Received: 5 August 2020 | Revised: 29 October 2020 | Accepted: 23 November 2020

DOI: 10.1111/jsr.13261

REGULAR RESEARCH PAPER

Journal of Sleep Research

Sleep and emotion processing in paediatric posttraumatic stress disorder: A pilot investigation

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Funding information
Dr Benca receives consulting fees from Eisai, Genomind, Jazz and Merck. None of these funding sources had a direct effect in the design, analysis or interpretation of the study results, or in preparation of the manuscript.

Summary

Emotion processing abnormalities and sleep pathology are central to the phenomenology of paediatric posttraumatic stress disorder, and sleep disturbance has been linked to the development, maintenance and severity of the disorder. Given emerging evidence indicating a role for sleep in emotional brain function, it has been proposed that dysfunctional processing of emotional experiences during sleep may play a significant role in affective disorders, including posttraumatic stress disorder. Here we sought to examine the relationship between sleep and emotion processing in typically developing youth, and youth with a diagnosis of posttraumatic stress disorder . We use high-density electroencephalogram to compare baseline sleep with sleep following performance on a task designed to assess both memory for and reactivity to negative and neutral imagery in 10 youths with posttraumatic stress disorder, and 10 age- and sex-matched non-traumatized typically developing youths. Subjective ratings of arousal to negative imagery (ΔArousal = post-sleep minus pre-sleep arousal ratings) remain unchanged in youth with posttraumatic stress disorder following sleep (mean increase 0.15, CI −0.28 to +0.58), but decreased in TD youth (mean decrease −1.0, 95% CI −1.44 to −0.58). ΔArousal, or affective habituation, was negatively correlated with global change in slow-wave activity power (ρ = −0.58, p = .008). When considered topographically, the correlation between Δslow-wave activity power and affective habituation was most significant in a frontal cluster of 27 electrodes (Spearman, ρ = −0.51, p = .021). Our results highlight the importance of slow-wave sleep for adaptive emotional processing in youth, and have implications for symptom persistence in paediatric posttraumatic stress disorder. Impairments in slow-wave activity may represent a modifiable risk factor in paediatric posttraumatic stress disorder.

KEYWORDS

emotion processing, high-density electroencephalogram, memory, paediatric posttraumatic stress disorder, slow-wave activity

Stephanie Jones and Anna Castelnovo contributed equally to this manuscript

1 | INTRODUCTION

Approximately 5% of youth will develop paediatric posttraumatic stress disorder (pPTSD) by the age of 18 years, with prevalence estimates rising to 40% in cases of sexual or physical abuse (McLaughlin et al., 2013). While early intervention has the potential to reduce enduring psychosocial dysfunction, current treatments for pPTSD are suboptimal, leaving a significant portion of youth unrecovered. Novel, effective treatments are urgently required, but the development of such treatments requires more research into the basic mechanisms involved in the development and maintenance of PTSD, as well as factors that impact treatment response.

Both emotion processing abnormalities and sleep pathology are central to the phenomenology of PTSD across the lifespan. Dysfunctions in emotion processing—including heightened reactivity to, biased processing towards, and diminished habituation responses to aversive stimuli—have been implicated in the development and maintenance of PTSD (Lissek & van Meurs, 2015; Seligowski et al., 2015). Additionally, up to 90% of those with PTSD report sleep disturbance, and sleep pathology has been linked to the development, maintenance and severity of the disorder (Kovachy et al., 2013). Given the bidirectional relationship between sleep and emotional brain function, it has been proposed that dysfunctional processing of emotional experiences during sleep may play a significant role in affective disorders, including posttraumatic stress disorder (Goldstein & Walker, 2014).

Recently, theoretical conceptions of sleep and emotion processing have suggested that sleep helps reset the brain to an emotionally optimized baseline each day. According to the sleep to forget, sleep to remember hypothesis, rapid eye movement (REM) sleep in particular serves to actively strip emotional memories of their associated emotional intensity while simultaneously strengthening the informational content (Walker & van der Helm, 2009). In support of the role of REM in regulating reactivity, subjective ratings of emotional arousal in response to scared and angry faces was reported to decrease across an afternoon nap containing REM sleep, and increase across a similar sleep interval that did not contain REM (Gujar, McDonald, Nishida, & Walker, 2011). In a related study, participants who viewed emotional imagery before sleep reported reduced subjective arousal and diminished amygdala responses to the same imagery the following day (van der Helm et al., 2011). Decreased amygdala reactivity was found to be greatest in participants with low prefrontal cortical gamma (30–40 Hz) during REM sleep, supporting a specific role for REM sleep in the process of emotional habituation. However, although these data support a role for REM sleep in the process of affective habituation—"the process by which emotional stimuli become less impactful over time"—whether REM sleep exclusively facilitates this process remains unclear. Indeed, some evidence suggests that REM sleep may serve to reinforce reactivity to emotionally salient stimuli rather than support habituation (Baran et al., 2012). Evidence suggests that non-REM (NREM) slow-wave sleep (SWS) may also play a significant role in overnight habituation. For example, using a split night design, Wagner attempted to isolate the distinct influences of REM and SWS on memory consolidation and emotional habituation, and found that emotional arousal *decreased* across early-night SWS relative to REM, while arousal increased slightly across REM periods and wake. Other recent studies also support a broader role for NREM sleep in emotional memory and affective restructuring. For example, overnight emotional attenuation following the viewing of a stressful film was shown to be associated with the percent of SWS early in the night, (Talamini et al., 2013) and NREM sleep during a daytime nap enhanced electromyographic habituation to negative stimuli (Pace-Schott et al., 2011).

