Early childhood adversity associations with nightmare severity and sleep spindles

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Abstract

Objective: Childhood adversity figures prominently in the clinical histories of children and adolescents suffering from a panoply of physical, mental and sleep disorders, including especially posttraumatic stress disorder. But the nature and prevalence of early adversity in the case of idiopathic nightmare-prone individuals has 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 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 trauma and nontrauma forms of adversity were both elevated for nightmare-prone participants; for 0-6 years, nontrauma 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 sleep spindle anomalies—lower slow spindle density especially—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.

Keywords: nightmares, stress-acceleration theory, adverse childhood experience, post-traumatic stress disorder, parasomnias, sleep

Introduction

Idiopathic nightmares—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 in prevalence through adulthood, even though they remain more prevalent among females. They are considered to be distinct from other DSM5 psychiatric disorders, but nonetheless constitute a risk factor for post-traumatic stress disorder (PTSD) and suicidal behavior, and are comorbid with anxiety, depression and insomnia (for review see (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 had experienced abuse and family adversity assessed between birth and age 4. 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).

A critical period for emotional maturity

One recent theory, the Stress Acceleration Hypothesis of nightmares (4), suggests that early adversity triggers a neural cascade of events that leads to later nightmares. Adversity may

interfere with a critical period of brain plasticity occurring around ages 3-4—when amnesia for early life experiences normally sets in. By this theory, adversity presumably 'accelerates,' or temporally advances, infantile amnesia, producing unusually good memory for some events prior to age 4, 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 4 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 speak to an etiological role for this early critical period.

The Traumatic Antecedents Questionnaire

Both the age of onset and the commonest types of adversity are assessed by the TAQ (10, 11), a self-report instrument that quantifies 8 common adversity domains (e.g., *neglect, separation*) for each of 4 age ranges (*0-6, 7-12, 13-18, 19+)*. Validational 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. Further, to quantify possible separate links to traumatic and non-traumatic forms of adversity, we derived two new TAQ measures—the Traumatic Adversity (TAQ-TA) and Non-Traumatic Adversity (TAQ-NTA) scales—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 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 results from adverse events that are more clearly traumatic.

Sleep spindle indicators of adversity

Sleep spindles are an EEG hallmark of non-rapid eye movement (NREM) sleep, i.e., waxing-and-waning, 0.5-3.0 sec 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, e.g., abrupt increases in density between ages 3-4 (16). They predict children's emotional–behavioral development (17) and are linked to neurodevelopment disorders of anxiety (18), schizophrenia (19) and depression (20). Early changes in spindle characteristics, such as density and frequency at age 5, robustly predict later developmental changes, such as positive emotional and behavioral characteristics at age 9 (17). We demonstrated, in nightmare participants, relationships between sleep spindle characteristics and pathology 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 10 (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).

Hypotheses

We tested 4 hypotheses, involving 10 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 greater lifetime adversity—independent of comorbid psychopathology; 2 they will report more adversity at age 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 recall frequency; 4) sleep spindle anomalies—slow density and fast frequency—will be associated with early (age 0-6) separation for nightmare-prone individuals. Secondarily, we explored relationships between groups and the TAQ domain scores (lifetime, age 0-6 only).

Method

Participants

Men and women aged 18-50, drawn from our laboratory database, who participated in studies of nightmares and recruited by advertisements, posters or word-of-mouth, were screened using a telephone questionnaire. They were excluded if they reported current major sleep disorders other than nightmares, psychiatric disorder, neurological disease, major medical problem, medication use, language deficit, or legal conflict. Participants were neither clinical patients nor seeking treatment.

Participants previously recruited for 4 different research studies of nightmare-prone individuals were included in the present study. Three of these 4 studies involved laboratory

polysomnograms during a morning nap; all required the completion of a questionnaire battery that included measures of depression, anxiety and alexithymia. The 4 studies included: 1) a protocol (N=28) in which participants underwent pre-sleep word association and semantic priming tasks; 2) a protocol (N=42) in which participants underwent a pre-sleep word association task; 3) a protocol (N=4) in which participants underwent transcranial alternating current stimulation (tACS) during REM sleep in an attempt to induce lucid dreaming; and 4) a protocol (N=66) in which participants underwent brain imaging procedures (SPECT or single proton emission computed tomography) but no laboratory PSG. Each protocol included both nightmare and control subjects. Despite the differing procedures, all participants complete the same TAQ.

