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Polysomnographic sleep patterns of non-depressed, non-medicated children with generalized anxiety disorder

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Abstract

Background—Polysomnographic (PSG) studies of children with psychiatric illness have primarily focused on depressed samples. Children with generalized anxiety disorder (GAD) report high rates of sleep problems yet investigation of objective sleep patterns in non-depressed children with GAD are unavailable. Identification of unique clinical features linking early GAD with sleep disturbance, including possible HPA activation during the pre-sleep period, is needed to inform understanding of effective treatments.

Method—Thirty non-medicated, pre-pubescent children (ages 7–11 years) were assessed including 15 children with GAD and 15 matched healthy controls. Anxious children had GAD as their primary diagnosis and did not meet criteria for secondary mood disorders. All participants underwent structured diagnostic assessment and laboratory-based polysomnography (PSG). State anxiety and salivary cortisol were assessed prior to light out on the PSG night.

Results—Children with GAD showed significantly increased sleep onset latency and reduced latency to rapid eye movement (REM) sleep compared to controls. Marginal differences in form of reduced sleep efficiency and increased total REM sleep also were found in the GAD group. Pre-sleep anxiety and cortisol levels did not differ between the two groups.

Limitations—A small sample size, time-limited assessment of cortisol, and possible first night effects should be considered.

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Conflicts of Interest None.

Contributors Dr. Alfano designed the study, oversaw data collection, conducted analyses and wrote the manuscript. Ms. Reynolds conducted data collection, assisted with analyses and edited the manuscript. Ms. Scott assisted with data collection and scores sleep studies. Dr. Mellman assisted in the design of the study, and wrote the manuscript. Dr. Dahl assisted with the design of the study and edited the manuscript. All authors contributed to and have approved the final manuscript.

Conclusions—Results of this study provide initial evidence of PSG-based differences in children with GAD compared to controls. Follow-up studies are needed to explore the course of sleep alterations and whether targeting sleep problems early in the course of GAD might improve clinical outcomes.

Keywords

Generalized Anxiety Disorder; Children; Depression; Sleep; Polysomnography; Cortisol

Introduction

Generalized anxiety disorder (GAD), characterized by pervasive, uncontrollable worry, somatic complaints, and negative affectivity, is one of the most commonly occurring affective disorders in all age groups (Costello et al., 2004; Wittchen et al., 1994). GAD is a chronic disorder associated with increased service utilization, disability and health care costs, as well as reduced quality of life (Barrera and Norton, 2009; Kessler et al., 1999; Roy-Byrne, 1996). Relapse rates, even among successfully treated patients, are among the highest of any anxiety disorder (Bruce et al., 2005; Yonkers et al., 2003). A substantial proportion of patients also meet diagnostic criteria for major depressive disorder (MDD) with evidence for concurrent and sequential comorbidity between childhood anxiety and later depression (Moffitt et al., 2007, a; Pine et al., 2001; Pine et al., 1998). The extensive comorbid, genetic and clinical overlap between GAD and depression (Kendler, 1996; Weissman et al., 2005; Beesdo et al., 2010; Moffitt et al., 2007, b) has fueled interest in identifying early biologic markers and risk factors that might signal more precise pathways toward these individual disorders.

Insomnia is also a prominent feature of GAD and is included as a symptom criterion in DSM-IV (APA, 2000). Up to 70% of adults with GAD report problems falling and/or staying asleep (Monti and Monti, 2000) which result in functional impairments beyond the effects of anxiety alone (Ramsawh et al., 2009). Polysomnographic (PSG) findings in this population confirm the presence of sleep initiation/maintenance difficulties including reduced total sleep time (TST), prolonged sleep onset latency (SOL), increased wake minutes after sleep onset (WASO), and reduced sleep efficiency (SE; Arriaga and Paiva, 1991; Papadimitriou et al., 1988; Rosa et al., 1983; Saletu et al., 1994). The transition from wake to sleep would appear particularly vulnerable for individuals with GAD since chronic worry, rumination, muscle tension, and feelings of hyperarousal are characteristic of the disorder while diminishing control of cognitive activity and physiologic arousal are essential for sleep onset (Ogilvie, 2001). Although research examining specific clinical symptoms and precise mechanisms linking GAD and insomnia is limited, such findings might inform choice and/or development of effective intervention strategies for this population.