Given the centrality of both sleep and emotion processing disruptions to the pathophysiology of PTSD, in this study we sought to examine these phenomena for the first time in youth and to explore which features of sleep, if any, are most strongly associated with emotional processing in this population. We use high-density (hd) electroencephalogram (EEG) to compare a baseline night of sleep with a night of sleep following performance on a task designed to assess both memory for and reactivity to negative and neutral imagery in a small sample of youth with diagnosis of PTSD and age- and sex-matched non-traumatized typically developing (TD) youth. Based on the above-mentioned literature, we hypothesized that youth with PTSD would show: (a) heightened reactivity to emotional imagery during the pre-sleep encoding relative to their TD counterparts; (b) that reactivity to previously viewed emotional imagery the following day would remain unchanged in youth with PTSD, and that this failure to downscale emotion reactivity would be related to microstructural changes in REM or SWS. Due to the lack of prior research in this population and our small sample size, our study was exploratory in nature, and all findings should be considered preliminary.

2 | METHODS

2.1 | Participants

The sample consisted of 10 age- and sex-matched healthy non-traumatized TD youth and 10 youth with PTSD between the ages of 10 and 18 years. Youth with PTSD were recruited from local mental health facilities as part of a longitudinal study on the neural correlates of PTSD (Keding & Herringa, 2015; Wolf & Herringa, 2016). Non-traumatized TD youth matched for age and sex were recruited from the community. A comprehensive clinical battery was used to assess all participants for past and current psychopathology and trauma exposure using the following assessment tools: Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS; Kaufman et al., 1997); Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994); Mood and Feelings Questionnaire (MFQ; Costello and Angold, 1988); Screen for Child Anxiety-Related Emotional Disorders (SCARED; Birmaher et al., 1997); Stressful Life Events Schedule Adolescent Report (SLES; Williamson et al., 2003); and Weschler Abbreviated Scale

of Intelligence-II (Wechsler, 2011) in order to assess IQ. For the clinical group, a PTSD diagnosis was determined using DSM-IV criteria by combination of the KSADS and Clinician-Administered PTSD Scale for Children and Adolescents (CAPS-CA; Weathers et al., 2001). Youth in the clinical group also completed the UCLA PTSD Reaction Index (PTSD-RI; Steinberg et al., 2004). Exclusion criteria for all participants included IQ < 70 or unstable medical condition. Additional exclusion criteria for youth with PTSD included active suicidality, history of psychotic disorder, bipolar disorder or OCD; recent (past 4 weeks) substance abuse or dependence; and recent use of psychotropic medication (past 4 weeks; 6 weeks for fluoxetine). TD subjects were free of any history of mental or neurological illness. The University of Wisconsin Health Sciences Institutional Review Board approved all procedures.

2.2 | Procedure

The study consisted of two counterbalanced overnight sleep visits to the laboratory (baseline, task) separated by a 1–3-week interval. On the task night, all youth performed an emotional learning task immediately before sleep, and again within 1 hr of natural wake time following an ad libitum interval of sleep the next morning. A schematic and description of the emotional learning task, adapted from Prehn-Kristensen et al. (2013), is shown in Figure S1. Stimuli were 248 pictures taken from the International Affective Picture System (IAPS; Lang et al., 2008). Based on normative IAPS data and previous studies, pictures were separated into two valence categories: "Negative" and "Neutral". During the pre-sleep portion of the task, participants viewed 140 target stimuli: 70 negative images intermixed with 70 neutral images. Each trial began with the appearance of a fixation cross (500 ms) followed by the target picture (1,500 ms). The Self-Assessment Manikin Scale for Arousal (SAM; Bradley & Lang, 1994) appeared after the target image, and participants were asked to rate the degree of emotional arousal they experienced while processing the picture by choosing one of nine responses (ranging from 1 = very low to 9 = very high). Subjects were informed that they would be asked to recall images in the morning. In the post-sleep phase, youth viewed 200 images (100 negative and 100 neutral). This set included 92 of the previously viewed images (targets) intermixed with 108 novel images (foils). Participants again assessed arousal, and indicated whether they recognized the picture from the previous encoding session ("old") or not ("new").

2.3 | Sleep recordings

On the baseline night, subjects' sleep was evaluated with standard polysomnographic equipment for the diagnosis of sleep disorders along with hdEEG (256 channels). On the task night, hdEEG (Electrical Geodesics) was recorded, along with electromyogram and electrooculogram. Sleep was scored by a registered sleep technologist according to AASM guidelines (Berry et al., 2012), and reviewed by a board-certified sleep physician. hdEEG processing methods were identical to those described in Jones et al. (2014). All data processing and statistical analyses were performed using MATLAB.

2.4 | Behavioural data

To assess affective habituation, our dependent variable was the average change in subjective arousal rating to target images (ΔArousal = subjective arousal rating during the post-sleep recognition phase minus arousal rating during the pre-sleep encoding phase). A negative score indicates images were rated as less arousing in the post-sleep compared with pre-sleep phase. Because each subject provided a rating for each image, we used a mixed-effects linear model to assess the significance of any group (PTSD or TD youth), valence (negative versus neutral image type), or group by valence interaction effects on ΔArousal while controlling for the possible random effects of individual subjects or specific images: ΔArousal ~ Group * Valence + (1|Subject) + (1|PictureID). For memory, recognition accuracy was calculated using "hit rate" (the number of times a subject correctly identified a target image as having been seen before; e.g. correctly responded "old"), as well as "false alarms" (the number of times a subject incorrectly identified a foil as having been seen before; e.g. incorrectly said "old"). Mixed-effects linear models considered hit rate and false alarm separately as functions of valence and group with error terms for subject and picture ID: thus, the models HitRate ~ Group * Valence + (1|Subject) + (1|PictureID) and FalseAlarm ~ Group * Valence + (1|Subject) + (1|PictureID) were both evaluated. Because each subject provided arousal ratings and an old/new determination for 200 images in the morning, 92 of which were repeated from the previous night, our sample size of 10 PTSD and 10 TD youth was sufficient to detect potential group by image valence interactions on memory and affective habituation using the mixed-effect linear models, even though the same sample was not sufficiently powered to detect group by night (baseline versus task night) interactions in sleep variables.

