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Longitudinal associations between PTSD and sleep disturbances among World Trade Center responders

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ABSTRACT

Objective/Background: Post-traumatic stress disorder (PTSD) is characterized by substantial disruptions in sleep quality, continuity, and depth. Sleep problems also may exacerbate PTSD symptom severity. Understanding how PTSD and sleep may reinforce one another is critical for informing effective treatments.

Patients/methods: In a sample of 452 World Trade Center 9/11 responders (mean age = 55.22, 89.4% male, 66.1% current or former police), we examined concurrent and cross-lagged associations between PTSD symptom severity, insomnia symptoms, nightmares, and sleep quality at 3 time points ~1 year apart. Data were analyzed using random intercept cross-lagged panel models.

Results: PTSD symptom severity and sleep variables were relatively stable across time (intraclass correlation coefficients: 0.63 to 0.84). Individuals with more insomnia symptoms, more nightmares, and poorer sleep quality had greater PTSD symptom severity, on average. Within-person results revealed that greater insomnia symptoms and nightmares at Time 1 were concurrently associated with greater PTSD symptoms at Time 1. Insomnia symptoms were also concurrently associated with PTSD symptoms at Times 2 and 3, respectively. Cross-lagged and autoregressive results revealed that PTSD symptoms and nightmares predicted nightmares at the next timepoint.

Conclusions: Overall, results suggest PTSD and sleep problems may be linked at the same point in time but may not always influence each other longitudinally. Further, individuals who experience more sleep disturbances on average may suffer from more debilitating PTSD. Evidence-based treatments for PTSD may consider incorporating treatment of underlying sleep disturbances and nightmares.

1. Introduction

Posttraumatic stress disorder (PTSD) is characterized by substantial disruptions in sleep quality, continuity, and depth [1]. Sleep problems also may exacerbate existing PTSD symptom severity or increase risk for developing PTSD after trauma exposure [2–4]. Approximately 50–90% of those with PTSD report insomnia symptoms [5–7] and 50–70% report frequent nightmares [8,9].

Abbreviations: PTSD, posttraumatic stress disorder; SNS, sympathetic nervous system; HPA, hypothalamic pituitary adrenal; WTC, World Trade Center; DSM, Diagnostic and Statistical Manual of Mental Disorders; SCID, Structured Clinical Interview for DSM-IV; RI-CLPM, Random-intercept cross lagged panel model; ICC, intra-class correlations; IDAS, Inventory of Depression and Anxiety Symptoms; PCL-5, PTSD Checklist for DSM-5; CFI, Comparative Fit Index; TLI, Tucker-Lewis Index; RMSEA, Root Mean Squared Error of Approximation; SRMR, Standardized Root Mean Square Residual; REM, Rapid eye movement.

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Meta-analyses of polysomnography-determined sleep disturbances in PTSD show those with PTSD exhibit more fragmented and less restorative sleep compared to those without PTSD [1]. Furthermore, sleep problems such as poor sleep quality, insomnia, and nightmares often do not remit after gold standard treatments for PTSD, highlighting the reinforcing nature of these symptoms in predicting recovery [10–12]. Given the large economic consequences of both sleep disturbances and anxiety disorders such as PTSD [13,14], understanding associations between these variables is of societal and public health importance. Insight into how PTSD and sleep disturbances unfold and potentially reinforce one another across time will inform more precise treatment sequencing, improve treatment outcomes, and reduce risk of relapse. To this end, the goal of the current study was to examine how PTSD, insomnia, nightmares, and daily sleep quality were associated across time in a trauma-exposed sample of World Trade Center responders.

Several theoretical models provide strong support for a robust link between disturbed sleep and PTSD. For example, the hyperarousal model of insomnia states that individuals with insomnia demonstrate increased arousal of multiple physiological symptoms both during the day and at night [15]. PTSD as a disorder is also characterized by elevated arousal and dysregulation of the hypothalamic pituitary adrenal (HPA)-axis and sympathetic nervous system (SNS) [16]. According to the trauma-induced insomnia model, trauma exposure can sensitize the central and peripheral nervous system, leading to physiological hyperarousal that disrupts the sleep-wake cycle [17]. Disturbed sleep may in turn prevent sufficient extinction of fear-based memories in PTSD, impair coping abilities, and disrupt emotion regulation, all of which may exacerbate PTSD symptom severity [2,18].

Evidence from prospective longitudinal studies reveals that sleep and PTSD are bidirectionally associated. In their 2010 systematic review of prospective studies on PTSD and sleep, Babson and Feldner found that: 1) traumatic events interfere with sleep, 2) PTSD is associated with the onset of self-reported sleep problems, and 3) sleep problems interfere with PTSD recovery [19]. A recent systematic review similarly indicated a bidirectional association between PTSD symptom severity and sleep at the daily level [20]. Specifically, shorter sleep and poorer quality sleep predict greater next-day PTSD symptom severity, and greater PTSD symptom severity predict nightmares and poorer sleep quality that night [20]. Other recent studies in military personnel and veterans demonstrate that sleep problems predict PTSD severity several months or years later, and that PTSD severity also predicts future sleep complaints, albeit to a lesser degree [18,21–24]. Together, these studies suggest that sleep disturbances and PTSD symptom severity may reinforce one another, and/or represent a potentially overlapping phenotype of psychopathology [25].