Similar to their adult counterparts, nearly 90% of children with GAD experience difficulty sleeping based on parent and self-reports (Alfano et al., 2006; Alfano et al., 2007; Alfano et al., 2010). Objective sleep patterns in this population have unfortunately received limited attention however and the extent to which subjective complaints correspond with actual sleep patterns is relatively unknown. It cannot be assumed however that children share the

same sleep patterns as adult GAD patients. Example comes from the trait-like sleep abnormalities found in adults with MDD (Reynolds and Kupfer, 1987), including reduced latency to rapid eye movement (REM) sleep, increased REM sleep, and decreased slow wave sleep (SWS) that have not been consistently found in depressed children (see Lofthouse et al., 2009). The precise nature of these developmental differences is unclear but could relate to a more powerful homeostatic sleep drive during childhood that overrides sleep disruption (Carskadon, 2002; Dahl, 1996).

One published study examined the sleep of youth with different DSM-IV anxiety disorders including GAD [as well as panic disorder, separation anxiety disorder (SAD), and social anxiety disorder (SOC)] during two consecutive nights of PSG (Forbes et al., 2008). Anxious children and adolescents exhibited a significantly greater number of awakenings than depressed youth and decreased slow wave sleep compared to both depressed and healthy children. As compared to the first PSG night, the anxious group exhibited prolonged SOL on the second night, whereas latency to REM sleep decreased in the depressed and control groups from night one to night two. Together with a greater percentage of missing data in the anxious group on night two, results suggest that anxious children may have more difficulty adapting to the sleep lab setting. Also, because anxious youth with comorbid MDD were included in the sample, the extent to which findings generalize to purely anxious children remains to be examined.

Based on the disorder's clinical presentation, examination of hypothalamic-pituitary-adrenal (HPA) axis activity may be of importance for understanding the mechanisms and development of sleep disturbance in GAD. Increased levels of cortisol which index HPA functioning are linked with an anxious temperament and have been shown to predict later internalizing problems (Goldsmith and Lemery, 2000). HPA activity and sleep also follow a circadian pattern and are reciprocally related whereby disruption in one system may create vulnerability in the other (Buckley and Schatzberg, 2005; Van Cauter and Spiegel, 1999). Two studies have reported atypical patterns of cortisol secretion among anxiety-disordered youth in the hours prior to and during the sleep period (Feder et al., 2004; Forbes et al., 2006). In one study, pre-sleep cortisol was significantly elevated in anxious children as compared to adolescents, reflecting possible adaptation of the HPA axis to chronic anxiety (Forbes et al., 2006). However, in both studies, children with a variety of anxiety disorders were examined.

The aim of the current study was to examine the PSG sleep characteristics of children with a primary diagnosis of GAD in comparison to a matched control group of healthy children. Based on the sleep alterations found in depressed patients and the fact that GAD and MDD commonly co-occur, anxious children with secondary mood disorders were excluded. In the absence of objective sleep findings in youth with GAD, we hypothesized that PSG sleep patterns would mirror those found in adult patients (including reduced TST, increased SOL, increased WASO, and reduced SE). We also examined self-reported state anxiety and salivary cortisol prior to sleep on the PSG night, hypothesizing increased levels of anxiety and neurophysiologic arousal (HPA activity) in GAD participants compared to controls.

Methods and Materials

Participants

The sample included 30 children (7 to 11 years) including 15 with primary GAD and 15 controls. Children were recruited through an anxiety specialty clinic at a pediatric hospital using community flyers and print advertisements for a research study about “behavior and emotion” without mention of sleep. A total of 32 children were evaluated in order to attain a sample of 15 children with GAD. Seven children were deemed ineligible at the initial assessment due to the presence of depression or other primary disorders ($n=4$), suspected sleep-disordered breathing ($n=3$), or $IQ < 80$ ($n=1$). Of the remaining 25 eligible children, 8 families decided not to complete the PSG study due to child anxiety or scheduling problems and 2 studies were lost due to computer problems. The 10 eligible GAD children without PSG data were compared to the final GAD sample ($n=15$) 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.

Control children were recruited as part of the same project and were chosen for inclusion in the current study based on similarity of demographic characteristics to the GAD group as shown in Table 1. Control children underwent the same assessment procedures as anxious youth including diagnostic interviews and sleep assessments. Control children were free of psychiatric or sleep disorders as well as any clinically-significant emotional/behavioral problems.

