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Polysomnographic Sleep Characteristics of Generally-Anxious and Healthy Children Assessed in the Home Environment

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Abstract

Background—Using laboratory-based polysomnography (PSG) we recently provided evidence of significantly prolonged sleep onset latency (SOL) and reduced latency to rapid eye movement (REM) sleep among non-depressed children with generalized anxiety disorder (GAD) compared to healthy age-matched controls. In the current study we conducted unattended ambulatory PSG in a new sample of children with GAD and controls in order to examine sleeping characteristics in the home environment.

Method—Thirty-two children (ages of 7–11 years) including 16 children with primary GAD and 16 controls receiving no psychotropic medications were studied. The anxious group had a primary diagnosis of GAD without secondary mood disorders and controls were free of any medical or psychiatric diagnoses. All participants underwent structured diagnostic assessments and completed one night of home-based polysomnography (PSG).

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Contributors

Dr. Patriquin conducted the analyses and wrote/revised drafts of the introduction, methods, and results. Dr. Mellman provided consultation on the project, including providing revisions/edits to the present manuscript. Dr. Glaze also provided consultation on the project, collected PSG data, and provided revisions/edits to the present manuscript. Dr. Alfano is the principal investigator for the study and the grant. She wrote the discussion and provided extensive feedback and edits on the manuscript drafts. All authors contributed to and have approved the final manuscript.

Conflict of Interest

The authors have no conflicts of interest to disclose.

Results—Children with GAD exhibited significantly higher sleep efficiency (SE) and fewer rapid eye movement (REM) sleep periods compared to controls. Self-reported somatic arousal during the pre-sleep period was negatively correlated with the percentage of total REM sleep among controls, but positively correlated with REM sleep percentage in the GAD group.

Limitations—A small sample size and one night of PSG only.

Conclusions—Home-based PSG recording do not provide evidence of disrupted sleep patterns in children with GAD. Contextual factors that better elucidate differences between laboratory and home-based sleep findings are suggested as important directions for future research.

Keywords

Generalized Anxiety Disorder; Children; Sleep; Polysomnography; Pre-Sleep Arousal; First Night Effects

Introduction

Subjective reports reveal upwards of 90% of children with generalized anxiety disorder (GAD) to experience sleep-related problems including longer sleep onset latency (SOL), nightmares, and difficulty waking in the morning (Alfano et al., 2006; Alfano et al., 2007; Alfano et al., 2010). Research utilizing objective measures of sleep in this population is comparatively rare. A previous study from our research group investigated the laboratory-based polysomnographic (PSG) sleep characteristics of pre-adolescent children with GAD (without comorbid depressive disorders) compared to an age-matched healthy control group (Alfano et al., 2013). We found evidence of significantly longer SOL and reduced latency to rapid eye movement (REM) sleep as well as marginally significant reductions in sleep efficiency (SE) and increased total REM sleep among children with GAD.

A notable limitation of this work is the inclusion of only one PSG night and hence the possibility of “first-night effects” (FNE). The term FNE refers to the dynamics of habituation to PSG assessment in the sleep laboratory environment and is a well-documented phenomenon in adults (Agnew et al., 1966; Webb and Campbell, 1979). Such effects, which include prolonged SOL, decreased SE, reduced total sleep time (TST), decreased amounts of REM sleep, prolonged latency to the first REM sleep period, and increased wake time after sleep onset (WASO) (Agnew et al., 1966; Webb and Campbell, 1979) are typically observed during a first night of PSG recording in a sleep laboratory but diminish or disappear over subsequent night/s. Commonly hypothesized causal factors include sleeping in a strange environment, discomfort/lack of mobility caused by sensors, being observed by experimenters/staff, and/or physiologic reactivity to a novel situation.

