Published in final edited form as:

Child Health Care. 2019 ; 48(4): 372–393. doi:10.1080/02739615.2019.1630283.

Sleep Patterns and Anxiety in Children Interact to Predict Later Suicidal Ideation

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Abstract

Elevated levels of childhood anxiety pose risk for suicide; however, factors that accentuate this risk are unknown. Seventy-one children participated in a longitudinal study investigating anxiety and sleep in childhood (between 7–11 years) and later suicidal ideation (SI; $M = 3.3$ years later). Sleep was assessed via subjective reports and objective measures (actigraphy and polysomnography). Children with greater anxiety symptoms were at greater risk for later SI when sleep disturbances were present in childhood. Results suggest that sleep disruption may amplify SI risk in anxious children.

Introduction

Suicide is the second leading cause of death among young people across the world (World Health Organization, 2018) and is a serious public health concern. Suicidal ideation (SI) and suicide attempts are also frequent during adolescence, with nearly 30% of adolescents reporting a lifetime history of SI and nearly 10% reporting a lifetime history of suicide attempts (Evans, Hawton, Rodham, & Deeks, 2005). Importantly, many children and teens do not seek or receive help for SI or suicidal behaviors (Gould et al., 2009; Pisani et al., 2012; Schmeelk-Cone, Pisani, Petrova, & Wynam, 2012). Identifying malleable risk factors that can be identified before the onset of suicidal behaviors or ideation by health officials and professionals is critical for early prevention. Anxiety is one risk factor that has been cross-sectionally associated with suicidal behaviors in childhood and adolescence (Hill, Castellanos, & Pettit, 2011). However, studies examining longitudinal associations between anxiety and SI are limited, as are studies examining synergistic risk factors that may buffer or heighten risk for SI in anxious children. The current study examines how sleep disturbances, a common symptom in anxious children that is independently associated with risk for suicide, moderates associations between pre-pubertal anxiety symptoms and later experiences of SI.

Anxiety and Suicide

Results from several investigations suggest that anxious individuals are at heightened risk for SI. In a population-based sample of adult primary care patients with anxiety (i.e., met DSM-IV criteria for at least one anxiety disorder and expressed moderate anxiety symptoms), 26% expressed passive SI, 16% held thoughts of suicide within the month of assessment, and 18% expressed a previous history of non-lethal suicide attempt (Bomyea et al. 2013). In adults, Sareen and colleagues (2005) found that of their participants who endorsed suicidal attempt and suicidal ideation, 55% and 37% of those participants respectively had at least 1 anxiety disorder (i.e., met DSM-III-R criteria). In adolescence, research has demonstrated that subthreshold-anxious and clinically-anxious adolescents are 1.79 and 2.76 times more likely to endorse SI respectively than their non-anxious peers (Balazs et al., 2013). In addition to these compelling cross-sectional associations, a recent meta-analytic study concluded that anxiety was a significant longitudinal predictor of SI and attempts but not suicide completion (Bentley, Franklin, Ribeiro, Kleiman, Fox, & Nock, 2016). In an in-patient psychiatric sample of adolescents, Goldston and colleagues (1999) identified trait anxiety symptoms as associated with suicide attempts 5 years later. Another longitudinal study in adolescents observed that teens with current SI were significantly more likely to have previously endorsed anxiety symptoms in childhood than their non-suicidal peers (Reinherz, 1995). Further, in adolescents aged 7–14 years treated with cognitive-behavioral therapy for a primary anxiety disorder who were followed-up 7–19 years later, 27.3% endorsed either current or lifetime SI, with treatment non-responders being significantly more likely to have endorsed SI at follow-up (Wolk et al., 2015). Importantly, studies have shown that this relationship between anxiety and later suicide risk exists over and above other comorbidities (e.g., depression; Boden et al., 2007).

Conversely, other studies have not found the presence of an anxiety disorder prior to puberty to pose increased suicide risk (Weissman et al., 1999; Wolk & Weissman, 1996). In addition, despite evidence for longitudinal relations between anxiety and suicide attempts and ideation, effect sizes are generally small (Bentley et al., 2016). Other limitations of this body of research include the fact that few studies have examined anxiety symptoms prior to the onset of puberty, and many studies have examined anxiety diagnoses or symptoms in isolation (i.e., without accounting for comorbid symptoms). As anxiety is highly comorbid with a range of other conditions and symptoms, it is unknown whether and how anxiety might interact with other established risk factors in the development of later SI. As a result, our current understanding of which anxious children are most at-risk for later SI is inadequate. One established risk factor that likely impacts later SI and disproportionately impacts anxious children is sleep disturbances. Investigation into how particular sleep patterns early in life may accentuate or buffer suicide risk in anxious children and adolescents is needed.

Anxiety and Sleep Disturbances in Childhood and Adolescence

Sleep patterns are an important risk factor to consider when examining psychiatric outcomes in anxious children for several reasons. First, a majority of children with clinical levels of anxiety experience subjective sleep complaints (McMakin & Alfano, 2015), including difficulties at bedtime (e.g., problems initiating sleep, bedtime resistance), maintaining sleep

throughout the night, and poor sleep quality (e.g., feeling rested upon waking in the morning). Sleep onset is also mildly prolonged based on assessments using actigraphy (Alfano, 2018). Second, the presence of sleep disturbances is associated with more severe forms of anxiety and greater impairments in functioning (Alfano, Ginsburg & Kingery, 2007; Chase & Pincus, 2011). Additionally, certain aspects of sleep architecture have been shown to correlate with symptom profiles among anxious children. Palmer and Alfano (2017a) found a greater percentage of rapid eye movement (REM) sleep to correspond with greater depressive symptoms whereas lower percentages of non-REM (NREM) stage 3 sleep (i.e., slow wave sleep) was associated with greater negative affect for anxious children but not healthy controls. These collective findings suggest both subjective and objective sleep characteristics to hold meaning for the emotional experiences of anxious children. Lastly, better understanding of potentially synergistic relationships between anxiety and sleep across the transition from childhood to adolescence, a period when both sleep and emotional systems are changing, has been highlighted as a critical area for research inquiry (Alfano, 2018; McMakin & Alfano, 2015).

