A Preliminary Multimethod Comparison of Sleep Among Adolescents With and Without Generalized Anxiety Disorder

Benjamin C. Mullin, Laura Pyle, Dustin Haraden, Justin Riederer, Natalie Brim, David Kaplan & Douglas Novins

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Introduction

Obtaining sufficient sleep is essential to healthy development. However, intermittent sleep disturbance is a common experience for children and adolescents (A. Kahn et al., 1989; Morrison, McGee, & Stanton, 1992; Ohayon, Roberts,
Zulley, Smime, & Priest, 2000; Owens, Spirito, McGuinn, & Nobile, 2000; Paavonen et al., 2000). Among youth, sleep may be disturbed in a variety of ways, from difficulty initiating or maintaining sleep, to nightmares, to simply obtaining an insufficient quantity of sleep. Correlational studies indicate that reductions in children’s sleep quantity or quality are associated with increased daytime sleepiness (Sadeh, Raviv, & Gruber, 2000), diminished school performance (Dewald, Meijer, Oort, Kerkhof, & Bögels, 2010), inattention, impulsivity, and poor behavioral regulation (Paavonen et al., 2009; Sadeh, Gruber, & Raviv, 2002; Steenari et al., 2003). Similarly, youth who have been experimentally deprived of sleep show decrements in attention (Beebe et al., 2008; Fallone, Acebo, Arnedt, Seifer, & Carskadon, 2001) and executive functions (Beebe et al., 2008). Emotional functioning, in particular, may be linked to sleep in a bidirectional fashion (M. Kahn, Sheppes, & Sadeh, 2013). In adults, a growing literature indicates that strong emotions disturb sleep but also that inadequate sleep degrades one’s ability to regulate daytime emotions (see review in Walker & van der Helm, 2009). Youth with anxiety disorders, who struggle to regulate high-arousal negative emotions (Suveg & Zeman, 2004), therefore may be especially vulnerable to chronic sleep disturbance.

Sleep disturbance and anxiety appear connected among youth, yet drawing conclusions is challenging given the small number of studies and the variation in methodologies and study findings. In this brief review of literature, we distinguish between subjective and objective measures of sleep. By subjective, we refer to measures that rely on a child’s or parent’s judgment regarding the quantity or quality of sleep. This primarily includes questionnaires and prospective sleep diaries. By objective, we refer to measures that estimate sleep without any judgments required by children or parents, such as polysomnography (PSG) and actigraphy (see Table 1 for definitions of key sleep terms).

To be clear, these latter methods do not provide unbiased, direct measurements of sleep but do estimate sleep based on physiological data rather than subjective impressions.

**Subjective Measures of Sleep Among Youth With Anxiety Disorders**

Parents of children with anxiety disorders endorse high rates (up to 85%) of child sleep disturbance (Alfano, Pina, Zerr, & Villalta, 2010), especially trouble sleeping, being over-tired, sleeping less than most children (Alfano, Beidel, Turner, & Lewin, 2006; Alfano, Ginsburg, & Kingery, 2007; Alfano et al., 2010; Chase & Pincus, 2011; Kendall & Pimentel, 2003; Masi et al., 2004), and waking throughout the night (Ivanenko, Crabtree, Obrien, & Gozal, 2006). Important to note, the degree of sleep disturbance has been shown to correlate cross-sectionally with overall parent-rated (Alfano et al., 2007) and child-rated (Chase & Pincus, 2011) anxiety severity. However, rates of sleep disturbance are lower (54%) when looking at the self-report of children with anxiety disorders (Alfano et al., 2010). In a study employing a week of sleep diaries, children with anxiety disorders endorsed going to bed later, and obtaining less total sleep than healthy children, but did not endorse more difficulty falling or staying asleep (Hudson, Gradisar, Gamble, Schniering, & Rebelo, 2009).

Although these studies illuminated potential connections between sleep and pediatric anxiety, they relied on parent-report of children’s sleep patterns (Alfano et al., 2006; Alfano et al., 2010) or the use of items from general clinical measures, rather than actual sleep questionnaires, to assess sleep (Alfano et al., 2006; Alfano et al., 2007; Alfano, Zakem, Costa, Taylor, & Weems, 2009; Caporino et al., 2015).

**Objective Measures of Sleep Among Youth With Anxiety Disorders**

Relatively fewer studies have assessed sleep in anxious youth using objective measures. One study employed PSG to compare two subsequent nights of sleep among children and adolescents with a primary anxiety disorder, those with primary depression, and healthy controls (Forbes et al.,