Participants in the nightmare (NM) group (N=73; 21 M, 52 F) reported ≥2 non-substance-induced nightmares/week for ≥6 months; the control (CTL) group (N=67; 22 M, 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±4.19y; CTL: 23.9±4.28y; t_{138}=-0.735, p=.463) or males (NM: 24.9±4.17y; CTL: 25.5±4.39y; t_{41}=0.455, p=.651) or females (NM: 24.3±4.22y; CTL: 23.2±4.06y; t_{95}=-1.320, p=.190) separately. Participants completed informed consent forms approved by the *CIUSSS-NIM–Hôpital du Sacré-Coeur de Montréal* ethics committee and given a modest financial compensation.

Subgroups of 49 NM participants (32F; 24.3±3.73y) and 41 CTL (26F; 23.6±3.94y; age: t_{88}=0.780, p=.438) were polysomnographically recorded during a 2-hr morning nap. Of these, 34 NM (21F: 23.85±3.6y) and 29 CTL (18F; 23.90±3.8y; t_{61}=0.047, p=.963) had results from sleep spindle analysis that were available for inclusion.

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 (23) or associative memory tasks with PSG and REM sleep dream collection. Task results are reported elsewhere (24, 25).

Home sleep/dream logs: Daily logs, the best prospective measure of sleep timing and nightmare severity (26), were collected with an interactive voice mail acquisition system (27); participants called and keyed in responses to recorded questions about their previous night of sleep and dreaming (~5 min/call).

Questionnaires

140 participants completed the Traumatic Antecedents Questionnaire (TAQ; 11), the Beck Depression Inventory-II (BDI-II; 28), and the Sleep Disorders Questionnaire-Abbreviated version (SDQ-A; 29). From the SDQ-A, 4 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*). Further, 139 participants completed the State Trait Anxiety Inventory (STAI; 30) and 69 the Toronto Alexithymia Scale (TAS; 31) with its 3 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; 32). 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 5 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 to 6 years), middle/late childhood (7 to 12 years), adolescence (13 to 18 years), and adulthood (19+ years); lifetime scores include all 4 age ranges (0 to 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 8 adversity domains—*neglect, separation, emotional, physical, sexual, witnessing adversity, exposure to alcohol/drugs*, *other*—and 2 resilience domains—*competence, safety* (not assessed here)—are calculated for each of 4 age ranges providing 32 scores plus 8 domain lifetime scores. *Global (lifetime)* score is calculated as the sum of 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†,6,7,9-11†,12-20†, 21-27†,28-40	35/31	5
1. *Neglect*	lifetime, 0-6, 7-12, 13-18, 19+	2†,6,7,21,27†	5/3	5
2. *Separation*	lifetime, 0-6, 7-12, 13-18, 19+	10,11†,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†,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†,23,24,28-39	17/16	5
Non-trauma adversity (NTA)[e]	lifetime, 0-6, 7-12, 13-18, 19+	6,7,9-11†,12,14,16-19,21,22,25-27†,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; †item not used for adult (19+) age range.

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, e.g., *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, e.g., *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 rated >1 and provide scores per age range plus a Lifetime total. The subscales thus add 10 scores.

To limit the number of group comparisons from the TAQ battery, our hypotheses specify 10 primary endpoints (Table 1), i.e., *lifetime* and *0-6* scores for *global, TA* and *NTA* and correlations with *Separation* at 4 age ranges. Because most score distributions were non-Gaussian, natural logs (score+1) were calculated for each. Significance thresholds were set at $p<.01$ for hypotheses and $p<.05$ for secondary exploratory analyses of TAQ domain scores.