2.5 | Sleep

This study was designed to assess within-group changes in sleep microstructure between a baseline night of sleep and a night following an emotional learning task, and was not sufficiently powered to address between-group differences in sleep macrostructure or microstructure. As such, changes in sleep macrostructure and all-night absolute spectral power density (μV^2) were considered separately for each group using paired *t*-tests (rather than using an ANOVA design to assess possible group by night interactions). Absolute topographical power maps in NREM and REM sleep were analysed using statistical non-parametric mapping (SNPM), using a supra-threshold cluster test (*t*-value threshold = 2) of all possible combinations (n = 1,024) to identify significant clusters of electrodes (Nichols &

Holmes, 2002). After choosing an appropriate threshold *t*-value (for consistency a *t*-value threshold = 2 was chosen for all frequency ranges), topographic power maps were randomly shuffled for each group between nights (baseline versus task) in all possible combinations. The size of the largest contiguous cluster above the threshold for each reshuffling was then used to create a maximal cluster size distribution. The supra-threshold cluster *p*-value was then determined by comparison of the actual cluster size (the cluster above threshold for the real subject grouping) against the maximal cluster size distribution.

2.6 | Sleep and behaviour correlations

To assess the relationships between sleep and affective habituation and sleep and memory, we correlated ΔArousal and hit rate with within-subject percentage change in power density between nights [Δpower = (task − baseline) * 200/(task + baseline)] for all frequency bands. Power and Δpower for each electrode were correlated with both ΔArousal and hit rate using Pearson's correlation coefficient and displayed on topographic maps. To control for multiple comparisons, significant correlations were determined again using a SNPM cluster test (cluster threshold of $r = .47$, with 50,000 permutations). We also did a post hoc confirmatory analysis of the significant cluster by averaging Δpower across the electrodes included in the cluster and ΔArousal using a non-parametric Spearman rank correlation. Although we attempted to correct for the problem of multiple comparisons where necessary (e.g. across topographical images), it should also be noted that we did not strictly correct for the issue of multiple testing given the small sample size and exploratory nature of this study.

3 | RESULTS

Participant characteristics are summarized in Table 1. The groups did not significantly differ in sex distribution, age, pubertal stage or handedness as assessed by unpaired *t*-tests. IQ was lower in PTSD youth (mean ± *SD*, PTSD 100.50 [± 3.42]; TD, 113.30 [± 3.49]; $p = .017$). Within the PTSD group, the most common index trauma was sexual abuse, followed by witnessing violence and traumatic death of a loved one. PTSD symptoms averaged 44.7 based on the PTSD-RI, which is indicative of severe PTSD.

TABLE 1 TD and PTSD youth demographics

	TD	PTSD
N	10	10
Age (years)	14.67 ± 0.94 range: 10.00–17.92	14.52 ± 0.95 range: 10.00–17.54
Tanner stage	3.10 ± 0.39 range: 1–5	3.23 ± 0.46 range: 1–5
IQ	113.30 ± 3.49 range: 97–139	100.50 ± 3.42 range: 89–121
Left-handed (*n*)	0	0
Index trauma (*n*)	–	Sexual abuse (3), witnessing violence (3), traumatic death of loved one (2), accident (2), physical abuse (1)
Comorbid diagnoses (*n*)	–	Major depressive disorder (4), ADHD (4), generalized anxiety disorder (3), separation anxiety disorder (2), social anxiety disorder (1), conduct disorder (1), social phobia (1)
PTSD duration (months)	–	36.70 (± 7.72)
PTSD-RI	–	44.70 (± 6.04)
CAPS-CA	–	60.45 (± 8.00)
MFQ	4.40 (± 0.79)	21.75 (± 3.13)
SCARED	7.65 (± 1.65)	32.89 (± 6.01)
Past psychiatric medication	–	Stimulant (3), antidepressant (3), benzodiazepine (1)

The sample consisted of 10 healthy non-traumatized youth and 10 youth with PTSD between the ages of 10 and 18 years. PTSD and TD youth were age-matched within ± 3 months.

The healthy and PTSD groups did not significantly differ in sex distribution, age, Tanner stage, or handedness. Numbers in parentheses with '±' represent *SEM*.

CAPS-CA, Clinician-Administered PTSD Scale for Children and Adolescents; MFQ, Mood and Feelings Questionnaire; PTSD, posttraumatic stress disorder; PTSD-RI, posttraumatic stress disorder reaction index; SCARED, Screen for Child Anxiety-Related Emotional Disorders; TD, typically developing.

3.1 | Overnight change in arousal shows distinct pattern in PTSD and TD youth

Although the distribution of arousal rating seems to differ between groups (Figure S2), on average, subjective arousal scores during pre-sleep encoding were similar for both groups (mean ± *SD*, TD negative 5.84 ± 2.27, neutral 2.49 ± 1.91; PTSD negative 5.42 ± 3.19, neutral 1.89 ± 1.66), as were the post-sleep arousal scores (mean ± *SD*, TD negative 4.72 ± 2.26, neutral 1.77 ± 1.19; PTSD negative 4.79 ± 3.17, neutral 1.60 ± 1.42). The average change in arousal scores showed a significant group by valence interaction ($\chi^2 = 9.4364$, $p = .002127$). As shown in Figure 1(a), the TD group significantly decreased average arousal to negative images (mean decrease −1.0, 95% CI −1.44 to −0.58) and to neutral images (mean decrease −0.74, CI −1.17 to −0.32), indicating that habituation had occurred. In contrast, responses in PTSD youth remained effectively unchanged, to both negative (mean increase

