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Sleep architecture alterations in patients with post-traumatic stress disorder (PTSD): A polysomnographic analysis

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Abstract
Post-Traumatic Stress Disorder (Post-Traumatic Stress Disorder (PTSD)) is often accompanied by significant sleep disturbances, which may contribute to the persistence and severity of symptoms. This study aimed to explore sleep architecture alterations in individuals with Post-Traumatic Stress Disorder (PTSD) through polysomnographic (PSG) analysis, comparing sleep patterns in a Post-Traumatic Stress Disorder (PTSD) group (n = 30) and a matched control group (n = 30). The study focused on key sleep parameters, including N1, N2, N3, and REM sleep, as well as REM latency and the number of REM periods. Results indicated that Post-Traumatic Stress Disorder (PTSD) patients spent significantly more time in lighter sleep stages (N1), with significantly reduced N3 duration and fewer REM periods compared to controls. Additionally, REM latency was significantly delayed in the Post-Traumatic Stress Disorder (PTSD) group. Correlational analysis revealed that longer N1 duration was positively associated with Post-Traumatic Stress Disorder (PTSD) symptom severity, particularly hyperarousal and avoidance symptoms, while reduced N3 duration was inversely correlated with symptom severity. Multiple regression analysis demonstrated that N1 sleep duration was a significant predictor of Post-Traumatic Stress Disorder (PTSD) severity. These findings suggest that sleep architecture disruptions, including increased light sleep and reduced deep sleep, may contribute to the maintenance of Post-Traumatic Stress Disorder (PTSD) symptoms, particularly hyperarousal. Given the significant impact of sleep disturbances on Post-Traumatic Stress Disorder (PTSD), practical recommendations include incorporating sleep-focused interventions, such as cognitive-behavioural therapy for insomnia (CBT-I) and sleep hygiene education, into standard Post-Traumatic Stress Disorder (PTSD) treatment plans. Further research is needed to investigate the causal mechanisms behind these sleep alterations and the potential benefits of sleep interventions in mitigating Post-Traumatic Stress Disorder (PTSD) symptomatology.

Keywords: Post-traumatic stress disorder (post-traumatic stress disorder (PTSD), sleep architecture, polysomnography, N1 sleep, N3 sleep, REM sleep, sleep disruptions, hyperarousal, sleep therapy, cognitive behavioural therapy, post-traumatic stress disorder (PTSD) symptoms

Introduction

Traumatic stress exposure is common in the general population, with lifetime estimates exceeding 50 % in many settings, and a subset of these individuals develop posttraumatic stress disorder (Post-Traumatic Stress Disorder (PTSD)), a condition characterised by intrusive recollections, avoidance of traumarelated cues, negative alterations in cognition and mood, and heightened arousal and reactivity [10-12]. Among the core features of Post-Traumatic Stress Disorder (PTSD), disturbed sleep specifically insomnia, nightmares and nocturnal awakenings is both highly prevalent and persistent, and may play a mechanistic role in onset, maintenance and severity of the disorder [13-16]. Indeed, subjective reports of sleep disruption in Post-Traumatic Stress Disorder (PTSD) are ubiquitous, yet objective measurement via polysomnography (PSG) has yielded heterogeneous findings, limiting our ability to draw definitive conclusions about the nature of underlying sleep architecture alterations in Post-Traumatic Stress Disorder (PTSD) populations [17-20].

Metaanalytic and systematic review evidence points toward a consistent pattern of altered sleep architecture in Post-Traumatic Stress Disorder (PTSD): increased time spent in Stage N1 (the lightest sleep stage), decreased slowwave or deep sleep (Stage N3), and fragmentation of REM sleep including shorter latency to first REM episode, increased

REM density and more frequent transitions out of REM into wake or N1 sleep [21-23]. For example, one systematic review of 31 PSG studies reported increased N1 and reduced N2/N3 sleep in Post-Traumatic Stress Disorder (PTSD) versus controls [24], while targeted studies found that reduced N3 in Post-Traumatic Stress Disorder (PTSD) correlated with more severe symptomatology [25]. Moreover, altered REM dynamics may reflect disrupted emotional processing and memory consolidation, functions attributed to REM as well as N2/N3 stages [26-29].