Few studies overall have specifically examined the presence of FNE in pediatric samples. A notable distinction between adult and pediatric sleep studies is the presence of a parent in the same room as the child throughout the PSG night. One of the largest studies included 87 healthy children between 6 and 15 years of age (Coble et al., 1984). Consistent with findings in adults, an increase in TST and decrease in SOL were found during a second night of laboratory-based PSG. Children also exhibited increased SE, decreased WASO, decreased latency to REM sleep, and increased total REM sleep on night two. In a more recent study

utilizing two nights of lab-based PSG among anxious, depressed, and control groups of children and adolescents (Forbes et al., 2008), only the depressed and control groups demonstrated a decrease in REM sleep latency on night two. Additionally, SOL was similarly prolonged across both PSG nights in the anxious group. These findings suggest that, whereas depressed and control children show adaption to the sleep laboratory after one night, anxious youth continue to experience difficulty sleeping in this setting across multiple nights. These findings may be specific to the sleep lab setting or, alternatively, reflect a biological sensitivity to novel situations/ contexts among children with GAD (Ellis and Boyce, 2008).

Consistent with the possibility of adaption effects in the laboratory environment, we found significantly prolonged SOL and reduced SE among children with GAD compared to healthy controls assessed in a sleep lab (Alfano et al., 2013). However, children with GAD also exhibited significantly reduced latency to REM sleep and increased total REM sleep, which are not typical FNE findings. These results raise additional question as to whether REM-based alterations found among children with GAD may in fact be characteristic of the disorder and override the presence of adaptation effects observed in the lab environment. To further examine these questions, the current study compared a new sample of children with GAD to non-anxious healthy control children based on one night of unattended ambulatory PSG performed in the children's home environment. Advantages of home-based PSG include assessment in the natural sleeping environment and greater convenience for families. Based on the characteristic presence of subjective sleep complaints in this population, we expected similar results as reported previously (Alfano et al., 2013). We also examined the presence of pre-sleep arousal in both groups and expected altered sleep patterns in the GAD group to be associated with greater levels of cognitive and somatic arousal during the pre-sleep period.

Methods and Materials

Participants

Thirty-two children (7 to 11 years old) including 16 with primary GAD and 16 healthy controls comprised the current sample. Children were recruited through a university-based clinic in Houston, TX using community flyers and print advertisements for a research study about "behavior and emotion" without the mention of sleep. A total of 23 anxious children were evaluated to participate. However four children were found ineligible at the initial assessment due to the presence of separation anxiety disorder or other primary disorders ($n = 2$), suspected sleep-disordered breathing ($n = 1$), or $IQ < 80$ ($n = 1$). Of the remaining 19 children, 3 families were excluded: 2 participants were unable to complete the PSG due scheduling problems and 1 participant reported symptoms of/had suspected sleep disordered breathing. The 3 eligible GAD children without PSG data were compared to the final GAD sample ($n = 16$) in terms of demographic variables (see Table 1) as well as severity of GAD (ADIS-C/P Clinical Severity Scores and the PSWQ-C; see below). No significant differences were detected.

A total of 17 control children were enrolled and completed the study. However, one child had unusable PSG data due to equipment malfunction (resulting in a sample of $n=16$

controls). Control children were administered the same assessment procedures as were the GAD group including diagnostic interviews and sleep assessments. Control participants did not have psychiatric or sleep disorders or any clinically-significant emotional/behavioral problems.

All participants resided with a parent/primary caretaker and were enrolled in a regular classroom setting. Exclusion criteria for both GAD and control groups included: a) current/lifetime history of a psychotic, pervasive developmental, bipolar, attention-deficit/hyperactivity, bipolar, oppositional defiant, substance abuse, tic, eating or conduct disorder; b) present use of treatment services for emotional, behavioral, or sleep problems, including either behavioral or pharmacological interventions; c) regular use of any medications known to impact sleep (e.g., anti-histamines, melatonin); e) chronic medical illness that requires a daily medication regimen (e.g., diabetes, asthma).

Measures

The Anxiety Disorders Interview Schedule for DSM-IV – Child Version (ADIS-C/P; Silverman and Albano, 1996) was used to determine clinical diagnoses. The ADIS-C/P is a well-validated measure for the assessment of anxiety in children (Silverman et al., 2001). A Ph.D. level psychologist or trained doctoral level graduate student administered the ADIS separately to the child and parent. All cases were reviewed with a licensed clinical psychologist prior to assigning final diagnoses. ADIS-C/P clinician severity ratings (CSRs; range 0 – 8) are used to categorize disorders as primary (most severe/disabling) or secondary. Reliability for a GAD diagnosis was excellent ($\kappa = .90$). Within the GAD group, six (38%) participants had secondary diagnoses including specific phobia ($n = 2$), social anxiety disorder ($n = 2$), and separation anxiety disorder ($n = 2$). Based on ADIS-C/P interviews, 56% of GAD participants and 44% of their parents endorsed that their child had ‘difficulty sleeping’ related to worry.