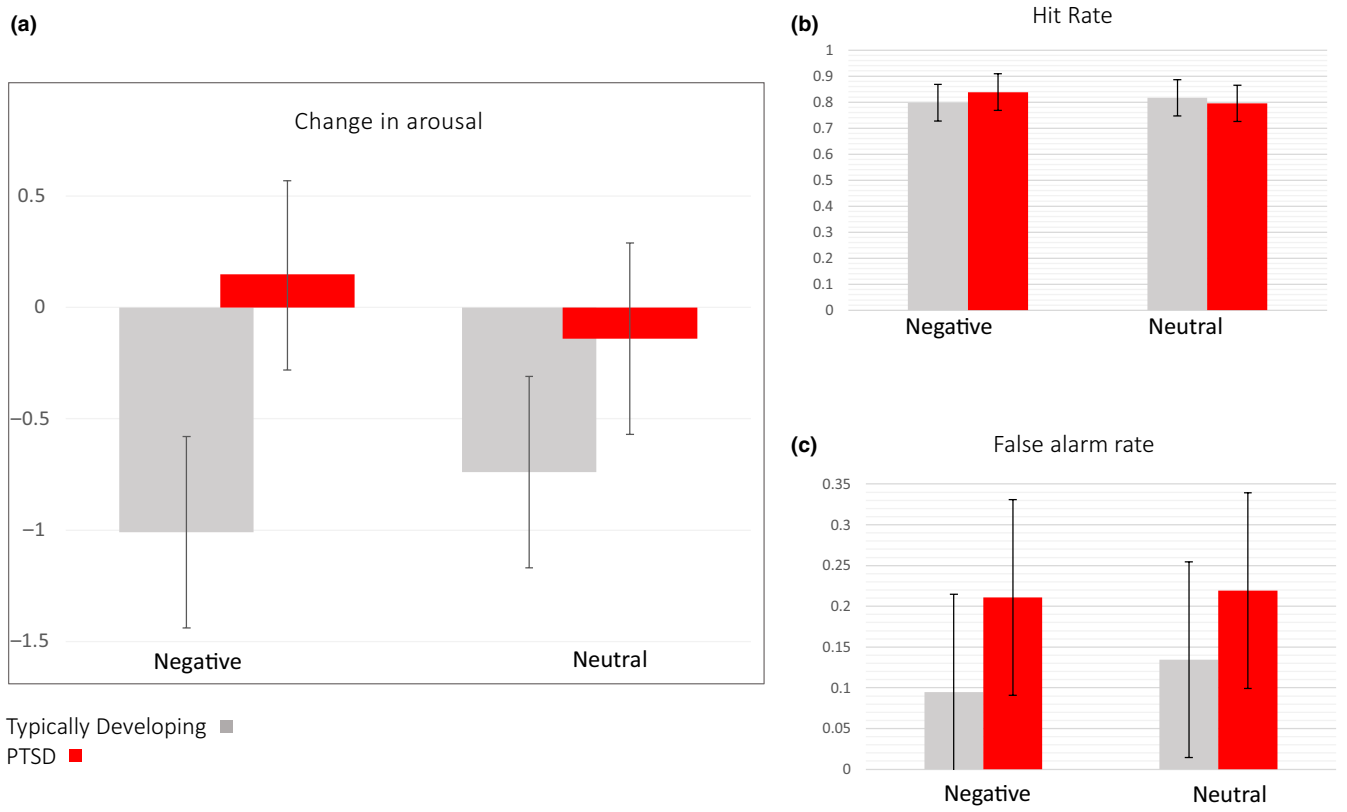

FIGURE 1 Overnight change in affective habituation, but not memory, distinguishes posttraumatic stress disorder (PTSD) and typically developing (TD) youth. Hit rate (percentage of old pictures correctly labelled as "old") and false alarms (percentage of new pictures incorrectly labelled as "old") shown as a function of valence (emotional and neutral) in TD (grey) and PTSD (red) youth. (a). Mean values of hit rate for emotional (left) and neutral (right) images did not differ significantly by group ($\chi^2 = 0.0391$, $p = .8432$) or by valence ($\chi^2 = 0.2036$, $p = .6518$), and there was no significant groups by valence interaction ($\chi^2 = 3.2367$, $p = .07201$). (b) Mean values of false alarm as a function of valence did not differ significantly by group ($\chi^2 = 1.4767$, $p = .2243$) or by valence ($\chi^2 = 1.5823$, $p = .2084$), and there was no group by valence interaction ($\chi^2 = 1.371$, $p = .2416$). (Error bars represent 95% confidence interval.) (c) Mean overnight change in subjective arousal ratings highlight distinct response patterns in PTSD and TD youth. ΔArousal (morning−evening) shown as a function of group (TD and PTSD) and valence category (Emotional and Neutral). A negative score indicates images were rated as less arousing in the post-sleep phase. As shown on right side of (c), the TD group (shown in grey) significantly *decreased* average arousal to emotional images (mean decrease −1.0, 95% CI −1.44 to −0.58). In contrast, responses in PTSD youth to emotional images remained effectively unchanged (mean increase 0.15, 95% CI −0.28 to + 0.58), suggesting that, after a period of sleep, PTSD youth did not habituate to the emotional content of the imagery. TD youth also significantly decreased responses to neutral images (mean decrease −0.74, CI −1.17 to −0.32). PTSD youth arousal to neutral images was also effectively unchanged (mean decrease −0.14 CI −0.57 to 0.29). (Error bars represent 95% confidence interval)

0.15, CI −0.28 to +0.58) and neutral images (mean decrease −0.14 CI −0.57 to +0.29).

3.2 | Memory recall does not differ in PTSD and TD youth

As shown in Figure 1(b), mean values of hit rate (negative images PTSD = 0.83 ± 0.10, TD = 0.79 ± 0.09; neutral images PTSD = 0.79 ± 0.15, TD = 0.81 ± 0.07) did not differ significantly by group (χ^2=0.0391, p = .8432) or by valence (χ^2=0.2036, p = .6518). Similarly, mean values of false alarm (negative images PTSD = 0.2107 ± 0.28; TD = 0.09 ± 0.08; neutral images PTSD = 0.21 ± 0.23; TD = 0.13 ± 0.09) did not differ significantly by group ($\chi^2 = 1.4767$, $p = .2243$) or by valence ($\chi^2 = 1.5823$, $p = .2084$), and there was no group by valence interaction ($\chi^2 = 1.371$, $p = .2416$; Figure 1c).