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**TABLE 1**

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polysomnography (PSG)</td>
<td>The “gold standard” method of estimating sleep. PSG involves simultaneous measurement of several physiological channels (brain waves, respiration, heart rate, eye movements, and limb movements) that are analyzed to estimate sleep and wakefulness. PSG allows for the identification of particular sleep stages.</td>
</tr>
<tr>
<td>Actigraphy</td>
<td>A method for estimating sleep that typically involves having participants wear a sensitive accelerometer embedded in a watch (actigraph) on his or her nondominant hand. The actigraph measures and stores time-locked data on gross motor movement, which is then downloaded to a computer and converted into estimates of sleep and wake using established algorithms.</td>
</tr>
<tr>
<td>Sleep Onset Latency</td>
<td>The number of minutes it takes for an individual to fall asleep at bedtime.</td>
</tr>
<tr>
<td>Slow Wave Sleep</td>
<td>A form of non-REM sleep, typically identified as Stage 3 or 4, featuring high-amplitude low frequency brain waves.</td>
</tr>
<tr>
<td>Rapid Eye Movement (REM)</td>
<td>A stage of sleep featuring high-frequency brain activity, muscle atonia, and bursts of rapid eye movements. REM sleep is believed to play an important role in emotional memory and emotion regulation.</td>
</tr>
<tr>
<td>Sleep Efficiency</td>
<td>The percentage of time in bed during which an individual is actually sleeping (i.e., minutes of sleep/minutes in bed X 100).</td>
</tr>
<tr>
<td>Wake After Sleep Onset</td>
<td>The sum total minutes of wakefulness during the night.</td>
</tr>
</tbody>
</table>
Anxious participants had more awakenings than the depressed participants, and less slow-wave sleep and longer sleep onset latencies than the depressed or control participants. That study also found evidence that several objective markers of sleep disturbance were connected to cross-sectional reports of anxiety and depression. Another recent study compared children with and without generalized anxiety disorder (GAD) using PSG, finding longer sleep onset latencies, and reduced latency to rapid eye movement (REM) sleep, among the participants with GAD relative to controls (Alfano, Reynolds, Scott, Dahl, & Mellman, 2013). In contrast, a similar study using home-based PSG found that participants with GAD actually had higher sleep efficiency and fewer REM sleep episodes per night than controls (Patriquin, Mellman, Glaze, & Alfano, 2014). These results hinted that perhaps the abnormalities detected in laboratory-based PSG studies actually reflected difficulties that anxious participants might have had adjusting to novel sleep environments, rather than being true markers of chronic sleep dysfunction.

Studies employing actigraphy, which is less intrusive than PSG and allows participants to more easily follow their typical sleep schedules, might help clarify this issue. A recent actigraphy study of children with and without GAD found only marginally longer sleep onset latencies among the GAD participants but no other actigraphic evidence of sleep disturbance despite significant endorsement of sleep disturbance by children with GAD and their parents on questionnaire measures (Alfano, Patriquin, & Reyes, 2015). In another actigraphy study of children and adolescents with a primary anxiety disorder, primary depression, and controls, the anxious youth showed longer sleep onset latencies than controls, and less wake after sleep onset and higher sleep efficiency than depressed participants but not controls (Cousins et al., 2011).

Links Between Sleep Duration and Anxiety

Piecing together the findings from prospective and experimental studies, we can begin to infer potential causal relationships between nighttime sleep and subsequent anxiety. For example, using actigraphy and ecological momentary assessment sampling of mood, Cousins et al. (2011) found that among youth with anxiety disorders, increased wakefulness after sleep onset resulted in higher negative affect ratings the next day. This effect was not present for controls or those with primary depression. Another study of healthy adolescents found that partial sleep restriction (allowing only 6.5 hr in bed per night for 5 consecutive nights) resulted in increased self-ratings of tension/anxiety relative to a “healthy sleep” condition (10 hr in bed per night for 5 nights; Baum et al., 2013). Similarly, adults subjected to 24 hr of sleep deprivation reported higher ratings of anxious arousal and general distress than those who slept normally (Babson, Trainor, Feldner, & Blumenthal, 2010). Two studies have also reported that experimental fragmentation of sleep (i.e., inserting periods of wakefulness throughout the night) produces heightened negative affect the following day (Finan, Quartana, & Smith, 2015; Martin, Engleman, Deary, & Douglas, 1996), suggesting that the continuity of sleep may also be linked to the subsequent expression of anxiety and depression.

In sum, studies employing subjective sleep measures have most consistently reported difficulties falling and staying asleep and shorter overall sleep durations among anxious youth relative to controls, whereas the majority of studies employing objective measures have documented longer sleep onset latencies among anxious youth but few other reliable findings. Interpreting findings is made more difficult due to methodological inconsistencies across studies (use of parent-vs. child-report, composite measures vs. dedicated sleep measures, etc.), and differences in samples (child vs. adolescent, single diagnosis vs. “mixed” anxiety samples). Most existing studies in this area have used either a subjective or objective method to assess sleep but rarely both; thus interpreting subjective-objective discrepancies across studies is difficult due to the use of different samples. The existing literature also focuses primarily on children rather than adolescents, yet adolescents are at a greater risk of developing insomnia than children (Fricke-Oerkermann et al., 2007) and in general have higher rates of insufficient sleep (Gradisar, Gardner, & Dohnt, 2011; McKnight-Eily et al., 2011; Wolfson & Carskadon, 1998). Further studies of anxious adolescents implementing both subjective and objective measurement of sleep, as well as studies evaluating prospective relationships between various forms of sleep disturbance and anxiety symptomatology, are needed. Clarifying the presence and nature of sleep disturbance among anxious youth will assist in the development of appropriate, targeted interventions.

