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Original Article

Early childhood adversity associations with nightmare severity and sleep spindles☆

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A B S T R A C T

Objective: Childhood adversity figures prominently in the clinical histories of children and adolescents suffering from a panoply of physical, mental or sleep disorders, including posttraumatic stress disorder. But the nature and prevalence of early adversity in the case of idiopathic nightmare-prone individuals have received little study. We characterize the types and frequencies of self-reported childhood adversity for nightmare-prone individuals using the developmentally sensitive Traumatic Antecedents Questionnaire (TAQ) and assess relationships between *separation adversity* and sleep spindles.
Method: The TAQ was administered to 73 non-treatment-seeking volunteers with frequent idiopathic nightmares and 67 healthy controls. Nightmare severity, anxiety, depression, alexithymia and past and present sleep disorders were also assessed. Sleep was recorded with polysomnography (PSG) for 90 participants and sleep spindles were assessed for 63.
Results: Nightmare-prone participants scored higher on most TAQ measures, including adversity at 0–6 years of age. TAQ-derived scales assessing traumatic and nontraumatic forms of adversity were both elevated for nightmare-prone participants; for 0–6 year estimates, nontraumatic adversity was associated with nightmares independent of trauma adversity. Group differences were only partially mediated by current psychopathology symptoms and were largely independent of nightmare frequency but not of nightmare distress. Adversity/nightmare relationships were graded differentially for the two study groups. Separation adversity at 0–6 years of age correlated with current sleep spindle anomalies—in particular, lower slow spindle density—an anomaly known to index both psychopathology and early nightmare-onset.
Conclusions: Self-reported adversity occurring as young as 0–6 years of age is associated with nightmare severity and sleep spindle anomalies. Adversity-linked nightmares may reflect pathophysiological mechanisms common also to the nightmares of pre-clinical and full-blown post-traumatic stress disorder.

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1. Introduction

Idiopathic nightmares—frequent dysphoric dreams that have no known cause—are a common REM sleep parasomnia with an approximately 5% prevalence [1,2]. These nightmares often begin in early childhood, rise in prevalence through adolescence and decrease again through adulthood, even though they remain more prevalent among females. They are considered to be distinct from other DSM5 psychiatric disorders, but nevertheless constitute a risk factor for post-traumatic stress disorder (PTSD) and suicidal behavior, and are comorbid with anxiety, depression, and insomnia (for review see Ref. [1]).

1.1. Nightmares and early adversity

Just as traumatic experiences frequently lead to the trauma-replication nightmares of PTSD [3], adverse childhood experiences have been linked with later nightmares [4–6]. One longitudinal study (N = 6050) [5] found that nightmares assessed prospectively by the mother when the child was 2.5, 3.5, 4.8 and 6.8 years of age were associated with abuse and family adversity assessed between birth and age four. An even more striking retrospective study (N = 5020) [6] found that more nightmare-prone adults than controls had endured maternal separations longer than a month as infants. Such studies signal that even idiopathic nightmares may be influenced by early childhood adversity; maternal separation is a particularly troubling precursor that both human and animal research suggests influences later brain development and pathology [7].

1.2. A critical period for emotional maturity

One recent theory, the Stress Acceleration Hypothesis of nightmares [4], suggests that early adversity triggers a cascade of neural events that leads to later nightmares. Adversity may interfere with a critical period of brain plasticity occurring around ages 3 to 4—when amnesia for early life experiences normally sets in. According to this theory, adversity presumably ‘accelerates,’ or temporally advances, infantile amnesia, producing unusually good memory for some events prior to age four, including an unwanted infiltration of early dysphoric feelings and memory fragments into nightmares. This approach explains findings from both epidemiological [8] and clinical [9] studies that nightmare-prone adults have better memory for experiences prior to age four than do those with few nightmares. However, most studies that have examined adversity in relation to nightmares have assessed neither the specific age of adversity onset nor the many types of adversity potentially affecting young children and thus do not address an etiological role for this early critical period.

1.3. The Traumatic Antecedents Questionnaire

Both the age of onset and the most common types of adversity are assessed by the TAQ [10,11], a self-report instrument that quantifies eight common adversity domains (eg, *neglect*, *separation*) for each of four age ranges (*0–6*, *7–12*, *13–18*, *19+*). Validation studies [12,13] show strong correlations between the TAQ and a) the more common Childhood Trauma Questionnaire [12], b) a diagnosis of PTSD [13], and c) gray matter density in limbic/paralimbic regions [13].

In the present study, we used the TAQ to assess nightmare-prone individuals and age- and sex-matched comparison participants. To quantify possible separate links to traumatic and non-traumatic forms of adversity, we derived two new TAQ measures, the Traumatic Adversity (TA) and Non-Traumatic Adversity (NTA) scales. These were comprised of items tapping either traumatic or non-traumatic adverse experiences as described by the DSM-IV PTSD Criterion A. This distinction was drawn on the basis of the suggestion that idiopathic and post-traumatic nightmares stem from similar underlying mechanisms but vary in the severity of their expression [1]; on this basis, less severe idiopathic nightmares might be expected to result from less severe adverse events while more severe post-traumatic nightmares result from adverse events that are more clearly traumatic. We recognize that items comprising the NTA scale may nonetheless be subjectively traumatic to an individual depending upon both Internal factors, such as inherited and learned resilience, and external factors, such as event context and available social support. Conversely, such factors may also determine that a given DSM-specified trauma may not be traumatic in all cases. In the absence of standardized instruments for quantifying these multi-factorial influences on subjective trauma, we consider the development of our TA and NTA TAQ subscales as an initial exploratory step in attempting to distinguish among degrees of trauma severity.