3.3 | Sleep macrostructure

Paired *t*-tests revealed no significant differences between task and baseline nights in the PTSD or TD groups on any variable of sleep macrostructure, with one exception: in the TD group, wake after sleep onset (WASO) was significantly higher on the baseline night relative to the task night, suggesting that TD youth had more consolidated sleep following the presentation of images on the task night. The same was not true for the PTSD group. Macrostructural variables for both the task and baseline recordings are summarized for each group in Table 2.

3.4 | Sleep microstructure

3.4.1 | Changes in REM sleep from baseline to task night

As shown in Figure 2(a), both groups showed evidence of changes in high-frequency power during REM sleep, albeit in opposite directions. In PTSD youth, a significant increase in high frequency (22.5–27 Hz, 29.6–33 Hz, 33–37 Hz, $p = .048, .009, .036$) was observed during the sleep following task performance relative to baseline sleep (Figure 2a). In contrast, in TD youth, a small increase in the gamma range (27.8–29.6 Hz, $p < .039$) was observed on the baseline night relative to the task night (Figure 2a). Despite the global increases in high-frequency activity, no consistent topographic changes in EEG power emerged in either group.

3.4.2 | Changes in NREM sleep from baseline to task night

Both groups showed the classical pattern of all-night NREM spectral activity (Figure 2b), with greatest power in the slow-wave frequency band (1–4.5 Hz) and a second peak in the sigma band (12–15 Hz). As shown in Figure 2(b) when averaged across channels, PTSD youth showed a significant increase in high-frequency activity in the beta/gamma range (19–39 Hz, $p < .003$ Hz) on the task night relative to the baseline night, as well as a significant decrease in the slow-wave activity (SWA) range (0.5–2.5 Hz, $p < .026$). In TD youth, a broad band decrease in high-frequency beta/gamma (16–36 Hz, $p < .002$) was observed on the task night relative to the baseline night (Figure 2b).

TABLE 2 Macrostructural sleep variables

Measure	Group	PSG night	Task night
AHI	PTSD	2.03 (0.74)	
	TD	2.93 (0.55)	
TST	PTSD	436.49 (33.92)	425.61 (38.52)
	TD	452.42 (19.59)	451.79 (19.91)
WASO	PTSD	64.35 (14.17)	75.20 (17.19)
	TD	45.50 (4.76)	36.10 (4.38)
AI	PTSD	11.00 (1.03)	11.01 (1.10)
	TD	10.95 (0.89)	11.35 (1.42)
SE	PTSD	90.84 (1.81)	89.73 (2.27)
	TD	93.19 (0.71)	94.44 (0.63)
N1%	PTSD	3.64 (0.87)	4.00 (0.64)
	TD	4.05 (0.87)	2.73 (0.55)
N2%	PTSD	54.61 (1.49)	47.47 (2.32)
	TD	57.09 (1.60)	58.23 (0.98)
N3%	PTSD	22.95 (2.12)	20.79 (2.21)
	TD	21.93 (2.25)	20.44 (1.92)
REM%	PTSD	18.81 (1.93)	17.72 (2.17)
	TD	16.96 (1.61)	18.59 (1.83)
REML	PTSD	159.85 (18.10)	164.65 (32.73)
	TD	141.15 (21.60)	135.30 (14.55)

Mean values (± standard error of the mean, $n = 10$ per group). Percentage values for sleep stages are expressed per total sleep time (TST).

AHI, apnea–hypopnea index; AI, arousal index; PSG, polysomnography; PTSD, posttraumatic stress disorder; REM, rapid eye movement; REML, rapid eye movement onset latency; SE, sleep efficiency; TD, typically developing; TST, time in bed; WASO, wake after sleep onset.

3.4.3 | Topographical changes in NREM SWA and gamma in PTSD youth

As shown in Figure 2(c), in PTSD youth, a large cluster of electrodes ($N = 110$, $p = .036$) was reduced in the SWA band during NREM sleep on the task night relative to the baseline night, consistent with the observed global change in low frequency spectral power. In addition to the marked decrease in SWA, a cluster of right-lateralized posterior electrodes ($N = 20$, $p = .011$) was significantly increased in the gamma band during NREM on the task night in PTSD youth (Figure 2d). In TD youth, no significant regional differences were observed in any frequency band (Figure 2a,b, EEG topography).

3.4.4 | Correlation of sleep microstructure with affective habituation

Despite the robust differences between PTSD and TD youth in gamma power changes between the task and baseline nights, we identified no relationship between NREM or REM gamma changes with average ΔArousal for negative images. However, we did find a negative correlation between average ΔArousal for negative images and global changes in NREM SWA power ($\rho = -0.58$, $p = .008$). A topographic investigation identified a significant negative correlation between ΔSWA power in a frontal cluster of electrodes (27 channels, $p = .045$) and ΔArousal, such that as SWA power decreased (% decrease on task night relative to baseline night) emotional arousal failed to normalize (failed to change from a high to a low value) as shown on the left of Figure 3. When ΔSWA power was averaged across electrodes in the significant cluster, a Spearman correlation confirmed a robust relationship with ΔArousal ($\rho = -0.51$, $p = .021$), as shown in the scatterplot on the right of Figure 3. Data shown are for negative images that were correctly remembered, but the results were similar when considering all negative images.