Sleep and Suicide

Several longitudinal studies and meta-analyses provide evidence for subjective sleep disturbances (e.g., insomnia, nightmares) as an independent risk-factor for suicidality in adults despite different methodologies, sample demographics, and when controlling for the effects of depression and other comorbid symptoms (Bernert et al., 2015; Pigeon et al., 2012; Nadorff, Anestis, Nazam, Harris, & Winer, 2014; Fujino et al., 2005; Tanskanen et al., 2001; Turvey et al., 2002). In children and adolescents, previous studies suggest that those who endorse SI or suicidal attempt were more likely to have sleep problems compared to those who did not endorse SI or attempt (Franic, Kralj, Marcinko, Knez, & Kardum, 2014; Gex, Narring, Ferron, & Michaud, 1998; Liu, 2004; Vignau et al., 1997). However, published literature examining longitudinal relationships between sleep disturbances and suicidality in children and adolescents are unfortunately limited (Liu & Buysse, 2006). One study using a large ($N = 6,504$) sample of adolescents found that sleep difficulties (i.e., difficulty falling asleep, nocturnal wakefulness) predicted both suicidal attempts and ideation concurrently one and five years later (Wong & Brower, 2012). One other longitudinal study using an adolescent sample has similarly found significant relationships between sleep disturbances and both SI and non-lethal suicide attempt several years later, even when controlling for age, gender, depressive symptoms, aggressive behavior, and substance abuse (Wong et al., 2011).

Importantly, the aforementioned longitudinal studies relied on subjective reports of sleep without additionally incorporating objective measures (i.e., actigraphy, polysomnography). Research using polysomnography among adults has shown marked differences in the sleep patterns of those endorsing suicidality and those who do not, with the most reliable differences emerging for patterns of REM sleep. For example, one study found greater REM sleep and phasic REM activity (along with a longer sleep onset latency) among depressed adults endorsing suicidality as compared to depressed adults without a history of suicide (Sabo, Reynolds III, Kupfer, & Berman, 1990). Similar findings have been reported among adults with schizophrenia (Lewis et al., 1996) and untreated depression (Agargun &

Cartwright, 2003). Additional research using polysomnography found current SI in adult patients with either major depressive disorder or bipolar disorder was significantly associated with less NREM sleep and higher nocturnal wakefulness when controlling for depressive symptoms (Bernert et al., 2017).

Within adolescent populations, research using objective sleep data is more limited and somewhat mixed. Increased slow wave sleep and the interaction between slow wave and altered REM sleep during adolescence has been found to predict suicidality 10–15 years later (Goetz, Wolk, Coplan, Ryan, & Weissman, 2001). Comparatively, Dahl and colleagues (1990) examined polysomnography sleep measures in adolescents with major depressive disorder with and without SI. The adolescents with SI had significantly longer sleep onset latencies than their non-suicidal peers, but differences in REM latency, REM density, or REM time were not found (Dahl et al., 1990). Other research comparing polysomnography sleep measures in adolescents with major depressive disorder did not find differences between those who endorsed SI and those who did not (Rao et al., 1996).

The Current Study

In sum, a growing number of studies provide evidence that both anxiety and aspects of sleep are prospectively associated with risk for SI. However, interactions between childhood anxiety and sleep in predicting suicidal ideation have yet to be examined, and studies examining both subjective and objective sleep patterns and longitudinal predictors of SI in childhood are limited. Investigation of early sleep patterns that might modify SI risk for anxious children and adolescents could help identify those most vulnerable. This longitudinal study aimed to address these gaps using both subjective reports and objective measures of sleep in a sample of pre-pubertal children with a range of anxiety symptoms. These participants underwent a second assessment approximately 3 to 4 years later. It was hypothesized that greater anxiety symptoms at Time 1 would predict elevated risk for SI at Time 2, and that the presence of subjective sleep disturbances in childhood would amplify SI risk. Also, based on findings in adults showing greater nighttime wakings (Wong & Brower, 2012), increased sleep onset latency (Dahl et al., 1990; Wong & Brower, 2012), and increased REM sleep (Agargun & Cartwright, 2003; Lewis et al., 1996) to forecast SI risk, we hypothesized the same relationships in our sample of children.

Methods

Participants included 71 children who completed a comprehensive sleep and psychiatric assessment between the ages of 7–11 years, and a follow-up assessment approximately 3–4 years later ($M = 3.34$, $SD = 1.63$, range 1–7). All children were pre-pubertal at Time 1 as determined by the Pubertal Development Scale (PDS; Carskadon & Acebo, 1993). Participant demographic characteristics are displayed in Table 1.

