MIND, BRAIN, AND EDUCATION

Dissonance Between Parent-Selected Bedtimes and Young Children's Circadian Physiology Influences Nighttime Settling Difficulties

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ABSTRACT— Nighttime settling difficulties (i.e., bedtime resistance, sleep-onset delay) occur in about 25% of young children and are associated with attentional, behavioral, and emotional problems. We examined whether the timing of internal (endogenous) circadian melatonin phase (i.e., dim light melatonin onset; DLMO) and its relationship with parent-selected bedtimes were related to nighttime settling behaviors. Fourteen regularly napping preschoolers (8 females; 30–36 months) participated in a 6-day protocol (parent-report of nighttime settling, actigraphic assessment of sleep onset latency, evening salivary DLMO). Average DLMO clock time was 07:40 p.m. ± 00:48 minutes, occurring 29 minutes ± 32 minutes prior to bedtime (lights-out). Children with later DLMOs had longer sleep-onset latencies ($r = .62$) and poorer success in falling asleep ($r = -.59$). Children whose bedtimes were closer to their DLMO had longer sleep-onset latencies ($r = .72$) and increased bedtime resistance ($r = -.54$). We conclude that dissonance between parent-selected bedtimes and children's circadian physiology may contribute to the development of nighttime settling difficulties in early childhood.

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Sleep problems are a frequent parental complaint in clinical practice. Findings from large-scale studies estimate that approximately 25% of young children experience some type of sleep disturbance (e.g., bedtime resistance, difficulties falling asleep, prolonged nighttime or early terminal awakenings) (Beltramini & Hertzig, 1983; Bruni, Lo Reto, Miano, & Ottaviano, 2000; Lozoff, Wolf, & Davis, 1985), which often persist into later childhood (Kataria, Swanson, & Trevathan, 1987). Early sleep difficulties are associated with concurrent attentional, emotional, and behavioral problems (Bruni et al., 2000; Hvolby, Jorgensen, & Bilenberg, 2008; Lavigne et al., 1999; Minde, Faucon, & Falkner, 1994) and predict the onset of such problems in later childhood and adolescence, even after controlling for the stability of sleep disturbance and demographics (Friedman, Corley, Hewitt, & Wright, 2009; Gregory & O'Connor, 2002; O'Callaghan et al., 2010). Finally, parents of children with sleep problems often complain of interruptions and changes in their own sleep patterns, chronic fatigue, and spousal discord (Chavin & Tinson, 1980).

Specifically, settling at night is a challenge for many young children. Bedtime resistance (i.e., "curtain calls," tantrums about bedtime, calling out after being put to bed) and difficulties falling asleep are commonly reported by parents (Beltramini & Hertzig, 1983; O'Callaghan et al., 2010). Interestingly, prior research findings suggest such settling difficulties increase across early childhood, with the most dramatic changes occurring between infancy and age 3 years. In one longitudinal study, bedtime resistance occurred in 14% of infants, 42% of 3-year-olds, and 50% of 5-year-olds. Similarly, 26% of infants in comparison to 61% of 3-year-olds and 66% of 5-year-olds took longer than 30 minutes to fall asleep at night. Thus, early childhood likely represents

a sensitive window in the development of nighttime settling difficulties.

Current theoretical models suggest sleep is regulated by two intrinsic biological processes, a sleep–wake dependent homeostatic process and a clock-like circadian process (Achermann & Borbely, 2003; Borbely, 1982; Daan, Beersma, & Borbely, 1984; Jenni & LeBourgeois, 2006). The homeostatic process accounts for the accumulation of sleep pressure with sustained wakefulness and for its dissipation during sleep. In contrast, the circadian process oscillates with a near 24-hour cycle typically promoting arousal during the daytime (highest in the early evening) and sleep during the night (highest in the early morning). The increase in homeostatic pressure during wakefulness is thus opposed by the increase in circadian alertness across the day, thereby allowing maintenance of relatively constant levels of alertness throughout the waking episode. In the late evening, sleep propensity is facilitated by the combination of high homeostatic sleep pressure and an increase in circadian sleep tendency, thus producing a "sleep gate." During the night, an increase in circadian sleep tendency counteracts the declining homeostatic sleep pressure to ensure sleep continuity. Although the interaction of both processes plays an important role in determining levels of alertness and sleep tendency across the 24-hour day, the focal point of this article is the circadian process and its influence on nighttime settling in young children (i.e., bedtime resistance, sleep-onset delay).