Despite such strong evidence that sleep and PTSD are bidirectionally related, there are several limitations of the current literature on this topic. First, most studies do not examine nightmares as a specific sleep disturbance, despite the fact nightmares are some of the most commonly reported and most debilitating symptoms among those with PTSD [8,9]. Second, most studies on PTSD and sleep have been conducted among younger adults or military samples, and proximal to trauma exposure [18,21–24]. These results may not generalize to other trauma-exposed populations, such as first responders (e.g., police, firefighters, emergency medical technicians, paramedics). First responders experience frequent sleep disturbances [26], as well as some of the highest rates of occupational trauma exposure (~80%) [27] and a higher prevalence of PTSD (~10–15%) than the general population [27,28]. After the September 11th, 2001 (9/11) World Trade Center (WTC) attacks in particular, first responders and other adults reported substantial increases in stress [29]. However, trauma-related consequences also may unfold many years after trauma exposure, not only right after the index event. Despite this, most studies do not examine the interplay of trauma, PTSD, and sleep across longer periods of time using appropriate statistical techniques, which may lead to inaccurate or incomplete conclusions about the nature of PTSD and sleep disturbances.

To address these gaps, the goals of the current study were to examine associations between insomnia, nightmares, sleep quality, and PTSD symptom severity at three timepoints across two years among a sample of trauma-exposed World Trade Center responders. At the within-person level, we hypothesized both concurrent and cross-lagged associations between sleep and PTSD symptom severity. Specifically, in terms of *concurrent* associations, we hypothesized that greater insomnia symptoms, more nightmares, and poorer sleep quality would be associated with greater PTSD symptom severity at each time point. In terms of *cross-lagged* associations, we hypothesized that greater insomnia symptoms, more nightmares, and poorer sleep quality at the previous time point would predict greater PTSD symptom severity at the subsequent time point, and vice versa. Based on mixed previous research and conflicting theoretical perspectives (as reviewed above) [17–24,30], we did not have specific hypotheses about one direction (i.e., PTSD to sleep vs. sleep to PTSD) being stronger than the other. Finally, at the between-person level, we hypothesized that individuals with greater insomnia symptoms, more nightmares, and poorer sleep quality on average would have greater PTSD symptom severity on average.

2. Materials and method

2.1. Participants

All 452 participants in the World Trade Center (WTC) Personality and Health Study were included in the present project [31]. They were recruited from Stony Brook University WTC Health Program, which monitors over WTC responders from Long Island, NY who responded to the September 11th, 2001 (9/11) WTC attacks [31]. Participants were enrolled in the study in 2017. Exclusion criteria included linguistic, cognitive, or physical limitations that would prevent completion of study procedures, such as inability to understand survey questions, attend a baseline appointment, or complete surveys on a mobile device at home. The Institutional Review Board of Stony Brook University approved the study and all participants provided informed consent. Participants primarily identified as male (89.4%, $n = 404$) White (90.0%, $n = 406$), non-Hispanic (92.4%, $n = 412$), and currently working full time (41.8%, $n = 189$). The majority were current or former police (66.1%, $n = 291$). At baseline (Time 1), 34 (7.7%) participants had a current diagnosis of PTSD as determined by the Structured Clinical Interview for DSM-IV (SCID). At Time 2 and Time 3, 23 (6.4%) and 9 (2.8%) participants, respectively, had a current diagnosis of PTSD as determined by the SCID. Additional participant demographic information at baseline is presented in Table 1.

2.2. Procedure

Once participants signed informed consent and enrolled in the study, they proceeded to complete the baseline (i.e., Time 1 [T1]) assessment which included a battery of self-report questionnaires, including the PTSD, insomnia symptoms, and nightmares measures. Participants also completed a Structured Clinical Interview for DSM-IV (SCID) [32], and device training on how to access the survey application on a mobile phone for completion of the daily sleep measure. The baseline visit was followed by 2-weeks of daily

diary assessment of sleep (completed in the morning upon awakening). Approximately one (Time 2 [T2]) and two (Time 3 [T3]) years later, participants completed the self-report questionnaires, including the PTSD, insomnia symptoms, and nightmare measures, and the daily diary portion to assess sleep quality again.

2.3. Measures

2.3.1. PTSD symptom severity

PTSD symptom severity in the past month was assessed using the 20-item PTSD Checklist (PCL-5) [33]. Response options ranged from 0 (not at all) to 4 (extremely). The two sleep-related items from the PCL-5 ("Repeated, disturbing dreams of the stressful experience" and "Trouble falling or staying asleep") were removed to reduce confounding effects with the other sleep variables. The remaining 18 items were summed to obtain a total score ranging from 0 to 72, with higher scores indicating greater symptom severity. The PCL-5 scores have good reliability and convergent validity [34,35]. In the current study, the PCL-5 score (18 items) demonstrated excellent internal consistency (α = 0.95 at each timepoint).