Inclusion/Exclusion Criteria

Participants were recruited at Time 1 to capture a range of both clinical and subclinical anxiety symptoms. To be eligible to participate at Time 1, children were required to live with a primary caretaker, be fluent in English (and have a caretaker who was fluent in English),

and could not be taking medications or over the counter supplements known to impact anxiety or sleep (e.g., melatonin, selective serotonin reuptake inhibitors). Participants also could not have any chronic medical conditions that might impact their sleep (e.g., atopic dermatitis) or any diagnosed or suspected organic sleep disorders (e.g., sleep apnea, restless leg syndrome). Because the purpose of the study was to investigate relationships between anxiety and sleep specifically, children were also excluded if they had any lifetime or current history of depressive disorders. In addition, children with psychotic, pervasive developmental, or bipolar disorders, and/or any lifetime presence of suicidal behaviors or ideation were ineligible at Time 1. Parents reported on all of these exclusionary criteria during a phone screening assessment prior to their participation. Participants also underwent polysomnography to further assess for the presence of any sleep disorders, and completed testing to ensure that they did not have an IQ below 80 (which was confirmed using the Wechsler Abbreviated Scale of Intelligence; Wechsler, 1999). Diagnostic status and psychiatric history was further assessed using the Anxiety Disorders Interview Schedule for the DSM-IV for Children and Parents (Silverman & Albano, 1996).

Procedure

All study procedures at Time 1 were approved by the Institutional Review Board at the University of Houston. Study procedures for the assessment at Time 2 were approved as a separate follow-up study by the Institutional Review Board. At Time 2, participants were recruited from a larger sample of 96 children who completed a Time 1 assessment as part of one of two studies with nearly identical assessment procedures. One study targeted children with anxiety disorders or healthy children ($n = 25$) and the other study targeted children with subclinical levels of anxiety ($n = 46$). All families at Time 1 were recruited using flyers, postcard mailings using public school databases, and through public/community events.

At Time 1, all children and one parent were consented/assented and then completed an initial assessment that included parent-reported questionnaires about the child's sleep and psychiatric symptoms, a diagnostic interview to determine any clinical diagnoses, and abbreviated IQ testing. All children then completed 6–7 days of actigraphy sleep monitoring and one night of at-home polysomnography sleep monitoring. At Time 2, families from Time 1 were contacted and asked if they would like to participate in a separate follow-up study to assess their child's current level of functioning. Interested participants were re-consented/assented and completed one in-person assessment. During this assessment, participants completed both parent and child-reported questionnaires, along with the same diagnostic interview as Time 1. A Ph.D.-level psychologist or a trained doctoral-level graduate student conducted all assessments at both time points.

Assessments

Suicidal ideation (SI).—No children reported SI or self-harm behaviors at Time 1. This was verified during the diagnostic interview, and also by parent-report on the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). At Time 2, participants were dichotomized based on whether they reported any occurrence of SI. This presence of current or previous SI was assessed via either self-report from participants on the Children's Depression Inventory (CDI; Kovacs, 1992) which were then followed-up by a clinician, or

were reported during the diagnostic interview. During the semi-structured diagnostic interview, participants were provided with an initial prompt to assess their experience of depressive symptoms. If participants indicated the presence of depression, they were given a series of follow-up yes or no questions regarding specific symptoms they had experienced, including the experience of suicidal thoughts over the last two weeks. All participants also completed the full 27-item CDI as part of the general study procedures, including one item assessing suicidal ideation. This item on the CDI provides participants with an option to choose one of the following statements describing their feelings and thoughts over the last two weeks: a) *I do not think about killing myself*, b) *I think about killing myself, but I would not do it*, and c) *I want to kill myself*. Children were also provided the option to decline to answer this question. For all participants selecting options b, c, or who declined to answer, further SI assessment was conducted through a clinician-administered risk assessment. Any reports of SI (by the parent or participant) during follow-up questions or the diagnostic interview were immediately reviewed with a licensed clinical psychologist to determine risk and next steps. Participants with confirmed suicidal ideation based on the diagnostic interview, the CDI item, and/or these follow-up questions were coded as 1 (*endorsed SI*) and participants without suicidal ideation were coded as 0 (*did not endorse SI*).

Sleep patterns.

Subjective reports.: Parents reported on their child's sleep patterns at Time 1 using the Children's Sleep Habits Questionnaire (CSHQ; Owens, Spirito, & McGuinn, 2000). The CSHQ includes 33 items that assess a range of various sleep problems. Parents were asked to report how often certain sleep difficulties occurred in a recent typical week of their child's sleep from 1 (*rarely; 0–1 times a week*), 2 (*sometimes; 2–4 times a week*), or 3 (*usually; 5–7 times a week*). The CSHQ includes subscales that assess the following sleep problems: *bedtime resistance* ($\alpha = .61$, 6 items; e.g., "Child needs parent in the room to fall asleep"), *sleep onset delay* (assessed with one reversed scored item, "Child falls asleep within 20 minutes after going to bed"), *sleep anxiety* ($\alpha = .74$, 4 items; e.g., "Child has trouble sleeping away from home"), *sleep duration* ($\alpha = .74$, 3 items; e.g., "Child sleeps too little"), *daytime sleepiness* ($\alpha = .82$, 8 items; e.g., "Child takes a long time to become alert in the morning"), *sleep disordered breathing* ($\alpha = .51$, 3 items; e.g., "Child snores loudly"), *night awakenings* ($\alpha = .31$, 3 items; e.g., "Child awakes more than once during the night"), and *parasomnias* ($\alpha = .13$, 7 items; e.g., "Child awakens alarmed by a frightening dream"). Reliability analyses indicated that removing one item from the night awakening subscale ("Child moves to someone else's bed during the night") resulted in an increase in Cronbach's alpha from .31 to .61. As a result, the final scores from this subscale included the remaining 2 items only. Due to the exclusionary criteria of the current sample (i.e., no organically based sleep disorders), along with the low internal consistencies, we did not include the parasomnias or sleep disordered breathing subscales. We also calculated a *total sleep disturbance* score ($\alpha = .79$).