Melatonin is one of the primary outputs of the circadian system; the master biological clock located in the SCN of the hypothalamus stimulates the pineal gland, which is responsible for regulating the release of this hormone (Moore & Klein, 1974; Rosenwasser, 2009). Although the daily rhythm of melatonin levels is driven by the internal circadian clock, they can be suppressed by light exposure at night (Lewy, Wehr, Goodwin, Newsome, & Markey, 1980). Endogenous levels of melatonin concentration begin to rise before sleep, peak during the night or early morning, and fall to daytime levels before morning rise time (Lewy & Markey, 1978; Wehr, 1991). In humans, increased secretion of melatonin initiates a cascade of physiological events that are sleep promoting (e.g., thermoregulatory and neural changes). The timing of the 24-hour sleep–wake cycle is in close sync with the intrinsic melatonin rhythm due to the strong modulatory influence of the circadian system (Shochat, Luboshitzky, & Lavie, 1997).

Melatonin is commonly used to quantify internal biological time. Circadian melatonin phase (i.e., dim light melatonin onset; DLMO) is a well-established, reliable phase marker of the circadian system. Based upon saliva or blood samples taken at frequent time points (e.g., every 30 or 60 minutes), the DLMO is defined as the time when melatonin levels rise above a specific threshold. In healthy adults, DLMO closely corresponds with the onset of sleep time (Burgess & Eastman, 2005). The phase angle of entrainment can be defined as the time between endogenous circadian phase and a recurring environmental or behavioral event (e.g., interval between DLMO and bedtime, midpoint of sleep, or rise time). Factors known to influence the phase angle of entrainment include the intrinsic period of the circadian clock, prior light exposure history, and older age (Wright, Gronfier, Duffy, & Czeisler, 2005). Currently, little is known about the timing of the internal circadian clock and its relationship with parent-selected bedtimes in young children.

An extensive literature on the role of the circadian system in coordinating physiology and behavior is available in adolescents and adults, and knowledge of the biological limits imposed by the master clock on individuals' ability to adapt to different environmental circumstances is expanding. Findings from well-controlled laboratory studies in which the homeostatic and circadian systems are disentangled (forced desynchrony) indicate that attempting to sleep at an inappropriate internal circadian time leads to prolonged sleep latencies, decreased sleep efficiency, and lower total sleep time (Dijk & Czeisler, 1995). In the real world, social demands, such as early school start times, shift work, and long work hours, challenge the ability of adolescents and adults to obtain sufficient sleep and to maintain optimal sleep–wake rhythms (Carskadon, 2004). Common consequences, including decreased work productivity, increased motor vehicle accidents, and decrements in mood, attention, and behavior, provide evidence of the dissonance—or poor fit (Thomas & Chess, 1977)—between the circadian timekeeping system and social expectations/demands (Pack et al., 1995; Wolfson & Carskadon, 1998).

Young children, too, face environmental demands that may result in poor fit with the biological limits imposed by their circadian system (Jenni & O'Connor, 2005). Unlike most adults, who when permitted are likely to select a sleep schedule reflective of their endogenous circadian phase (Duffy, Dijk, Hall, & Czeisler, 1999), the time children are expected to try to fall asleep in the evening is largely determined by their parents or caregivers. The question of appropriate bedtimes in early childhood is a common point of discussion in the media, parenting groups, educational settings, and the scientific community. Whether variability in the "fit" between circadian physiology and imposed bedtimes contributes mechanistically to nighttime settling difficulties (i.e., bedtime resistance, sleep-onset delay) in young children is an unanswered question.

In this work, we studied healthy, typically developing 30–36-month-old children with no reported sleep problems. We chose this age group because it offers a sensitive window on the development of nighttime settling difficulties. Also, given that inconsistent napping may produce differing levels of sleep pressure in the evening (Achermann & Borbely, 2003), we studied only children who regularly took daytime naps. Children participated in an in-home salivary DLMO protocol after 5 days of actigraphy monitoring while sleeping

on a parent-selected sleep schedule. We obtained subjective and objective measures of nighttime settling. With these data, we examined whether the timing of the circadian system (DLMO) and its relationship to parent-selected bedtimes were associated with young children's nighttime settling behaviors. We hypothesized that children with later DLMOs and parent-selected bedtimes closer to their DLMOs would take longer to fall asleep at night and have poorer reported success in going to bed (bedtime resistance) and falling asleep (sleep-onset delay).