4 | DISCUSSION

To our knowledge, this study represents the first report of sleep EEG assessment in pPTSD. There are two notable findings. First, relative to a pre-sleep baseline, youth with PTSD show no affective habituation to negative imagery, while TD youth do—yet the two groups have similar factual recall for these images. Second,

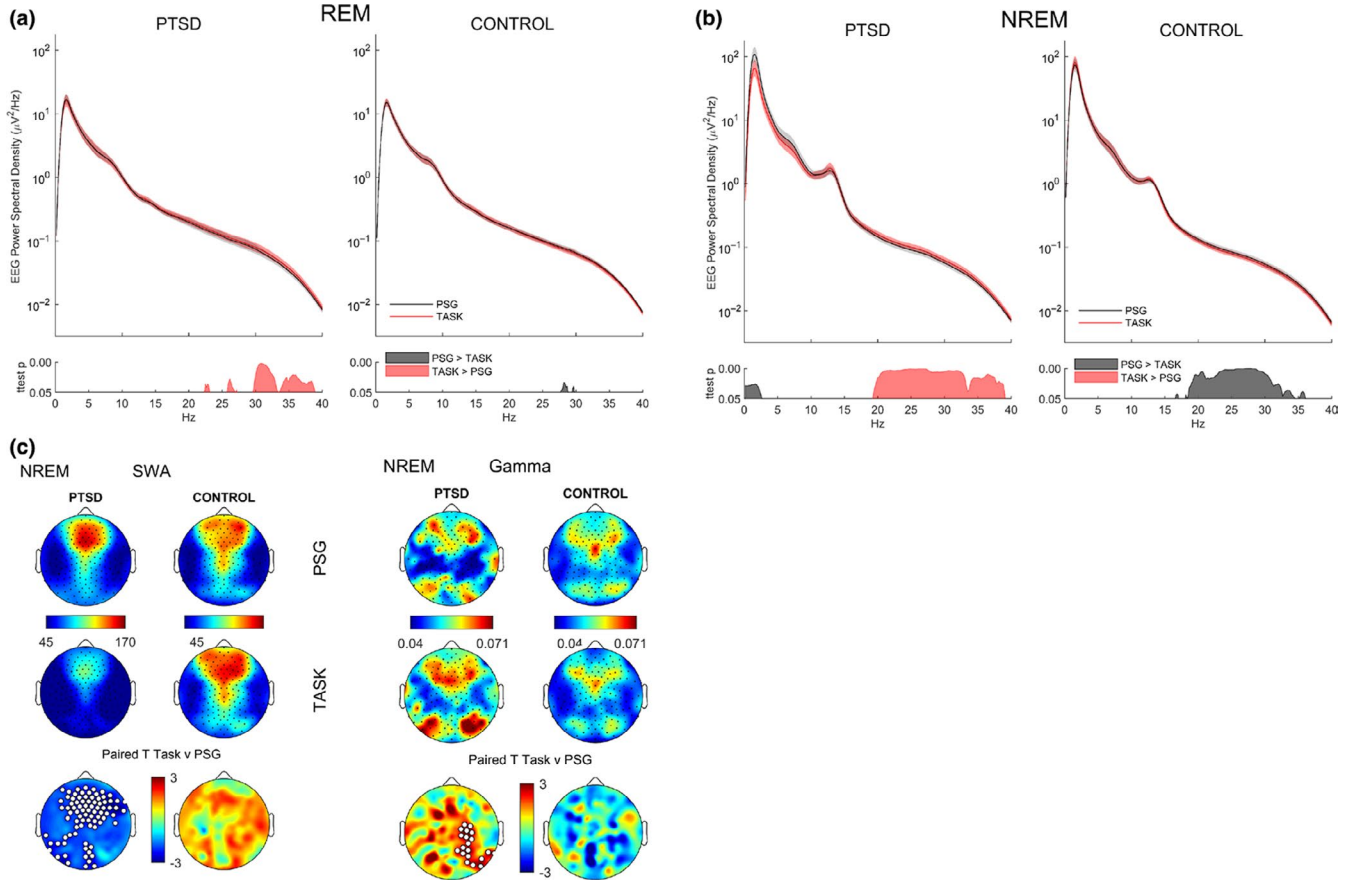

FIGURE 2 Analysis of sleep microstructure reveals distinct patterns of sleep in typically developing (TD) and posttraumatic stress disorder (PTSD) youth after task performance. (a and b) Spectral analysis of all-night electroencephalogram (EEG) power during rapid eye movement (REM) and non-(N)REM sleep on the task night versus the baseline night in PTSD and TD youth. (a) Highlights opposite changes in high-frequency power during REM sleep in both groups. In PTSD youth, a significant increase in high-frequency activity is evident during REM following task performance relative to baseline sleep; while in TD youth, a small decrease in the gamma range is evident. (b) Highlights a similar pattern of high-frequency increase in PTSD and decrease in TD youth during NREM sleep on the task night relative to the baseline night. In PTSD youth, a decrease in slow-frequency activity is also evident on the task night. In contrast, in TD youth increased high-frequency activity is present on the *baseline* night relative to the task night, suggesting more consolidated sleep after task performance. Spectral density plots for the global average across all electrodes in (a) NREM and (b) REM sleep for PTSD (left) and TD (right) youth. Uncorrected *p*-values for the comparison between task night (black) and baseline night (red) are shown below each plot, respectively. (c and d) Topographical analysis of NREM sleep EEG in PTSD youth reveals a broadly distributed decrease in slow-wave activity (SWA) on the task night relative to the baseline night as well as a regional increase in gamma EEG power. (c) Top: average NREM sleep EEG topographies in SWA (1–4.5 Hz) for PTSD and TD youth on baseline (polysomnography) night. Middle: average NREM SWA for PTSD and TD youth on task night. Lower: topographic distribution of the change in SWA during NREM sleep between the baseline and the task night. Blue values represent a decrease on EEG power on the task night relative to the baseline night. White dots indicate the cluster of 27 electrodes showing decreased SWA on the task night ($p < .01$, statistical non-parametric mapping, supra-threshold cluster test controlling for multiple comparisons). (d) Top: topographical averages for NREM gamma (25–40 Hz) for PTSD and TD youth on baseline. Middle: topographical averages of each group for gamma on task night. Lower: topographic distribution of the change in gamma power during NREM sleep between the baseline and the task night. Red values represent an increase in EEG power on the task night relative to the baseline night. White dots indicate the cluster of 20 electrodes showing an increase in power on the task night ($p < .01$)

this post-sleep impairment in affective habituation was unrelated to macrostructural sleep patterns in youth with PTSD as these did not change from the baseline to the task night. Third, impairments in post-sleep affective habituation were not associated with any feature of REM sleep but were correlated with reductions in SWA, particularly over frontal regions. Together, these findings point to novel cortical sleep mechanisms that may underlie enhanced reactivity to threat and potentially threat-extinction impairments in pPTSD.