Actigraphy.: Actigraphy-based sleep patterns were assessed in childhood using a wrist-based accelerometer (Micro Motionlogger Actigraph Sleep Watches; Ambulatory Monitoring Inc., Ardsley, NY). Participants wore the watch for 6–7 days, and pushed an event button each evening when they got into bed each night, and again in the morning when

they got out of bed. Participants were also asked to complete a sleep diary each morning and evening, which is a necessary validation check for actigraphy to confirm sleep-wake times and identify any artifacts or irregularities in the data (Meltzer et al., 2012). Data was visually inspected to omit any epochs where the watch was not worn. Consistent with other studies using actigraphy in pediatric populations, data was scored using the zero-crossing mode and the Sadeh algorithm in one minute epochs (Meltzer et al., 2012; Sadeh et al., 1994). The variables derived from the actigraphy estimates include total sleep time (TST, or average minutes of sleep duration), sleep onset latency (SOL, or the number of minutes taken to fall asleep), and wake after sleep onset (WASO, or the number of minutes spent awake at night between sleep onset and sleep offset time). Based on recommendations for using actigraphy in pediatric research, only participants with a minimum of 5 nights of valid data were included in the present analyses (Acebo et al., 1999).

Polysomnography. Children completed one night of unattended, at-home polysomnography monitoring, which included 6 channels of EEG (frontal, central, and occipital regions), along with EOG, facial EMG, electrocardiogram, respiratory inductance plethysmography, pulse oximetry, and tibial EMG. Some participants ($n = 25$) were prepared for the polysomnography recording at a pediatric sleep center using NicoletOne ambulatory equipment (Natus, Inc.) and some participants ($n = 46$) were prepared for the recording at their home using RemLogic Embletta equipment (Natus, Inc.). All participants slept at home (based on prescribed bed and wake times) and simultaneously wore actigraph watches. Some participants were missing polysomnography data due to data recording errors, or when actigraphy data indicated that polysomnography did not capture the entire sleep period (i.e., either fell asleep prior to the start of the recording, or slept in after the recording was set to end). These participants ($n = 21$) were not included in analyses using polysomnography variables. All recordings were reviewed and scored by registered sleep technicians with pediatric experience and reviewed by a certified sleep medicine specialist. Recordings were scored in 30-second epochs using the criteria set forth by the American Academy of Sleep Medicine scoring rules (Iber, Ancoli-Israel, Chesson, & Quan, 2007). For the current study, the polysomnography derived variables included the percentage of total sleep time that was spent in N3 sleep (N3%) and percentage of total sleep time that was spent in REM sleep (REM%).

Psychopathology symptoms.

Clinical interview. At both Time 1 and Time 2, participants completed the Anxiety Disorders Interview Schedule for DSM-IV for Children and Parents (ADIS-C/P; Silverman & Albano, 1996), which has excellent psychometric properties (Lyneham, Abbott, & Rapee, 2007; Silverman, Saavedra, & Pina, 2001). Separate interviews were conducted with a caregiver and the child by a Ph.D.-level psychologist or a trained doctoral level graduate student according to standardized procedures. All cases were reviewed with a licensed clinical psychologist prior to assigning any diagnoses. A summary of the diagnoses at Time 1 and Time 2 are provide in Table 1.

Anxiety symptoms. Child anxiety symptoms were assessed at Time 1 via parent-report on the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). Specifically, parents

reported on how much a series of statements described their child now or in the last 6 months from 0 (*not true of their child*) to 2 (*very true or often true of their child*). For the current study, we used the DSM-oriented anxiety subscale. However, this subscale includes one item about sleep which was removed for the purposes of the current study. The remaining 8 items were averaged to create a total anxiety symptoms subscale ($\alpha = .81$).

Depressive symptoms. Child depressive symptoms at Time 1 were assessed using parent-reported scores on the DSM-oriented depressive symptoms subscale of the CBCL (Achenbach & Rescorla, 2001). Similar to the anxiety subscale, parents responded to statements based on how well they described their child now or in the last 6 months from 0 (*not true of their child*) to 2 (*very true or often true of their child*). For the current study, we removed four items from this subscale that pertained to sleep, and 2 items that pertained to self-harm and SI. The remaining 7 items were averaged to create a total depressive symptoms subscale ($\alpha = .41$).

Analysis Plan

First, a series of chi-squares and t-tests were conducted to determine any potential significant differences between participants reporting and not reporting SI at Time 2. We also examined bivariate associations among other variables of interest and demographic characteristics. Finally, based on the dichotomous nature of our SI outcome, a series of logistic regression models predicting the presence of SI were conducted. All analyses included sleep patterns at Time 1 (either subjective, actigraphy-measured TST, SOL, or WASO, or polysomnography-measured N3% or REM%), anxiety symptoms (based on the CBCL) at Time 1, and the interaction between sleep and anxiety symptoms. These analyses were conducted using the PROCESS 3.1 macro plugin for SPSS (Hayes, 2013). Due to the small number of cases with SI, and the lack of prospective data on sleep and suicide in anxious youth, we set the PROCESS criteria to probe the simple slopes for all interactions with a p value of less than .20 to examine exploratory relationships and minimize the possibility of Type II error. All analyses controlled for the time lapse between the two assessments, along with depressive symptoms at Time 1.

Results

Preliminary Analyses

A total of 16 participants (22.54%) reported that they experienced SI between Time 1 and Time 2. A summary of the differences between participants who reported SI compared to those who did not endorse SI on all demographic characteristics and variables of interest is reported in Table 1 and Table 2. Initial comparisons indicated that participants who did and did not report SI at Time 2 did not differ significantly on any Time 1 sleep variables or Time 1 anxiety or depressive symptoms.