METHODS

Participant Recruitment and Screening

We recruited families in Providence, RI, via flyers, laboratory website advertisements, and face-to-face contact at community events. Parents completed a brief telephone screening interview and a set of questionnaires that more fully assessed inclusion/exclusion criteria. Study inclusion required children to be 30 to 36 months old at the time of assessment and regularly following a biphasic sleep schedule (nighttime sleep episode of at least 10.5 hours and one daytime nap of at least 45 minutes time in bed) in which they reportedly fell asleep ≥3 days per week during the nap opportunity. Exclusionary criteria included the following: (1) regular bed sharing; (2) a bedtime/rise time sleep schedule >2 hours between weekdays and weekends; (3) travel beyond two time zones within 3 months before assessments; (4) regular use of medications affecting sleep, alertness, or the circadian system; (5) sleep problems; (6) developmental disabilities, neurologic/metabolic disorders, chronic medical conditions, lead poisoning, or head injury involving loss of consciousness; (7) a conceptual age of ≤37 weeks or >42 weeks; (8) low birth weight (<5.5 lbs); or (9) a family history (first degree) of diagnosed narcolepsy, psychosis, or bipolar disorder.

Of the 78 children screened, 32 met criteria. Sixteen children enrolled, and 15 completed the study. One child was unable to produce adequate saliva during the training segment, and another child was excluded after data collection because of a "missed" DLMO (description below); thus, this analysis includes 14 children. All parents signed a Brown University IRB-approved consent form. Parents received $50 in cash; children received small non-monetary gifts and a $50 savings bond at study completion.

Participants

Participants were 14 healthy children (8 females; 6 first-born; 12 Caucasian, 1 African American, 1 mixed-race), ages 30 to 36 months ($M = 34$; $SD = 2$). Four attended full-time daycare, one attended part-time daycare, three had in-home childcare provided by a nonfamily member, two were cared for by an extended-family member at their home, and four were taken care of exclusively by their parents.

Protocol

Parents completed the Children's Sleep–Wake Scale (CSWS, see below for details) approximately 3 weeks prior to the start of the 6-day study. During days 1–5, researchers made in-home visits to build rapport with families and to train children in providing adequate saliva samples. Children followed a parent-selected sleep schedule during this segment leading up to the DLMO assessment on day 6. Throughout the study, children did not consume substances (e.g., caffeine, medications) affecting the sleep or circadian systems, and their sleep was monitored continuously with diaries and wrist-worn actigraphy. Data were collected September–December 2008 ($n = 8$) and February–May 2009 ($n = 6$). Participants were not studied during the 7 days following daylight saving time changes.

Measures

Parental Daily Reports

We e-mailed or telephoned parents daily to inquire about their children's behavior, sleep patterns, and compliance with study procedures during the previous 24 hours. Questions asked about bedtime (lights-out), rise time, and napping schedule, as well as whether their child went to daycare, was sick, ingested caffeine/medication, and wore the actigraph.

Children's Sleep–Wake Scale

The children's sleep–wake scale (CSWS) is a 25-item pencil-and-paper research instrument assessing sleep quality in 2–8-year-old children across five behavioral dimensions: Going to Bed (5 items), Falling Asleep (5 items), Maintaining Sleep (5 items), Reinitiating Sleep (5 items), and Returning to Wakefulness (5 items). Using a 1-month reference interval, caregivers reported the frequency of their child's sleep behaviors using a 6-point response set (*always*, *frequently-if not always*, *often*, *sometimes*, *not often*, *never*). Scores on each of the five CSWS subscales and the CSWS total sleep quality scale (average of subscales) range from 1 (*poor sleep quality*) to 6 (*good sleep quality*). Psychometric assessments (LeBourgeois, Hancock, & Harsh, 2001; LeBourgeois & Harsh, 2001) showed (1) CSWS subscale and total scale scores have good reliability for research instruments ($\alpha = .72$ to $\alpha = .93$) and (2) CSWS subscale scores correspond adequately with caretaker diary ratings ($r = .52$ to $r = .66$) and actigraphic estimates of sleep quality ($r = .36$ to $r = .63$). Because of our interest in young children's nighttime settling, this analysis focused on the Going to Bed (e.g., when it's time for bed child makes repeated requests; child complains about going to bed; child "puts off" or delays bedtime) and Falling Asleep (e.g., child has

trouble falling asleep; child falls asleep quickly after lights-out) subscales of the CSWS.