Robust differences were evident when considering the changes in sleep following task performance relative to a baseline night of sleep in each of the groups separately. In PTSD youth, sleep depth and quality was markedly impaired following task performance as indexed by a significant global decrease in SWA and a significant increase in high-frequency beta/gamma activity suggestive of cortical arousal (Fernandez-Mendoza et al., 2016). It is reasonable to speculate that the viewing of the IAPS images before sleep was ultimately responsible

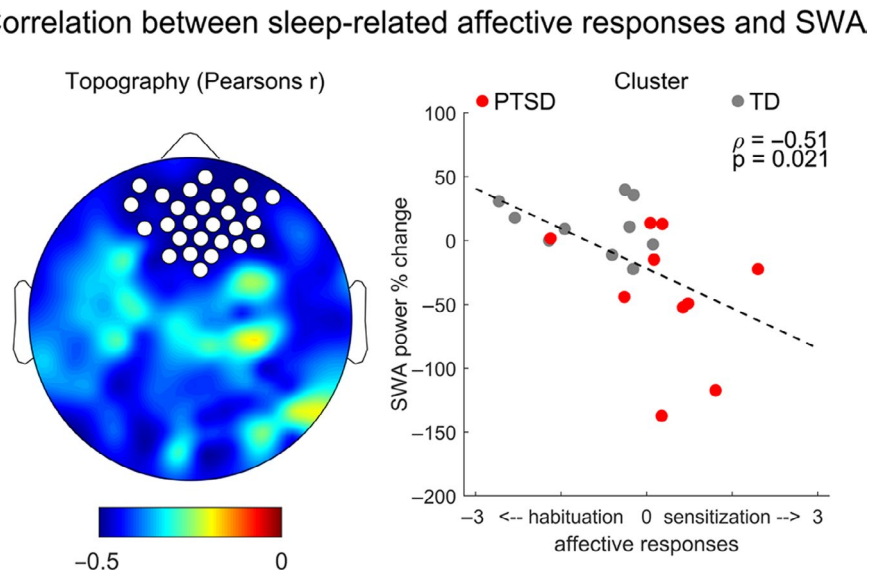

FIGURE 3 Topographical correlation between affective habituation and slow-wave activity (SWA). A decrease in SWA is associated with a failure of affective habituation (Left). A frontal cluster of channels in the SWA band (white dots) is significantly correlated (statistical non-parametric mapping [SNPM] cluster threshold of $r = -.47$, $N = 21$) with affective arousal such that as SWA decreased on the task night relative to the baseline night (negative % change values), affective habituation did not occur (failed to change from a high to a low value; $\rho = -0.51$, $p = .021$). Right: a scatter plot showing coefficient of correlation (r) between arousal and SWA power (μV^2) in significant cluster (white dots indicate cluster of 27 electrodes showing decreased SWA on the task night; $p < .01$, SNPM, supra-threshold cluster test controlling for multiple comparisons). Red dots (posttraumatic stress disorder; PTSD) and grey dots (typically developing; TD) represent an individual subject's % SWA change and the average overnight change in affective habituation

for the decrease in sleep quality and sleep depth following task performance in PTSD youth. Indeed, emotional distress and negatively valenced imagery before sleep has deleterious effects on subjective as well as objective sleep quality (Kahn et al., 2013). Notably, however, when considering the subjective rating of arousal during the pre-sleep encoding period, there was no evidence that the images were any more distressing to PTSD compared with TD youth.

In the morning after a period of sleep, TD youth habituated images in both valence categories such that an image viewed as arousing during the pre-sleep encoding was rated as significantly less arousing after sleep. In contrast, PTSD youth did not reduce subjective arousal ratings to either valence category. Given that a global change in SWA was correlated with affective habituation, one possible interpretation of this relationship is that in PTSD youth the images proved disruptive to sleep, and this reduction in sleep depth and quality then exacerbated next day emotional reactivity, which appears as a failure to habituate. Indeed, both naturalistic and experimental evidence indicates that inadequate sleep in both adults and youth is associated with increased negative affect, amplified responsivity to aversive stimuli, and diminished emotion regulation capacity and mood disturbance (Palmer & Alfano, 2017). In addition to these behavioural effects, the functional integrity of the networks subserving emotion are also impaired following sleep loss or restriction. One night of sleep restriction leads to a 60% amplification of amygdala activation in response to negative emotional stimuli relative to a rested condition, along with a concomitant decrease in medial prefrontal cortical activity, a region known to exert top-down control of the amygdala (Goldstein & Walker, 2014; Yoo et al., 2007). Importantly, this failure of affective habituation appears to be specifically related to SWA and not to a general impairment in sleep quality and/or elevated arousal. For example, gamma power during sleep, generally considered an index of a more "wakeful" sleep and an index of elevated central adrenergic activity (Cape & Jones, 1998), was robustly elevated in PTSD youth on the task night relative to the baseline night, and reduced in TD youth. However, neither the change in gamma power in NREM from baseline to task, nor REM gamma on the task night was related to the change in emotional reactivity. Because we do not have additional assessments of emotional reactivity (e.g. skin conductance), it is not possible to disentangle whether we are seeing a failure to habituate across the sleep period, or if the lack of habituation is related to a general higher level of reactivity to aversive stimuli. The pattern of responses to never before seen emotional images in PTSD youth in the morning was remarkably similar to that seen in the pre-sleep period, but the significance of this is unclear.