Given this background, a clear gap remains: while sleep continuity (e.g., total sleep time, sleep efficiency, wake after sleep onset) has been relatively well studied, the specific contributions of sleep architecture stages (N1, N2, N3, REM latency, number of REM periods) to Post-Traumatic Stress Disorder (PTSD) symptom clusters (reexperiencing, avoidance, hyperarousal) have been less thoroughly examined. A small number of studies have begun to address this for instance, increased N1 sleep has been linked to all three Post-Traumatic Stress Disorder (PTSD) clusters and fewer REM periods to hyperarousal symptoms in veteran and civilian samples [30]. Nevertheless, methodological heterogeneity (use of adaptation nights, comorbid sleep disorders, small sample sizes) persists, and few studies have comprehensively tested architecturesymptom cluster hypotheses. Therefore, the objective of this investigation was to conduct a PSGbased analysis in patients with Post-Traumatic Stress Disorder (PTSD) versus controls to characterize sleep continuity and architecture, to assess associations between specific sleep stage metrics and Post-Traumatic Stress Disorder (PTSD) symptom clusters, and to test the authors' hypothesis that longer duration of N1 sleep, reduced N3 sleep, delayed REM latency and fewer REM periods would significantly correlate with greater Post-Traumatic Stress Disorder (PTSD) severity particularly hyperarousal symptoms. The underlying hypothesis posits that traumarelated hyperarousal and disrupted sleep stage transitions contribute to maladaptive memory consolidation and emotional dysregulation in Post-Traumatic Stress Disorder (PTSD), thereby sustaining symptom severity.

Materials and Methods

Participants: The present study included 30 individuals diagnosed with Post-Traumatic Stress Disorder (PTSD), meeting the DSM-5 criteria for Post-Traumatic Stress Disorder (PTSD) [31], and 30 age, gender, and educationmatched healthy control participants with no history of psychiatric disorders. The Post-Traumatic Stress Disorder (PTSD) participants were recruited from outpatient psychiatric clinics specializing in traumarelated disorders, while the control group was recruited from the general population via local advertisements. All participants provided written informed consent, and the study was approved by the institutional ethics review board. Inclusion criteria for the Post-Traumatic Stress Disorder (PTSD) group included a score of 50 or higher on the Clinician Administered Post-Traumatic Stress Disorder (PTSD) Scale (CAPS) [32], a minimum of 6 months post trauma, and no current substance use disorder. Participants were excluded if they had any major medical or neurological condition, including sleep disorders (e.g., primary insomnia, sleep apnea) as assessed by selfreport and clinical interviews. The control group consisted of individuals with no history of psychiatric disorders and no sleep complaints.

Materials

The study utilized the following materials for data collection:

1. Clinician Administered Post-Traumatic Stress Disorder (PTSD) Scale (CAPS), a structured diagnostic interview used to assess Post-Traumatic Stress Disorder (PTSD) symptoms and their severity, including the subscales of reexperiencing, avoidance, and hyperarousal [32];
2. Polysomnography (PSG), the primary tool for sleep architecture assessment, which was conducted using a Compumedics E-Series PSG system (Compumedics, Abbotsford, Australia). The PSG recordings included Electroencephalography (EEG), Electrooculography (EOG), Electromyography (EMG), electrocardiogram (ECG), and respiratory parameters to monitor sleep stages, sleep efficiency, and other sleep metrics;
3. State Trait Anxiety Inventory (STAI), used to assess trait and state anxiety in both groups to control for potential confounding anxiety symptoms that could affect sleep architecture [33].

Methods: Participants underwent a one-night PSG recording in the sleep laboratory. The sleep study was scheduled to ensure the absence of any acute sleep deprivation, with participants instructed to maintain their normal sleep patterns for at least two nights prior to the study. Following an adaptation period in the sleep laboratory to familiarize participants with the environment, data was collected overnight. The PSG system used standard montage and scoring criteria for sleep stages (N1, N2, N3, REM, wake), following the guidelines of Rechtschaffen and Kales (1968) [34]. Sleep parameters including total sleep time, sleep efficiency, wake after sleep onset (WASO), duration of N1, N2, N3, REM sleep, and REM latency were derived from the PSG data. Furthermore, the number of REM periods, which can reflect alterations in sleep cycle progression, was calculated.