All participants were required to reside with a parent/primary caretaker and be enrolled in a regular classroom setting. Exclusion criteria included: a) current/lifetime history of a depressive, psychotic, pervasive developmental, bipolar, eating or conduct disorder; b) use of medications known to impact sleep or cortisol (e.g., anti-depressants, stimulants, corticosteroids); c) $IQ < 80$; d) chronic medical condition requiring a daily medication regime; e) diagnosis, evidence or indicators of sleep-disordered breathing (e.g., snoring, snorting, gasping during sleep); f) a body mass index (BMI) ≥ 25 ; and g) current treatment services for an emotional, behavioral or sleep problem.

Measures

Clinical diagnoses were determined based on the Anxiety Disorders Interview Schedule for DSM-IV – Child Version (ADIS-C/P; Silverman and Albano, 1996) which is well validated for the assessment of anxiety in children (Silverman and Saavedra, 2001). Children and one parent were interviewed separately by a Ph.D. level psychologist. 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 primary GAD diagnosis in the current study was excellent ($\kappa = 1.0$). Seven (47%) participants had secondary diagnoses including SOC ($n=3$), attention deficit/hyperactivity disorder ($n=2$), SAD ($n=1$), specific phobia ($n=1$), and oppositional defiant disorder ($n=1$). Based on ADIS-C/P interviews, 82% of GAD participants and 88% of their parents endorsed ‘difficulty sleeping’ related to worry.

A General Information Form used to collect demographic and family information was completed by the (interviewed) parent. The form included assessment of whether either parent had ever experienced, been diagnosed with or treated for depression. Five (33.3%) GAD families and two (13.3%) control families had a parent with a history of depression. Children also completed the Penn State Worry Questionnaire for Children (PSWQ-C; Chorpita et al., 1997) as well as the State Trait Anxiety Inventory for Children-State scale (STAIC; Spielberger, 1973) on the PSG night.

Pubertal status was determined based on parental report on the Pubertal Development Scale (PDS) developed by Carskadon and Acebo (Clarkson 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 .80 to 2.6 ($M=1.4$; $SD=.47$). See Table 1.

Study Procedures

The study protocol was approved by the appropriate 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 forms along with sample electrodes to take home. Consenting families (all mothers except for two fathers) underwent interviews, completion of parent and child measures, and abbreviated IQ testing (Wechsler, 1999).

Within two weeks of the diagnostic assessment, families were asked to arrive at the sleep laboratory at 18:00 for one night of PSG (typically a Friday). 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. The decision to conduct one PSG night only was informed by previous research showing evidence of poor adaptation to the sleep lab among anxiety-disordered children (Forbes et al., 2008). Children were given time to get settled, relax, read, watch quiet movies, or talk before preparation for the PSG during which a parent/guardian slept in the same room. Total cortisol concentrations were assessed by collecting a saliva sample at 30 minutes prior to and at lights out. Timing and procedures for saliva collection were strictly controlled and participants abstained from eating, drinking, or teeth brushing during the hour prior to the first saliva sample.

Polysomnography

Standard multichannel PSG was conducted using Medcare amplifiers and Rembrandt 9.0 Sleep Acquisition Software. Polysomnographic technicians with extensive training working with children conducted and scored sleep studies. Technicians were instructed to allow participants to sleep for a maximum of 8.5 hours (510 minutes) from the time of sleep onset in the lab (i.e., to ensure that sleep architecture was not truncated in the event of prolonged sleep onset). Technicians were blind to child diagnostic status. Electroencephalogram (EEG; frontal, central, and occipital regions), electrooculogram (EOG), electromyogram (EMG; submental, right/left tibial), electrocardiogram (ECG) and oximetry data were collected.

Sleep scoring was conducted in 30-second epochs based on the criteria of the American Academy of Sleep Medicine (Iber et al., 2007).

The following PSG variables were computed: total recording time (TRT=time from 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); sleep onset latency (SOL=time from lights out to the first epoch of any sleep stage); wake minutes after sleep onset (WASO=wake minutes between sleep onset and final awakening); percentage of REM and non-REM sleep stages 1-3; REM latency (LREM=minutes from sleep onset to the first epoch of REM sleep); REM periods (REMP=periods of REM sleep with interruptions of NREM<15 min); and REM density [REMD= total number of REMs (i.e., minimal 25 μ V excursions within 200 milliseconds) divided by total REM minutes] (Benson and Zarcone, 1993; Mellman et al., 2002).