1.4. Sleep spindle indicators of adversity

Sleep spindles are an EEG hallmark of non-rapid eye movement (NREM) sleep, ie, waxing-and-waning, 0.5–3.0 s duration oscillations in the 10–16 Hz range occurring predominantly in stages N2 and N3 sleep (for review see [14]). They are age-related [15], demonstrating clear early developmental changes, eg, abrupt increases in density between ages 3–4 [16]. They predict children's emotional-behavioral development [17] and are linked to neurodevelopmental disorders of anxiety [18], schizophrenia [19] and depression [20]. Early changes in spindle characteristics, such as lower density at age five, robustly predict later developmental changes, such as positive emotional and behavioral characteristics at age nine [17]. We demonstrated, in nightmare participants, relationships between sleep spindle characteristics and pathological indicators such as anxiety and depression [21]. Two spindle features in particular, slow spindle density and fast spindle frequency, clearly discriminated nightmare participants from controls [21], especially individuals for whom nightmares had started younger than age ten [22]. Accordingly, spindle anomalies among adults may reflect the effects of early childhood adversity. Here, we assess these two sensitive sleep spindle markers (slow density; fast frequency) in relation to early adversity in nightmare-prone and control participants. In light of the finding that early parental separation correlates with adult nightmares [6], we expected that these markers would correlate with self-reported parental separation in the earliest TAQ age range (0–6 years).

1.5. Hypotheses

We tested four hypotheses encompassing ten primary endpoints about how nightmare-prone individuals will differ from controls in adversity history and in how adversity and sleep spindles are related. 1) they will report a greater lifetime adversity, independent of comorbid psychopathology; 2) they will report more adversity at ages 0–6, especially for non-traumatic adversity; 3) relationships between adversity and nightmare severity (recall frequency, distress) will be graded, independent of psychopathology, and more robust for distress than for recall frequency; and 4) sleep spindle anomalies—slow density and fast frequency—will be associated with early (ages 0–6) separation for nightmare-prone individuals. Second, we explored relationships between groups and the TAQ domain scores (lifetime, ages 0–6 only).

2. Method

2.1. Participants

Men and women aged 18–50, drawn from our laboratory database, who participated in studies of nightmares and were recruited by advertisements, posters or word-of-mouth, were screened using a telephone questionnaire. They were excluded if they reported any current major sleep disorder (other than nightmares), psychiatric disorder, neurological disease, major medical problem, medication use, drug/alcohol abuse, trauma, recent death of a loved one, language deficit, legal conflict, recent (last six months) night shift work or trans-meridian travel. Participants were self-declared to be neither clinical patients nor seeking treatment.

Participants had previously been recruited for four different research studies of nightmare-prone individuals. Three of these studies involved laboratory PSG during a morning nap, and one involved a waking state brain imaging procedure. All required participants to complete a questionnaire battery containing psychopathology measures that assess difficulties in emotion regulation frequently comorbid with nightmare severity, ie, depression, anxiety, and alexithymia. Alexithymia, in particular, has been shown by our group and others (review in Ref. [23]) to correlate in a graded fashion with nightmare distress. The four study cohorts included: 1) 42 participants (21 NM [11F], 21 CTL [13F]) undergoing a pre-sleep word association task; 2) 28 participants (14 NM [10F], 14 CTL [9F]) undergoing a pre-sleep word association task and a semantic priming task; 3) four participants (4 NM [3F]) undergoing transcranial alternating current stimulation (tACS) during REM sleep; and 4) 66 participants (34 NM [28F], 32 CTL [23F]} undergoing waking state, single-photon emission computed tomography (SPECT) imaging but no laboratory PSG.

Participants in the final nightmare (NM) group (N = 73; 52 F) reported ≥2 non-substance-induced nightmares/week for ≥6 months; the control (CTL) group (N = 67; 45 F) reported <1 nightmare/month for ≥5 years. Groups were matched for age, sex and language preference; age did not differentiate the total sample (NM: 24.5 ± 4.19 y; CTL: 23.9 ± 4.28 y; $t_{138} = -0.735$, $p = 0.463$) or males (NM: 24.9 ± 4.17 y; CTL: 25.5 ± 4.39 y; $t_{41} = 0.455$, $p = 0.651$) or females (NM: 24.3 ± 4.22 y; CTL: 23.2 ± 4.06 y; $t_{95} = -1.320$, $p = 0.190$) considered separately. Participants completed informed consent forms approved by the *CIUSSS-NIM–Hôpital du Sacré-Coeur de Montréal* ethics committee and were given a modest financial compensation.

Subgroups of 49 NM participants (32F; 24.3 ± 3.73 y) and 41 CTL participants (26F; 23.6 ± 3.94 y; age: $t_{88} = 0.780$, $p = 0.438$) were polysomnographically recorded during a 2-hr morning nap. Of these, 63 participants from studies one and two above (study one: 20 NM, 19 CTL; study two: 14 NM, 10 CTL) had results from sleep spindle analysis that were available for inclusion; two and four CTL participants respectively were dropped from spindle analyses because of technical difficulties. There was no difference in age between these NM (N = 34; 21F: 23.85 ± 3.6 y) and CTL (N = 29; 18F; 23.90 ± 3.8 y) groups ($t_{61} = 0.047$, $p = 0.963$).

2.2. Procedures

Participants first completed 1-week home sleep/dream logs. At the laboratory, they completed questionnaires and a protocol with either negative picture viewing and brain imaging but no PSG [24] or associative memory tasks with PSG and REM sleep dream collection. Task results are reported elsewhere [25,26].

Home sleep/dream logs: Daily logs, the best prospective measure of sleep timing and nightmare severity [27], were collected with an interactive voice mail acquisition system [28]; participants called and keyed in responses to recorded questions about their previous night of sleep and dreaming (~5 min/call).

2.3. Questionnaires

One hundred forty participants completed the Traumatic Antecedents Questionnaire (TAQ; [11]), the Beck Depression Inventory-II (BDI-II; [29]), and the Sleep Disorders Questionnaire-Abbreviated version (SDQ-A; [30]). From the SDQ-A, four items dealing with whether participants, as children, experienced nightmares, bad dreams, bizarre dreams or sleepwalking were rated on 5-point scales (*1 = never*, *2 = rarely*, *3 = sometimes*, *4 = usually*, *5 = often*). One hundred thirty-nine participants completed the State-Trait Anxiety Inventory (STAI; [31]) and 69 the Toronto Alexithymia Scale (TAS; [32]) with its three component subscales, Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF) and Externally Oriented Thinking (EOT). Nightmare severity was assessed with retrospective nightmare frequency and distress measures: one item assessing #times/week recalling nightmares (dysphoric dreams that awaken from sleep) and the Nightmare Distress Questionnaire (NDQ; [33]). Secondary measures included retrospective items *bad dreams* (dysphoric dreams that do not awaken) and non-nightmare dreams/week frequencies, whether nightmares began after a past event/when that event occurred, and prospective measures of nightmare distress (from home logs; mean distress ratings (5-point scales) during and after dreams).