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 4 electrooculogram (EOG) leads to evaluate sleep stages. In addition to electrode derivations for standard polysomnography (PSG), we recorded 4 EMG leads (chin, corrugator supercilii, dominant arm and leg), and 3 ECG leads to monitor heart rate. Recordings were accomplished with a Grass M15 Neurodata Acquisition System (-6dB filters with cut offs 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 (33) 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 5 participants, C3 and C4 derivations were excluded twice each (resulting total N=61), and F4 and O2 derivations were excluded once each (resulting 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 spindles 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: χ^2=0.98, p=1.00 Fisher exact). Spindles were detected automatically on 6 artifact-free derivations (F3, F4, C3, C4, O1, O2; reference: A1+A2) for 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).

Statistical Analyses

TAQ *global (lifetime)* and *global (0-6)* primary endpoints were assessed with 2x2, Group (NM, CTL) x Sex, ANOVAs/ANCOVAs holding significant confounders constant. *Domain (lifetime)* and *domain (0-6)* secondary endpoints were assessed with 2x2, Group x Sex, MANOVA/MANCOVA with 8 *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 oneway MANOVAs with Group as 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<.01 was selected for significance testing.

Results

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 ($p<.005$) and higher prospective estimates of distress during and after dreams ($p<.001$). They also scored higher on all pathological indicators ($p<.05$) except TAS-DDF and TAS-EOT.

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

Measure	Control Group Mean	*SD*	Nightmare Group Mean	*SD*	Group N (CTL/NM)	t	*p*
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	*<.001*
• Bad dreams/week	0.23	*0.36*	2.66	*1.38*	67/73	-14.03	*<.001*
• Nightmares/week	0.03	*0.08*	1.22	*1.22*	67/73	-7.99	*<.001*
• Nightmare Distress (NDQ)	6.64	*5.02*	16.38	*8.02*	67/73	-8.69	*<.001*
Retrospective (SDQ-A): frequency:							
• Nightmares	2.25	*0.91*	3.21	*0.97*	67/73	-5.97	*<.001*
• Bad dreams	2.42	*0.92*	3.29	*0.98*	67/73	-5.40	*<.001*
• Bizarre dreams	2.82	*1.06*	3.64	*1.09*	67/73	-4.54	*<.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	*<.001*
• Distress: after dreams	2.37	*1.94*	4.59	*2.27*	65/70	-6.08	*<.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	*<.001*
• Alexithymia (TAS)-Total	42.79	*8.61*	47.86	*10.20*	34/35	-2.23	*0.029*
o DIF	13.85	*3.91*	17.20	*5.41*	34/35	-2.94	*0.005*
o DDF	11.62	*3.77*	12.94	*4.79*	34/35	-1.28	*0.207*
o 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.

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

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*/ Z†	*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†	12.67	*21.73*	8.358	*7.71*	0.933	*0.351*	49	41
REM Latency†	43.80	*30.30*	45.83	*23.06*	-0.158	*0.874*	42	32
Total Sleep Time*	67.60	*23.14*	70.40	*25.37*	-0.547	*0.586*	49	41
Sleep Efficacy*,£	73.27	*22.03*	72.12	*22.54*	0.245	*0.807*	49	41
Min Stage1†	13.08	*7.14*	15.24	*9.08*	-0.993	*0.321*	49	41
Min Stage2†	30.99	*14.80*	30.41	*17.62*	0.835	*0.404*	49	41
Min Stage3†	11.45	*13.72*	13.29	*13.15*	-0.858	*0.391*	49	41
Min NREM*	55.52	*19.01*	58.95	*22.29*	-0.788	*0.433*	49	41
Min REM†	12.08	*9.53*	11.45	*9.12*	0.419	*0.676*	49	41
%Stage1†	23.52	*18.68*	25.47	*19.59*	-0.502	*0.615*	49	41
%Stage2*	45.44	*14.65*	42.10	*15.76*	1.043	*0.300*	49	41
%Stage3†	14.87	*16.69*	16.97	*16.91*	-0.788	*0.430*	49	41
%NREM*	83.84	*11.03*	84.54	*12.47*	-0.282	*0.778*	49	41
%REM†	16.16	*11.03*	15.46	*12.47*	0.780	*0.435*	49	41
MinREM in REM*	14.10	*8.79*	14.67	*7.64*	-0.296	*0.768*	42	32
Min NREM in REM†	2.86	*3.92*	1.50	*2.13*	1.107	*0.268*	42	32
Min Wake in REM†	0.51	*2.47*	0.23	*0.40*	0.941	*0.346*	42	32
# REM periods†	1.18	*0.67*	0.98	*0.65*	1.491	*0.136*	49	41
# Fragments in REM†	2.93	*2.31*	2.44	*1.32*	0.477	*0.634*	42	32
REM Efficacy*	80.91	*24.67*	88.67	*16.99*	-1.524	*0.132*	42	32