CURRENT STUDY

The overall purpose of this preliminary study was to employ multiple methodologies to characterize the nature of sleep disturbance and its relevance to clinical symptomatology among adolescents with and without GAD. There is some evidence that youth with GAD experience the highest levels of sleep disturbance among the pediatric anxiety disorders (Alfano et al., 2006; Alfano et al., 2010). GAD includes insomnia as a diagnostic symptom (American Psychiatric Association, 2013), as well as other key features such as poorly controlled worry and restlessness that may interfere with sleep. This study had three aims. Our first aim was to investigate differences in sleep among participants using subjective (sleep diary and questionnaire) and objective (actigraphy) measures of sleep collected over a period of 1 week. Based on the existing literature, we anticipated finding evidence of greater sleep disturbance among the GAD participants, primarily increased sleep onset latency, wake after sleep onset, and reduced sleep efficiency via sleep...
diary, and higher scores on sleep disturbance subscales from the sleep questionnaire. With respect to the actigraphy data, we anticipated finding longer sleep onset latencies among GAD compared to healthy participants but no other significant differences. Our second aim was to investigate relationships between objective sleep parameters and self- and parent-report of anxiety and depression, assessed using questionnaires. Based on previous literature documenting the anxiogenic and depressogenic effects of shortened sleep duration and the fragmentation of sleep, we hypothesized that objective measures of decreased sleep duration and problems with sleep initiation (sleep onset latency) and maintenance (sleep efficiency) would be associated with increased levels of anxiety and depression among the entire sample. Finally, our third aim was to investigate temporal relationships between sleep duration and next-day self-report of anxiety over the course of the recording period. We hypothesized that reduced sleep duration would be predictive of higher levels of morning anxiety, particularly among participants with GAD.

**METHOD**

**Participants**

This study utilized a case-control design comparing adolescents with GAD to healthy controls. For both GAD cases and controls, participants needed to be between 12 and 18 years of age. Exclusion criteria included current or lifetime diagnoses of sleep disorders other than insomnia, psychotic disorders, and problems with sleep initiation (sleep onset latency) and maintenance (sleep efficiency) would be associated with increased levels of anxiety and depression among the entire sample. Finally, our third aim was to investigate temporal relationships between sleep duration and next-day self-report of anxiety over the course of the recording period. We hypothesized that reduced sleep duration would be predictive of higher levels of morning anxiety, particularly among participants with GAD.

**METHOD**

**Participants**

This study utilized a case-control design comparing adolescents with GAD to healthy controls. For both GAD cases and controls, participants needed to be between 12 and 18 years of age. Exclusion criteria included current or lifetime diagnoses of sleep disorders other than insomnia, psychotic disorders, or current use of hypnotic medications. The adolescents in this study were part of a larger study that included magnetic resonance imaging (MRI), thus all participants were subject to MRI-related exclusion criteria, including having non-removable metallic items on their bodies, having a fear of enclosed spaces, or being pregnant. Controls were also excluded if they met lifetime or current criteria for any Diagnostic and Statistical Manual of Mental Disorders (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000) diagnoses. Participants were recruited from the outpatient psychiatry and adolescent medicine clinics of a large, urban pediatric hospital. Potential cases were identified through a medical records search, identifying patients who met age requirements and who had previously received a diagnosis of GAD or who had not received any psychiatric diagnoses. These patients were sent recruitment letters describing the study. Recruitment flyers were also posted in community centers within the Denver Metro area.

Forty-seven adolescents were consented for this study. Of those, participation was discontinued for four individuals following the clinical assessment due to not meeting criteria for GAD (n = 3) or for meeting criteria for a psychiatric diagnosis while in the control group (n = 1). Forty-three adolescents (ages 12–18 years; see Table 2)—26 with a primary diagnosis of GAD and 17 healthy controls without any psychopathology—completed this study. All participants were living with primary caregivers and were currently attending school in a regular classroom setting. The GAD group included participants who met DSM-IV-TR criteria for a primary diagnosis of GAD, all of whom were in treatment,

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>Participant Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GAD&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Gender (% Female)</td>
<td>59.26</td>
</tr>
<tr>
<td>Race (% White)</td>
<td>84.62</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>2</td>
</tr>
<tr>
<td>Non-Hispanic</td>
<td>24</td>
</tr>
<tr>
<td>M Age in Years</td>
<td>15.13 (1.92)</td>
</tr>
<tr>
<td>Pubertal Status</td>
<td></td>
</tr>
<tr>
<td>Pre/Early</td>
<td>3</td>
</tr>
<tr>
<td>Mid/Late/Post</td>
<td>17</td>
</tr>
<tr>
<td>Missing</td>
<td>6</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>21.09 (4.14)</td>
</tr>
<tr>
<td>Body Mass Index Percentile</td>
<td>52.2 (31.9)</td>
</tr>
<tr>
<td>Mean SCARED Total Score–Self-Report</td>
<td>31.57 (16.95)</td>
</tr>
<tr>
<td>Mean SCARED Total Score–Parent-Report</td>
<td>24.95 (15.01)</td>
</tr>
<tr>
<td>Mean MFQ Total Score–Self-Report</td>
<td>19.00 (10.97)</td>
</tr>
<tr>
<td>Mean MFQ Total Score–Parent-Report</td>
<td>14.71 (9.25)</td>
</tr>
<tr>
<td>% Taking Psychiatric Medication</td>
<td>76.92</td>
</tr>
</tbody>
</table>

<sup>a</sup>n = 26.
<sup>b</sup>n = 17.