As expected, bivariate analyses indicated that anxiety and depressive symptoms were significantly associated with one another ($r = .52, p < .001$). Total subjective sleep disturbances were associated with anxiety ($r = .49, p < .001$) and depressive symptoms ($r = .42, p < .001$). Anxiety symptoms were associated with several subscales on the CSHQ as

well, including greater bedtime resistance ($r = .39, p = .001$), sleep onset delay ($r = .45, p < .001$), sleep anxiety ($r = .50, p < .001$), and greater daytime sleepiness ($r = .34, p = .049$). Depressive symptoms were associated with a longer subjective sleep onset delay ($r = .29, p = .02$), greater sleep anxiety ($r = .37, p = .002$), daytime sleepiness ($r = .33, p = .01$), and total sleep disturbances ($r = .42, p < .001$). There were no significant associations among symptoms and the polysomnography and actigraphy sleep variables.

Bivariate associations with demographic characteristics indicated that age at Time 1 was associated with less total sleep time measured via actigraphy ($r = -.27, p = .02$), and age at Time 2 was associated with shorter subjective sleep duration ($r = .30, p = .01$). There were no differences in any variables of interest based on gender, with the exception of SI at Time 2. Girls were significantly more likely to report SI compared to boys, $X^2(1) = 5.78, p = .02$ (13 of the 16 SI cases were girls, adjusted residual = 2.4).

Prospective Associations between Sleep, Anxiety, and SI

Logistic regression models were examined with Time 1 anxiety symptoms, Time 1 sleep variables (CSHQ, actigraphy, or polysomnography), and the interaction between Time 1 anxiety and sleep. Models also controlled for the variable time lapse (3 to 4 years) between the two assessments, as well as Time 1 depressive symptoms.

Subjective sleep.—For parent-reported total subjective sleep disturbance, the overall model was significant $X^2(5) = 15.81, p = .007$. The model as a whole explained between 20.21% (Cox and Snell R squared) and 30.68% (Nagelkerke R squared) of the variance in SI. A significant interaction emerged between Time 1 sleep and anxiety symptoms (Beta = .66, SE = .25, $p = .008$, 95% CI: .17, 1.16), where anxiety predicted a greater likelihood of reporting SI at Time 2 for those with high levels of subjective sleep disturbances (Effect_{high} = 6.28, SE_{high} = 2.62, $p_{high} = .016$, 95% CI_{high}: 1.14, 11.42; Effect_{low} = -2.98, SE_{low} = 2.25, $p_{low} = .19$, 95% CI_{low}: -7.39, 1.44). This interaction is displayed in Figure 1. Follow-up analyses of the simple slopes using the Johnson-Neyman technique indicated that anxiety symptoms were a significant predictor of later SI when sleep disturbances on the CSHQ were at values of 46.47 or above, which is just above the established clinical cut-off score of 41 (Owens, Spirito, & McGuinn, 2000).

Follow-up analyses examined the individual subscales of the CSHQ in predicting SI. For parent-reported sleep onset delay, the overall model was significant: $X^2(5) = 14.34, p = .01$. The model as a whole explained between 19.06% (Cox and Snell R squared) and 28.12% (Nagelkerke R squared) of the variance in SI. A significant interaction emerged between parent-reported sleep onset delay and anxiety symptoms (Beta = 9.03, SE = 4.03, $p = .025$, 95% CI: 1.12, 16.93). Anxiety symptoms predicted a greater likelihood of reporting SI at Time 2 for those with longer sleep onset delay (Effect_{long} = 8.05, SE_{long} = 3.60, $p_{long} = .025$, 95% CI_{long}: .99, 15.11; Effect_{short} = -.98, SE_{short} = 1.82, $p_{short} = .59$, 95% CI_{short}: -4.54, 2.59). This interaction is displayed in Figure 2.

No findings emerged for the bedtime resistance or sleep anxiety subscales of the CSHQ, however several marginal interactions emerged between anxiety and parent-reported sleep duration, night wakings, and daytime sleepiness. For parent-reported sleep duration, the

interaction with anxiety symptoms was trending ($Beta = 2.34$, $SE = 1.51$, $p = .12$, 95% CI: $-.61, 5.30$), although the overall model was not significant. The model explained between 11.72% (Cox and Snell R squared) and 17.97% (Nagelkerke R squared) of the variance in SI. Analyses of the simple slopes indicated that for shorter sleep duration but not for longer sleep duration, anxiety was a marginal predictor of later SI ($Estimate_{short} = 4.40$, $SE_{short} = 2.29$, $p_{short} = .055$, 95% CI $_{short}$: $-.09, 8.90$; $Estimate_{long} = .55$, $SE_{long} = 1.47$, $p_{long} = .70$, 95% CI $_{long}$: $-2.33, 3.44$). For parent-reported night wakings, the overall model was significant: $X^2(5) = 13.64$, $p = .018$. The model explained between 17.71% (Cox and Snell R squared) and 26.88% (Nagelkerke R squared) of the variance. A marginal interaction between anxiety symptoms and reports of night wakings emerged ($Beta = 8.03$, $SE = 4.74$, $p = .09$, 95% CI: $-1.26, 17.33$), such that anxiety was marginally associated with a greater likelihood of endorsing SI for those who experienced greater night wakings, but not for those with reports of low levels of night wakings ($Estimate_{high} = 8.55$, $SE_{high} = 4.73$, $p_{high} = .07$, 95% CI $_{high}$: $-.72, 17.81$; $Estimate_{low} = .51$, $SE_{low} = 1.44$, $p_{low} = .72$, 95% CI $_{low}$: $-2.30, 3.32$). For parent-reported daytime sleepiness, the overall model was marginal, $X^2(5) = 9.62$, $p = .087$, and explained between 12.84% (Cox and Snell R squared) and 19.49% (Nagelkerke R squared) of the variance in SI, and the interaction was trending ($Beta = .73$, $SE = .43$, $p = .09$, 95% CI: $-.11, 1.58$). Analyses of the simple slopes suggested that anxiety symptoms were associated with later SI for those reporting high levels of daytime sleepiness, but not for those with low levels of daytime sleepiness ($Effect_{high} = 5.15$, $SE_{high} = 2.44$, $p_{high} = .03$, 95% CI $_{high}$: $.38, 9.93$; $Effect_{low} = -.45$, $SE_{low} = 1.87$, $p_{low} = .81$, 95% CI $_{low}$: $-4.12, 3.22$). Figures for these marginal interactions can be found in Supplemental File 1.