Demographic and family information was collected via parent-report on a general information form. The general information form included assessment of whether either parent had ever experienced, been diagnosed with, or had been treated for depression. Two (12.5%) GAD families had a parent with a history of depression. No control families had a parent with a history of depression.

As part of a battery of measures, children completed a measure of pre-sleep arousal during the initial diagnostic assessment. The Pre-Sleep Arousal Survey for Children (PSAS-C; Gregory et al., 2008) is a 16-item measure used to assess a child’s current physiologic, mental, and overall arousal prior to sleep onset. Responses are indicated on a 5-point scale and items are summed to form somatic, cognitive, and total scores. In the current study, PSAS-C total and subscale scores demonstrated acceptable to good internal consistency: somatic ($\alpha = .75$), cognitive ($\alpha = .87$), and total ($\alpha = .87$).

Pubertal status was determined based on parent-report on the Pubertal Development Scale (PDS; Carskadon and Acebo, 1993). A score of 3 or below is interpreted as pre-pubertal status in both boys and girls. PDS scores in the current sample ranged from 1.00 to 2.40 ($M = 1.50$; $SD = .38$). Children’s body mass index (BMI) was also assessed. See Table 1.

Study Procedures

The study protocol was approved by the Institutional Review Board and all families were paid for their time and efforts. Informed consent included a detailed description of all study procedures and presentation of equipment to be used on the PSG night. Parents and children were required to sign consent/assent forms and were given a copy of the forms. At the time of consent, sample sensors were demonstrated and participants were shown pictures of children wearing PSG equipment. Consenting families (all biological mothers except one adoptive mother) underwent interviews, completion of parent and child measures including the PDS, PSAS-C, and abbreviated IQ testing (Wechsler, 1999).

Within two weeks of the initial diagnostic assessment, participants completed one night of at-home PSG monitoring through an AASM-accredited sleep center. Participants wore wrist actigraphs during the 7 nights immediately prior to the PSG night to ensure that children were not sleep deprived and that shifted sleep schedules that might impact sleep architecture were not present. Although not presented here, actigraphy data indicated adequate amounts of and patterns of sleep prior to the PSG night. The PSG was conducted in the participant's home on the Friday or Saturday night following actigraphy data collection in order to minimize disruptions in school and family schedules. On the PSG night, participants arrived to a pediatric sleep center (at approximately 18:00) at a major medical school-based pediatric hospital where a registered PSG technologist placed sensors on the participant and demonstrated the equipment. Participants were instructed to follow their typical bedtime and routines at home. Families returned to the sleep center the next day (at approximately 8:00) to return the sleep equipment.

Ambulatory Polysomnography

Ambulatory PSG was conducted using the NicoletOne Ambulatory electroencephalogram (EEG) equipment. Registered PSG technologists with extensive training and experience with pediatric PSG performed the preliminary scoring of the PSG according to AASM scoring rules; a board certified sleep medicine specialist (DGG) reviewed the entire study and generated the final report. Since the current study was primarily interested in sleep staging, and because participants are pre-screened for any signs of sleep-disordered breathing, PSG assessment included 6 channels of electroencephalogram (EEG; frontal, central, and occipital regions), right and left electrooculogram (EOG), 1 channel of electromyogram (EMG; submental and right/left tibial), two-lead electrocardiogram (ECG), respiratory inductance plethysmography, and pulse oximetry only. Sleep scoring was conducted in 30-second epochs based on the criteria of the American Academy of Sleep Medicine (Iber et al., 2007). All technicians were naïve to child group status.