Note: Body mass index was calculated using the Centers for Disease Control and Prevention online calculator (https://nced.cdc.gov/dnpabmi/calculator.aspx). SCARED = Screen for Child Anxiety Related Emotional Disorders; MFQ = Moods and Feelings Questionnaire.
and 20 of 26 (77%) were taking psychiatric medications. Of those, 17 were taking a selective serotonin reuptake inhibitor (SSRI), three were taking an atypical antipsychotic, two were taking a stimulant, and one a tricyclic antidepressant. We did not inquire about the stability of medication usage for our participants, although no participants had any medication changes during their participation in the study. Secondary diagnoses for the GAD participants included major depressive disorder (n = 9), attention deficit/hyperactivity disorder (n = 5), dysthyemic disorder (n = 5), panic disorder (n = 5), social anxiety disorder (n = 5), specific phobia (n = 2), and obsessive-compulsive disorder (n = 1). This high rate of comorbidity among our GAD participants is consistent with previous large epidemiologic samples of adolescents (Merikangas et al., 2010). One healthy participant was taking a stimulant medication for attention but did not meet lifetime or current criteria for any disorder.

Procedure

The Colorado Multiple Institutional Review Board approved this study. Data collection took place only during the school year. The study consisted of two study visits: At the first visit, participants and a parent completed informed assent/consent documents, a joint diagnostic interview, and numerous questionnaire measures. Participants were then asked to complete 7 consecutive nights of sleep monitoring at home, wearing an actigraph and keeping a sleep diary. Each participant completed 5 weeknights and 2 weekend nights, though not always in the same order. Participants then returned 1 week later to return the actigraph and complete MRI scanning (data not described here).

Clinical Assessment

The MINI International Neuropsychiatric Interview for Children and Adolescents version 6.0 (Sheehan et al., 2010) was administered to each adolescent–parent dyad together by a licensed clinical psychologist (the first author) to evaluate the presence or absence of Axis I disorders from the DSM-IV-TR (American Psychiatric Association, 2000). The MINI International Neuropsychiatric Interview for Children and Adolescents is a brief, structured interview that has previously shown sensitivity and specificity across diagnoses, comparable to more lengthy diagnostic instruments (Sheehan et al., 2010). Symptoms were considered present when endorsed by either the parent or the adolescent. Adolescents were also interviewed without the parent present, where they were given the opportunity to change any responses that they were not comfortable voicing in front of their parent.

Anxiety symptoms were further assessed using the self- and parent-report versions of the Screen for Child Anxiety Related Emotional Disorders (SCARED) (Birmaher et al., 1997). This widely used 41-item instrument screens for DSM-IV-TR anxiety disorders and has excellent psychometric properties across clinical (Birmaher et al., 1999) and community (Hale, Raaijmakers, Muris, & Meeus, 2005) samples, including good discriminant validity between anxiety disorders and other childhood disorders (Birmaher et al., 1999). In this study, we employed the total score as a measure of current anxiety severity. A cutoff score of 25 on the self-report version has been shown to optimally capture youth in the clinically anxious range. (Birmaher et al., 1999). In our study, the SCARED demonstrated high internal consistency for both self-report (α = .96) and parent-report (α = .96). Severity of depressive symptoms was measured using the self- and parent-report Moods and Feelings Questionnaire (MFQ). The MFQ is a 34-item questionnaire that assesses depressive symptoms on a scale from 0 to 2 points (Angold, Costello, Messer, & Pickles, 1995), generating a total score. This measure has been shown to have sensitivity to depression throughout childhood and adolescence (Messer et al., 1995) and adequate ability to discriminate between major depression and anxiety disorders (Davis et al., 2006). Cutoff scores of 29 on self-report and 25 on parent-report have been found to accurately distinguish between depressed and nondepressed youth (Kent, Vostanis, & Feehan, 1997). The MFQ demonstrated high internal consistency across self-report (α = .92) and parent-report (.93). The SCARED and MFQ were completed during the first study visit.

Pubertal Development

Pubertal maturation was assessed using the Self-Rating Scale of Pubertal Development (Carskadon & Acebo, 1993). Items on the scale assess for external signs of pubertal development, including changes in skin, body hair, voice (male), and menarche. This scale has demonstrated good reliability and reasonable concordance with hormonal indices of pubertal development (Shirtcliff, Dahl, & Pollak, 2009).

Sleep Diary

Each morning, adolescent participants completed an online sleep diary, collected using the secure Research Electronic Data Capture (Harris et al., 2009) web-based application hosted at the University of Colorado, Denver. The diary required participants to report on the previous night’s bedtime, sleep onset time, number of awakenings, total time awake during the night, overall quality of sleep on a 6-point scale from 1 (horrible) to 6 (excellent), and anxiety upon waking in the morning from 1 (calm, relaxed) to 6 (severe). This online format allowed diary entries to be time stamped, and only entries made before noon each day were included in the data set. To maximize our response rate we sent automated text messages to participants each morning, reminding them to go online and complete their sleep diaries. When study staff noticed that a sleep diary had not been completed, a call was placed to the family reminding them to complete subsequent diaries. Across our 43
participants, we had only 4 nights of missing data (i.e., 98.7% completion rate), with only one participant exceeding more than 1 night of missing data. We had previously decided to exclude from analysis any participant contributing fewer than 5 nights of data, however none of our participants were below this threshold.

Note that the first five participants (all from the GAD group) completed the study using a version of sleep diary that did not include the question assessing morning anxiety, thus those data are missing from analyses involving that variable.