Traumatic Antecedents Questionnaire (*TAQ*). The TAQ [11] uses 40 self-report items to assess lifetime and age-specific exposure to diverse traumatic and non-traumatic adverse experiences (Table 1). Each TAQ item is rated by participants with five response options (*0 = never or not at all*; *1 = rarely or a little bit*; *2 = occasionally or moderately*; *3 = often or very much*; *DK = don't know*) for four age ranges: early childhood (0–6 years), middle/late childhood (7–12 years), adolescence (13–18 years), and adulthood (19+ years); lifetime scores include all four age ranges (0–19+ years). Each domain score is calculated as the average of designated items rated >1 (excluding DK responses); total scores within or across age ranges are calculated as a sum of these >1 averages. Each of eight adversity domains—*neglect*, *separation*, *emotional*, *physical*, *sexual*, *witnessing adversity*, *exposure to alcohol/drugs*, *other*—and two resilience domains—*competence*, *safety* (not assessed here)—are calculated for each of four age ranges providing 32 scores plus eight domain lifetime scores. *Global (lifetime)* score is calculated as the sum of the *domain lifetime* scores.

Two new subscales assessed traumatic vs. non-traumatic forms of adversity. The Trauma Adversity (TA) subscale contained 17 items reflecting experiences fitting DSM-IV PTSD Criterion A trauma, eg, *I saw dead bodies*; *I was beaten, kicked or punched by someone close to me*; *Someone forced me to have sex against my will*. The Non-Trauma Adversity (NTA) subscale contained 17 items reflecting adverse experiences not identifiable as traumatic in the DSM-IV, eg, *My parents were divorced or separated*; *The rules in my family were unclear and inconsistent*; *I abused alcohol and/or drugs*. Both subscales are calculated as the sum of items that were rated >1 and provide scores per age range plus a *lifetime* total. The subscales thus add ten scores.

To limit the number of group comparisons from the TAQ battery, our hypotheses specify ten primary endpoints (Table 1), ie, *lifetime* and *0–6* scores for *global*, *TA* and *NTA* and correlations with *Separation* at four age ranges. Because most score distributions were non-Gaussian, natural logs (score+1) were calculated for each. Significance thresholds were set at $p < 0.01$ for hypotheses and $p < 0.05$ for secondary exploratory analyses of TAQ domain scores.

Table 1
Scale and Age Range composition of the Traumatic Antecedents Questionnaire.

Adversity domains	By age[a]	Items in domain scale[b]	#items Ch/Ad[c]	#vars[d]
Global (G)	lifetime, 0–6, 7–12, 13–18, 19+	2[f],6,7,9–11[f],12–20[f], 21–27[f],28–40	35/31	5
1. Neglect	lifetime, 0–6, 7–12, 13–18, 19+	2[f],6,7,21,27[f]	5/3	5
2. Separation	lifetime, 0–6, 7–12, 13–18, 19+	10,11[f],12,14	4/3	5
3. Emotional	lifetime, 0–6, 7–12, 13–18, 19+	9,16–19	5/5	5
4. Physical	lifetime, 0–6, 7–12, 13–18, 19+	28–30	3/3	5
5. Sexual	lifetime, 0–6, 7–12, 13–18, 19+	35–38	4/4	5
6. Witnessing	lifetime, 0–6, 7–12, 13–18, 19+	20[f],22–24,31,34	6/5	5
7. Substances	lifetime, 0–6, 7–12, 13–18, 19+	25,26	2/2	5
8. Other	lifetime, 0–6, 7–12, 13–18, 19+	13,15,32,33,39,40	6/6	5
Trauma adversity (TA)[e]	lifetime, 0–6, 7–12, 13–18, 19+	13,15,20[f],23,24,28–39	17/16	5
Non-trauma adversity (NTA)[e]	lifetime, 0–6, 7–12, 13–18, 19+	6,7,9–11[f],12,14,16–19,21,22,25–27[f],40	17/15	5

[a] Primary endpoints of present study in underlined text.
[b] Items and domains from the Traumatic Adversity Questionnaire Scoring Instructions, public version (www.traumacenter.org/products/instruments.php); Competence (items #3,4) and Safety (items #1,5,8) domains were excluded.
[c] Number of items comprising childhood (Ch: 0–6, 7–12, 13–18)/adult (Ad: 19+) domains.
[d] Number of distinct variables in domain.
[e] TA and NTA scales are new to the present study.
[f] Item not used for adult (19+) age range.

Sleep recordings. The subset of 90 participants (49 NM; 41 CTL) who underwent PSG slept in a bedroom with audio-visual surveillance and 2-way intercom. Biosignals included EEG, EMG and four electrooculogram (EOG) leads to evaluate sleep stages. In addition to electrode derivations for standard PSG, we recorded four EMG leads (chin, corrugator supercilii, dominant arm and leg), and three ECG leads to monitor heart rate. Recordings were accomplished with a Grass M15 Neurodata Acquisition System (−6 dB filters with cutoffs at 0.30 and 100 Hz) and were archived under the control of Stellate Harmonie 5.4 software (Natus Medical Inc., San Carlos, CA). PSG tracings were scored according to current American Academy of Sleep Medicine standards [34] by an experienced PSG technician. Standard sleep variables (REM min, %REM, NREM min, %NREM, TST) were calculated with in-house software. Dream reports were collected and self-rated by participants after awakenings but are not considered further here.