When considering the correlation between SWA and affective habituation, the relationship was strongest in a cluster of electrodes over the frontal cortex. In light of this regional relationship, we speculate that impaired affective habituation and the regional decrease in SWA in PTSD youth may arise as a consequence of reduced engagement of prefrontal cortical regions during stimulus encoding. This, in turn, may result in a reduced need for SWA and its associated plasticity functions during the subsequent sleep period. SWA is broadly associated with synaptic remodelling and cortical plasticity (Tononi & Cirelli, 2014). SWA increases or decreases in cortical

regions that are more or less active, respectively, during previous waking in animals (Vyazovskiy et al., 2011) as well as in humans (Huber et al., 2004). Functional magnetic resonance imaging studies of adults with PTSD, and other psychiatric disorders featuring anxiety, consistently demonstrate hypoactivity of prefrontal regions implicated in cognitive-emotional control during emotion processing tasks (Pitman et al., 2012). Our group and others have found similar evidence in pPTSD, including reduced coupling between the amygdala and medial prefrontal cortex during viewing of threat imagery (Herringa, 2017; Keding & Herringa, 2015; Wolf & Herringa, 2016).

Given the theoretical and, to a lesser extent, experimental work supporting a role for REM sleep in emotional brain function (Goldstein & Walker, 2014), and the prevailing view that REM sleep is prominently disrupted in PTSD (Germain, 2013), the lack of correlation with REM power, yet robust correlation between SWA power and affective habituation, was unexpected. However, although the role of REM sleep in emotional memory has been widely explored, the specific role sleep plays in subjective reactivity to emotional stimuli and overnight habituation has received less attention, and the existing data do not support an exclusive role for REM in reducing emotional reactivity. Indeed, REM sleep may reinforce reactivity to emotionally salient stimuli rather than support adaptive reappraisal (Baran et al., 2012; Talamini et al., 2013; Werner et al., 2015). Moreover, a number of studies support a role for SWS in the adaptive affective reappraisal of complex stimuli (Hauner et al., 2013; Kleim et al., 2014; Kobayashi et al., 2016). In clinical samples, reduced SWS following written narrative exposure therapy for PTSD was associated with diminished therapeutic efficacy (Kobayashi et al., 2016), while increased SWS predicted success of exposure psychotherapy for simple phobia (Kleim et al., 2014).

With respect to memory, we did not detect a performance difference between PTSD relative to TD youth for either valence type. At first glance, this lack of an emotional memory advantage in PTSD seems surprising in light of work showing that individuals with PTSD have a tendency to encode negative or neutral stimuli as subjectively more aversive. However, although some data support an emotional memory advantage in PTSD relative to comparison groups, a number of studies report no memory differences despite underlying differences in neural activation during encoding (Hayes et al., 2012). Given the sample size, however, we were not powered to fully consider group differences, so these findings should be considered in that context. We did not find a relationship between any feature of sleep microstructure and recognition memory for stimuli in either valence category. Despite some evidence in adults that specific features of NREM and REM may aid declarative and emotional memory, respectively (Diekelmann et al., 2009), data in youth are both scant and discrepant (Bolinger et al., 2018; Prehn-Kristensen et al., 2009, 2017). Of note, this task was not optimized to test memory effects because we did not have a method of controlling for encoding strength, nor did we use short-term memory baseline after encoding. Our findings, albeit preliminary, suggest that sleep function may play a preferential role in affective reappraisal, but not factual encoding, for emotional imagery.

5 | LIMITATIONS

A central strength of this study includes the use of an emotional learning task combined with sleep hdEEG, which allows for a regional analysis of neural activity not possible with traditional polysomnography studies. Our analyses were conducted in an unmedicated, otherwise healthy population of PTSD and TD youth, suggesting that results are not confounded by factors related to medication or sleep and/or medical disorders. However, the sample size used in these analyses was modest, which increases the risk of false-positives and may overestimate the magnitude of effects. We attempted to mitigate this issue by using a within-subjects design to assess sleep and the use of non-parametric statistical methods, which make few assumptions about the distribution of the data and are more robust to outliers. Additionally, the study was not sufficiently powered to explore group by night interactions on sleep variables, so we cannot determine if the sleep changes observed between task and baseline nights were significantly different between groups. As such, the results of this study, while novel and of potential clinical importance, should be considered preliminary and replicated in larger samples of youth. Finally, our study did not include a trauma-exposed comparison group without PTSD, which will be important in future work to assess specificity of findings to PTSD versus trauma exposure per se.

6 | CONCLUSION

The relationship between sleep SWA and affective dysregulation in youth with PTSD highlights the importance of SWS for adaptive emotional processing, and has potentially broad-ranging implications for understanding the persistence of symptoms in PTSD. Viewed more directly, these data also have potential relevance for the success and/or timing of therapeutic interventions. For example, in vivo exposure therapy requires a child to face a feared stimulus for a sustained period while experiencing heightened feelings of arousal. Habituation to a feared stimulus, the goal of exposure therapy, may be less likely to occur if the underlying cortical sleep mechanisms are not acting to downscale the affective content of memory. Given significant inter-relationships between sleep, PTSD and emotion processing and their respective and combined contributions to functional impairment, a deeper understanding of how one impacts the other has the potential to inform the development of novel sleep-focused therapies for the treatment of PTSD in youth. Indeed, if these data are replicated in a larger sample, slow-wave enhancement may represent a promising interventional tool.

ACKNOWLEDGEMENT

The authors would like to sincerely thank Sydney Notterman for her help with recruitment and data collection for this study.

CONFLICT OF INTEREST

The authors have no conflicts relevant to this manuscript.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

How to cite this article: Jones S, Castelnovo A, Riedner B, et al. Sleep and emotion processing in paediatric posttraumatic stress disorder: A pilot investigation. *J Sleep Res*. 2021;30:e13261. https://doi.org/10.1111/jsr.13261