The following PSG variables were computed: total recording time (TRT = time from parent-reported lights out to lights on); total sleep time (TST = actual sleep minutes from sleep onset to the final awakening); sleep efficiency (SE = $TST/TRT \times 100$); wake minutes after sleep onset (WASO = wake minutes between sleep onset and final awakening); duration (minutes) and percentage of total sleep time for all non-REM (1–3) and REM sleep stages; REM latency (REML = minutes from sleep onset to the first epoch of REM sleep); and REM periods (REMP = periods of REM sleep with interruptions of NREM <15 min). Sleep

onset latency (SOL = time from lights out to the first epoch of any sleep stage) also was examined although calculation relied on parent-report of light-out. Additional parameters examined included breathing, oxygen saturation levels, and periodic limb movements. For all 32 participants included in the study, integrity of the sensors was maintained and the entire study from lights out to lights on could be reliably scored and evaluated.

Statistical Analyses

All analyses were conducted using SPSS 21.0 statistical software. We first compared the two groups based on all variables presented in Table 1. The groups differed significantly on paternal education and it was therefore entered as a covariate in all analyses. Also, since prior findings indicate that sex moderates sleep outcomes in depressed children (Roberts et al., 2006), sex-based differences were examined for all PSG variables across GAD and control groups while controlling for parental depression status using ANCOVA. No significant differences were found. Between group (GAD vs. control) differences were examined for PSG sleep variables and pre-sleep arousal controlling for parental depression status using ANCOVA. Race/ethnicity was also entered as a covariate based on established differences in rates of sleep problems (Alfano et al., 2010; Quan et al., 2003). Lastly, Pearson product-moment correlations were used to examine relationships between cognitive and somatic forms of presleep arousal and PSG parameters within the GAD and control groups.

Results

PSG Sleep Variables × Group

ANCOVA analyses revealed that children with GAD had significantly higher SE percentages [$F(1, 28) = 5.60, p = .03$] and significantly fewer REM periods [$F(1,28) = 4.49, p = .04$] than control children. Additionally, there was a trend for children with GAD to exhibit shorter SOL [$F(1,28) = 3.69, p = .07$]. No other significant group differences were found. See Table 2 for results.

Pre-sleep Arousal

Group comparisons indicated that GAD and control groups did not significantly differ in terms of pre-sleep cognitive ($M_{GAD} = 17.94, M_{control} = 14.71$) or somatic ($M_{GAD} = 14.69, M_{control} = 11.87$) arousal. However, Pearson product-moment correlations indicated pre-sleep cognitive and somatic arousal scores were significantly related to PSG variables in both groups.

Control group—Within the control group, pre-sleep somatic arousal was significantly positively associated with lights off time [$r(13) = -.54, p = .04$] and negative associated with percentage of REM sleep [$r(13) = -.65, p = .01$].

GAD group—In the GAD group, pre-sleep somatic arousal was significantly positively associated with percentage of REM sleep [$r(14) = .67, p = .01$]. Also, pre-sleep cognitive arousal was significantly negatively correlated with TRT [$r(14) = -.53, p = .04$] and TST [$r(14) = -.65, p = .01$].

Discussion

The current study extends previous research in clinically-anxious children studied in the sleep laboratory setting (Alfano et al., 2013; Forbes et al., 2008). Using a cohort of children not studied previously, we examined differences in PSG-based sleep parameters during overnight assessment in the home environment. None of the children with GAD were taking psychotropic medication or had secondary depressive diagnoses and analyses controlled for parental depression given evidence of objective sleep differences in depressed patients and their first-degree relatives (Giles et al., 1989; Giles et al., 1998). We also controlled for child race/ethnicity status given higher rates of sleep disturbances in minority youth (Alfano et al., 2010; Quan et al., 2003) and paternal education due to significant differences between the groups.

Surprisingly, no differences were found between the groups with the exception that children with GAD had significantly better SE and fewer REM periods than controls. The difference in SE appears to be partly attributable to both reduced SOL and reduced WASO in the GAD group; although the latter difference was non-significant and the group difference in SOL was marginally significant. Still, on average, control children required approximately twice as long to initiate sleep than children with GAD. It should be noted however that calculation of SOL relied directly upon parent report of lights out, making this a somewhat subjective assessment of sleep initiation. We also note that although the group difference was not statistically significant, lights out was more than 30 minutes later among children with GAD. Greater variability in lights out time was observed in the anxious group as well. These findings suggest parents of children with GAD may be less consistent with regard to child bedtimes, perhaps in response to greater bedtime resistance which has been found in previous studies (Alfano et al., 2010). Evidence for less consistency between weekend and weekday sleep schedules among clinically-anxious youth than controls was found in a previous study utilizing one-week sleep diaries (Hudson, Gradisar, Gamble, Schniering & Rebelo, 2009). A delayed bedtime might better align with the homeostatic sleep drive of some children, potentially resulting in shorter SOL. Conversely, since inconsistent sleep schedules, independent of total sleep, have been linked with emotional/behavioral difficulties in children (Sadeh, Raviv & Gruber, 2000; Wolfson & Carskadon, 1998), maintenance of a stable sleep-wake cycle may be particularly critical for anxious children based on the presence established emotion regulatory deficits (e.g., Carthy, Horesh, Apter & Gross, 2010). Expanding sleep-based assessments to include evaluation of sleep timing and rhythmicity is therefore a needed next step.