Sleep spindle detection. Spindles were recorded from F3, F4, C3, C4, Cz, O1, and O2 all referenced to A2 and referenced offline to A1+A2. Due to the presence of artifacts for more than 80% of the nap in five participants, C3 and C4 derivations were excluded twice each (resulting in a total of $N = 61$), and F4 and O2 derivations were excluded once each (resulting in a total of $N = 62$) from spindle detection. Raw digitized signals were bandpass-filtered from 11 to 16 Hz using a linear phase finite impulse response (FIR) filter (−3 dB at 11.1 and 15.9 Hz). Forward and reverse filtering was performed to obtain zero phase distortion and double the filter order. The root mean square (RMS) of the filtered signal was then calculated with a 0.25-s time window and thresholded at the 95th percentile (Martin et al., 2013). A spindle was identified when at least two consecutive RMS time-points exceeded this threshold, and the spindle duration met the criterion of 0.5 s. Spindle frequency was calculated as the number of zero-crossings divided by time in seconds. Because our analyses showed a clear division between slow and fast spindle distributions in the 12.70–12.80 Hz bin, a cut-off of 12.8 Hz was used to distinguish slow (10.0–12.79 Hz) from fast (12.8–16.0 Hz) spindles. Spindles were assessed in the EEG recordings of 34 NM (21F) and 29 CTL (18F) participants (sex ratio: $\chi^2 = 0.98$, $p = 1.00$ Fisher exact). Spindles were detected automatically on six artifact-free derivations (F3, F4, C3, C4, O1, O2; reference: A1+A2) for all scored epochs of NREM stage N2. Per-derivation spindle densities were computed as the count of slow or fast spindles divided by time (min) in artifact-free N2 in the corresponding channel (Supplementary Methods).

2.4. Statistical analyses

TAQ *global (lifetime)* and *global (0–6)* primary endpoints were assessed with 2 × 2, Group (NM, CTL) x Sex, ANOVAs/ANCOVAs holding significant confounders constant. *Domain (lifetime)* and *domain (0–6)* secondary endpoints were assessed with 2 × 2, Group x Sex, MANOVA/MANCOVA with eight *domain* scores as multiple dependent measures; univariate tests assessed individual *domain* scores. Spearman correlations assessed graded associations between TAQ and spindle measures. TA and NTA scores were assessed with one-way MANOVAs with Group as an independent variable, *TA (lifetime)*, *TA (0–6)*, *NTA (lifetime)*, and *NTA (0–6)* scores as a multivariable, and logNM or NDQ as covariables. A corrected p-value threshold of $p < 0.01$ was selected for significance testing.

3. Results

3.1. Demographics

As shown in Table 2, the NM group gave higher retrospective estimates of nightmare and bad dream recall, nightmare-induced distress ($p < 0.05$), frequencies of nightmares, bad dreams, bizarre dreams and sleepwalking as children ($p < 0.005$) and higher prospective estimates of distress during and after dreams ($p < 0.001$). They also scored higher on all pathological indicators ($p < 0.05$) except TAS-DDF and TAS-EOT.

Sleep Structure. Groups did not differ (Table 3).

3.2. Adversity: lifetime

A Group (NM, CTL) by Sex ANOVA with *global (lifetime)* adversity as dependent measure revealed a Group effect ($F_{1,136} = 17.379$, $p < 0.00006$, $eta^2 = 0.113$) showing higher NM (1.322 ± 0.497) than CTL (0.916 ± 0.497) means, but no Sex effect ($F_{1,136} = 1.067$, $p = 0.304$, $eta^2 = 0.008$) or Group x Sex interaction ($F_{1,136} = 0.375$, $p = 0.541$, $eta^2 = 0.003$). The Group effect was not eliminated by covarying BDI-II, TAS, and DIF scores ($F_{1,62} = 10.590$, $p = 0.002$, $eta^2 = 0.146$).

A Group by Sex MANOVA with eight *domain (lifetime)* dependent measures produced a multivariate Group effect (Hotelling's $T = 0.185$, $F_{8,129} = 2.978$, $p = 0.004$, $eta^2 = 0.156$) and no Sex or Group x Sex effects (both $p > 0.185$). Fig. 1A shows five univariate Group effects (*Emotional*: $p = 0.003$; *Physical*: $p = 0.0004$; *Witnessing*: $p = 0.008$; *Substances*: $p = 0.003$; *Other*: $p = 0.007$) and

Table 2
Comparisons between Control and Nightmare groups on dream, nightmare and psychopathology measures.

Measure	Control Group		Nightmare Group		Group N (CTL/NM)	t	*p*
	Mean	*SD*	Mean	*SD*			
Age							
Total sample	23.93	4.28	24.45	4.19	67/73	−0.74	0.463
Sleep recorded sample	23.63	3.94	24.09	3.63	41/49	−0.78	0.438
Spindle analysis sample	23.90	3.80	23.85	3.60	29/34	0.05	0.963
Retrospective recall/distress:							
Dreams/week	3.64	2.02	5.64	2.51	67/73	−5.16	**<0.001**
Bad dreams/week	0.23	0.36	2.66	1.38	67/73	−14.03	**<0.001**
Nightmares/week	0.03	0.08	1.22	1.22	67/73	−7.99	**<0.001**
Nightmare Distress (NDQ)	6.64	5.02	16.38	8.02	67/73	−8.69	**<0.001**
Retrospective (SDQ-A): frequency:							
Nightmares	2.25	0.91	3.21	0.97	67/73	−5.97	**<0.001**
Bad dreams	2.42	0.92	3.29	0.98	67/73	−5.40	**<0.001**
Bizarre dreams	2.82	1.06	3.64	1.09	67/73	−4.54	**<0.001**
Sleepwalking	1.22	0.74	1.77	1.22	67/73	−3.22	**0.002**
Prospective (sleep log) distress:							
Distress: during dreams	3.29	2.13	6.04	1.81	65/71	−8.07	**<0.001**
Distress: after dreams	2.37	1.94	4.59	2.27	65/70	−6.08	**<0.001**
Psychopathology measures:							
Anxiety (STAI)-State	30.37	6.63	36.10	10.90	60/62	−3.52	**0.001**
Anxiety (STAI)-Trait	34.97	8.66	39.74	11.39	66/73	−2.76	**0.007**
Depression (BDI-II)	5.76	5.27	11.16	10.18	67/73	−3.89	**<0.001**
Alexithymia (TAS)-Total	42.79	8.61	47.86	10.20	34/35	−2.23	**0.029**
DIF	13.85	3.91	17.20	5.41	34/35	−2.94	**0.005**
DDF	11.62	3.77	12.94	4.79	34/35	−1.28	0.207
EOT	17.32	3.40	17.71	4.03	34/35	−0.44	0.665

NDQ = Nightmare Distress Questionnaire, SDQ-A = Sleep Disorders Questionnaire-Abbreviated version: childhood items ('As a child, did you have…'), STAI = State Trait Anxiety Inventory, BDI-II = Beck Depression Inventory-II, TAS = Toronto Alexithymia Scale, DIF = TAS Difficulty Identifying Feelings subscale, DDF = TAS Difficulty Describing Feelings subscale, EOT = TAS Externally Oriented Thinking subscale. p-values in bold <0.05.