Cortisol

Salimetrics oral swabs and storage tubes were used to collect saliva. Participants held oral swabs under their tongue for one minute prior to placement in a labeled plastic tube. Samples were frozen at -80° C within 10 minutes of collection and stored frozen until batched and shipped on dry ice to Salimetrics (State College, PA) for analysis by duplicate using a highly sensitive enzyme immunoassay. The test used 25 μ l of saliva and had a sensitivity range of from .003 to 1.8 μ g/dl.

Statistical Analyses

All analyses were conducted using SPSS 19.0 statistical software. Parental depression status was entered as a covariate for all group comparisons using analyses of covariance (ANCOVA). Although sex has been found to moderate objective sleep outcomes in depressed children (Roberts et al., 2006) sex-based differences for PSG variables across the groups were non-significant.

Results

PSG Sleep Variables $\times$ Group

As shown in Table 2, bedtime (lights out time) did not differ between the groups. However, lights on occurred significantly later in the GAD group. In addition, children with GAD evidenced significantly prolonged SOL and reduced REML compared to controls. Group differences in REML are depicted in Figure 1. SE was decreased and percentage of REM sleep was increased among children with GAD at a level approaching significance.

Pre-Sleep State Anxiety and Salivary Cortisol

A repeated-measure ANCOVA was used to examine main and interaction effects for pre-sleep total concentrations of cortisol. Main effects for time [$F(1, 25)=1.37, p>.05$] and group [$F(1, 25)=3.16, p>.05$] were non-significant. In addition, state anxiety at bedtime did not differ between the GAD and control groups [$F(1, 26)=2.65, p>.05$]. Group means and SD are provided in Table 3.

Post-Hoc Analyses of REM Latency

Because REM sleep is influenced by a number of factors including circadian phase and homeostatic sleep processes, we were interested in determining whether the significant group difference in SOL might account for a reduced latency to the first REM period in children with GAD. An ANCOVA using SOL as a covariate was conducted comparing REML between the two groups. The result just failed to reach statistical significance [$F(1, 26)=3.89, p=.059$].

Second, we compared children within the GAD group in terms of presence/absence of parental depression, demographic characteristics, and self-reported worry (PSWQ-C) based on a median split of REML (median=129 minutes; $SD=39.0$). A significant difference emerged for sex only; a greater percentage of anxious girls (78%) compared to boys (17%) exhibited REML less than 129 minutes [$\chi^2(1)=5.40, p<.05$].

Discussion

The current study sought to provide needed information about the objective sleep patterns of children with primary GAD in comparison to a matched sample of healthy children. In light of the developmental complexities inherent in studying sleep our sample was limited to pre-pubertal children to minimize the effects of circadian sleep changes typically occurring during adolescence. Also, based on evidence of objective sleep abnormalities in depressed patients as well as their first-degree relatives (Giles et al., 1989; Giles et al., 1998), children with comorbid depression were excluded and current/lifetime depression in either parent was included as a covariate. Lights on time in the morning occurred significantly later in GAD participants, presumably as a result of an increased amount of time required to initiate sleep among this group. REML also was significantly shortened compared to controls, and trends for reduced SE and a greater percentage of REM sleep were detected in the GAD group. Differences in state anxiety and salivary cortisol during the 30 minutes prior to bedtime were non-significant.

SOL was relatively prolonged in all children in our study, likely reflecting the presence of first night effects in the lab. Nonetheless, children with GAD required almost twice as long as controls to initiate sleep, corroborating frequent complaints of insomnia in this population (Alfano et al., 2006; Alfano et al., 2007; Alfano et al., 2010). The range of factors can interfere with the ability to initiate sleep including anxiety/worry, hypervigilance, and somatic arousal, each of which has been shown to characterize GAD patients. Although we did not find evidence of increased anxiety or neurophysiologic arousal during the pre-sleep period, it is possible that our sample size was inadequate and/or that the narrow window during which cortisol was assessed was insufficient to capture meaningful differences. Further, having a parent sleep in the same room may have served to reduce stress levels considerably among anxious children in particular. We did not assess cognitive activity/worry at bedtime, yet because adult GAD patients commonly identify their worries as interfering with sleep (Bélanger et al., 2005; Gendron et al., 1998) this may be an important area for future research in children as well. In a previous study of children with various anxiety diagnoses, significantly greater levels of pre-sleep cognitive rather than somatic arousal were reported (Alfano et al., 2010).

