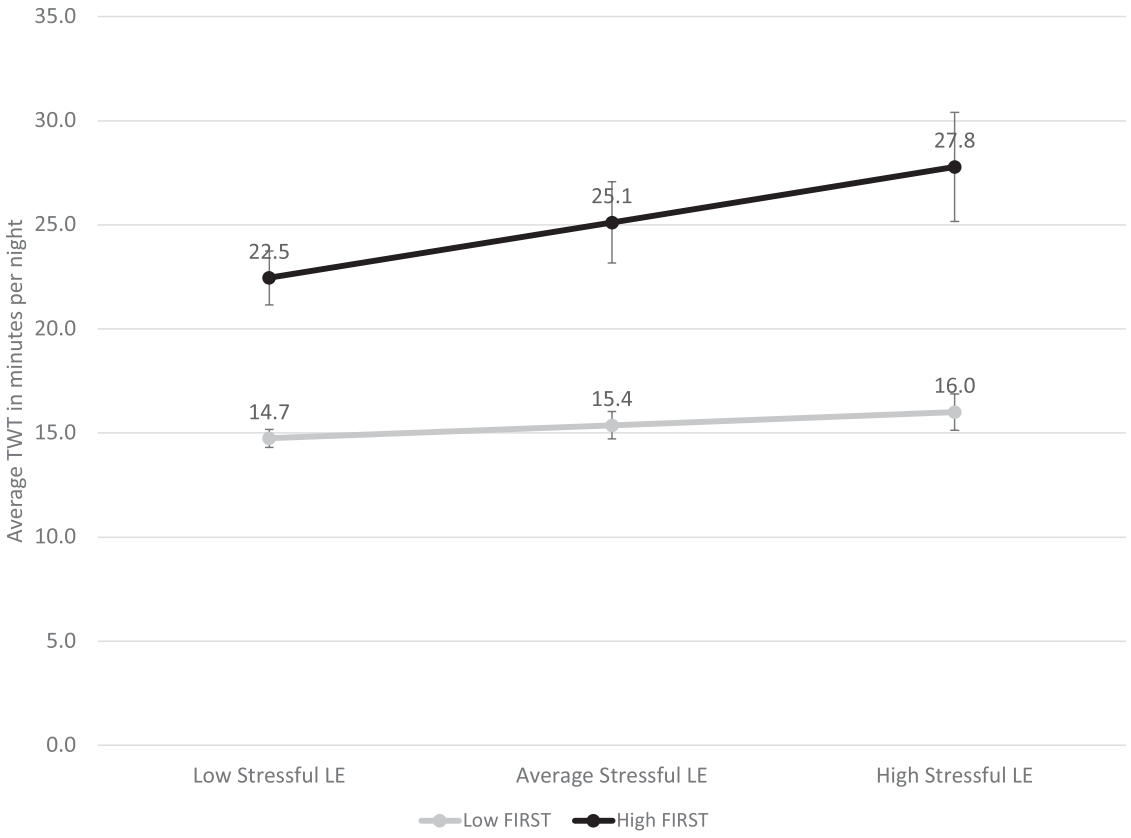

Figure 2. Model-based estimated from two-way interaction between FIRST scores and stressful life events on TWT. This figure represents differences in average total wake time based on varying levels of stressful life events (LE) among participants with high and low sleep reactivity. Stressful life events represent relative deviations in stress from each person's own mean levels. Error bars were estimated using 95% confidence intervals.