Table 3
Sleep architectural findings for participants taking a morning nap. Groups did not differ on any measure.

	NM	*SD*	CTL	*SD*	χ^2/T[a]/Z[b]	p	NM-N	CTL-N
Sex (M:F)	17:32		15:26		0.035	0.852	49	41
Age	24.27	3.73	23.63	3.94	0.780	0.438	49	41
Sleep Latency[b]	12.67	21.73	8.358	7.71	0.933	0.351	49	41
REM Latency[b]	43.80	30.30	45.83	23.06	−0.158	0.874	42	32
Total Sleep Time[a]	67.60	23.14	70.40	25.37	−0.547	0.586	49	41
Sleep Efficacy[a,c]	73.27	22.03	72.12	22.54	0.245	0.807	49	41
Min Stage 1[b]	13.08	7.14	15.24	9.08	−0.993	0.321	49	41
Min Stage 2[b]	30.99	14.80	30.41	17.62	0.835	0.404	49	41
Min Stage 3[b]	11.45	13.72	13.29	13.15	−0.858	0.391	49	41
Min NREM[a]	55.52	19.01	58.95	22.29	−0.788	0.433	49	41
Min REM[b]	12.08	9.53	11.45	9.12	0.419	0.676	49	41
%Stage 1[b]	23.52	18.68	25.47	19.59	−0.502	0.615	49	41
%Stage 2[a]	45.44	14.65	42.10	15.76	1.043	0.300	49	41
%Stage 3[b]	14.87	16.69	16.97	16.91	−0.788	0.430	49	41
%NREM[a]	83.84	11.03	84.54	12.47	−0.282	0.778	49	41
%REM[b]	16.16	11.03	15.46	12.47	0.780	0.435	49	41
MinREM in REM[a]	14.10	8.79	14.67	7.64	−0.296	0.768	42	32
Min NREM in REM[b]	2.86	3.92	1.50	2.13	1.107	0.268	42	32
Min Wake in REM[b]	0.51	2.47	0.23	0.40	0.941	0.346	42	32
# REM periods[b]	1.18	0.67	0.98	0.65	1.491	0.136	49	41
# Fragments in REM[b]	2.93	2.31	2.44	1.32	0.477	0.634	42	32
REM Efficacy[a]	80.91	24.67	88.67	16.99	−1.524	0.132	42	32

Min: minutes; REM: Rapid Eye Movement sleep; NREM: Non-REM sleep.
[a] t-test.
[b] Mann-Whitney U test.
[c] Based on recording length (includes sleep latency, sleep period and last awakening duration).

either trends or no effects for three (*Separation*: p = 0.094; *Sexual*: p = 0.079; *Neglect*: *p* = .272). The NM group scored higher than the CTL group in all instances. There were no univariate Sex effects (all p > 0.076) and one marginal Group x Sex interaction (*Separation*: p = 0.020; all other p > 0.192) indicating highest means for the female NM group. Covarying BDI-II, TAS and DIF did not eliminate the multivariate Group effect (T = 0.411, $F_{8,55}$ = 2.829, p = 0.011, eta^2 = 0.291) or three of the univariate effects (*Emotional*: p = 0.037; *Physical*: p = 0.005; *Witnessing*: p = 0.051), but did reduce or eliminate *Sexual* (p = 0.612), *Substances* (p = 0.095) and *Other* (p = 0.078) domains while rendering *Separation* highly significant (p = 0.007). The Group x Sex interaction was eliminated (p > 0.124).

3.3. Adversity: 0–6 years

A Group x Sex ANOVA using *global* (*0–6*) score as dependent measure revealed a Group main effect ($F_{1,136}$ = 5.135, p = 0.025,

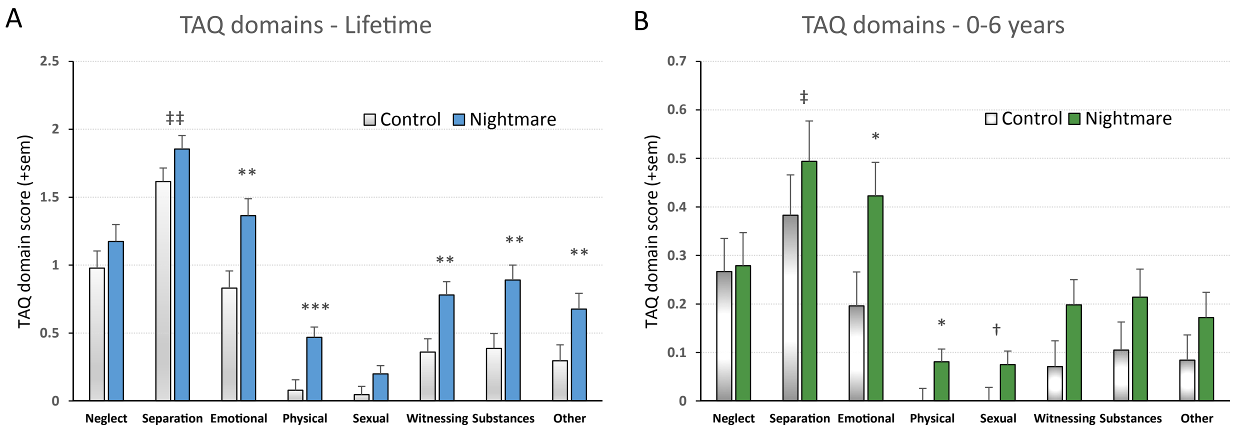

Fig. 1. Traumatic Antecedents Questionnaire (TAQ) domain scores (log(score)+sem) for A) Lifetime and B) 0–6 years of age. ***$p < 0.001$; **$p < 0.01$; *$p < 0.05$; †$p = 0.058$; ‡‡$p = 0.02$, Sex X Group interaction; ‡$p = 0.056$ Sex X Group interaction.