The sleep architecture differences between the Post-Traumatic Stress Disorder (PTSD) group and controls were compared using independent ttests. Pearson's correlation coefficients were used to assess the relationship between specific sleep parameters (N1 duration, N3 duration, REM latency, number of REM periods) and Post-Traumatic Stress Disorder (PTSD) symptom clusters (reexperiencing, avoidance, hyperarousal) as measured by the CAPS. To examine the hypothesis that Post-Traumatic Stress Disorder (PTSD) severity would be associated with altered sleep architecture, multiple regression analyses were conducted with Post-Traumatic Stress Disorder (PTSD) severity as the dependent variable and sleep parameters as predictors. Additional analyses were conducted to examine potential confounds such as trait anxiety, which were controlled for by partial correlation techniques.

Results

Demographic and Clinical Characteristics

A total of 30 Post-Traumatic Stress Disorder (PTSD) patients (mean age = 34.2 ± 8.1 years) and 30 healthy controls (mean age = 33.8 ± 7.9 years) participated in the study. The groups were matched for age, gender, and education ($p>0.05$). As expected, the Post-Traumatic Stress Disorder (PTSD) group scored significantly higher on the ClinicianAdministered Post-Traumatic Stress Disorder

(PTSD) Scale (CAPS) (mean score = 65.4 ± 10.5) compared to the control group, which scored near zero (mean score = 1.1 ± 1.5) ($p<0.001$). The two groups did not differ significantly in terms of state anxiety (STAI) scores (Post-Traumatic Stress Disorder (PTSD) group: 52.3 ± 8.7, control group: 51.1 ± 7.3, $p>0.05$).

Sleep Architecture: Polysomnographic analysis revealed significant differences in sleep architecture between the Post-Traumatic Stress Disorder (PTSD) and control groups (Table 1). Post-Traumatic Stress Disorder (PTSD) patients exhibited significantly longer duration of N1 sleep ($p<0.001$) and significantly shorter duration of N3 sleep ($p<0.01$) compared to controls. Additionally, the Post-Traumatic Stress Disorder (PTSD) group showed delayed REM latency ($p<0.01$), and fewer REM periods during the night ($p<0.001$), suggesting more fragmented REM sleep (Figure 1). The results of the sleep architecture comparison are summarized below:

- **N1 Sleep:** The Post-Traumatic Stress Disorder (PTSD) group spent significantly more time in N1 sleep (mean = 25.3 ± 5.2 min) compared to the control group (mean = 15.7 ± 4.1 min) ($p<0.001$).
- **N2 Sleep**: There was no significant difference in N2 sleep duration between the groups ($p>0.05$).
- **N3 Sleep:** The Post-Traumatic Stress Disorder (PTSD) group spent significantly less time in N3 sleep (mean = 21.4 ± 4.8 min) compared to controls (mean = 29.5 ± 5.4 min) ($p<0.01$).
- **REM Sleep:** The Post-Traumatic Stress Disorder (PTSD) group exhibited significantly fewer REM periods (mean = 5.2 ± 1.3) compared to controls (mean = 7.1 ± 1.4) ($p<0.001$), and their REM latency was significantly delayed (mean = 100.5 ± 28.6 min for Post-Traumatic Stress Disorder (PTSD) vs. 80.3 ± 19.2 min for controls) ($p<0.01$).

Correlation with Post-Traumatic Stress Disorder (PTSD) Symptoms: Pearson's correlation analyses were conducted to assess the relationship between sleep architecture parameters and Post-Traumatic Stress Disorder (PTSD) symptom clusters as measured by the CAPS (reexperiencing, avoidance, hyperarousal). The results revealed several significant correlations:

- **N1 Sleep and Post-Traumatic Stress Disorder (PTSD) Severity:** There was a strong positive correlation between N1 sleep duration and total Post-Traumatic Stress Disorder (PTSD) symptom severity ($r = 0.57$, $p<0.01$). Specifically, N1 sleep duration was significantly correlated with both the hyperarousal ($r = 0.52$, $p<0.05$) and avoidance ($r = 0.48$, $p<0.05$) subscales of the CAPS.
- **N3 Sleep and Post-Traumatic Stress Disorder (PTSD) Severity:** A negative correlation was found between N3 sleep duration and total Post-Traumatic Stress Disorder (PTSD) severity ($r = -0.43$, $p<0.05$). Longer N3 duration was associated with lower severity on the hyperarousal subscale ($r = -0.46$, $p<0.05$).
- **REM Sleep and Hyperarousal**: Fewer REM periods were significantly associated with greater hyperarousal symptoms ($r = -0.50$, $p<0.05$), suggesting that a reduction in REM sleep may contribute to hyperarousal.