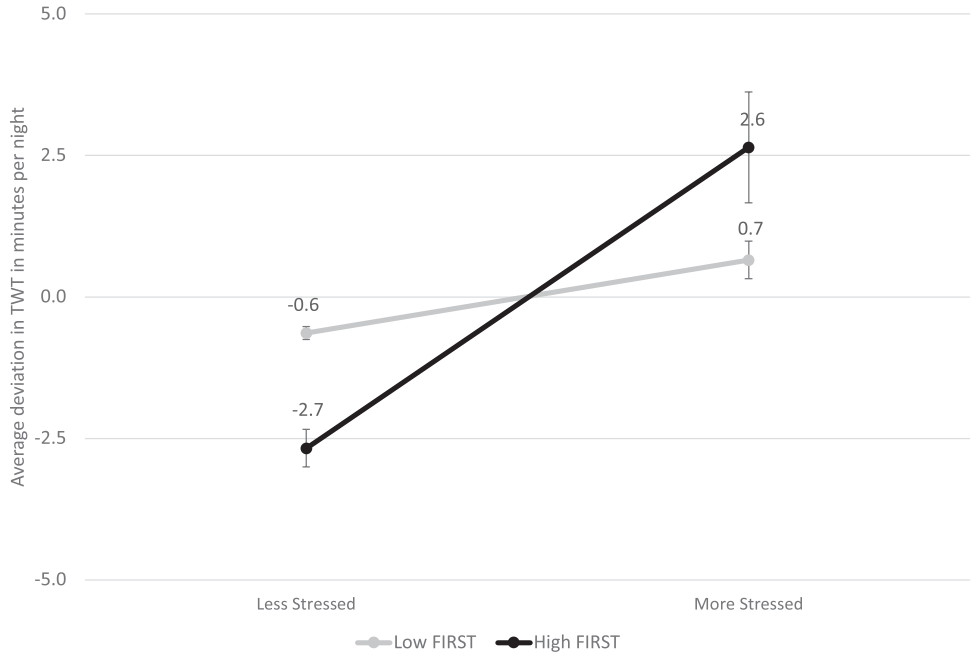

Figure 3. Model-based estimated from two-way interaction between FIRST scores and stressful life events on weekly deviations in TWT. This figure represents average deviation (from each person's mean levels) in TWT based on different levels of stress among participants with high and low sleep reactivity. Stressful life events represent relative deviations in stress from each person's own mean levels. Error bars were estimated using 95% confidence intervals.

Table 2. Regression estimates from models assessing the effects of stressful life events and sleep reactivity (FIRST) on total wake time (TWT). First scores were coded both as a continuous variable (Model 1) and a categorical variable (Model 2). For Model 2, beta coefficients for main effect and two-way interactions for FIRST represent relative change in TWT for high sleep reactive participants compared to low sleep reactive participants (i.e. Low FIRST as reference group).

	b	95% Confidence Interval		*P*-value
MODEL 1 (continuous)				
Stressful life events	1.49	1.30	1.69	<.001
FIRST	0.43	0.31	0.54	<.001
Stressors × FIRST	0.08	0.05	0.12	<.001
MODEL 2 (categorical)				
Stressful life events	1.30	1.00	1.60	<.001
FIRST	4.29	2.98	5.60	<.001
Stressors × FIRST	0.42	0.18	0.81	.04

Discussion

The sleep reactivity hypothesis of insomnia states that individuals with a greater vulnerability to stress-related sleep disturbance are more likely, when stressed, to experience sleeplessness in the short term and are at greater risk for developing insomnia disorder in the long term. While the relationship between sleep reactivity and stress has been previously observed using prospective data, this is the first study to assess acute sleep continuity disturbance and stress simultaneously (with high density sampling) over the course of an extended time interval. The goals of the present study were to estimate whether sleep reactivity (1) predicted the incidence of acute insomnia and (2) moderated the association between greater stressful life events and greater sleep continuity disturbance. As predicted, high sleep reactivity was associated with a greater likelihood of developing acute insomnia. There was also a main effect of stressful life events on total sleeplessness (i.e. TWT). That is, on weeks where participants reported high levels of stressful life events, they were also more likely to endorse greater TWT. Findings also supported our hypothesis that the relationship between greater stressful life events and greater insomnia symptoms was stronger among participants who endorsed high sleep reactivity.

As previously reported, the incidence rate of new-onset acute insomnia in this sample was 27% per annum [20]. Among those participants who developed acute insomnia, 64% reported high sleep reactivity at baseline (compared to 48% among those who remained good sleepers; see Figure 1). This finding is consistent with the three factor (3P) etiological model of insomnia and supports that some individuals possess a lower threshold for experiencing sleeplessness in the face of stress. Put differently, when stress (a precipitating factor) emerges, acute insomnia is more likely to occur in the presence of high sleep reactivity (a predisposing factor). The results from the present study are significant as they provide the first evidence that sleep reactivity precedes and predicts *acute* insomnia, whereas previous studies have focused on its relation to insomnia disorder (i.e. chronic insomnia). Not surprisingly, stressful life events, in and of itself, predicted greater sleep continuity disturbance. This finding aligns with research demonstrating that—even among good sleepers—exposure to stressful events results in greater nocturnal wakefulness (TWT) [16, 35, 36]. Findings from the current study also indicate that the effect of stressful life events on sleep was stronger among participants with high sleep reactivity. That is, the results suggest that individuals with higher sleep reactivity may be at an increased likelihood of developing insomnia symptoms when they experience a greater number of stressful life events. These individual differences further support the perspective that there may be an underlying general vulnerability (predisposition) toward insomnia [24]. One possible explanation for this variability is that individuals with high sleep reactivity are more susceptible to the effects of stress due to an elevated basal level of physiologic arousal, increased arousal responding (i.e. greater amplitude), and/or extended arousal responding (i.e. longer duration). This phenomenon may be tied to altered HPA-axis functioning, associated with increased evening and nocturnal cortisol concentrations [37], higher resting heart rate [38], reduction of slow wave and rapid eye movement sleep [39], disruptions to the dopaminergic or serotonergic systems [26], and/or "non-dipping" (i.e. lack of normal decline) in nocturnal blood pressure [40] in response to stress. Another possible explanation is that highly sleep reactive individuals may experience cognitive hyperarousal in terms of intrusive thoughts, increased rumination, or simply elevated levels of mental activity [41]. These phenomena may account for why sleep reactivity confers a vulnerability for insomnia.