Sleep Diary

Parents completed a 26-item daily sleep diary. Evening questions asked about children's mood, stress level, nap time(s), caffeine/medicine intake, actigraph off times, bedtime, and lights-out time. Morning questions inquired about children's night awakenings, sleep quality, and rise time. We used sleep diary entries to verify compliance with study rules and to support scoring of actigraphy data.

Actigraphy

Actigraphy is a noninvasive tool for objectively assessing sleep patterns under nonlaboratory conditions (Acebo & LeBourgeois, 2006). The actigraph (model AW64) was worn on the child's nondominant wrist and provided continuous recordings of sleep–wake states by measurement of motor activity (MiniMitter Company, Bend, OR, USA). Actiware-Sleep V5.02 software was used to estimate-minute actigraph epochs for sleep and wakefulness from activity levels produced in the surrounding 2-minute interval. This algorithm was applied to portions of the record identified as sleep through a combination of diary reports and actigraph event markers set by parents at "lights-out" and "lights-on." Standard scoring rules were applied to each sleep episode—sleep start was the first of three epochs of sleep after lights-out, and sleep end was the last of five epochs of sleep before lights-on. Sleep episodes were excluded when the (1) actigraph was off for all/part of the sleep period, (2) concurrent diary report was not available, or (3) the episode included external motion (e.g., sleeping in a car). We present actigraphic estimates of several sleep parameters, including lights-out time, sleep start time, sleep end time, sleep duration (number of minutes between sleep start and sleep end), and sleep-onset latency (number of minutes between lights-out and sleep start averaged across study days 1–5). Actigraphic estimate of lights-out time was also used to derive the bedtime phase angle of entrainment measure. Actigraphy data from 5 days prior to DLMO were usable for all children but one who had 4 days of data.

Salivary Dim Light Melatonin Assessment

On the day of the in-home salivary melatonin assessment, children skipped their routine nap opportunity and entered dim light conditions (light levels ranged from 0.46 to 10.90 lux) at least 1 hour before the first sample, and remained in dim light throughout the saliva collection episode. Lux levels were measured while obtaining each saliva sample with a light meter (Extech Instruments, Spring Hill, Florida, USA) held approximately 5 cm adjacent to the child's eye and directed in the angle of gaze. Children provided saliva samples (~2 mL) every 30 minutes for 6 hours concluding one hour past their average bedtime (12 samples total) from parental reports during study days 1–5. With assistance from a researcher, children rinsed their mouths with water, and if needed, gently brushed their teeth with water before samples occurring after eating (>15 minutes before each saliva sample). Saliva samples were collected by having children chew on a braided dental cotton roll (Henry Schein Inc., Denver, Pennsylvania, USA) for 1–2 minutes. Samples were immediately centrifuged (LabEssentials, Inc. Monroe, Georgia, USA), refrigerated, and frozen (−20 °C) within 12 hours. Assays were performed at the Bradley Hospital Sleep and Chronobiology Laboratory (Providence, RI, USA) using radioimmunoassay (ALPCO Diagnostics, Salem, New Hampshire, USA), minimum detection 0.2 pg/ml. The intra- and inter-assay coefficients of variation for evening time levels of salivary melatonin were 4.1% and 6.6%, respectively.

Data Processing and Analysis

Circadian DLMO phase was defined as the clock time evening salivary melatonin concentrations increased and remained above a 4 pg/mL threshold using linear interpolation between successive samples. This threshold is a well-accepted standard with children and adolescents (Carskadon, Acebo, Richardson, Tate, & Seifer, 1997) and was derived from reports that salivary melatonin concentrations are about 40% of plasma levels in healthy young adults (10 pg/mL is the most common DLMO threshold for plasma melatonin) (Deacon & Arendt, 1994). The DLMO was "missed" if at least two data points did not remain above the 4 pg/mL threshold. The bedtime phase angle of entrainment was computed as the time interval between DLMO and actigraphic estimate of lights-out time averaged across study days 1–5 (Figure 1). Children's success in initiating sleep at night was estimated objectively with actigraphy (sleep-onset latency) and subjectively with the Going to Bed (bedtime resistance) and the Falling Asleep (sleep-onset delay) subscales of the CSWS.