It also warrants mention that rates of child and parent-reported sleep problems (based on ADIS-C/P interviews) were considerably lower in the current sample than those found in previous studies based on subjective (Alfano et al., 2007; 2010) as well as laboratory-based sleep assessments (Alfano et al., 2013) of children with GAD. The reason for this finding is unclear, particularly since post-hoc comparisons between the current anxious sample and the sample examined by Alfano and colleagues (2013) did not reveal any meaningful differences based on clinical variables or pubertal development. Nonetheless, the overall lower incidence of subjectively-reported 'trouble sleeping' found in the current study necessitates replication of home-based sleep findings in other samples.

The current set of results suggests at least some of our previous findings may be related to adaption effects associated with sleeping in a laboratory environment. That is, similar to adults, children tend to exhibit an increase in SOL and a reduction in SE during the first night spent in a sleep laboratory (Coble et al., 1984). Nevertheless, anxious youth do not necessarily adapt to sleeping in this environment even during additional nights (Forbes et al., 2008) making ambulatory sleep monitoring a valuable methodological approach for examining the objective sleep patterns of this population of children. By the same token, unattended home sleep studies are not without limitations. For example, since neither observation nor video recording are available, it is unknown whether participants followed their usual sleep routines during the overnight assessment or whether some children might have shared a room or co-slept with parents or siblings on the PSG night. Co-sleeping has been shown to reduce objective measures of stress in infants (e.g., cortisol; Beijers et al., 2013) and may alter older children's sleep patterns as well.

More difficult to interpret are differences in REM-based sleep parameters between the current study and a previous laboratory-based investigation of sleep where children with GAD exhibited decreases in REM sleep latency and increases in total REM sleep – the reverse pattern associated with FNE. In fact, anxious youth assessed with PSG at home had significantly fewer REM periods than controls. Notably, REM sleep is directly linked with negative emotionality and fear extinction (Pace-Schott et al., 2009; Spoormaker et al., 2010) and alterations in REM sleep, including decreases in REM sleep latency and increases in overall REM sleep are a consistent finding in adults with affective disorders and their first-degree relatives (e.g., Giles et al., 1989; Modell et al., 2005). Interestingly, we did observe differential associations between REM sleep and pre-sleep somatic arousal, where somatic arousal correlated positively with REM sleep percentage in the GAD group but negatively with REM sleep percentage among controls. This finding may help reconcile REM-based differences found across studies. Specifically, children with GAD may experience substantially higher levels of physiologic arousal when attempting to fall asleep in a sleep-laboratory, which could in turn impact subsequent REM sleep. Higher levels of cognitive and physiologic arousal have been associated with increases in REM sleep (e.g., Kobayashi et al., 1998) and a recent study by our group found higher heart rate in anticipation of an ambiguous laboratory task predicted increased levels of pre-sleep somatic arousal in children with GAD (Patriquin, Clementi & Alfano, 2013). Situations that are ambiguous, uncertain, and/or unfamiliar are indeed remarkably challenging for children with GAD (Read et al., 2013). Future studies should use home-based assessments of sleep and pre-sleep arousal among anxious as well as depressed youth should therefore be considered.

Limitations of this study included a small sample size, one night of PSG recording only, subjective report of lights off/on, and limited assessment of parental psychopathology (e.g., depression). Parental depression was controlled for statistically but is intrinsically related to group status and cannot therefore be fully controlled for (Miller and Chapman, 2001). In general, group comparisons were underpowered to detect anything but large effects.