The current study has several important strengths and limitations to note. The strengths include: (1) the formal aggregation of a good sleeper sample (using prospective and retrospective corroboration of sleep status); (2) the focus on [new onset] acute rather than chronic insomnia; (3) the use of a dense sampling approach over a long monitoring interval (daily and weekly assessments for up to one year); (4) the study sample size; and (5) the examination of sleep reactivity as a moderator between stressful life events and insomnia, particularly in a good sleeper sample where the measures were obtained prospectively. Regarding the latter point, the use of a good sleeper sample was ideal for testing the sleep reactivity model as it provides a group of individuals who do not have a prior history of sleep continuity disturbance. Therefore, their perceptions of the likelihood that they will experience insomnia during times of stress are unbiased.

The limitations of the study include: (1) the high exclusion/attrition rate that resulted from the loss of participants due to ineligibility, noncompliance, withdrawal, disenrollment, and/or removal from the dataset owing to the irregular completion of sleep diaries; (2) the lack of diversity in the sample (i.e. the sample was primarily white, female, and older); (3) the limited scope by which stressful life events were assessed, and specifically, that we were not able to control for the duration of each acute stressor that was endorsed (i.e. we did not assess the onset/offset of each acute stressor) or the variability in how

participants reported their experience of each acute stressor (e.g. how stressful was it for them?); (4) the lack of covariates in our primary analytic models; and (5) the observed differences in TWT were relatively small (i.e. even at the highest levels of stress the estimated group difference in TWT was about 12 min, see Figure 2). This last point may reflect the period or interval nature of insomnia, in general, and acute insomnia, in specific. For example, an individual with sleep initiation or maintenance problems of 30 min and duration for 3 days per week would technically meet the criteria for insomnia but only have a weekly average TWT of approximately 12 min. Thus, the relatively small difference at the weekly level may obscure what is more significant insomnia. This being true, it may also be the case that even at these relatively small levels of insomnia, the cumulative effect that they have over time may be a sufficient risk factor for insomnia disorder. While the use of a good sleeper sample is primarily considered a strength of the study, it likely resulted in incidence rates and means that were lower than what would be expected in a general population [36] or clinical sample [42]. Moreover, the magnitudes of the effects between TWT and other variables is likely also underestimated due to an overall ceiling effect. Regarding covariates, the present paper did not examine whether the interactive effects of stressful life events and sleep reactivity on insomnia vary as a function of other variables (e.g. gender, age, race, mood). The focus of this paper is on providing a proof of concept for the sleep reactivity hypothesis; however, future research should consider the relative contribution of these additional covariates.

In summary, findings from the current study indicate that a greater number of stressful life events are related to greater levels of sleep continuity disturbance, which confirms what previous studies have found regarding sleep and stress. Results from the current study also suggest that individuals with higher sleep reactivity may be particularly at risk for acute bouts of insomnia when they are experiencing a relatively greater number of stressful life events. These findings have the potential to enhance a clinician's ability to identify those individuals most at risk for developing insomnia in the face of stress, which can lead to downstream improvements in the prevention and treatment of insomnia. Specifically, these data support the utility of the FIRST instrument and potentially the importance of addressing sleep reactivity prospectively to diminish risk for acute and chronic insomnia. Although sleep reactivity is considered a trait-like construct, research has shown that implementing a brief modified Cognitive Behavioral Therapy for Insomnia preventive program may reduce individual sleep reactivity [43]. Therefore, lowering one's vulnerability to stress-related sleep disturbance may be an important approach to prevent the onset of insomnia. While it may not be possible to eliminate the onset of acute insomnia, given that stress necessarily provokes sleeplessness, with this knowledge (i.e. whether someone is sleep "reactive") we can minimize the severity and/or duration of the acute insomnia episode by recommending more targeted interventions for how to minimize or buffer the effects of stress on sleep, such as reducing time in bed (i.e. sleep restriction) or engaging in stress reduction strategies (e.g. mindfulness based stress reduction). While these are possible therapeutic strategies that may prove to be helpful, further research is needed to evaluate their effectiveness in mitigating bouts of acute insomnia, and specifically whether stress-focused or sleep-focused interventions are more helpful.

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Data Availabilty Statement

The data underlying this article will be shared on reasonable request to the corresponding author.

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