Actigraphy.—For actigraphy-measured SOL, the interaction with anxiety symptoms was trending ($Beta = .13$, $SE = .09$, $p = .17$, 95% CI: $-.05, .31$), although the overall model was not significant. The model explained between 10.12% (Cox and Snell R squared) and 15.19% (Nagelkerke R squared) of the variance in SI. Analyses of the simple slopes indicated that anxiety was marginally associated with SI when SOL duration was longer ($Effect_{long} = 2.88$, $SE_{long} = 1.71$, $p_{long} = .09$, 95% CI $_{long}$: $-.46, 6.22$; $Effect_{short} = .04$, $SE_{short} = 1.93$, $p_{short} = .98$, 95% CI $_{short}$: $-3.73, 3.82$; see figure in Supplemental File 1). No findings emerged for the models examining actigraphy-measured TST or WASO.

Polysomnography.—For REM%, the interaction term between REM% and anxiety symptoms was trending ($Estimate = .33$, $SE = .22$, $p = .13$, 95% CI: $-.10, .76$). However, the overall model was not significant. The model explained between 14.87% (Cox and Snell R squared) and 21.79% (Nagelkerke R squared) of the variance in SI. Analyses of the simple slopes indicated that anxiety symptoms were a significant predictor of later SI for those experiencing high levels of REM sleep but not those with lower levels of REM sleep ($Estimate_{high} = 3.90$, $SE_{high} = 1.90$, $p_{high} = .04$, 95% CI $_{high}$: $.17, 7.63$; $Estimate_{low} = -.42$, $SE_{low} = 1.95$, $p_{low} = .82$, 95% CI $_{low}$: $-4.25, 3.40$; see figure in Supplemental File 1). No findings emerged for N3%.

Discussion

Our study fills a critical gap in the literature by examining both objective and subjective sleep characteristics in conjunction with childhood anxiety symptoms in the prediction of SI risk during the transition from childhood to adolescence. Overall findings suggest that certain aspects of childhood sleep, especially parent-reported sleep disturbances, act synergistically with anxiety symptoms to heighten risk for SI years later. These results converge with other longitudinal data revealing anxiety and sleep disturbances to independently forecast later SI, and extend this work by showing anxious children who also experience comorbid sleep problems to be particularly vulnerable. As both anxiety and sleep problems represent malleable risk factors, these findings also highlight potential opportunity for early identification and prevention.

In terms of subjective sleep reports, results indicated childhood anxiety symptoms were associated with later SI only for children who experienced high levels of sleep disturbance. Notably, an anxiety by sleep interaction was prognostic when total sleep disturbance scores on the CSHQ were above the established clinical cut-off, supporting the clinical utility of this measure. Follow-up analyses of the CSHQ subscales further revealed long sleep onset delays, and marginal evidence for short sleep duration, greater nighttime wakings, and/or daytime sleepiness to pose SI risk in the face of elevated anxiety levels. While some of these interactions or simple slopes were only statistically marginal, it is notable that the patterns of findings were consistent across these subscales.

There are a range of mechanisms that could explain why subjective sleep disturbances amplify anxiety-based risk for SI. For example, negative emotional experiences (Palmer & Alfano, 2017b), problems with cognitive control including increased catastrophizing and rumination (Noone et al., 2014; Carney et al., 2006), and impaired social functioning (Beattie et al., 2015), are exacerbated when sleep is disrupted. Importantly, the current sample was pre-pubertal at Time 1, and sleep-anxiety links may be particularly problematic during the transition to adolescence when emotional systems are developing. Indeed, research has suggests that sleep problems during the early adolescent period may be a time when emotional health is most sensitive to the effects of poor sleep (Kelly & El-Shiekh, 2014). Consequently, it is possible that sleep disturbances may result in cascading negative emotional effects that become more severe overtime. It is also possible that sleep disturbances exacerbate anxiety symptoms in children (Alfano et al., 2007), or lead to the emergence of new symptoms overtime (e.g., depressive symptoms).

Another possible mechanism for associations found among anxiety symptoms, subjective sleep disturbances and SI is articulated in Joiner's interpersonal-psychological theory of suicide (Joiner, 2005). This theory states that desire to die by suicide may result from perceptions of decreased belongingness and isolation. Many anxious children experience difficulties with social and interpersonal functioning (Beidel & Alfano, 2011) and it increasingly clear that poor sleep similarly also corresponds in a range of social impairments including less time spent engaging in social activities (Beattie et al., 2015; Palmer & Alfano, 2017b). As a result, a combination of anxiety and impairing sleep problems may result in poorer social relationships and ultimately feeling less belongingness. However, as

belongingness was not assessed in the current study, this is speculative and these connections should be tested in future investigations.