$eta^2 = 0.036$) whereby the NM group had higher scores (0.364 ± 0.336) than the CTL group (0.214 ± 0.253), but no other effects. The Group effect was eliminated by covarying BDI-II, TAS, and DIF ($F_{1,62} = 1.910$, $p = 0.172$, $eta^2 = 0.030$).

A Group x Sex MANOVA with the eight *domain (0–6)* scores as dependent measures produced no multivariate Group effect ($T = 0.097$, $F_{8,129} = 1.561$, $p = 0.143$, $eta^2 = 0.088$) or Sex or Group X Sex effects (all $p > 0.467$) but did reveal univariate Group effects or trends for *Emotional* ($p = 0.022$, $eta^2 = 0.038$), *Physical* ($p = 0.028$, $eta^2 = 0.035$), *Sexual* ($p = 0.058$, $eta^2 = 0.026$) and *Witnessing* ($p = 0.090$, $eta^2 = 0.021$) domains, with NM > CTL in all instances (Fig. 1B). A single, marginal, Group X Sex interaction for *Separation* ($p = 0.060$, $eta^2 = 0.026$) showed NM females scored higher than other groups. Covarying BDI-II, TAS, and DIF left a single effect, *Emotional* ($p = 0.048$, $eta^2 = 0.062$), and a trend for *Witnessing* ($p = 0.087$, $eta^2 = 0.046$), but eliminated the Sex X Group *Separation* interaction ($p = 0.255$, $eta^2 = 0.021$). Thus, the pattern of findings for ages 0–6 largely mirrored that for the Global scales although differences were less robust and more closely linked to psychopathology.

3.4. Adversity: traumatic vs. non-traumatic

One-way ANCOVAs compared Groups on either TA or NTA while covarying NTA and TA respectively—for both *lifetime* and *0–6 years* measures. For *NTA* (*lifetime*) with *TA* (*lifetime*) covaried, there was a Group effect ($F_{1,137} = 5.912$, $p = 0.016$, $eta^2 = 0.041$) while for *TA* (*lifetime*) with *NTA* (*lifetime*) covaried, there was a slightly larger Group effect ($F_{1,137} = 8.011$, $p = 0.005$, $eta^2 = 0.055$). However, for *NTA* (*0–6*) with *TA* (*0–6*) covaried, there was a marginal Group effect ($F_{1,137} = 3.714$, $p = 0.056$, $eta^2 = 0.026$) while for *TA* (*0–6*) with *NTA* (*0–6*) covaried, there was no Group effect ($F_{1,137} = 1.271$, $p = 0.262$, $eta^2 = 0.009$). In brief, whereas *lifetime* traumatic and non-traumatic adversity are both associated with nightmares, for early childhood (0–6 years) measures non-traumatic adversity shows the association independent of traumatic adversity.

3.5. Graded adversity-nightmares relationships

Relationships between nightmare severity—i.e., frequency (logNM) and distress (NDQ)—and adversity were calculated with Pearson correlations (Table 4). For the NM group, NDQ correlated positively with all *lifetime* and *0–6* scores (all $p < 0.05$), whereas for the CTL group correlations were weaker and less numerous (two of six at $p < 0.02$) but nevertheless positive in all cases. For the whole sample, all TAQ correlations with NDQ attained $p < 0.0003$.

In contrast, TAQ correlations with logNM obtained only for *TA* (*lifetime*) in the CTL group ($p = 0.04$) and for no measures in the NM group (all $p > 0.35$). For the whole sample, logNM correlations were substantial for five of six coefficients (all $p < 0.05$).

Table 4 also reveals that NDQ and logNM are strongly intercorrelated for the whole sample ($r = 0.438$, $p < 0.0000001$), less so for the CTL group ($r = 0.281$, $p = 0.021$) and not at all for the NM group ($r = 0.046$, $p = 0.700$).

In sum, nightmare severity is related to adversity in a graded fashion, with distress associated, in the NM group, with both traumatic and non-traumatic adversity and for both lifetime and 0–6 years measures. In the CTL group, nightmare distress and frequency correlate differentially with NTA and TA respectively—but only for lifetime adversity.

3.6. Nightmare severity: frequency vs. distress

Whether observed group differences in adversity are associated more closely with nightmare distress or frequency was evaluated using one-way ANCOVAs. Covarying logNM produced a substantial multivariate Group effect ($T = 0.098$, $F_{4,134} = 3.290$, $p = 0.013$, $eta^2 = 0.089$) and three (of four) univariate effects: *TA* (*lifetime*) ($F_{1,137} = 7.760$, $p = 0.006$, $eta^2 = 0.054$), *TA* (*0–6*) ($F_{1,137} = 0.689$, $p = 0.408$, $eta^2 = 0.005$), *NTA* (*lifetime*) ($F_{1,137} = 7.696$, $p = 0.006$, $eta^2 = 0.053$) and *NTA* (*0–6*) ($F_{1,137} = 5.043$, $p = 0.026$, $eta^2 = 0.036$); NM > CTL in all cases. In contrast, covarying NDQ produced no multivariate effect ($T = 0.041$, $F_{4,134} = 1.381$, $p = 0.244$, $eta^2 = 0.040$) and a marginal univariate effect for *TA* (*lifetime*): $F_{1,137} = 3.465$, $p = 0.065$, $eta^2 = 0.025$ (all other $p > 0.288$). Thus, adversity-based group differences are more closely tied to nightmare-induced distress than to frequency of nightmare recall.

3.7. Adversity relationships with sleep spindles

As specified by hypothesis four, a closer examination of *separation* domain scores revealed correlations with slow spindle densities for the NM, but not the CTL, group (Table 5; Fig. 2). Correlations for the NM group were negative between *separation* (*lifetime*) and spindles in central derivations (C3: $r = -0.453$, $p = 0.008$; C4: $r = -0.496$, $p = 0.003$); fewer slow spindles were associated with higher adversity scores. By age, similar relationships obtained for 0–6 years (C3: $r = -0.473$, $p = 0.005$; C4:

Table 4
Pearson correlations (upper) and p-values (lower) between measures of nightmare severity (frequency: logNM; distress: NDQ) and Traumatic Antecedents Questionnaire (TAQ) subscales for Lifetime and age range 0–6.