2.3.2. Insomnia symptoms

Insomnia symptoms were assessed using the Inventory of Depression and Anxiety Symptoms (IDAS) [36]. The IDAS is a multidimensional measure of depression and anxiety that contains 10 specific symptom scales, including one 6-item scale on insomnia symptoms in the past two weeks. The IDAS Insomnia scale asks questions about the extent to which respondents experienced long sleep latency (e.g., "I had trouble falling asleep"), awakenings during the night (e.g., "I woke up frequently during the night"), early morning awakenings (e.g., "I woke up early and couldn't get back to sleep" and "I woke up much earlier than usual"), poor sleep quality (e.g., "I slept very poorly") and sleep duration ("I slept less than usual"). All item responses consisted of a scale from 1 (not at all) to 5 (extremely) and were summed together to create a total score ranging from 6 to 30. The IDAS has been shown to have good convergent validity and reliability in previous studies [36]. In the current study, the IDAS demonstrated excellent internal consistency (α = 0.88–0.89).

2.3.3. Nightmare frequency

Nightmare frequency was assessed using a single item: "How many nights in the past week have you experienced a nightmare?" on a scale of 0–7.

2.3.4. Daily sleep quality

During the 2-weeks of daily diary assessment, participants reported on their previous night's sleep each morning with one item asking about their subjective sleep quality: "My sleep quality last night was …" with responses on a Likert scale ranging from 1 = very poor to 5 = very good. At each time point, responses to this item were averaged across the 2 weeks to obtain a mean for each person.

2.4. Statistical analysis plan

Random-intercept cross lagged panel models (RI-CLPM) were run using the R package *lavaan* [37]. R code is available in Supplementary Materials. RI-CLPM were used to test whether sleep

Table 1
Participant Characteristics at Baseline (Time 1).

	M (SD) or n (%)
Age	55.22 (8.73)
Sex	
Male	404 (89.4%)
Female	48 (10.6%)
Race	
White	406 (90.0%)
Black	32 (7.1%)
Asian	4 (0.9%)
American Indian/Alaskan Native	1 (0.2%)
Other or multicultural	8 (1.8%)
Ethnicity	
Hispanic	34 (7.6%)
Non-Hispanic	412 (92.4%)
Education	
Less than high school degree	3 (0.7%)
High school graduate	65 (14.4%)
Some college	192 (42.5%)
College graduate	115 (25.4%)
Professional academy graduate	25 (5.5%)
Some graduate or professional schooling	16 (3.5%)
Masters/doctoral or other advanced degree	36 (8.0%)
Current employment status	
Working full time	189 (41.8%)
Homemaker	1 (0.2%)
Full time student	2 (0.4%)
Working part time	52 (11.5%)
Laid off	2 (0.4%)
Retired	174 (38.5%)
Physical or psychiatric disability	28 (6.2%)
Leave of absence	1 (1.2%)
Unemployed	3 (0.6%)
PTSD (% with PTSD diagnosis based on SCID)	34 (7.7%)
Responder type	
Police officer	291 (66.1%)
Other (Firefighter/emergency medical services/construction/public health)	149 (33.9%)

Note. Percentages represent valid percentages (i.e., variables with missing data were not included in the total calculation).
SCID = Structured Clinical Interview for DSM-IV.

variables predicted changes in PTSD symptom severity over time and, conversely, whether PTSD symptom severity predicted sleep variables. RI-CLPM are an extension of the traditional cross-lagged panel model (CLPM) [38–40]. The main benefit of the RI-CLPM over the traditional CLPM is that it decomposes the variance of longitudinal data into between-person differences and within-person fluctuations across time. Between-person effects can help understand stable, trait-like characteristics of individuals and within-person effects can help understand how people change across time compared to their average. With the RI-CLPM, the trait-like characteristics can be captured by the random intercept term, and the state-like fluctuations can be captured by the autoregressive and cross-lagged effects, allowing for more accurate inferences of directionality of symptoms across time [38–40].

First, we calculated intra-class correlations (ICC) for sleep variables and PTSD symptom severity. The ICC is the ratio of the variance explained by differences between persons to the total variance (i.e., between-person plus within-person variance). Next, we used RI-CLPM where observed sleep variables and PTSD symptom scores were regressed on their own latent factor (each loading constrained at one). The resulting 6 latent factors were used to identify autoregressive, cross-lagged paths, and cross-sectional associations (i.e., T1 correlation and correlated change). The residual variances of the observed variables were constrained at zero, which allowed the latent factor structure to capture the within- and between-person variance. Next, we added two random intercepts (one for each sleep variable, the other for PTSD symptom severity) with factor loadings constrained at one. These random intercepts represent the stable trait-like differences in sleep variables or PTSD symptom severity between individuals. The correlation between the random intercepts reflected how stable between-person differences in sleep variables were associated with stable between-person differences in PTSD symptom severity (Fig. 1). Autoregressive paths reflect how prior differences from expected scores predicted within-person deviations in sleep variables and PTSD symptom severity at the next timepoint. The cross-lagged paths reflect to what extent sleep variables and PTSD symptom severity are associated across time and indicated whether deviations from expected scores in sleep variables predicted deviations from expected scores in PTSD symptom severity (and vice versa) at the next assessment wave. Cross-sectional paths are correlations between variables at the same timepoint (i.e., correlations at T1, and then correlated changes at T2 and T3).