**t-test; †Mann-Whitney U test; £based on recording length (includes sleep latency, sleep period and last awakening duration); Min: minutes; REM: Rapid Eye Movement sleep; NREM: Non-REM sleep*

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<.00006, eta^2=.113) showing higher NM (1.322±.497) than CTL (.916±.497) means, but no Sex effect ($F_{1,136}$=1.067, p=.304, eta^2=.008) or Group x Sex interaction ($F_{1,136}$=0.375, p=.541, eta^2=.003). The Group effect was not eliminated by covarying BDI-II, TAS, and DIF scores ($F_{1,62}$=10.590, p=.002, eta^2=.146).

A Group by Sex MANOVA with 8 *domain (lifetime)* dependent measures produced a multivariate Group effect (Hotelling's T=.185, $F_{8,129}$=2.978, p=.004, eta^2=.156) and no Sex or Group x Sex effects (both p>.185). Figure 1A shows 5 univariate Group effects (*Emotional*: p=.003*; Physical*: p=.0004*; Witnessing:* p=.008*; Substances:* p=.003*; Other:* p=.007) and trends or no effects for 3 (*Separation*: p=.094; *Sexual*: p=.079; *Neglect: p=.272*). The NM group scored higher than the CTL group in all instances. There were no univariate Sex effects (all p>.076) and one marginal Group x Sex interaction (*Separation:* p=.020; all other p>.192) indicating highest means for the female NM group. Covarying BDI-II, TAS and DIF did not eliminate the multivariate Group effect (T=.411, $F_{8,55}$=2.829, p=.011, eta^2=.291) or 3 of the univariate effects (*Emotional:* p=.037; *Physical:* p=.005; *Witnessing:* p=.051), but did reduce or eliminate *Sexual* (p=.612), *Substances* (p=.095) and *Other* (p=.078) domains while rendering *Separation* highly significant (p=.007). The Group x Sex interaction was eliminated (p>.124).

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=.025, eta^2=.036) whereby the NM group had higher scores

(.364±.336) than the CTL group (.214±.253), but no other effects. The Group effect was eliminated by covarying BDI-II, TAS and DIF ($F_{1,62}$=1.910, p=.172, eta^2=.030).