Statistical analyses were performed with PASW Statistics Package 17.0 (SPSS Inc., Chicago, IL, USA). Summary measures are presented as mean ± standard deviation. Pearson correlations were used to assess associations between continuous variables and independent *t*-tests were used to examine sex and daycare status differences in circadian parameters.

RESULTS

Sleep Patterns Prior to the DLMO Assessment

Children followed parent-selected sleep schedules during the five days before the DLMO assessment. Aggregated data across this interval from parental daily reports showed children's average bedtime (lights-out) was 08:08 p.m. ± 00:38 minutes, rise time was 07:03 a.m. ± 00:29 minutes, and time in

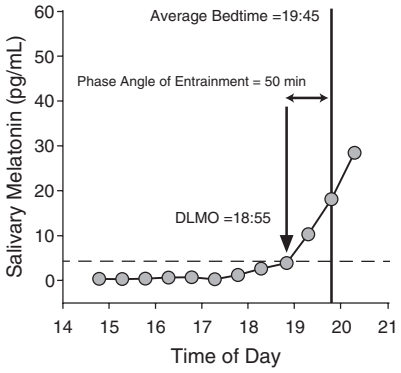

Fig. 1. Evening melatonin profile for an individual child. Average bedtime during the 5 days prior to the salivary dim light melatonin onset (DLMO) assessment was 19:45 DLMO was 18:55 based on a 4 pg/mL threshold (dotted line). The bedtime (lights-out) phase angle of entrainment (interval between DLMO and parent-selected bedtime) was 50 minutes.

bed was 10 hours 54 minutes ± 44 minutes. Based upon actigraphy, average lights-out time (event marker press) was 08:09 p.m. ± 00:37 minutes, sleep start time was 08:37 p.m. ± 00:43 minutes, sleep end time was 06:55 a.m. ± 00:32 minutes, and sleep duration was 10 hours 26 minutes ± 46 minutes. An important part of this study was to verify children were maintaining a biphasic sleep schedule (falling asleep during daily nap opportunities) during the 5 days prior to the DLMO assessment. Actigraphy showed 9 children fell asleep at naptime on all 5 days, and 5 children fell asleep 3 of 5 days (3.9 ± 1.9 days; 86% of days), thereby verifying children were following a biphasic sleep pattern.

Circadian Parameters

Melatonin assessment results in this group of healthy, napping, 30- to 36-month-old children indicated an average DLMO of 07:40 p.m. ± 00:48 minutes, which occurred 29 minutes ± 32 minutes prior to bedtime (phase angle of entrainment). DLMO time and phase angle of entrainment were not associated with age, sex, daycare status, or number of days napping.

Measures of Nighttime Settling

Actigraphy indicated children's average sleep-onset latency during the five days prior to the DLMO assessment was 31.1 minutes ± 10.9 minutes. Parental reported scores were 4.3 ± 0.7 for the Going to Bed and 4.6 ± 0.7 on the Falling Asleep subscales of the CSWS (Figure 2).

Associations Between Circadian Parameters and Nighttime Settling Measures

Figure 2 shows scatterplots and correlation coefficients for all examined associations. In terms of the clock hour of circadian phase, children with later DLMOs took longer to fall asleep after lights-out (sleep-onset latency) as estimated by actigraphy ($r = .62, p = .018$) and were rated by their parents as having poorer success in falling asleep ($r = -.59, p = .027$) but not going to bed ($r = -.39, p = .166$). Children with more narrow bedtime phase angles of entrainment also had longer sleep-onset latencies ($r = .72$, $p = .004$) and were reported by their parents as less successful in going to bed at bedtime ($r = -.54$, $p = .047$) but not falling asleep after lights-out ($r = -.26$, $p = .370$). Individual differences in DLMO and bedtime phase angle of entrainment were not associated with parental reports of children's success in maintaining sleep (arousals, awakenings), reinitiating sleep during the night, or waking in the morning as assessed by the CSWS.