Joiner's (2005) interpersonal-psychological theory of suicide may also be one possible explanation for the discrepancies between some of the sleep findings. Specifically, marginal findings emerged suggesting that parent-reported sleep duration and night wakings moderated the anxiety-SI relationship, whereas similar objectively measured sleep patterns (TST, WASO) did not. According to the interpersonal-psychological theory of suicide, risk of suicide is greater in those who perceive the self as a burden to others. It is possible that parent-reported sleep disturbances more accurately assess the burden of the child's sleep disturbance on the family, as parents may be more aware of these sleep patterns when they result in some type of disturbance in their own sleep (e.g., their child wakes up throughout the night and requires a parent to help them fall back asleep). As a result, parental reports of sleep problems may be a proxy for perceived burdensomeness of these disturbances. Again, while these connections are largely speculative as burdensomeness was not directly assessed, further examination of the association between parental reports and children's perceptions of their sleep problems as a burden on others may be an important direction for future research. Further, a combination of these potential mechanisms may best explain associations between anxiety, sleep, and suicidal ideation. A recent study of adolescents identified serial indirect effects from anxiety to depressive symptoms to perceived burdensomeness, and finally, to suicide ideation (Hill, del Busto, Buitron, & Pettit, 2018). It is possible that sleep disturbances impact that pathway at multiple points.

It is also important to note that previous studies using the CSHQ have also identified that the subscales do not directly map on to polysomnography or actigraphy-measured variables (Markovich, Gendron, & Corkum, 2014). Discrepancy between subjective and objective sleep findings may therefore be due to the two methodologies measuring different aspects of sleep disturbances. For example, while actigraphy-measured TST and WASO identify the total time asleep or awake (in minutes), the subjective reports may instead be assessing how problematic these aspects of sleep are for overall sleep health which can be biased by individual differences in sensitivity to sleep need and the disruptiveness of night wakings.

The findings from the present study also contribute to research using polysomnography to determine relationships between sleep and SI. Previous studies have reported associations between increased REM sleep duration and risk for SI in adults (Sabo et al., 1990; Lewis et al., 1996), but not in adolescents (Dahl et al., 1990; Rao et al., 1996). Consistent with prior findings in adolescents, REM% in the present pre-pubertal sample did not significantly predict later ideation. However, it is important to note that the interaction between REM% and anxiety symptoms was trending, and follow-up analyses of the simple slopes indicated that the relationship between anxiety symptoms and SI was significant for those experiencing higher levels of REM%. Longitudinal data on objective sleep characteristics and suicide risk is limited, particularly for anxious children and adolescents, and the findings of this study provide preliminary evidence for these processes. However, this marginal finding should be interpreted with caution until replicated using a larger sample.

The present study has a few limitations that should be acknowledged and considered when planning future research. First, the results of this study should be interpreted with caution due to the small sample size, and further replication is needed in understanding the processes underlying sleep disturbances and SI in anxious children. Consistent with prior research (Center for Disease Control and Prevention, 2018), the majority of our participants reporting SI were also primarily female, but our small sample size limited power to test differences by gender. Future studies should further investigate how gender interacts with both childhood anxiety and sleep in predicting later risk. Additionally, our measurement of suicidality was not comprehensive. While all reports of SI were confirmed by a trained interviewer and were reviewed with a licensed clinical psychologist, the nature of our suicide assessment necessitated dichotomizing participants based on whether they reported any SI or no SI. Future studies should measure SI using more comprehensive methods to capture variation in SI severity, such as the Beck Scale for Suicide Ideation (Beck & Steer, 1991) which has demonstrated excellent psychometric properties (Batterham et al., 2015). Additionally, children with SI at Time 1 were excluded from study participation, thus the study was unable to examine children with ideation prior to study entry. Future research should assess suicide risk with greater precision to facilitate more accurate examination of time of onset and course of SI with regard to sleep problems.

The polysomnography data acquired included only a single night of assessment. This limits the assessment as the results may have been biased by first night effects (i.e., discomfort due to electrode placement) or sleep patterns that were not typical or normative of the participants. However, it is important to note that the disruptive effects may have been alleviated slightly as participants slept in their own homes (as opposed to a hospital or sleep laboratory) for the night of the assessment. Lastly, while our sample was somewhat diverse, participants in this study were still predominantly Caucasian/White, limiting the external validity with which the findings may be generalized to other populations. It is highly recommended that future studies seek more diverse participants by exploring these processes in different racial or ethnic groups.

Implications for Practice

Identification of early risk for suicide is critical, particularly in high-risk samples (i.e., those with elevated anxiety symptoms). Results from the current study suggest that children with higher pre-pubertal anxiety symptoms were most likely to report SI years later, but only when high levels of sleep disturbance were also present. Based on these findings and previous literature, it is suggested that the incorporation of sleep assessments such as the CSHQ may be an important evidence-based tool in assessing risk for SI. Further, behavioral treatments for childhood sleep disturbances (CBT-I) have been shown to be effective for those who are anxious (Gregory & Sadeh, 2016). Thus, identifying and treating sleep disturbances prior to the onset of adolescence may be an effective treatment in reducing the risk of SI in populations at heightened risk for suicidality.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Funding information:

This work was supported by the National Science Foundation under Grant 1813574 (PI: Palmer); National Institutes of Health under Grant K23 MH081188 (PI: Alfano); and National Institutes of Health under Grant R21 MH099351 (PI: Alfano).

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Figure 1. Interaction between anxiety symptoms and subjective sleep disturbances at Time 1 predicting SI at Time 2. The simple slope of high levels of sleep disturbances is significant ($p = .016$).

Figure 2. Interaction between anxiety symptoms and subjective sleep onset delay at Time 1 predicting SI at Time 2. The simple slope for a longer sleep onset delay is significant ($p = .025$).