Taken together, findings from home and laboratory-based PSG highlight the importance of context in assessing the sleep patterns of anxious youth. We did not find evidence of disrupted or inadequate sleep in children with GAD compared to controls in the current

study. On the contrary, we found significantly better SE in the anxious group. A benefit of home-based PSG is the ability to evaluate objective sleep parameters in the natural environment. We did not, however, collect information regarding other possibly relevant contextual factors (e.g., whether usual sleep routines were followed, if children were experiencing other stressors, parental sleep presence/co-sleeping) which should be considered in future studies. It is possible that the common presence of sleep complaints in children with GAD relate more directly to pre-sleep behaviors such as bedtime avoidance as opposed to disruption during actual sleep, which may in turn bring about less consistency in sleep-wake schedules. More comprehensive assessments that include contextually-relevant sleep information are warranted.

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Table 1

Demographic Characteristics of Children with GAD and Controls

	GAD (<i>n</i> = 16)	Control (<i>n</i> = 16)	<i>t</i>/χ^2 statistic	<i>p</i> value
Age in years (M/SD)	8.8(1.3)	8.4(1.3)	.80	.43
Female (n/%)	8(50.0)	8(50.0)	.00	1.00
Race/Ethnicity (n/%)			2.40	.30
Caucasian	8(50.0)	5(31.3)		
Hispanic/Latino	3(18.8)	7(43.8)		
Other/Mixed Race	5(31.3)	4(25.0)		
Household Income (n/%)			4.90	.30
\$20–40K	1(6.3)	4(25.0)		
\$40–60K	2(12.5)	1(6.3)		
\$60–80K	2(8.3)	5(31.3)		
\$80–100K	2(12.5)	1(6.3)		
>\$100K	9(56.3)	5(31.3)		
Marital Status (n/%)			1.14	.29
Married	13(81.3)	15(93.8)		
Maternal Education (n/%)			.82	.37
< College degree	2(12.5)	4(25.0)		
College degree or >	14(87.5)	12(75.0)		
Paternal Education (n/%)			4.50	.03
< College degree	5(31.3)	11(68.8)		
College degree or >	11(68.8)	5(31.3)		
BMI (M/SD)	17.0(2.9)	17.3(2.5)	.39	.71
PDS (M/SD)	1.5(.38)	1.5(.39)	.70	.49

Note: GAD=Generalized Anxiety Disorder; BMI=Body Mass Index; PDS=Pubertal Development Scale; BMI data were missing for GAD (*n* = 3) and control (*n* = 8) participants.

Table 2

Polysomnographic Variables in Children with GAD and Controls

	GAD (n = 16)	Control (n = 16)	F statistic	p value	Partial η^2
Lights Out (M/SD)	21:56(120.52)	21:20(63.1)	.15	.70	.01
Lights On (M/SD)	06:56(36.3)	07:08(52.5)	.66	.42	.02
TRT (M/SD)	578.9(61.7)	588.8(71.0)	.88	.35	.03
TST (M/SD)	510.8(48.6)	498.0(50.4)	.47	.50	.02
SE (%)	92.1(5.4)	86.7(7.6)	5.60	.03	.17
SOL (M/SD)	11.2(14.3)	25.2(27.4)	3.69	.07	.12
WASO (M/SD)	34.0(32.0)	54.3(41.5)	2.42	.13	.08
N1 (%)	2.7(1.7)	1.8(1.5)	.47	.50	.02
N2 (%)	50.2(6.6)	47.0(7.0)	1.80	.19	.06
N3 (%)	25.4(5.5)	26.8(5.3)	.06	.81	.00
REM (%)	21.8(3.3)	24.3(6.3)	3.12	.09	.10
REML	146.4(59.2)	134.2(59.6)	.71	.41	.03
REMP (M/SD)	4.7(1.01)	5.56(1.6)	4.49	.04	.14

Note: GAD = Generalized Anxiety Disorder; TRT = Total Recording Time; TST = Total Sleep Time; SE = Sleep Efficiency; SOL = Sleep Onset Latency; WASO = Wake after Sleep Onset; N1 = non-REM Stage 1; N2 = non-REM Stage 2; N3 = non-REM Stage 3; REM = Rapid Eye Movement Sleep; REML = REM Latency; REMP = REM periods.