DISCUSSION

To our knowledge, this is the first study to examine associations between circadian physiology and measures of nighttime settling in young children. We obtained a reliable and precise measure of circadian phase, DLMO, in the home environment by modifying standard laboratory procedures with adolescents and adults to be more child-friendly. We also employed multimodal assessments of children's nighttime settling. Tightly controlled procedures, wide variability in circadian and sleep parameters, and strong associations led to several important findings: (1) DLMO in habitually napping 30- to 36-month olds occurs at about 07:40 p.m., with a bedtime phase angle of entrainment of approximately 30 minutes; (2) children with later circadian phases take longer to fall asleep after lights-out as indicated by actigraphy and parental reports; and (3) children with shorter intervals between DLMO and bedtime have longer sleep-onset latencies and are more resistant to going to bed. Findings are discussed with regard to the benefits of assessing the hormone melatonin as a means to further understand young children's circadian physiology and its role in the development and maintenance of nighttime settling difficulties.

We approach the study of nighttime settling from the "Goodness of Fit" perspective (Thomas & Chess, 1977), which suggests children's behaviors are best understood within the context of intrinsic child factors (i.e., biological capabilities, individual characteristics) in their interaction with extrinsic factors (i.e., environmental demands, opportunities, stresses). In this study, we used bedtime phase angle of entrainment as a measure of the "fit" resulting from an interaction between children's intrinsic biological sleep capability (circadian phase; DLMO) and environmental demands (parent-selected bedtimes).

Circadian parameters in our group of healthy, regularly napping young children differed from those of other age groups. Specifically, young children's DLMO was on average