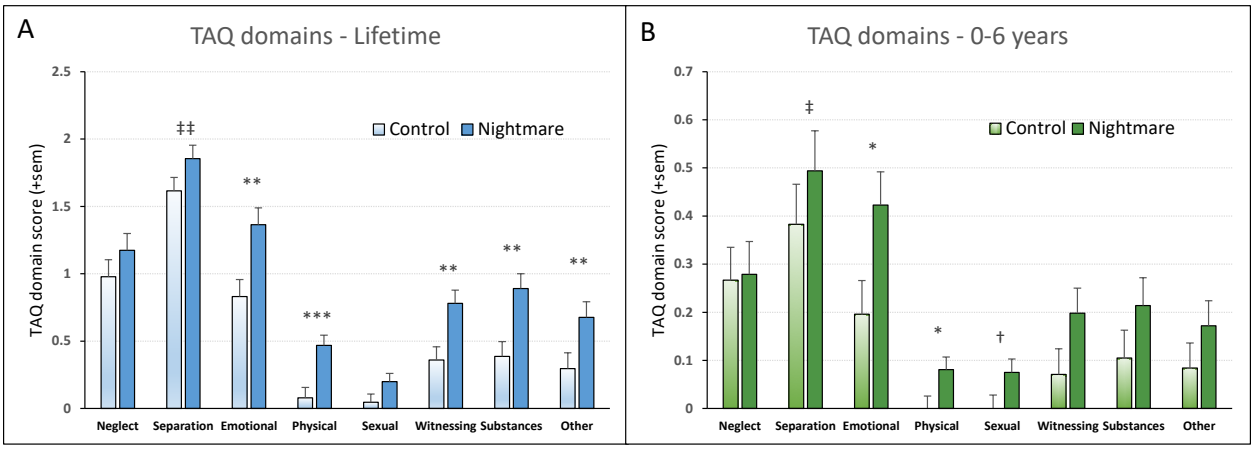

Figure 1. Traumatic Antecedents Questionnaire (TAQ) domain scores (log(score)+sem) for A) Lifetime and B) 0-6 years of age. ***p<.001; **p<.01; *p<.05; †p=.058; ‡‡p=.02, Sex X Group interaction; ‡p=.056 Sex X Group interaction.

A Group x Sex MANOVA with the 8 *domain (0-6)* scores as dependent measures produced no multivariate Group effect (T=.097, $F_{8,129}$=1.561, p=.143, eta^2=.088) or Sex or Group X Sex effects (all p>.467) but did reveal univariate Group effects or trends for *Emotional* (p=.022, eta^2=.038), *Physical* (p=.028, eta^2=.035), *Sexual* (p=.058, eta^2=.026) and *Witnessing* (p=.090, eta^2=.021) domains, with NM > CTL in all instances (Figure 1B). A single, marginal, Group X Sex interaction for *Separation* (p=.060, eta^2=.026) showed NM females to score higher than other groups. Covarying BDI-II, TAS and DIF left a single effect, *Emotional* (p=.048, eta^2=.062), and a trend, *Witnessing* (p=.087, eta^2=.046), but eliminated the Sex X Group *Separation* interaction (p=.255, eta^2=.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.

Adversity: Traumatic vs. Non-traumatic

Oneway ANCOVAs compared Groups on either TA or NTA while covarying NTA and TA respectively—and this 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=.016, eta^2=.041) while for *TA (lifetime)* with *NTA (lifetime)* covaried, there was a slightly larger Group effect ($F_{1,137}$=8.011, p=.005, eta^2=.055). However, for *NTA (0-6)* with *TA (0-6)* covaried, there was a marginal Group effect ($F_{1,137}$=3.714, p=.056, eta^2=.026) while for *TA (0-6)* with *NTA (0-6)* covaried, there was no Group effect ($F_{1,137}$=1.271, p=.262, eta^2=.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.

Graded adversity-nightmares relationships

Relationships between nightmare severity—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<.05) whereas for the CTL group, correlations were weaker and less numerous (2 of 6 at p<.02) but nonetheless positive in all cases. For the whole sample, all TAQ correlations with NDQ attained p<.0003.

In contrast, TAQ correlations with logNM obtained only for *TA (lifetime)* in the CTL group (p=.04) and for no measures in the NM group (all p>.35). For the whole sample, logNM correlations were substantial for 5 of 6 coefficients (all p<.05).

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**	
correlations	**logNM**	**NDQ**	**logNM**	**NDQ**	**logNM**	**NDQ**
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) Total	*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) total	*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 8 TAQ adversity subscales; NTA: Non-Traumatic Adversity subscale; TA: Traumatic Adversity subscale; Values in bold are p<.05

Table 4 also reveals that NDQ and logNM are strongly inter-correlated for the whole sample (r=.438, p<.0000001), less so for the CTL group (r=.281, p=.021) and not at all for the NM group (r=.046, p=.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.