Model fit was evaluated using several fit indices: 1) chi-square statistic (χ [2]) and p-value, 2) the Comparative Fit Index (CFI) value, 3) the Tucker-Lewis Index (TLI) value, 4) the Root Mean Squared Error of Approximation (RMSEA) value, and 5) the Standardized Root Mean Square Residual (SRMR) value. A non-significant χ [2] p-value, a CFI and TLI >0.95, and a RMSEA and SRMR <0.06 typically indicate a good model fit [41,42]. Two-tailed p-values less than 0.05 were considered statistically significant. All results reflect standardized regression coefficients.

Following guidelines from Hamaker et al., 2015 [39], after freely estimating all pathways, we tested more constrained models to maximize statistical power and model parsimony. For these models, we constrained the autoregressive (i.e., "b" paths in Fig. 1) and cross-lagged paths (i.e., "c" paths in Fig. 1) to be equal for sleep variables and PTSD symptoms across T1-T2 and T2-T3, given the expectation these associations would be similar across lags. Constrained models were then compared to unconstrained models using Akaike information criterion (AIC), Bayesian information criterion (BIC) and Chi-square (χ^2) difference tests values. In the case of a non-significant χ^2 test (i.e., model fit did not significantly worsen), we present the more constrained model.

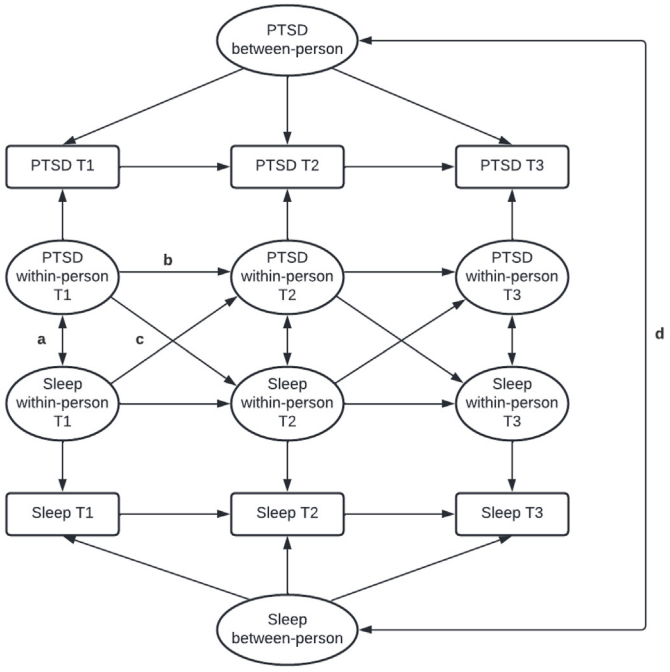

Fig. 1. Three-wave random intercept cross-lagged panel model between PTSD and sleep variables (insomnia symptoms, nightmares, and sleep quality were examined in separate models; "sleep" is a general term used to denote each of these variables): (a) cross-sectional paths, (b) autoregressive paths, (c) cross-lagged paths), (d) correlation between stable traits of sleep problems and PTSD symptom severity at the between-person level. Rectangles denote observed variables; ovals represent latent variables.

3. Results

3.1. Descriptive statistics

Depending on the measure, 2–4% of data from the original sample of 452 participants were missing at T1, 23–26% were missing at T2, and 18–38% were missing at T3 (see N by key study measure in Table 2). Attrition analyses revealed that there were no significant differences in terms of baseline age, PTSD diagnosis, insomnia symptoms, nightmares, or sleep quality between those participants who completed the T3 measures and those who dropped out before T3. However, those who dropped out before T3 were more likely to be male (odds ratio = 3.65, p = .034) and had greater baseline PTSD symptoms (M = 33.74 vs. M = 29.63, respectively, p = .036).

On average, responders completed 11.37 (SD = 3.30) of 14 possible daily sleep surveys to assess sleep quality at T1; 12.20 (SD = 2.72) of 14 at T2, and 12.08 (SD = 2.76) of 14 at T3. Means, standard deviations and correlations between primary variables at each time point are displayed in Table 2. In general, scores on each measure were strongly correlated with scores on the same measure across the three time points (rs = 0.82-0.83 for PTSD symptom severity; rs = 0.54-0.63 for nightmares; rs = 0.71-0.76 for sleep quality; rs = 0.65-0.67 for insomnia symptoms). ICC coefficients revealed a similar pattern. For PTSD symptom severity, the ICC was 0.84, indicating that stable differences in PTSD between responders explained 84% of the total variance in PTSD symptom severity across the three measurement waves, and within-person fluctuations explained the remaining 16% of the variance in PTSD symptom severity. For insomnia symptoms, nightmares, and sleep quality, the ICC was 0.67, 0.63, and 0.76, respectively.

Table 2
Means, standard deviations, N, ranges, and correlations with confidence intervals among key study variables at each time point.