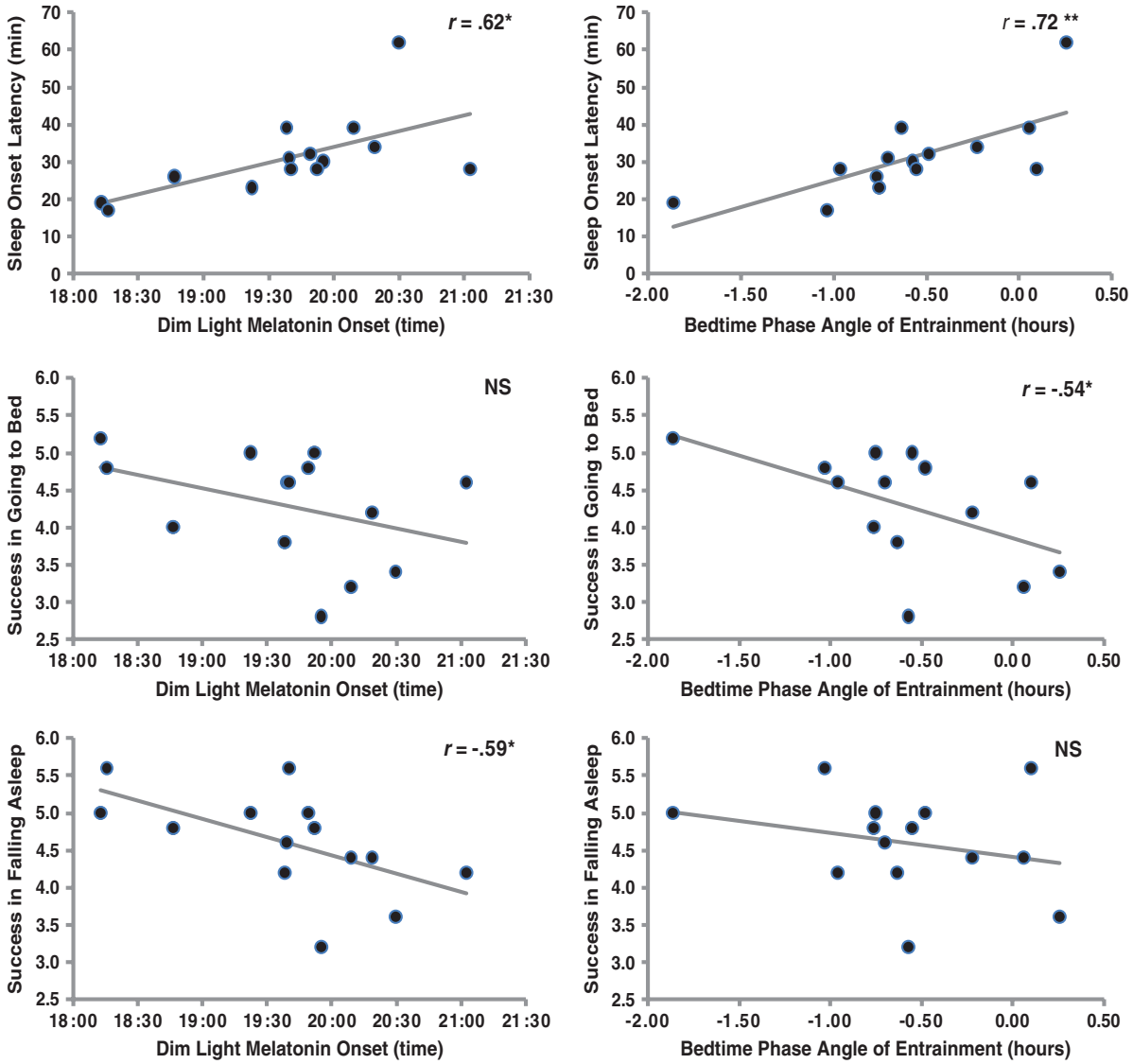

Fig. 2. Scatterplots of associations between circadian parameters and children's nighttime settling behaviors. Pearson correlations (*$p < .05$; **$p < .01$).

07:40 p.m., whereas prior studies have reported a DLMO of 08:33 p.m. in prepubertal children, 09:29 p.m. in fully mature adolescents, and 08:35 p.m. in adults aged 22–38 years (Taylor, Jenni, Acebo, & Carskadon, 2005; Wright et al., 2005). We also found the temporal relationship between DLMO and parent-selected bedtime was shorter in toddlers (30 minutes) than in previous reports of young adolescents (71 minutes), older adolescents (116 minutes), and adults (129 minutes)(Crowley, Acebo, Fallone, & Carskadon, 2006; Wright et al., 2005). Thus, although young children's earlier circadian phase may be permissive of a larger interval between DLMO and bedtime, our data suggest their parents select bedtimes that are closer to the increase in evening melatonin than do adolescents and adults.

In this study, children with later DLMOs were more likely to take longer to fall asleep after lights-out as measured with actigraphy and parent-reports. When taking into consideration the interaction between circadian phase and parent-selected bedtime, we found children being put to bed closer to their DLMOs were more likely to take longer to fall asleep, as estimated by actigraphy, and were more likely to be rated by parents as having more settling difficulties. These findings are consistent with classic circadian physiology principles of a "wake maintenance zone" (Strogatz, Kronauer, & Czeisler, 1986) or a "forbidden zone" for sleep (Lavie, 1986), when sleep is attempted at too early of a circadian time. Thus, inappropriate bedtimes relative to the child's internal circadian timing appear to be an important component contributing to

settling difficulties. Future studies manipulating the timing of sleep relative to circadian phase are needed to determine the effects on young children's nighttime settling difficulties. It is possible that the sleep-circadian mismatch we observed may contribute mechanistically to the development of insomnia, as theoretical models of insomnia specify that staying in bed while awake can lead to an association of the bed and bedroom with arousal, not sleep (Drake, Roehrs, & Roth, 2003; Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997). Thus, the practice of putting children to bed at too early of a circadian time may elicit an arousal response via classical conditioning, especially for children with an underlying vulnerability for insomnia (Drake, Friedman, Wright, & Roth, 2011).

Our findings on the relationship between the phase angle of entrainment and bedtime resistance in young children are novel. We found the DLMO-to-bedtime interval but not the clock time of circadian phase was associated with parent-report of success in going to bed, suggesting that the way children react to bedtime is the result of an interaction between their circadian physiology and environment. Young children are not able to choose a bedtime that is in sync with their endogenous circadian rhythms; typically, parents in many industrialized countries control their child's sleep schedule (Jenni & O'Connor, 2005). One can speculate that parents may set their children's bedtimes based on their own/family schedule, personal sleep requirements, circadian preference (i.e., morning or evening tendency), or beliefs about the sleep need of their child. Our data suggest that if young children are put to bed at a biologically nonoptimal time, they will not feel ready for bed and will resist (e.g., come out of the bedroom for another drink of water, call-out, refuse bedtime, tantrum). Interestingly, in the revised version of the International Classification of Sleep Disorders (ICSD-2; American Academy of Sleep Medicine, 2005), the rubric "dyssomnias, extrinsic sleep disorders" includes a limit-setting sleep disorder. Limit-setting sleep disorder involves bedtime struggles and prolonged sleep-onset time which may not be primarily driven by the caregivers' inability of setting limits, but rather by the poor fit between parental expectations (e.g., inappropriate bedtime) and individual biological characteristics of the child (e.g., circadian phase). These results are especially important for children with late circadian phases, as sleeping at their optimal circadian time while at the same time getting adequate sleep may be challenging. Currently, a convenient, cost-effective measure of DLMO is not available. However, if parents are aware of the circadian system's influence on nighttime settling, they may select a bedtime that aligns more closely with a child's individual physiology.