Nightmare severity: frequency vs. distress

Whether observed group differences in adversity are associated more closely with nightmare distress or frequency was evaluated using oneway ANCOVAs. Covarying logNM produced a substantial multivariate Group effect (T=.098, $F_{4,134}$=3.290, p=.013, eta^2=.089) and 3 (of 4) univariate effects: *TA (lifetime)* ($F_{1,137}$=7.760, p=.006, eta^2=.054), *TA (0-6)* ($F_{1,137}$=0.689, p=.408, eta^2=.005), *NTA (lifetime)* ($F_{1,137}$=7.696, p=.006, eta^2=.053) and *NTA (0-6)* ($F_{1,137}$=5.043, p=.026, eta^2=.036); NM > CTL in all cases. In contrast, covarying NDQ produced no multivariate effect (T=.041, $F_{4,134}$=1.381, p=.244, eta^2=.040) and a marginal univariate effect for *TA (lifetime)*: $F_{1,137}$=3.465, p=.065, eta^2=.025 (all other p>.288). Thus, adversity-based group differences are more closely tied to nightmare-induced distress than to recall.

Adversity relationships with sleep spindles

As specified by hypothesis 4, closer examination of *separation* domain scores revealed correlations with slow spindle densities for the NM, but not the CTL, group (Table 5; Figure 2). Correlations for the NM group were negative between *separation (lifetime)* and spindles in

central derivations (C3: r=-0.453, p=.008; C4: r=-.496, p=.003); fewer slow spindles were associated with higher adversity scores. By age, similar relationships obtained for 0-6 years (C3: r=-0.473, p=.005; C4: r=-.540, p=.001) and to a lesser extent 7-12 years (C3: r=-0.428, p=.013; C4: r=-.399, p=.019) but not 13-18 or 19+ years. For the CTL group, no correlations were observed. For spindle frequencies, no correlations exceeded p<.05 for either group, although a vast majority of the correlations were positive as predicted (Figure 2).

Table 5. Spearman correlations between density of slow sleep spindles and Separation domain scores on the Traumatic Antecedents Questionnaire for 4 age ranges and lifetime scores among Nightmare (N=34)[a] and Control (N=29)[b] participants. Bold values: p<.02; (r): Spearman rho-value; (p): p-value.

		Slow Spindle Density					
	Separation score	**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