	Nightmare		Control		Whole sample	
	logNM	NDQ	logNM	NDQ	logNM	NDQ
Correlations						
NDQ	0.046	–	**0.281**	–	**0.438**	–
Global Lifetime	0.091	**0.491**	0.137	**0.298**	**0.310**	**0.535**
Global 0–6	−0.010	**0.384**	0.131	0.209	**0.170**	**0.402**
Non-trauma (NTA) Lifetime	0.077	**0.470**	0.142	**0.301**	**0.294**	**0.509**
Non-trauma (NTA) 0-6	−0.055	**0.357**	0.129	0.201	0.135	**0.371**
Trauma (TA) Lifetime	0.083	**0.374**	**0.251**	0.105	**0.320**	**0.452**
Trauma (TA) 0-6	0.111	**0.297**	0.064	0.104	**0.201**	**0.309**
p-values						
NDQ	0.70005	–	**0.02133**	–	**0.00000**	–
Global Lifetime	0.44588	**0.00001**	0.26742	**0.01422**	**0.00019**	**0.00000**
Global 0-6	0.93384	**0.00080**	0.29032	0.08962	**0.04526**	**0.00000**
Non-trauma (NTA) Lifetime	0.51910	**0.00003**	0.25072	**0.01334**	**0.00042**	**0.00000**
Non-trauma (NTA) 0-6	0.64121	**0.00193**	0.29809	0.10321	0.11051	**0.00001**
Trauma (TA) Lifetime	0.48502	**0.00110**	**0.04008**	0.39625	**0.00012**	**0.00000**
Trauma (TA) 0-6	0.35113	**0.01084**	0.60796	0.40015	**0.01710**	**0.00021**

logNM: log(recalled nightmares per week+1); NDQ: Nightmare Distress Questionnaire; Global: sum of eight TAQ adversity subscales; NTA: Non-Traumatic Adversity subscale; TA: Traumatic Adversity subscale; Values in bold are $p < 0.05$.

$r = -0.540$, $p = 0.001$) and to a lesser extent 7–12 years (C3: $r = -0.428$, $p = 0.013$; C4: $r = -0.399$, $p = 0.019$) but not 13–18 or 19+ years. For the CTL group, no correlations were observed. For spindle frequencies, no correlations exceeded $p < 0.05$ for either group, although a vast majority of the correlations were positive as predicted (Fig. 2).

4. Discussion

Results largely support the four hypotheses, bolstering the notion that idiopathic nightmares constitute a reaction to early adversity. More speculatively, they support the possibility that nightmares induced by adversity—but not by any identifiable trauma—nonetheless share some pathophysiological mechanisms with PTSD nightmares.

Findings for hypothesis one (lifetime adversity) are consistent with several studies demonstrating links between past adversity and current nightmares [5,6,35]. Global adversity for nightmare-prone participants was almost twice that for controls, and seven of eight domain scores were markedly elevated. Consistent with the notion that nightmares are a pathology of dysphoric emotion regulation [4,36], our largest group domain difference (apart from *Other*) was for *Emotional adversity* ($p = 0.003$, $eta^2 = 0.064$). Further, group differences were largely independent of psychopathological symptoms. Global adversity, in particular, survived covarying depression and even alexithymia, which itself may reflect a problem with emotion regulation. Multiple studies (review in Ref. [23]), including the present one, have found that nightmare severity is associated with higher Alexithymia total scores and, in particular, higher scores on the Difficulty Identifying Feelings (DIF) alexithymia subscale. Unlike the Lifetime TAQ measures, however, age 0–6 measures were diminished by covarying psychopathology. Thus, even though nightmare-prone individuals reported more adversity as infants/toddlers as predicted, associations with psychopathology suggest the relationships may reflect either generalized effects of early adversity on multiple adult pathologies

Table 5
Spearman correlations between density of slow sleep spindles and Separation domain scores on the Traumatic Antecedents Questionnaire for four age ranges and lifetime scores among Nightmare (N = 34)[a] and Control (N = 29)[b] participants. Bold values: $p < 0.02$; (r): Spearman rho-value; (p): p-value.

Separation score		Slow Spindle Density					
		F3	F4	C3	C4	O1	O2
Nightmare (r)	0–6	−0.112	−0.147	−**0.473**	−**0.540**	−0.051	−0.039
	7–12	−0.086	−0.115	−**0.428**	−**0.399**	−0.039	−0.054
	13–18	−0.059	−0.158	−0.191	−0.153	0.029	0.017
	Adult	−0.155	−0.232	−0.103	−0.172	0.065	0.082
	Lifetime	−0.096	−0.159	−**0.453**	−**0.496**	0.007	−0.001
(p)	0–6	0.529	0.414	**0.005**	**0.001**	0.773	0.831
	7–12	0.628	0.525	**0.013**	**0.019**	0.827	0.764
	13–18	0.740	0.381	0.288	0.388	0.873	0.926
	Adult	0.382	0.194	0.569	0.330	0.716	0.651
	Lifetime	0.588	0.378	**0.008**	**0.003**	0.969	0.998
Control (r)	0–6	−0.201	−0.193	−0.137	−0.036	−0.117	−0.108
	7–12	−0.139	−0.024	−0.006	0.118	−0.009	−0.036
	13–18	0.054	0.080	0.148	−0.008	−0.206	−0.226
	Adult	−0.256	−0.173	−0.048	−0.007	−0.034	−0.033
	Lifetime	−0.256	−0.165	−0.054	−0.006	−0.123	−0.142
(p)	0–6	0.295	0.316	0.486	0.858	0.545	0.578
	7–12	0.471	0.903	0.977	0.558	0.963	0.855
	13–18	0.779	0.682	0.453	0.967	0.284	0.239
	Adult	0.180	0.371	0.808	0.973	0.860	0.865
	Lifetime	0.180	0.393	0.784	0.975	0.526	0.464

[a] N = 33 for F4, C3, O2.
[b] N = 28 for C3, 27 for C4; values in bold are $p < .05$.