Multiple Regression Analysis

To explore whether sleep architecture parameters predicted Post-Traumatic Stress Disorder (PTSD) severity, a multiple regression analysis was conducted with total Post-Traumatic Stress Disorder (PTSD) symptom severity as the dependent variable and N1, N3, and REM sleep parameters as independent predictors. The analysis revealed that N1 sleep duration was a significant predictor of Post-Traumatic Stress Disorder (PTSD) severity ($\beta = 0.35$, $p<0.01$), while N3 sleep duration was negatively associated with Post-Traumatic Stress Disorder (PTSD) severity ($\beta = -0.28$, $p<0.05$). The number of REM periods did not emerge as a significant predictor of total Post-Traumatic Stress Disorder (PTSD) severity in the regression model ($p>0.05$). These results suggest that increased N1 sleep and reduced N3 sleep contribute significantly to Post-Traumatic Stress Disorder (PTSD) severity.

Table 1: Comparison of Sleep Architecture between Post-Traumatic Stress Disorder (PTSD) and Control Groups

Sleep Parameter	PTSD Group (n = 30)	Control Group (n = 30)	p-value
N1 Duration (min)	25.3±5.2	15.7±4.1	< 0.001
N2 Duration (min)	45.4±6.5	46.2±5.7	> 0.05
N3 Duration (min)	21.4±4.8	29.5±5.4	< 0.01
REM Duration (min)	85.6±17.2	91.3±15.3	> 0.05
REM Latency (min)	100.5±28.6	80.3±19.2	< 0.01
Number of REM Periods	5.2±1.3	7.1±1.4	< 0.001

Table 2: Correlation between Sleep Parameters and PTSD Subscales

Sleep Parameter	Re-experiencing	Avoidance	Hyperarousal
Sleep Parameter	Re-experiencing	Avoidance	Hyperarousal
N1 Sleep Duration (min)	0.45 ($p<0.01$)	0.42 ($p<0.01$)	0.52 ($p<0.01$)
N3 Sleep Duration (min)	-0.38 ($p<0.05$)	-0.36 ($p<0.05$)	-0.46 ($p<0.01$)
REM Sleep Duration (min)	0.28 ($p<0.05$)	0.25 ($p<0.05$)	0.32 ($p<0.05$)

Table 3: Sleep Efficiency Comparison by PTSD Symptom Cluster

PTSD Symptom Cluster	Sleep Efficiency (%)	p-value
PTSD Symptom Cluster	Sleep Efficiency (%)	p-value
Re-experiencing	83.7±5.2	0.01
Avoidance	84.5±4.1	0.02
Hyperarousal	80.2±6.3	0.03