Close examination of the existing literature suggests age, secular, and cultural differences in the incidence of nighttime settling problems. For example, data collected as part of the New York Longitudinal Study of Temperament and Development showed bedtime resistance increased in a linear fashion from 26% at age 2 years to 50% at age 5 years. Likewise, sleep-onset difficulties (>30 minutes to fall asleep) were found in 43% of 2-year-olds, with a peak of 69% at age 4 years (Beltramini & Hertzig, 1983). However, data from the Zurich Longitudinal Studies indicated a peak in bedtime resistance at age 3 years (18.6%), with little to no change between ages 2–5 years (Jenni, Fuhrer, Iglowstein, Molinari, & Largo, 2005). Sleep-onset difficulties in this sample hovered around 9% in 2- to 5-year-olds. Interestingly, parent reports of bedtime resistance were higher and children's bedtimes were earlier in the 1970s than in the 1980s and 1990s (Iglowstein, Jenni, Molinari, & Largo, 2003). Jenni (2005) proposed such differences may be due to bedtimes that are not optimally aligned with the individual child's sleep biology. Our findings provide the first empirical evidence that nonoptimal circadian timing of sleep may strongly contribute to such age-, generation-, and culture-related differences in nighttime settling behaviors. That is, when bedtimes are not adjusted across early childhood to maintain optimal alignment of the sleep and circadian systems, children's intrinsic biology may drive "resistance" to going to bed and falling asleep at night.

Several study limitations deserve mention. First, this analysis included only children who napped on a regular basis, a decision driven by our concern that inconsistent or no napping would result in within- and across-child differences in evening sleep propensity. Our ability to understand circadian and homeostatic influences on nighttime settling as children transition from a biphasic to a monophasic sleep schedule would be strengthened by studying children longitudinally. Second, this study is correlational, thus, we cannot confidently draw causal inferences from our results. Future research should experimentally test the effects of different intervals between DLMO and bedtime on sleep-onset latency and bedtime resistance in children. Third, assessment of children's success in falling asleep is difficult—young children are not able to self-report and parents of good sleeping children may be unaware of sleep-onset latency without frequent bedroom "checks." Finally, this sample included children who were reportedly good sleepers with relatively stable sleep schedules, which may limit the generalizability of our findings. Examining circadian phase and its relation to bedtime in children with sleep-onset difficulties and/or children with more day-to-day variation in sleep patterns would be a fruitful next research direction.

Although our study did not permit direct examination of associations between circadian parameters, nighttime settling, and outcomes related to young children's potential for school success, our results have implications for parents and educators. Children with nighttime settling difficulties are more likely to sleep for shorter durations during the night (Jenni et al., 2005). Thus, they may be at higher risk for sleep restriction at a time when they are also

obtaining many important academic and social/emotional skills (e.g., complexity of language, vocabulary, basic counting, impulse control, listening to directions) that serve as the foundation for future educational success (Rouse, Brooks-Gunn, & McLanahan, 2005). In children, daytime sleepiness is often manifested by increased activity, aggressive behavior, impulsivity, acting-out behavior, poor concentration, and inattention (Fallone, Owens, & Deane, 2002). Given that nighttime settling difficulties commonly emerge in early childhood and may persist into the school years, early intervention is of the utmost importance. Our findings highlight the need to consider children's individual circadian physiology in designing such programs. In adolescents and adults, a rich literature is emerging on the performance, mood, and health consequences of poor fit between the circadian timing system and social demands. With procedures now in place for assessing the onset of the hormone melatonin in young children, we can begin to answer key questions linking circadian rhythms, sleep, and young children's school readiness.

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