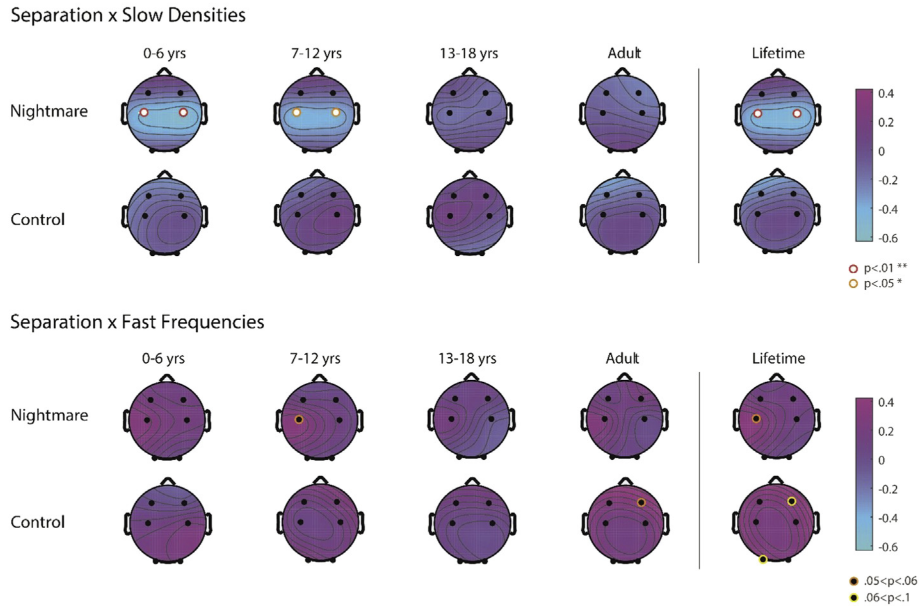

Fig. 2. Spearman correlations between Traumatic Antecedents Questionnaire Separation domain scores and slow sleep spindle density (top panel) and mean frequency of fast (13–16 Hz) sleep spindles (bottom panel); results are shown for 6 cortical derivations (F3, F4, C3, C4, O1, O2), 4 age ranges and Lifetime adversity. See Table 5 for descriptive details.

(reviews in [4,37]), or that current psychopathology biased participants' recall and/or reporting of early experiences.

Consistent with hypothesis two (0–6 adversity), scores for nightmare-prone participants were elevated for *0–6 years* (*lifetime*) and on three of eight domain scores for this age range: *Emotional*, *Sexual* and *Separation* (females only). Further, early non-trauma adversity was associated with the NM group to a greater extent than was early trauma adversity, a finding suggesting that nightmares may result when youngsters experience low-grade forms of adversity, such as emotional abuse, that the DSM does not necessarily define as traumatic. These results are consistent with the theory that preschool adversity sufficient to disrupt normal development of emotional mechanisms leads to nightmares [4].

Consistent with hypothesis three, relationships between adversity and nightmare severity were graded, relatively independent of psychopathology and most robust for nightmare distress. Distress correlated positively with several adversity measures—and this for both NM and CTL groups separately. Nightmare frequency correlated with adversity only for the whole sample combined, possibly due to parallel group differences in the two measures. These graded relationships parallel much prior research showing graded relationships between adversity and various pathologies—including sleep symptoms [38]. For example, distress and other PTSD symptoms escalate with the accumulation of adverse events [39]; the odds of developing PTSD increase incrementally with the increasing diversity of violence types accumulated [40]. That graded relationships occurred for both our NM and CTL groups suggests that accumulation of early adversities may constitute a risk factor for nightmares across a range of adversity severities; even very mild nightmare suffering may be associated with early adversity history. It bears repeating that NM participants in the present cohort were neither clinical patients nor seeking treatment. In this respect, our cohort likely consists of individuals whose nightmare problems are less severe than those of typical clinical samples. If so, our results support the possibility that early adversity may contribute to even the most mundane forms of idiopathic nightmares. It may also be the case that the participants in our study circumvented clinical suffering by more effective resilience or coping mechanisms, a possibility spelled out by the Differential Susceptibility framework for nightmare personality [41].

Finally, and consistent with hypothesis four, early *Separation* was associated with spindle anomalies that index both psychopathology [17,18] and nightmare genesis prior to age ten [22]. A higher *Separation* adversity at 0–6 years, and less so at 7–12 years, correlated robustly with a lower density of slow spindles over central derivations (C3, C4) for nightmare-prone, but not control, participants. This further supports the notion that anomalies in the early maturation of sleep spindles [16] contribute to long-term nightmare development [4].

Findings for fast spindle frequency only weakly supported hypothesis four in that, for the NM group, the measure did tend to correlate positively with *Separation* at 0–6 years ($p = 0.089$) and 7–12 years ($p = 0.054$), and also with lifetime *Separation* adversity ($p = 0.054$), but none of these correlations surpassed the $p < 0.01$ threshold correction.

Altogether, the findings add weight to a growing literature demonstrating that idiopathic nightmares are associated with prior adversity—even when this adversity occurs as young as 0–6 years of age and is not specifically traumatic in nature. More speculatively, the results support the possibility that idiopathic nightmares are not, in fact, idiopathic but may in some individuals be caused by an accumulation of adverse experiences. If so, such nightmares may share pathophysiological mechanisms with post-traumatic nightmares and should perhaps not be considered a psychiatric disorder completely distinct from PTSD. It may be more accurate to consider idiopathic nightmares as falling on a continuum of adversity-induced stress disorders which includes common nightmares at the mildest extreme, replicative PTSD nightmares at the most severe extreme, and nightmares characteristic of partial or preclinical PTSD between the two. Such a framework could readily explain the observation that idiopathic nightmares predict future PTSD and would harmonize a variety of epidemiological, clinical and laboratory work suggesting parallel clinical profiles for nightmare disorder, PTSD and partial PTSD. These parallels include similarities in sex and age prevalence [1,36], common comorbidities such as anxiety and suicidal behavior [42], and shared polysomnographic characteristics [43].

In sum, our findings demonstrate that early childhood adversity is associated with adult nightmare suffering and disruptions in basic sleep spindle expression. They are consistent with the possibility that nightmares are caused by such adversity and thus may share pathophysiological mechanisms with PTSD nightmares.

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Conflict of interest

None to declare.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2019.03.004.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2019.03.004.

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