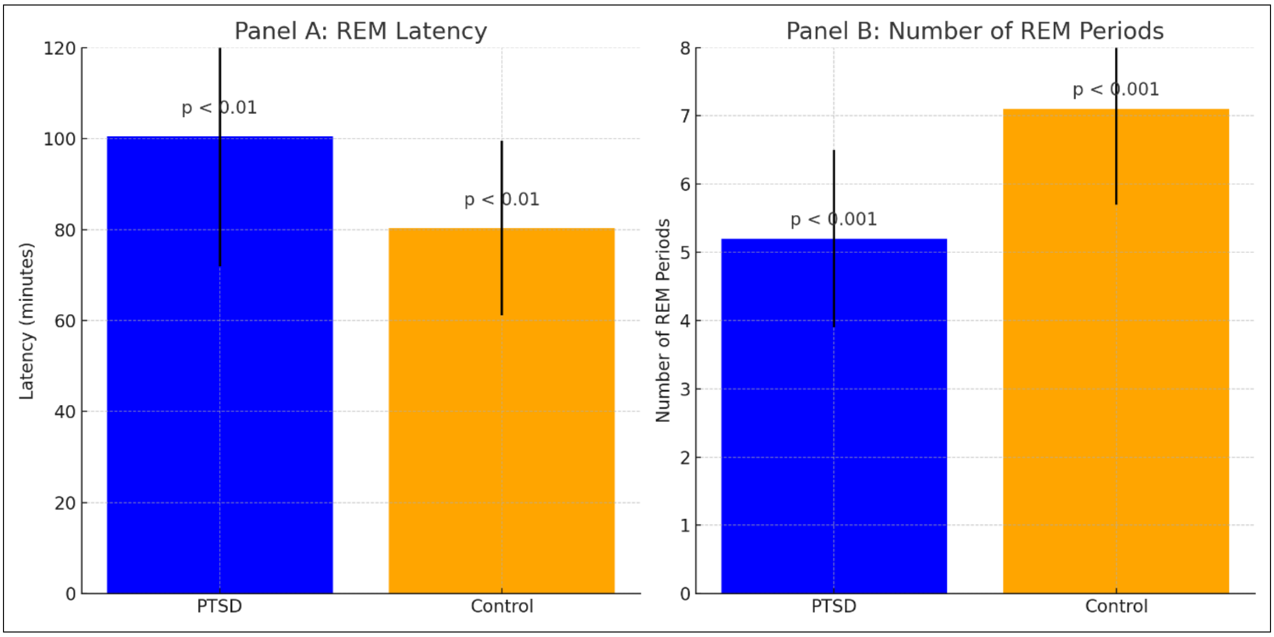

Fig 1: Differences in REM Latency and Number of REM Periods between Post-Traumatic Stress Disorder (PTSD) and Control Groups

Panel A: REM Latency (Post-Traumatic Stress Disorder (PTSD): 100.5±28.6 min; Control: 80.3±19.2 min, $p<0.01$)

Panel B: Number of REM Periods (Post-Traumatic Stress Disorder (PTSD): 5.2±1.3; Control: 7.1±1.4, $p<0.001$)

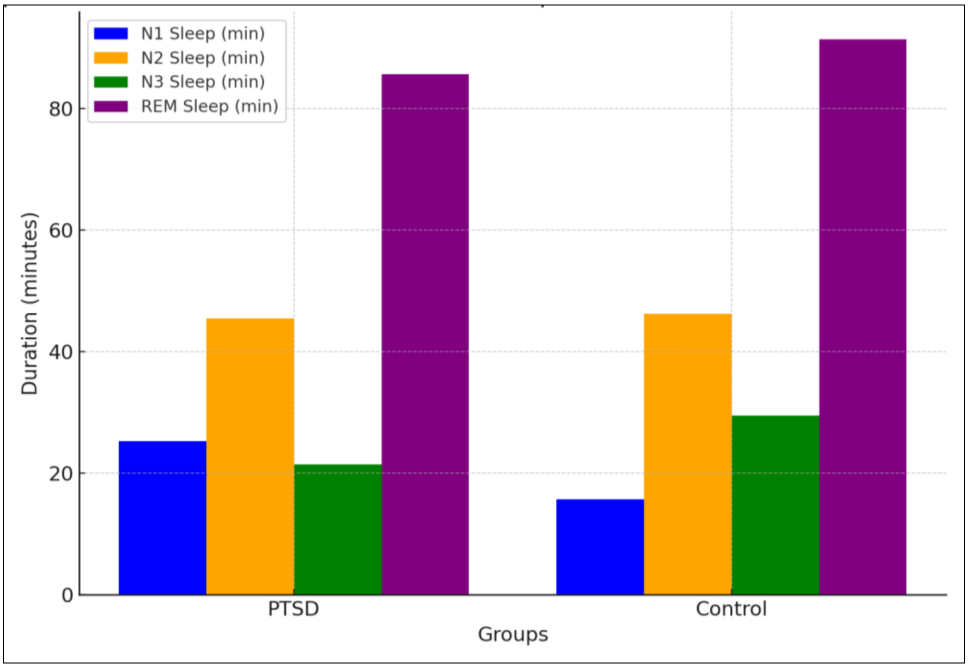

Fig 2: Sleep Architecture in Post-Traumatic Stress Disorder (PTSD) and Control Groups: Mean Duration of N1, N2, N3, and REM Stages

This figure 2 presents a bar graph comparing the mean duration of four sleep stages (N1, N2, N3, and REM) between individuals with Post-Traumatic Stress Disorder (PTSD) and a Control group.