Variable	*M*	*SD*	*N*	range	1	2	3	4	5	6	7	8	9	10	11
1. PTSD1	30.37	13.96	444	0–68											
2. PTSD2	28.34	12.57	348	0–72	.82** [.78, .85]										
3. PTSD3	29.18	12.75	363	0–59	.83** [.79, .86]	.83** [.79, .86]									
4. NIGHTM1	0.76	1.41	444	0–7	.54** [.47, .60]	.43** [.34, .51]	.45** [.37, .53]								
5. NIGHTM2	0.78	1.41	343	0–7	.50** [.42, .58]	.57** [.49, .64]	.49** [.40, .57]	.63** [.56, .69]							
6. NIGHTM3	0.81	1.40	361	0–7	.47** [.38, .55]	.48** [.39, .56]	.48** [.39, .55]	.54** [.46, .61]	.67** [.60, .72]						
7. SLPQUAL1	3.34	0.72	435	1–5	−.37** [-.45, −.29]	−.39** [-.48, −.30]	−.40** [-.48, −.31]	−.28** [-.36, −.19]	−.24** [-.34, −.14]	−.23** [-.33, −.13]					
8. SLPQUAL2	3.43	0.71	333	1.13–5	−.36** [-.45, −.26]	−.40** [-.49, −.30]	−.37** [-.46, −.26]	−.22** [-.32, −.11]	−.30** [-.40, −.19]	−.23** [-.33, −.12]	.76** [.71, .80]				
9. SLPQUAL3	3.44	0.72	281	1–5	−.35** [-.44, −.24]	−.38** [-.48, −.27]	−.37** [-.47, −.26]	−.17** [-.28, −.05]	−.17** [-.29, −.05]	−.14* [-.25, −.02]	.71** [.65, .77]	.75** [.69, .80]			
10. INSOM1	13.73	5.84	446	6–30	.55** [.48, .61]	.47** [.38, .55]	.50** [.42, .58]	.35** [.26, .43]	.32** [.22, .41]	.29** [.19, .38]	−.55** [-.61, −.48]	−.48** [-.56, −.39]	−.43** [-.52, −.33]		
11. INSOM2	12.81	5.60	349	6–30	.43** [.34, .51]	.54** [.46, .61]	.45** [.36, .53]	.29** [.19, .38]	.35** [.25, .44]	.29** [.18, .39]	−.51** [-.59, −.43]	−.54** [-.61, −.45]	−.44** [-.53, −.33]	.65** [.58, .71]	
12. INSOM3	13.24	5.78	369	6–30	.40** [.31, .49]	.47** [.38, .55]	.52** [.44, .59]	.29** [.20, .38]	.35** [.25, .45]	.31** [.22, .40]	−.56** [-.62, −.48]	−.58** [-.65, −.50]	−.53** [-.61, −.44]	.67** [.61, .72]	.65** [.58, .71]

Note. M and SD are used to represent mean and standard deviation, respectively. N is the number of observations for each measure. Range is the minimum and maximum actual values. Values in square brackets indicate the 95% confidence interval for each correlation. * indicates p < .05. ** indicates p < .01. INSOM = insomnia symptoms; NIGHTM = nightmares; PTSD = posttraumatic stress disorder symptoms (PCL-5 total score minus 2 sleep-related items); SLPQUAL = sleep quality. 1, 2, and 3 suffixes represent each time point.

3.2. Associations between PTSD symptom severity and insomnia symptoms

The overall model fit of the freely estimated (i.e., unconstrained) RI-CLPM between PTSD symptoms and insomnia symptoms was good: χ^2 (26 parameters, 1 *df*) = 2.492, *p* = .114, CFI = 0.999, TLI = 0.986, RMSEA = 0.057, SRMR = 0.015. After constraining the autoregressive and cross-lagged effects to be the same across lags, model fit indices revealed that the constrained model did not significantly worsen model fit (unconstrained model: AIC = 15049, BIC = 15156; constrained model: AIC = 15040, BIC = 15118; $\Delta\chi$ [2] = 5.278, *p* = .626; see Supplementary Fig. 1 for all regression estimates for the unconstrained model). Therefore, the constrained model was retained as the final model to maximize statistical power and model parsimony. The overall fit of this model was good: χ^2 (26 parameters, 8 *df*) = 7.770, *p* = .456, CFI = 1.000, TLI = 1.000, RMSEA = <0.001, SRMR = 0.027. At the between-person level, there was a strong positive association between stable traits of insomnia symptoms and PTSD symptom severity (β = 0.62, *p* < .001; Fig. 2). This indicates that, on average, responders who had more insomnia symptoms reported more severe PTSD symptom severity across the three measurement waves. Greater within-person fluctuations in insomnia symptoms at T1, T2, and T3, were concurrently associated with greater within-person fluctuations in PTSD symptom severity at T1 (β = 0.27, *p* < .001), T2 (β = 0.34, *p* < .001) and T3 (β = 0.34, *p* < .001), respectively. There were no significant autoregressive or cross-lagged associations between insomnia and PTSD, indicating that within-person fluctuations in PTSD symptoms and insomnia symptoms did not predict one another from one timepoint to the next (Fig. 2).

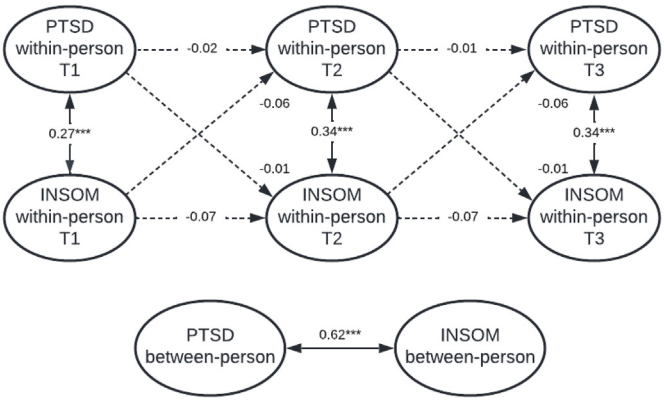

Fig. 2. Three-wave random intercept cross-lagged panel model between insomnia symptoms (INSOM) and PTSD symptom severity (PTSD), with autoregressive and cross-lagged paths constrained to be equal across waves. Values represent standardized regression estimates. Statistically significant lines are solid, and statistically non-significant lines are dotted. ***p < .001.