**Actigraphy**

Nighttime sleep was measured using Actiwatch-2 actigraphs (Philips Respironics, Murrysville, PA) worn over 7 nights on the participant’s nondominant wrist. Actigraphs provide an objective estimate of sleep/wake cycles based on the measurement of movement and have the advantage of measuring sleep in an individual’s typical sleep environment. Although there is a scarcity of research validating the use of actigraphy with pediatric samples (Meltzer, Montgomery-Downs, Insana, & Walsh, 2012), existing studies of youth and adults have found that when compared to PSG, actigraphs have strong sensitivity to detect sleep but weaker specificity in detecting wake (Meltzer, Walsh, Traylor, & Westin, 2012; Sadeh, Sharkey, & Carskadon, 1994). In the current study, movement data were summed into 1-min epochs, which were analyzed using the “medium sensitivity” setting in the Actiware 6.0 software (Respironics Inc., 2011). This software uses a validated algorithm to classify epochs as either sleep or wake and calculates variables such as sleep duration, sleep onset latency, minutes of wake after sleep onset, and sleep efficiency. Per this algorithm, sleep onset was defined as the first period of 10 consecutive immobile min, and sleep offset as the last 10 consecutive immobile min, between bedtime and wake time. Each actigraph also features an event marker button. Participants were instructed to push this button each night as they were ready to try to fall asleep (i.e., when they turned the lights off) and again in the morning as they got out of bed (i.e., lights on). Each actigraph record was manually checked. In cases of missing event markers, bedtime or wake time would be imputed from the sleep diary.

Compliance with wearing the actigraphs was also very high, and we had no instances of device failure. We had only 2 nights of missing data across our entire sample (99.3% compliance), thus all participants were included in analyses.

**Children’s Report of Sleep Patterns**

Subjective impressions of sleep were also captured using the Children’s Report of Sleep Patterns (CRSP), an instrument that has demonstrated good reliability and validity among both child (Meltzer, Biggs, et al., 2012) and adolescent (Meltzer et al., 2014) samples. Both the self- and parent-report versions were administered. The self-report version contains 62 items and the parent-report version 67 items. The CRSP contains three modules (Sleep Patterns, Sleep Hygiene, and Sleep Disturbance), each with individual subscales. In this study, we focused on the Sleep Disturbance module, which contains the subscales of Bedtime Fears/Worries, Restless Legs, Parasomnias, and Insomnia. We also examined the Sleepiness subscale to assess possible daytime consequences of inadequate or nonrestorative sleep. For all items contributing to the Sleep Disturbance module, respondents were asked to rate the frequency of behaviors for a “regular week” when the adolescent was not sick or on vacation. For the majority of items, a 5-point Likert scale was used. Potential ranges for each subscale were as follows: Bedtime Fears/Worries (2–10), Restless Legs (5–25), Parasomnias (2–10), Insomnia (5–25), and Sleepiness (5–25). Internal consistency was adequate for the Sleepiness scale and all Sleep Disturbance subscales (ranging from $\alpha = .77$ to .89) with the exception of the parent-report Restless Legs subscale ($\alpha = .54$). We did not calculate alpha coefficients for the Bedtime Fears/Worries or Parasomnias subscales, as they each contained only two items.

**Data Analysis**

For the first study aim, we employed independent samples $t$-tests to address our primary hypotheses regarding group differences across sleep parameters. Mann–Whitney $U$ tests were used for all variables with non-normal distributions. We used Pearson correlations to test our hypotheses for the second study aim regarding relationships between sleep parameters and anxiety and depression severity scores. For the third study aim, we employed a repeated measures mixed-effects model, allowing for correlation between repeated measures of sleep duration and sleep-diary-reported morning anxiety made on the same individual, and with an interaction between group and sleep duration, to test whether the two groups differed in the relationship between sleep duration and morning anxiety.

We used independent samples $t$-tests to evaluate whether GAD participants taking versus not taking psychiatric medications differed on any of the sleep parameters measured by actigraphy and sleep diaries. All analyses were performed using SPSS for Windows (version 23.0) with the exception of the mixed-effects analyses, which were performed using SAS.

**RESULTS**

**Sleep Diary**

Consistent with hypotheses, sleep onset latencies were longer among participants with GAD than controls (see Table 3). They also reported lower sleep efficiency, although this finding did not quite reach statistical significance ($p = .06$), and both groups endorsed relatively high sleep efficiency (91.5% and 94.7%, respectively). Participants with GAD rated their
sleep as significantly lower quality than controls and endorsed higher ratings of anxiety upon waking.

**Children’s Report of Sleep Problems**

Participants with GAD endorsed widespread problems with sleep. Relative to healthy controls the GAD participants reported higher levels of Bedtime Fears/Worries, Restless Legs, and overall Insomnia symptoms, as well as greater daytime sleepiness (see Figure 1a). Group differences were less consistent via parent report with only the Insomnia and Bedtime Fears/Worries subscales showing greater severity of ratings among the GAD participants (see Figure 1b).

**Actigraphy**

Significant differences across multiple sleep variables were found using actigraphy (see Table 4). Consistent with our hypothesis, participants with GAD took significantly longer to fall asleep than healthy controls, by an average of nearly 15 min per night. We did not find a significant difference with respect to sleep efficiency or wake after sleep onset. Contrary to hypotheses, participants with GAD slept longer each night than controls. This finding appeared to stem primarily from participants with GAD going to bed slightly earlier and waking later than their healthy peers on weekdays and weekends, though group differences on sleep timing did not reach significance thresholds.

**Associations Between Actigraphic Sleep Parameters and Clinical Symptoms**

We found a significant negative association between sleep efficiency and parent MFQ total score and a positive association between sleep onset latency and parent MFQ total score and parent SCARED total score (see Table 5). There were no significant associations between these sleep variables and self-report of anxiety or depression.