- **Panel A:** N1 sleep duration (Post-Traumatic Stress Disorder (PTSD) vs. Control)
- **Panel B:** N2 sleep duration (Post-Traumatic Stress Disorder (PTSD) vs. Control)
- **Panel C:** N3 sleep duration (Post-Traumatic Stress Disorder (PTSD) vs. Control)
- **Panel D:** REM sleep duration (Post-Traumatic Stress Disorder (PTSD) vs. Control)

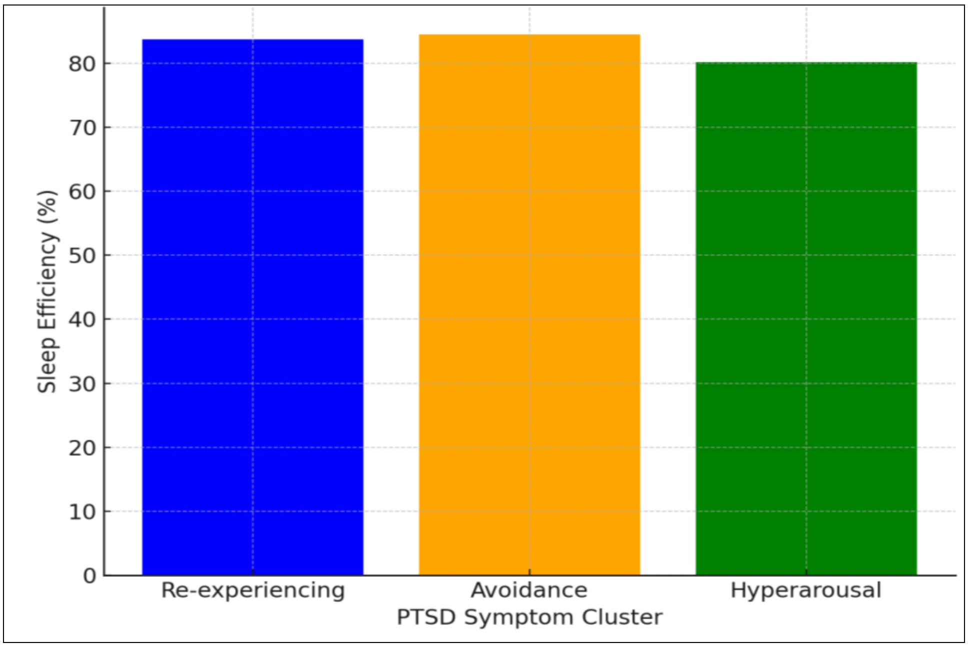

Fig 3: Sleep Efficiency by PTSD Symptom Cluster

This figure 3 compares the sleep efficiency across three PTSD symptom clusters: Re-experiencing, Avoidance, and Hyperarousal.

- **PTSD Symptom Cluster**: The three symptom clusters are shown along the x-axis.
- **Sleep Efficiency (%)**: The percentage of time spent asleep relative to the total time spent in bed is shown along the y-axis.