3.3. Associations between PTSD symptom severity and nightmares

The overall model fit of the freely estimated (i.e., unconstrained) RI-CLPM between PTSD symptoms and nightmares was good: χ^2 (26 parameters, 1 *df*) = 1.978, *p* = .160, CFI = 0.999, TLI = 0.990, RMSEA = 0.047, SRMR = 0.017. After constraining the autoregressive and cross-lagged effects to be the same across lags, model fit indices revealed that the constrained model did not significantly worsen model fit (unconstrained model: AIC = 11808, BIC = 11915; constrained model: AIC = 11804, BIC = 11882; $\Delta\chi$ [2] = 9.264, *p* = .234; see Supplementary Fig. 2 for all regression estimates for the unconstrained model). Therefore, the constrained model was retained as the final model. The overall fit of this model was good: χ^2 (26 parameters, 8 *df*) = 11.242, *p* = .188, CFI = 0.998, TLI = 0.996, RMSEA = 0.030, SRMR = 0.031. At the between-person level, there was a strong positive association between stable traits of nightmares and PTSD symptom severity (β = 0.76, *p* < .001). Greater within-person fluctuations in nightmares at T1 were concurrently associated with greater within-person fluctuations in PTSD symptoms severity at T1 (β = 0.16, *p* < .01; Fig. 3), but not at T2 or T3.

Autoregressive results revealed that more within-person fluctuations in nightmares at T1 and T2 were associated with more within-person fluctuations in nightmares at T2 and T3 (βs = 0.25 and 0.24, p = .008 and .006), respectively. Within-person fluctuations in PTSD symptoms were not associated with within-person fluctuations in PTSD symptoms at the subsequent timepoint. Cross-lagged results revealed that within-person fluctuations in PTSD symptoms at T1 and T2 were associated with within-person fluctuations in nightmares at T2 and T3 (βs = −0.15 and −0.18, p = .015 and .014), respectively. Nightmares were not associated with subsequent PTSD symptoms (Fig. 3).

3.4. Associations between PTSD symptom severity and daily sleep quality

The overall model fit of the freely estimated (i.e., unconstrained) RI-CLPM between PTSD symptoms and daily sleep quality was good: χ^2 (26 parameters, 1 df) = 0.213, p = .644, CFI = 1.000, TLI = 1.008, RMSEA = <0.001, SRMR = 0.006. After constraining the autoregressive and cross-lagged effects to be the same across lags, model fit indices revealed that the constrained model significantly worsened model fit (unconstrained model: AIC = 10039, BIC = 10146; constrained model: AIC = 10088, BIC = 10166; $\Delta\chi$ [2] = 62.547, p < .001; see Supplementary Fig. 3 for all regression estimates for the constrained model). Therefore, the freely estimated (i.e., unconstrained) model was retained as the final model. At the between-person level, there was a strong negative association between stable traits of daily sleep quality and PTSD symptom severity (β = −0.48, p < .001). There were no significant cross-sectional, autoregressive, or cross-lagged associations between daily sleep quality and PTSD symptom severity at the within-person level (Fig. 4).

4. Discussion

Sleep disturbances and PTSD represent significant threats to World Trade Center (WTC) responders' quality of life and well-being. To understand how these symptoms unfold and potentially influence each other across time, we examined concurrent and lagged associations between PTSD and sleep disturbances across two years in an at-risk group of WTC responders. We found that, on average, responders with more PTSD symptom severity had greater insomnia symptoms, nightmares, and poorer sleep quality. PTSD and most sleep measures were concurrently associated at most timepoints, and within-person changes in PTSD were associated with changes in nightmares across time. However, there were no cross-lagged, prospective associations between PTSD and other sleep variables. Together, these results confirm the partially overlapping nature of PTSD and sleep problems among trauma-exposed individuals. Our results support and extend previous research by examining these associations using a random-intercept CLPM, which allows for examination of stable traits and within-person changes in symptoms across time. An additional innovation of the current study is the opportunity to explore these associations in a group of individuals all exposed to a similar traumatic event, the 9/11 WTC attacks.

On average (i.e., at the between-person level), our results showed that responders with greater PTSD symptom severity reported more insomnia symptoms, nightmares, and poorer sleep quality. These findings align with previous work showing that individuals with PTSD report a very high prevalence of nightmares and insomnia [8]. In fact, sleep disturbances are now considered a core feature, rather than just a secondary symptom, of PTSD [25]. Sleep disturbances are highly prevalent in PTSD, are a risk factor for PTSD onset after trauma exposure, and often do not fully remit after PTSD treatment, highlighting their critical role in the onset and maintenance of the disorder [25]. Identifying those individuals who have comorbid PTSD and high levels of sleep disturbances may be one way to customize and maximize PTSD treatment gains.