We also found differences in the REM sleep patterns of the two groups in the form of reduced REML and a marginally increased percentage of REM sleep in GAD children. Girls with GAD evidenced significantly shorter REML than boys; a finding that has also been reported in depressed youth (Roberts et al., 2006). GAD shares extensive symptomatic and genetic overlap with MDD and, even though our GAD sample was non-depressed, the possibility that shared genetic factors in part account for this convergence of findings cannot be ruled out. A previous study of anxious children (Forbes et al., 2008) did not find evidence of REM sleep alteration. However differences could be explained by a singular focus on GAD which is associated with a greater frequency of sleep disruption than other anxiety disorders (Alfano et al., 2006; Alfano et al., 2010). It is also important to note that although REM abnormalities have generally not been identified in adults with GAD (Rosa et al., 1983; Saletu et al., 1994; Fuller et al., 1997; Reynolds et al., 1983), methodological limitations of this research including the use of inpatient samples, sub-clinical GAD symptoms, and/or former DSM GAD criteria which demonstrated poor diagnostic reliability should be considered (Andrews et al., 2010).

On the other hand, there is limited evidence to suppose that the pathophysiology of GAD is identical across the developmental spectrum and that the disorder reflects the same condition at different ages. Early-onset GAD (i.e., prior to adolescence) is a relatively unstable diagnosis as evidenced by considerable heterotypic continuity over time. For instance, childhood GAD predicts a range of later disorders including conduct disorder (Bittner et al., 2007), agoraphobia (Copeland et al., 2009) and depression (Pine et al., 2001; Pine et al., 1998) and unique trajectories are poorly understood. Such findings have fueled suggestion that childhood GAD might be better conceptualized as a non-specific prodromal rather than a distinct clinical condition (Barlow, 1988). In this view, REM sleep alterations could represent markers of risk (e.g., Giles et al., 1989) rather than neurobiological markers of GAD. Longitudinal studies are therefore needed to examine whether early sleep patterns might provide improved prediction of developmental trajectories and outcomes associated with pediatric GAD.

Despite a reduced latency to the first REM period, SWS was preserved in children with GAD. This finding is consistent with sleep macroarchitecture findings in depressed children but not with results from a mixed sample of anxious children (Forbes et al., 2008). The latter study included both children and adolescents however, and because SWS is strongly influenced by development, full neurobiological maturation may be required for alterations of this sleep parameter to manifest (Dahl, 1996). In addition, whereas SWS does not appear to reliably prognosticate depression risk in adults (Giles et al., 1989; Buysse et al., 1997) recent findings suggest that preservation of SWS in childhood may be protective for depression. In a long-term follow-up study of children at familial risk for MDD, those with greater amounts of SWS in childhood were less likely to develop depression as adolescents or young adults (Silk et al., 2007). Understanding of whether this relationship might extend to early GAD will require structured follow-up assessment into later periods of development.

Although our study included a well-characterized sample of pre-pubescent children, limitations exist. Our sample size was small and group comparisons were generally

underpowered to detect anything but large effects. Specific exclusion criteria used as well as the relatively affluent status of our sample may limit the generalizability of results. Assessment of cortisol provided only a 'snapshot' of HPA activity immediately prior to the sleep period. Although we assessed parental depression, structured clinical interviews were not used and presence of depression in other first degree relatives was not evaluated. Further, because parental depression status is intrinsically related to group status, which could not be randomly assigned, it was not possible to fully 'control' for this variable (Miller and Chapman, 2001). The use of only one night of PSG means that first night effects are possible. Lastly, PSGs were generally conducted on Friday evenings and there is evidence to suggest that objective sleep parameters vary on school days and weekends (Ohayon et al., 2004).