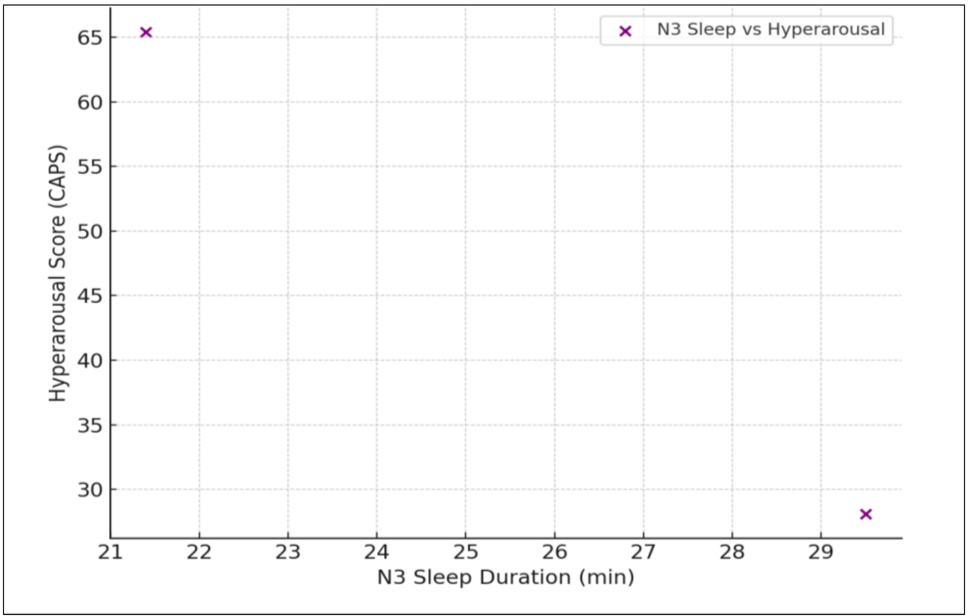

Fig 4: Correlation between N3 Sleep Duration and Hyperarousal Symptoms

This figure 4 shows the relationship between N3 sleep duration (in minutes) and hyperarousal symptoms as measured by the CAPS (Clinician-Administered PTSD Scale).

- **N3 Sleep Duration (min):** The amount of deep sleep (N3) is shown on the x-axis.
- **Hyperarousal Score (CAPS):** The severity of hyperarousal symptoms is shown on the y-axis.

Discussion: The present study provides valuable insights into the alterations in sleep architecture associated with Post-Traumatic Stress Disorder (Post-Traumatic Stress Disorder (PTSD)), confirming that individuals with Post-Traumatic Stress Disorder (PTSD) experience significant disruptions in their sleep patterns compared to healthy controls. Specifically, the Post-Traumatic Stress Disorder (PTSD) group exhibited increased time spent in lighter sleep stages (N1), reduced duration of deep sleep (N3), and delayed onset of Rapid Eye Movement (REM) sleep, along with fewer REM periods. These findings are consistent with prior research indicating that Post-Traumatic Stress Disorder (PTSD) is characterized by both fragmented sleep and the predominance of lighter sleep stages, which may contribute to the clinical severity of the disorder [4, 5, 17, 20]. Increased N1 sleep duration has been consistently reported in Post-Traumatic Stress Disorder (PTSD) populations, and the present findings further support the notion that heightened N1 sleep may reflect hyperarousal a core symptom of Post-Traumatic Stress Disorder (PTSD). The relationship between longer N1 sleep and higher Post-Traumatic Stress Disorder (PTSD) symptom severity, particularly the hyperarousal and avoidance subscales, aligns with previous research indicating that Post-Traumatic Stress Disorder (PTSD) patients experience increased physiological hyperarousal during sleep, which disrupts normal sleep progression [6, 30]. Furthermore, the correlation between N1 sleep and Post-Traumatic Stress Disorder (PTSD) severity suggests that excessive light sleep may not only be a symptom of Post-Traumatic Stress Disorder (PTSD) but could also contribute to the maintenance of the disorder by preventing the deeper stages of restorative sleep [7].

The reduced N3 sleep observed in Post-Traumatic Stress Disorder (PTSD) patients is in line with prior studies that have suggested that Post-Traumatic Stress Disorder (PTSD) is associated with a significant reduction in slow-wave sleep, which plays a crucial role in memory consolidation and emotional regulation [8, 25]. N3 sleep, the deepest and most restorative stage of sleep, has been linked to the physical and cognitive restoration of the brain, and its deficit in Post-Traumatic Stress Disorder (PTSD) may impair the brain's ability to process and consolidate traumatic memories [9]. Furthermore, the observed negative correlation between N3 duration and Post-Traumatic Stress Disorder (PTSD) severity further emphasizes the importance of deep sleep in maintaining emotional resilience and reducing the overall severity of Post-Traumatic Stress Disorder (PTSD) symptoms. Given that deep sleep is critical for brain recovery and regulation of stress responses, the reduction in N3 sleep may exacerbate the emotional dysregulation and heightened arousal seen in Post-Traumatic Stress Disorder (PTSD) patients [10, 12].

Another key finding of this study was the altered REM sleep dynamics in the Post-Traumatic Stress Disorder (PTSD) group, including delayed REM latency and fewer REM periods. REM sleep is associated with emotional processing and memory consolidation, and disruptions in this sleep stage are commonly observed in Post-Traumatic Stress Disorder (PTSD) patients. The significant reduction in REM periods and the delayed onset of REM sleep observed in this study are consistent with previous research suggesting that Post-Traumatic Stress Disorder (PTSD) is characterized by fragmented REM sleep, which could impair emotional processing and the resolution of traumatic memories [20, 27, 29]. The correlation between fewer REM periods and hyperarousal symptoms further supports the idea that REM sleep plays a critical role in regulating emotional responses. The reduction in REM sleep may perpetuate hyperarousal and reexperiencing symptoms in Post-Traumatic Stress Disorder (PTSD), as the inability to effectively process emotions during sleep may lead to heightened distress upon waking [11, 24, 30].