**Prospective Links Between Nightly Sleep Duration and Morning Anxiety**

In the entire cohort, there was not a significant relationship between sleep-diary-reported morning anxiety and the previous night’s sleep duration (see Table 6). In the model including the interaction of sleep duration and GAD status, each 1-hr increase...
in sleep duration resulted in a decrease in morning anxiety of 0.13 ± 0.09 additional points for GAD participants relative to controls. Although the interaction of sleep duration and group was not significant (p = .13), effect size calculations for each group revealed a very small average effect for sleep duration on morning anxiety in controls (Cohen’s $d = 0.08$) but a small to moderate effect for the GAD participants (Cohen’s $d = 0.39$). Figure 2 presents the predicted values for each group in this interaction model.

Because these analyses were likely underpowered to detect an interaction, we elected to run a sensitivity analysis with participants stratified by group. Repeated measures mixed-effects models were used to test the association of sleep duration

![TABLE 4](image)

Actigraphic Sleep Comparison Between Groups

<table>
<thead>
<tr>
<th>Effect</th>
<th>GAD</th>
<th>Control</th>
<th>Test Statistic</th>
<th>$d$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>1.92</td>
<td>0.47</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TST</td>
<td>-0.01</td>
<td>0.06</td>
<td>0.19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GAD</td>
<td>1.88</td>
<td>0.73</td>
<td>0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TST × GAD</td>
<td>-0.13</td>
<td>0.09</td>
<td>0.13</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Beta estimates and standard errors for TST were converted from minutes to hours for this table for ease of interpretation. TST = total sleep time; GAD = generalized anxiety disorder.

![TABLE 5](image)

Correlations Between Actigraphic Sleep Parameters and Anxiety and Depression Scores

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Sleep Duration</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>2. Sleep Efficiency</td>
<td>.19</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>3. Sleep Onset Latency</td>
<td>-.05</td>
<td>-.69**</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>4. SCARED Total—Self-Report</td>
<td>.12</td>
<td>.06</td>
<td>.14</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>5. SCARED Total—Parent-Report</td>
<td>.23</td>
<td>-.26</td>
<td>.38*</td>
<td>.64**</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>6. MFQ Total—Self-Report</td>
<td>.13</td>
<td>-.10</td>
<td>.24</td>
<td>.74**</td>
<td>.64**</td>
<td>—</td>
</tr>
<tr>
<td>7. MFQ Total—Parent-Report</td>
<td>.05</td>
<td>-.37*</td>
<td>.42**</td>
<td>.59**</td>
<td>.85**</td>
<td>.67**</td>
</tr>
</tbody>
</table>

Note: SCARED = Screen for Child Anxiety Related Emotional Disorders; MFQ = Moods and Feelings Questionnaire.

* $p < .05$. ** $p < .01$. 

![TABLE 6](image)

Parameter Estimates From Repeated Measures Mixed-Effects Model

<table>
<thead>
<tr>
<th>Effect</th>
<th>Estimate</th>
<th>SE</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>1.92</td>
<td>0.47</td>
<td>—</td>
</tr>
<tr>
<td>TST</td>
<td>-0.01</td>
<td>0.06</td>
<td>.19</td>
</tr>
<tr>
<td>GAD</td>
<td>1.88</td>
<td>0.73</td>
<td>.01</td>
</tr>
<tr>
<td>TST × GAD</td>
<td>-0.13</td>
<td>0.09</td>
<td>.13</td>
</tr>
</tbody>
</table>

Note: Beta estimates and standard errors for TST were converted from minutes to hours for this table for ease of interpretation. TST = total sleep time; GAD = generalized anxiety disorder.

![FIGURE 2](image)

Mixed effects model showing the relationship between sleep duration and group status on waking anxiety by group. GAD = generalized anxiety disorder. This figure shows the predicted mean waking anxiety level vs. sleep duration in the two groups across the range of observed sleep duration values in each group, using the results of the mixed-effects model with interaction. The solid line is the predicted mean value of waking anxiety and the shaded regions are 95% confidence intervals for the predicted values.

and morning anxiety in each group. There was no significant association in the control group ($p = .68$), but the association in the GAD group was significant ($p = .04$). Each 1-hr increase in
sleep duration was associated with a .15 ($SE = .07$) unit decrease in morning anxiety (Figure 2).

Medication Effects

Analyses comparing sleep parameters among GAD participants taking versus not taking psychiatric medications failed to reach significance. These can be found in Supplemental Table 1.

DISCUSSION

This preliminary study sought to evaluate the presence of sleep disturbance and its relationship to clinical symptomatology among adolescents with a primary diagnosis of GAD and healthy controls. We found evidence of sleep disturbance among adolescents with GAD across both subjective and objective measures, as well as evidence for connections between sleep and current anxiety and depression severity. Our prospective data also suggested a significant relationship between nightly sleep duration and subsequent morning anxiety that was specific to the GAD participants.