Using a framework that parses within- and between-person variance, we found some concurrent associations between PTSD symptom severity with insomnia and nightmares. These results align with other studies, which show that sleep disturbances and PTSD are strongly linked cross-sectionally [19,43,44]. Disturbed sleep may interfere with proper extinction of trauma-related memories or impair coping and emotion regulation abilities, aggravating PTSD symptom severity [2,45]. PTSD symptoms also may increase arousal and vigilance that interfere with the ability to fall asleep, stay asleep, or obtain restorative sleep [46]. Studies have shown that compared to healthy controls, individuals with PTSD spend less time in restorative slow wave sleep and rapid eye movement (REM) sleep, and more time awake after sleep onset [47]. This sleep profile may be partially attributed to awakenings from nightmares. PTSD may also lead to engagement in risky behaviors, such as excessive substance use [48], which can interfere with the ability to obtain good quality sleep. It is also possible that both PTSD symptom severity and sleep disturbances are

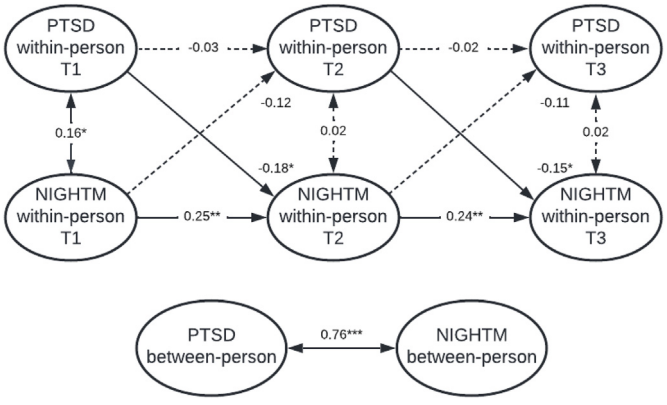

Fig. 3. Three-wave random intercept cross-lagged panel model between nightmare frequency (NIGHTM) and PTSD symptom severity (PTSD), with autoregressive and cross-lagged paths constrained to be equal across waves. Values represent standardized regression estimates. Statistically significant lines are solid, and statistically non-significant lines are dotted. ***p < .001. **p < .01. *p < .05.

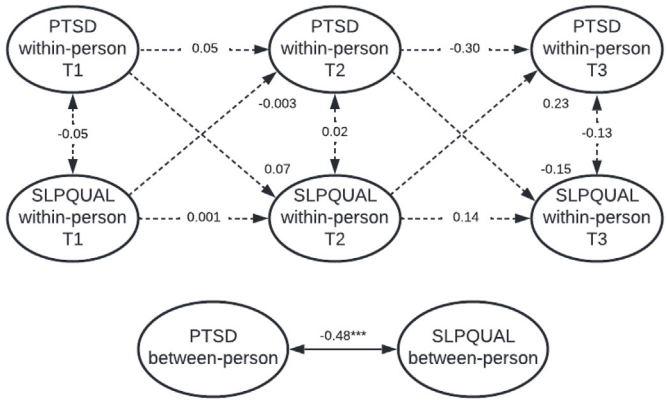

Fig. 4. Three-wave random intercept cross-lagged panel model between daily sleep quality (SLPQUAL) and PTSD symptom severity (PTSD), unconstrained model. Values represent standardized regression estimates. Statistically significant lines are solid, and statistically non-significant lines are dotted. ***p < .001.

exacerbated by a common third variable, such as hyperarousal of the HPA-axis, amygdala, or noradrenergic systems after trauma exposure or other stressful life events [25]. Additional experimental work is needed to untangle these effects.

Our cross-lagged results were generally null, and findings with PTSD and nightmares were somewhat unexpected. Higher than average PTSD symptoms were associated with fewer than average nightmares at the next time point. It may be that individuals with greater PTSD severity have found constructive ways to cope with nightmares, decreasing their frequency across time. Individuals with greater PTSD severity also may be more hesitant to report nightmares, which are often stigmatized [49]. Interestingly, no sleep variables predicted subsequent changes in PTSD. Compared to PTSD symptoms, sleep disturbances were less severe in our sample, which may have limited their predictive utility across time. In general, our mostly null cross-lagged results contrast with other studies, which have shown that PTSD symptom severity predicts subsequent sleep problems, and in particular, that sleep problems such as insomnia predict subsequent PTSD [21,22]. In fact, studies show poor sleep quality is associated with a 60% greater likelihood of developing subsequent PTSD among U.S. veterans, and that sleep complaints after trauma predict PTSD onset [4,50]. Disrupted sleep immediately before and/or after trauma exposure is now considered a causal risk factor for the development of PTSD [30].

It is also possible that our counterintuitive cross-lagged results may be attributed to several methodological explanations. Both PTSD symptom severity and sleep variables did not show much variation across time. In general, ICCs and correlations among the same measures across time points were high, suggesting these may be relatively stable characteristics of individuals. However, most previous longitudinal studies of PTSD and sleep have not decomposed effects into stable between-person traits and within-person fluctuations, as we were able to do in this study to provide a more accurate estimation of results [38,39]. Second, the length of time between assessment periods (approximately one year) also may have hindered our ability to detect predictive effects over time. Given that several years passed between the WTC attacks and when participants enrolled in the current study, it may be that this sample has recovered from the negative impacts of potentially traumatic events. Using an intensive longitudinal design over successive days may provide a better test of how PTSD and sleep influence each other on a micro-time scale. Third, we may have been limited by our measure of nightmares, which only assessed nightmare frequency and not severity. It is possible that nightmare severity may be impacted by PTSD than nightmare frequency. Finally, most previous studies have been conducted in veteran or military samples [18,21–24], the results from which may not generalize to this sample of 9/11 responders. Seeking psychological treatment for PTSD and sleep is still highly stigmatized in this population [51], which may have resulted in more intractable symptoms and an inability to detect within-person changes across time.