Table 1

Demographic Characteristics and Between-Group Comparisons for Participants Reporting (n =16) and Not Reporting (n = 55) SI

	SI	No SI	Entire Sample		
	M (SD) / n (%)	M (SD) / n (%)	M (SD) / n (%)	t(df) or χ^2 (df)	p
Age (Time 1)	9.5 (.89)	8.8 (1.49)	8.99 (1.40)	-2.21 (41.41)	.03
Age (Time 2)	12.75 (1.73)	12.18 (2.17)	12.31 (2.08)	-.96 (69)	.34
Sex				5.8 (1)	.02
Male	3 (18.75%)	29 (52.73%)	32 (45.07%)		
Female	13 (81.25%)	26 (47.27%)	39 (54.93%)		
Race				5.1 (4)	.28
White	12 (75%)	34 (61.81%)	46 (64.78%)		
Black/African-American	1 (6.25%)	12 (21.82%)	13 (18.30%)		
Asian	0 (0%)	4 (7.27%)	4 (5.63%)		
Biracial/Other	3 (18.75%)	5 (9.09%)	8 (11.27%)		
Ethnicity				.01 (1)	.93
Hispanic/Latino	6 (37.50%)	20 (36.36%)	26 (36.62%)		
Not Hispanic/Latino	10 (62.50%)	35 (49.30%)	45 (63.80%)		
Diagnoses (Time 1)				6.9 (1)	.01
No Diagnosis	11 (68.75%)	48 (87.27%)	59 (82.10%)		
Social Anxiety	1 (6.25%)	3 (5.45%)	4 (5.63%)		
Specific Phobia	1 (6.25%)	2 (3.64%)	3 (4.23%)		
GAD	4 (25%)	6 (10.91%)	10 (14.08%)		
MDD	0 (0%)	0 (0%)	0 (0%)		
ADHD	0 (0%)	0 (0%)	0 (0%)		
Other	1 (6.25%)	1 (1.82%)	1 (1.41%)		
Diagnoses (Time 2)				4.4 (1)	.04
No Diagnosis	5 (31.25%)	50 (90.9%)	55 (77.46%)		
Social Anxiety	3 (18.75%)	0 (0%)	3 (4.23%)		
Specific Phobia	1 (6.25%)	2 (3.64%)	3 (4.23%)		
GAD	5 (31.25%)	3 (5.45%)	8 (11.27%)		
MDD	3 (18.75%)	0 (0%)	3 (4.23%)		
ADHD	0 (0%)	1 (1.82%)	1 (1.41%)		
Other	1 (6.25%)	2 (3.64%)	3 (4.23%)		
Missing*	1 (6.25%)	0 (0%)	1 (1.41%)		

Notes. SI = suicidal ideation, GAD = generalized anxiety disorder, MDD = major depressive disorder, ADHD = attention-deficit/hyper activity disorder. Many participants met criteria for multiple disorders. Chi-square results for psychiatric diagnoses between the SI group and no SI group compare those who met criteria for any psychiatric disorder compared to those who did not meet criteria for any disorder.

* One participant is missing a final diagnosis at Time 2 due to reports of severe ideation during the diagnostic interview, and as a result the assessment was ended early.

Table 2

Means, Standard Deviations, and Between-Group Comparisons between Participants Reporting (n = 16) and Not Reporting (n = 55) SI on Time 1 Variables

	SI M (SD)	No SI M (SD)	Entire Sample M (SD)	t(df)	p	95% CI
Symptoms						
Anxiety Symptoms	.44(.52)	.22(.25)	.27 (.34)	-2.38(17.12)	.12	-.51, .06
Depressive Symptoms	.15(.14)	.09(.15)	.10 (.15)	-1.45(69)	.15	-.14, .02
Subjective Sleep						
Total Disturbances	44.63(10.07)	41.98(5.88)	42.59 (7.06)	-1.00(18.13)	.33	-8.19, 2.90
Sleep Onset Delay	1.44(.73)	1.31(.54)	1.34(.59)	-.73(68)	.47	-.46, .21
Sleep Duration	3.56(.89)	3.46(.97)	3.49(.94)	-.37(68)	.71	-.64, .44
Night Wakings	2.56(1.21)	2.29(.54)	2.36(.74)	-1.27(68)	.21	-.69, .15
Daytime Sleepiness	12.88(3.56)	12.52 (3.60)	12.60(3.57)	-.35(68)	.73	-2.40, 1.68
Bedtime Resistance	7.88(2.19)	7.44(2.04)	7.54(2.07)	-.73(68)	.47	-1.61, .75
Sleep Anxiety	5.56(2.22)	5.02(1.74)	5.14(1.86)	-1.03(68)	.31	-1.60, .51
Actigraphy						
SOL	20.44(14.87)	21.19 (10.30)	21.01 (11.42)	.22(62)	.83	-6.06, 7.56
TST	508.63(36.28)	505.37(42.54)	506.11 (40.99)	-.28(69)	.78	-26.64, 20.12
WASO	33.27(25.07)	30.58(23.09)	31.20(23.40)	-.40(67)	.69	-16.10, 10.72
PSG						
N3%	23.72 (6.32)	23.06 (7.91)	23.24 (7.47)	-.27(48)	.79	-5.55, 4.23
REM%	18.27 (4.64)	20.01 (6.78)	19.55 (6.29)	.85(48)	.40	-2.34, 5.83

Notes. SI = suicidal ideation, SOL = sleep onset latency (minutes), TST = total sleep time (minutes), WASO = wake after sleep onset (minutes), PSG = polysomnography, N3% = percentage of total sleep comprised of stage 3 sleep, REM% = percentage of total sleep comprised of REM sleep.