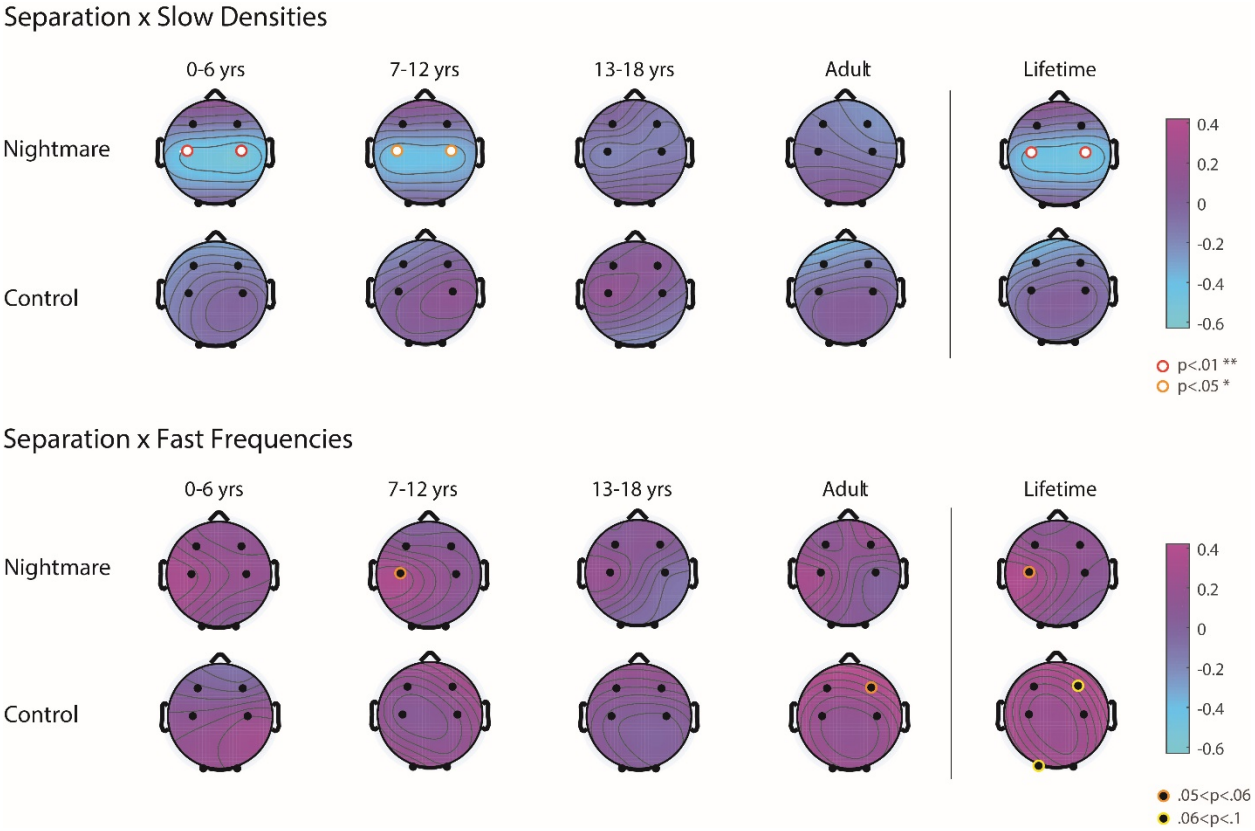

Figure 2. Spearman correlations between Traumatic Antecedents Questionnaire Separation domain scores and slow sleep spindle density (top panel) and mean frequency of fast (13-16 Hz) sleep spindles (bottom panel); results are shown for 6 cortical derivations (F3, F4, C3, C4, O1, O2), 4 age ranges and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

Discussion

Results largely support the 4 hypotheses, bolstering the notion that idiopathic nightmares constitute a reaction to early adversity. More speculatively, they support the possibility that adversity-induced nightmares share some pathophysiological mechanisms with PTSD nightmares.

Findings for hypothesis 1 (lifetime adversity) are consistent with several studies demonstrating links between past adversity and current nightmares (5, 6, 34). Global adversity for nightmare-prone participants was almost twice that for controls and 7 of 8 domain scores were markedly elevated. Consistent with the notion that nightmares are a pathology of dysphoric emotion regulation (4, 35), our largest group domain difference (apart from *Other*) was for *Emotional adversity* ($p=.003$, $eta^2=.064$). Further, group differences were largely independent of psychopathological symptoms. Global adversity, in particular, survived covarying depression and alexithymia. Unlike these Lifetime measures, however, age 0-6 measures were diminished by covarying psychopathology. Thus, even though nightmare-prone individuals reported higher 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 (reviews in 4, 36), or that current psychopathology biased participants' recall and/or reporting of early experiences.

Consistent with hypothesis 2 (0-6 adversity), scores for nightmare-prone participants were elevated for *0-6 years (lifetime)* and on 3 of 8 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 3, 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 (37). For example, distress and other PTSD symptoms escalate with the accumulation of adverse events (38); the odds of developing PTSD increase incrementally with the increasing diversity of violence types accumulated (39). 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.

Finally, and consistent with hypothesis 4, early *Separation* was associated with spindle anomalies indexing both psychopathology (17, 18) and nightmare genesis prior to age 10 (22). 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.

Findings for fast spindle frequency only weakly supported hypothesis 4 in that, for the NM group, the measure did tend to correlate positively with *Separation* at 0-6 years (p=.089) and 7-

12 years (p=.054), and also with lifetime *Separation* adversity (p=.054), but none of these correlations surpassed the p<.01 threshold correction.

Altogether, the findings add weight to a growing literature demonstrating idiopathic nightmares to be 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 pre-clinical 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, 35), common comorbidities such as anxiety and suicidal behavior (40), and shared polysomnographic characteristics (41).

In sum, 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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