Sleep Abnormalities Across Subjective and Objective Measures

Our finding of longer sleep onset latencies via actigraphy and sleep diary among our adolescents with GAD was consistent with two previous PSG studies (Alfano et al., 2013; Forbes et al., 2008) and one actigraphy study (Alfano et al., 2015) that focused on clinically anxious youth. We found no evidence to suggest that adolescents with GAD struggle to maintain sleep to a greater degree than controls, using actigraphy or sleep diaries. Sleep-onset insomnia, rather than problems maintaining sleep throughout the night, may be the most defining sleep disturbance in this population. This is somewhat different than what has been observed in adults with GAD for whom sleep maintenance problems predominate (Monti & Monti, 2000) yet is consistent with our questionnaire results in which participants with GAD reported significantly higher levels of bedtime fears than controls. Down-regulating negative emotions at bedtime is a critical aspect of initiating sleep (Baglioni, Spiegelhalder, Lombardo, & Riemann, 2010), thus sleep onset difficulties may reflect a broader deficit in emotion regulation in this disorder. Of interest, one recent study of adults with GAD reported that emotion regulation deficiencies fully mediated the relationship between GAD symptoms and sleep disturbance (Tsypes, Aldao, & Mennin, 2013).

Although we did find the aforementioned evidence of sleep disturbance among our adolescents with GAD using actigraphy, there was a disconnect between our objective and subjective sleep measures, with far greater sleep disturbance in the GAD group revealed in sleep diaries and in the CRSP questionnaire than via actigraphy. For example, unlike some previous studies actigraphy data indicated that our adolescents with GAD actually slept longer each night than their healthy peers by a considerable amount, yet these participants endorsed significantly lower sleep satisfaction ratings on sleep diaries and higher ratings of daytime sleepiness on the CRSP than controls. This discrepancy could be attributed to reporter bias, such that anxious individuals are more likely than nonanxious individuals to report problems in a variety of domains due to an influence of negative mood (Dalgleish & Watts, 1990). Another intriguing possibility is that adolescents with chronic anxiety actually have greater sleep need than healthy adolescents, thus they chronically wake up feeling unrestored. This hypothesis would be consistent with the observation that anxiety disorders (Hoehn-Saric & McLeod, 1988), and GAD in particular (Lyonfields, Borkovec, & Thayer, 1995; Thayer, Friedman, & Borkovec, 1996), involve an autonomic imbalance with hyperactivity of the sympathetic nervous system relative to the parasympathetic nervous system. Chronic heightened sympathetic activation is linked with excessive energy expenditure (Thayer & Lane, 2007), which may create greater need for restoration through sleep. In addition, there is reason to expect that the sleep obtained by anxious individuals is less restorative than for healthy individuals given some evidence of reduced slow wave activity in youth (Forbes et al., 2008) and adults with GAD (see review in Papadimitriou & Linkowski, 2005), and findings showing that apprehensive thoughts at bedtime are associated with reduced slow wave sleep throughout the night (Kecklund & Åkerstedt, 2004).

Cross-Sectional and Prospective Links Between Sleep and Anxiety

Our correlational analyses supported the hypothesis that actigraphic measures of disrupted sleep (extended sleep onset latency and poor sleep efficiency) would be cross-sectionally associated with both depression and anxiety; however, this was true only when using parent-report of clinical symptoms. These associations did not reach significance via self-report of symptoms, though the relationships were in the predicted direction, suggesting that this may be due to limited statistical power. Although these analyses do not indicate causality, they reinforce the notion that as severity of internalizing symptoms increases, so too do certain objective measures of sleep disturbance. Forbes et al. (2008) reported similar relationships between PSG sleep parameters and depression and anxiety symptom levels. Our prospective analyses allowed us to explore the temporal relationship between nighttime sleep duration and subsequent morning anxiety. Although we did not find significant relationships across the entire sample or in the interaction analysis, our sensitivity analysis did support our hypothesis of a relationship between decreased sleep duration and increased morning anxiety among our participants with GAD. This effect was
statistically significant, but its clinical significance is unclear. We employed a nonstandardized 6-point anxiety rating added to our sleep diary, with the hope of capturing some aspect of morning anxiety but without increasing the burden on participants. The downside of this approach is that scores are difficult to evaluate relative to more thorough, established anxiety measures. Our observed increase in anxiety of .15 points for every hour of sleep below the mean may indicate a rather marginal association. Alternatively, given our small sample size and measurement of anxiety at only a single time point, it may be that more standardized measurement of anxiety several times throughout the day would illuminate a clinically meaningful association between sleep duration and daytime anxiety. These results parallel a recent neuroimaging study of adults, which reported that sleep loss produces intensified activity in neural circuitry responsible for the anticipation of threat (i.e., the amygdala and insula; Goldstein et al., 2013) and that the degree of such vulnerability to sleep loss is related to trait anxiety. Previous studies have also found that sleep deprivation selectivity increases amygdala activation to threatening images (Yoo et al., 2007). It may be that anxious individuals are more sensitive to perturbations of sleep, experiencing increasing anxiety as a downstream consequence of the effects of sleep loss on brain regions responsible for threat processing.

The findings of this study indicate the potential utility of intervening directly with sleep among adolescents with GAD who present with sleep disturbance. Promoting good sleep hygiene practices such as maintaining a consistent bedtime, eliminating sleep disrupting electronics (e.g., smartphones, tablets) from the bedroom, and implementing a bedtime relaxation procedure may help these patients establish good sleep habits and reduce the arousal that appears to characterize sleep-onset insomnia (Alfano et al., 2010). Additional cognitive factors such as overestimating the effects of insufficient sleep and excessively attending for signs of sleepiness appear to underlie adult insomnia (Harvey, 2002) and may be treatment targets for adolescents with GAD and chronic sleep disturbance. Treatment protocols aimed at reducing these mechanisms of sleep disturbance among anxious youth are currently being tested (Cowie et al., 2014). Of interest, two recent studies suggest that standard cognitive behavioral therapy and pharmacotherapy for pediatric anxiety may themselves produce improvements in sleep-related problems even without directly targeting sleep symptoms (Caporino et al., 2015; Peterman et al., 2016). Given the interrelatedness of sleep disturbance and anxiety, it is not surprising that interventions that reduce anxiety might confer some benefits in the sleep domain. However, the actual benefits were small in one study (Caporino et al., 2015), and participants continued to report elevated sleep-related problems at posttreatment in the other study (Peterman et al., 2016). Although standard cognitive behavioral therapy for anxiety may prove sufficient for reducing mild sleep disturbance, it may be that patients with more significant or long-standing sleep symptoms require additional sleep-focused intervention.