4.1. Potential clinical implications

Results from our study have some tentative implications for informing the treatment of PTSD, insomnia, and nightmares. Nightmares and other sleep disturbances can affect PTSD treatment response, and sleep treatments may bolster PTSD recovery [52]. Despite empirical support for this idea, gold-standard treatments for PTSD (i.e., prolonged exposure [PE], cognitive processing therapy [CPT]) still do not explicitly target sleep disturbances. Several studies have shown that PE and CPT lead to simultaneous improvement in sleep and PTSD symptom severity, but neither treatments tend to result in full remission of sleep disturbances [53]. For example, among active-duty U.S. Army soldiers receiving PE for PTSD, insomnia and nightmares improved significantly from baseline to posttreatment, but many patients still reported clinically significant insomnia (>70%) and nightmares (>38%) posttreatment [54]. Other studies similarly show that sleep problems may reduce the speed of recovery in PTSD patients and that greater residual sleep symptoms predict smaller treatment gains among veterans receiving PE for PTSD [11,55]. Consistent with the current results, one promising pilot study among veterans with PTSD showed that an intervention targeting trauma-specific sleep disturbances resulted in large improvements in PTSD symptom severity, sleep quality, and insomnia severity [56]. Together, this work suggests treating sleep problems should be a core tenet of PTSD interventions, and doing so may be one way to maximize treatment gains and reduce PTSD relapse.

4.2. Limitations and future directions

Despite several strengths of this study (e.g., unique sample of responders, three waves of data collection across two years, multiple measures of sleep), there are some limitations worth mentioning. Most participants in our sample identified as White and male. While this matches the composition of most first responder units, future studies should further explore racial/ethnic and sex/gender differences in the context of the current study's research aims. For example, some work has shown women experience higher rates of some trauma types and more severe PTSD after trauma exposure [57,58]. Women are also at substantially increased risk for insomnia and nightmares compared to men [59,60].

Another limitation is possible attrition biases, as only 62–82% of the original sample participated in the final time point of data collection, depending on the specific measure. This may have resulted in a “healthy worker” selection bias [61], where the most severe cases dropped out. Although participants who dropped out before the final timepoint were comparable to completers on most measures, those who dropped out did have slightly more severe PTSD symptoms at baseline and were more likely to be male. Overall, our results revealed that, based on the SCID, most participants did not meet criteria for PTSD at baseline, suggesting this may have been a more resilient sample.

Finally, the measurement of sleep variables may have limited our ability to detect effects. Our nightmare variable consisted of a frequency count of nightmares, which may require more advanced non-linear modeling techniques to detect significant effects. Future research should also incorporate nightmare severity or daytime-related distress to complement nightmare frequency. Finally, although our insomnia measure consisted of a validated insomnia sub-scale, it contained items on sleep duration and sleep quality complaints, which are not part of the traditional diagnostic criteria for insomnia. Future research may consider using insomnia questionnaires that focus more on traditional symptoms and/or using diagnostic interviews to assess insomnia.

5. Conclusion

Both PTSD and sleep disturbances have devastating economic and public health impacts. Understanding how these variables are related to one another across time may help refine treatment initiatives and reduce social and economic costs. Our results suggest responders with more severe PTSD symptom severity also experience more nightmares, more insomnia symptoms, and poorer daily sleep quality, on average. At any given point in time, sleep problems and PTSD symptom severity also appear to be strongly linked. Without intervention, our results suggest these symptoms may

persist across time. Evidence based treatments for PTSD should consider explicitly addressing sleep problems, as these problems often do not remit after gold-standard treatments for PTSD, and sleep disturbances and nightmares represent some of the most debilitating concerns reported by those with PTSD.

CRediT authorship contribution statement

Danica C. Slavish: conceptualized the manuscript, ran all analyses, and drafted the manuscript. **Camilo J. Ruggero:** reviewed the manuscript and provided feedback, helped collect data for the larger study. **Madasen Briggs:** reviewed the manuscript and provided feedback. **Brett A. Messman:** reviewed the manuscript and provided feedback. **Ateka A. Contractor:** reviewed the manuscript and provided feedback. **Jiaju Miao:** reviewed the manuscript and provided feedback, helped collect data for the larger study. **Joshua R. Oltmanns:** reviewed the manuscript and provided feedback. **Monika A. Waszczuk:** reviewed the manuscript and provided feedback, helped collect data for the larger study. **Benjamin J. Luft:** reviewed the manuscript and provided feedback, helped collect data for the larger study. **Roman Kotov:** reviewed the manuscript and provided feedback, helped collect data for the larger study. All authors contributed and reviewed the final version of the paper.

Declaration of competing interest

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2022.11.021.

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