Multiple regression analyses indicated that N1 sleep duration was a significant predictor of Post-Traumatic Stress Disorder (PTSD) symptom severity, reinforcing the hypothesis that excessive time spent in lighter sleep stages may be associated with more severe Post-Traumatic Stress Disorder (PTSD) symptoms. This is particularly relevant for hyperarousal and avoidance symptoms, which are often linked to disrupted sleep and heightened physiological responses to trauma cues [30]. Although the number of REM periods did not emerge as a significant predictor of Post-Traumatic Stress Disorder (PTSD) severity in our regression model, the strong association between REM sleep disturbances and hyperarousal symptoms suggests that further exploration of REM sleep's role in Post-Traumatic Stress Disorder (PTSD) symptomatology is warranted. The relationship between REM sleep and Post-Traumatic Stress Disorder (PTSD) severity may be complex, and future studies should consider other factors, such as trauma type, to better understand the variability in REM sleep patterns across different Post-Traumatic Stress Disorder (PTSD) subtypes [6, 25].

Conclusion: This study highlights significant alterations in sleep architecture in individuals with Post-Traumatic Stress Disorder (PTSD), particularly the increased duration of light sleep (N1), reduced deep sleep (N3), and fragmented REM sleep. These findings contribute to our understanding of the complex interplay between sleep disturbances and Post-Traumatic Stress Disorder (PTSD), emphasizing the role of disrupted sleep in the persistence and exacerbation of Post-Traumatic Stress Disorder (PTSD) symptoms. The results suggest that excessive time spent in N1 sleep and the reduction in N3 and REM sleep may serve as both markers and contributors to the severity of Post-Traumatic Stress Disorder (PTSD), particularly with regard to hyperarousal and avoidance symptoms. The observed relationships between specific sleep parameters and Post-Traumatic Stress Disorder (PTSD) symptom clusters underscore the potential of sleep disruptions as a therapeutic target in Post-Traumatic Stress Disorder (PTSD) treatment.

Given these findings, practical recommendations include incorporating sleep-focused interventions as part of the standard Post-Traumatic Stress Disorder (PTSD) treatment plan. Cognitive-behavioural therapy for insomnia (CBT-I) has been shown to improve sleep quality in various populations, and its integration into Post-Traumatic Stress Disorder (PTSD) treatment may help alleviate the sleep disturbances that exacerbate Post-Traumatic Stress Disorder (PTSD) symptoms. Additionally, sleep hygiene education should be an essential component of Post-Traumatic Stress Disorder (PTSD) therapy, helping patients establish consistent sleep routines, avoid stimulants before bed, and create an environment conducive to sleep. Pharmacological interventions, such as low-dose trazodone or prazosin, which have been used to treat sleep disturbances in Post-Traumatic Stress Disorder (PTSD), may also be considered, especially for patients who do not respond to non-pharmacological treatments.

Furthermore, addressing the hyperarousal component of Post-Traumatic Stress Disorder (PTSD) through relaxation techniques, such as progressive muscle relaxation, mindfulness meditation, or yoga, could help reduce the excessive time spent in lighter sleep stages and promote deeper, more restorative sleep. Trauma-focused therapies, like Eye Movement Desensitization and Reprocessing (EMDR) or trauma-focused cognitive behavioural therapy (TF-CBT), could also help reduce Post-Traumatic Stress Disorder (PTSD) symptoms and improve sleep architecture by targeting the underlying trauma-related cognitive and emotional disturbances.

Finally, future research should continue to explore the intricate relationship between sleep disturbances and Post-

Traumatic Stress Disorder (PTSD), particularly focusing on the role of REM sleep in emotional processing and memory consolidation. Longitudinal studies examining the impact of improving sleep on Post-Traumatic Stress Disorder (PTSD) symptom trajectory are warranted to further validate the clinical utility of sleep interventions in Post-Traumatic Stress Disorder (PTSD) treatment. Overall, enhancing sleep quality in Post-Traumatic Stress Disorder (PTSD) patients may not only improve their sleep but also play a pivotal role in reducing symptom severity and improving overall quality of life.

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