**Limitations and Future Directions**

This study has several limitations. Because this is a preliminary study that was intended to gather pilot data, the sample size is relatively small, which left many of our analyses underpowered. For variables on which group differences were quite large, such as “sleep quality” assessed using the sleep diary, the achieved power was excellent (.96). However, for other variables with smaller but potentially significant between-group differences, such as “weekend morning wake time,” the achieved power was inadequate (.46). This may have resulted in cases of Type II error. Our sample was also heterogeneous, particularly in terms of age and comorbid conditions. With a small sample it becomes difficult to single out sleep abnormalities that are unique to GAD versus those imparted by comorbid conditions. That said, our findings were for the most part consistent with existing findings in youth with GAD (Alfano et al., 2006; Alfano et al., 2007; Alfano et al., 2015; Alfano et al., 2010; Alfano et al., 2013; Forbes et al., 2008; Kendall & Pimentel, 2003; Mast et al., 2004). In a follow-up study our group has under way, we are pursuing a larger sample and assessing a wider range of variables that may be relevant to understand sleep disturbance in this population, such as dysfunctional beliefs about sleep.

Another possible limitation of the current study is that the majority of our participants with GAD and one of our controls were taking psychiatric medications. When designing this study, we made a deliberate attempt to gather a representative sample of adolescents with GAD, and thus we anticipated that a significant portion would be treated with medication. Of the 20 GAD participants taking medication, 13 were taking SSRIs as monotherapy. Research suggests that the sleep-altering effects of SSRI medications are most consistent in reducing REM sleep and that the effects on sleep initiation and maintenance are inconsistent between agents and typically present only at the early initiation of such treatment (Wilson & Argyropoulos, 2005). Of the other medications taken by our participants, the potential effects on sleep are varied: Atypical antipsychotics often possess sedating properties that may reduce insomnia symptoms but also increase daytime sleepiness (Kane & Sharif, 2008), stimulants are known to cause difficulty with sleep initiation (Greenhill, Pliszka, & Dulcan, 2002), and tricyclic antidepressants do not consistently possess sleep-related side effects (Birmaher et al., 1998; Geller et al., 1993). In this study we did not collect a time line of medication usage and thus cannot rule out the possibility of very recent medication changes that might have been more likely to impact sleep. A previous study of anxious children and adolescents found no differences in the rate of sleep problems among those who were taking versus not taking...
psychiatric medications (Chase & Pincus, 2011). We did not find any differences among primary sleep parameters for GAD participants who were taking versus not taking medications. However, these analyses were underpowered, and it is certainly possible that medication usage impacted our results. We prioritized the overall representativeness of our sample relative to precluding possible medication effects in this study, but this issue warrants further investigation.

One other potential limitation involves our use of actigraphy. Although actigraphy possesses many important advantages, including allowing researchers to assess sleep unobtrusively while participants sleep in their own environment, its use is not well-established in pediatric populations. It appears to possess high sensitivity with regard to sleep but may overestimate wakefulness after sleep onset (Meltzer, Montgomery-Downs, et al., 2012; Meltzer, Walsh, et al., 2012; Short, Gradisar, Lack, Wright, & Carskadon, 2012). Our data indicated relatively high levels of wakefulness after sleep onset for both groups that was not reflected in sleep diaries, and thus may have been an overestimate. Even with the use of event markers, actigraphy relies heavily on manual entry of the time that participants attempted to fall asleep (i.e., bedtime). Thus, actigraphic measurement of sleep onset latency is of questionable validity. Further validation of actigraphy as a measure of sleep in pediatric and clinical populations is needed.

This study extends previous knowledge on the connections between sleep and anxiety among pediatric populations and suggests several future directions for research in this area. This is now the second study (see also Cousins et al., 2011) to report a prospective temporal relationship between nightly sleep parameters and subsequent mood among anxious youth. However, to make conclusions regarding causality, future studies might choose to employ sleep restriction/extension procedures and identify associated changes in anxiety symptoms and underlying processes (e.g., attentional biases toward threat), as well as examine changes in positive affect. In addition, it will be important for future studies to address whether anxious children and adolescents actually have a greater sleep need than their healthy peers. It may be possible to do this by examining relationships between objective sleep parameters and subjective and objective measures of sleepiness (e.g., multiple sleep latency test; Carskadon et al., 1986) and measures of neurobehavioral impairment (e.g., psychomotor vigilance task; Van Dongen, Maislin, Mullington, & Dinges, 2003). Sleep duration could be carefully manipulated, with examination of changes in these domains among anxious and healthy youth. Also, we would welcome future studies employing behavioral treatment of sleep disturbance among anxious youth, with subjective and objective measurement of sleep outcomes. From such trials, it may be possible to discern which changes across subjective or objective sleep measures are most closely connected with improved overall functioning.

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