Results of this initial study should be considered in the context of previous EEG sleep research in both children and adults. Evidence of REM-based sleep alteration in pre-pubertal children with GAD is a novel finding with potential importance for the developmental course of the disorder. Understanding whether these sleep patterns represents unique neurobiologic features of early GAD or prodromal marker of future depression risk could inform developmental models of the disorder. Moreover, since sleep and emotional functioning are bi-directionally related, particularly in children with affective disorders (Cousins et al., 2011), behavioral interventions that target problems related to sleep initiation might serve to produce more positive and sustainable treatment outcomes by mitigating added pressure on emotional systems (Forbes and Dahl, 2005). Based on data among adult GAD patients (Bélanger et al., 2005), replacing avoidance strategies with more effective approaches for managing sleep difficulties may be especially important to consider in this population.

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Figure 1. Latency in minutes to the first epoch of rapid eye movement (REM) sleep in children with GAD and healthy controls.

Table 1

Demographic Characteristics of Children with GAD and Controls

	GAD (n=15)	Control (n=15)	t/X² statistic	p value
Age in years (M/SD)	8.5(1.5)	8.9(1.3)	.77	.45
Female (n/%)	9(60)	9(60)	1.0	.65
Race/Ethnicity (n/%)			4.8	.44
Caucasian	10(66.7)	10(66.7)		
African Amer	1(6.7)	4(26.6)		
Asian Amer	1(6.7)	0(0)		
Hispanic/Latino	1(6.7)	0(0)		
Other/Mixed Race	2(13.2)	1(6.7)		
Household Income (n/%)			4.97	.42
\$59K	3(21.4)	3(20)		
\$60–99K	1(7.2)	2(13.3)		
>\$100K	10(71.4)	10(66.7)		
Marital Status (n/%)			3.69	.29
Married	10(71.4)	13(86.7)		
Maternal Education (n/%)			.409	.34
< College degree	5(35)	3(20)		
College degree or >	9(65)	12(80)		
Paternal Education (n/%)			1.05	.59
< College degree	4(29)	4(27)		
College degree or >	10(71)	11(73)		
BMI (M/SD)	15.9(1.3)	17.3(2.5)	1.7	.10
PDS (M/SD)	1.4(.41)	1.4(.53)	.36	.73

Note: GAD=Generalized Anxiety Disorder; BMI=Body Mass Index; PDS=Pubertal Development Scale; some demographic data were missing for one GAD participant.

Table 2

Polysomnographic Variables in Children with GAD and Controls

	GAD (n=15)	Control (n=15)	F statistic	p value	Partial η^2
Lights Out (M/SD)	21:26(41.0)	21:39(25.6)	1.97	.17	.07
Lights On (M/SD)	06:51(53.4)	06:36(54.2)	7.63	.01	.22
TRT (M/SD)	588.1(41.0)	561.6(33.4)	6.28	.02	.19
TST (M/SD)	485.1(39.1)	475.0(39.0)	.833	.37	.03
SE (%)	84.5(.06)	88.9(.06)	3.75	.06	.12
SOL (M/SD)	53.7(26.8)	28.9(28.0)	5.29	.03	.16
WASO (M/SD)	36.3(32.8)	31.9(21.3)	.433	.52	.02
N1 (%)	1.3(.86)	1.7(.96)	1.35	.26	.05
N2 (%)	49.8(4.5)	51.5(5.5)	.829	.37	.03
N3 (%)	24.9(5.5)	25.5(3.7)	.006	.94	.00
REM (%)	24.0(3.4)	21.3(3.2)	3.46	.07	.11
REML (M/SD)	112.5(39.0)	157.9(53.9)	5.72	.02	.18
REMP (M/SD)	4.3(.72)	3.9(.96)	1.128	.29	.04
REMD (M/SD)	1.82(.92)	1.68(.99)	.230	.64	.00

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=REM Latency; REMD=REM Density; REMP= REM periods.

Table 3

Pre-Sleep Anxiety and Salivary Cortisol in Children with GAD and Controls

	GAD (n=15)	Control (n=15)
STAIC	10.9(3.5)	8.5(4.3)
CORTISOL1	.032(.02)	.042(.02)
CORTISOL2	.028(.01)	.038(.02)

GAD=Generalized Anxiety Disorder; STAIC=State/Trait Anxiety Scale for Children; CORTISOL 1=salivary cortisol In µg/dl 30 min prior to lights out; CORTISOL 2=salivary cortisol in µg/dl at lights out. All comparisons $p>.05$.