

Pre-bedtime lighting as a predictor of sleep outcomes in preschool-aged children

Nell Adams

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Committee:
Michelle Garrison
Maida Chen

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Abstract

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Chair of Supervisory Committee:
Michelle Garrison
Health Services

Studies that examine the effects of light on subsequent sleep tend to be done in controlled laboratory settings, rather than in the lighting conditions humans sustain in day-to-day life; rarer still are environmental light/sleep studies in children. The purpose of this study was to examine the effects of evening environmental lighting on subsequent time to sleep onset latency (SOL), sleep efficiency (SE), and total sleep time (TST) in preschool-aged children. Children who screened positive for behavioral sleep problems (n=411) wore an actigraphy watch, which collected light and sleep data, for one week. Environmental light before bedtime was associated with SOL ($p < 0.01$), but not SE or TST. Light sustained up to, but not beyond, 35 minutes before bedtime predicted SOL. For the 15-minute period pre-bedtime, children whose upper quartile readings were 1 to 10 lux added 6 minutes ($p < 0.001$; 95% CI 3 to 9) to SOL compared to children whose upper quartile readings were < 1 lux. This trend continued with increasing lux exposure: at an upper quartile of ≥ 75 lux, children added 15 minutes to SOL ($p < 0.001$; 95% CI 7 to 24). Our suggestion that even slight differences in the relatively narrow range of at-home lighting (< 1 to 200 lux) in the 35 minutes before habitual bedtime can make a significant impact on the sleep onset latency of preschool-aged children, but that it may not be necessary for parents to restrict lighting > 35 minutes before habitual bedtime, adds new knowledge to the body of sleep research.

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Introduction

On average, preschool-aged children need 9 to 10 hours of nighttime sleep, although this may vary by child (Mindell & Owens, 2010; Simpkin et al., 2014). This amount of sleep is essential for normal growth and cognitive development. Insufficient sleep during this period is linked to short and long-term sequelae. Acute effects of under-sleeping may include mood disturbance and cognitive impairment (Bruni, Lo Reto, Miano, & Ottaviano, 2000; Mindell & Owens, 2010; Scher, Hall, Zaidman-Zait, & Weinberg, 2010). Long-term consequences, which can last into the teen years and even adulthood, may include obesity (Miller, Lumeng, & LeBourgeois, 2015), emotional and behavioral disorders (Hatzinger et al., 2010), and poor academic achievement (Akacem et al., 2016a). Sleep problems tend to present between two-and-a-half and five years of age. During this stage, most sleep disorders are behavioral, that is, related to the refusal or inability to initiate or maintain sleep (Kerr & Jowett, 1994). Parent surveys suggest that 20 to 40% of young children may be affected by behavioral sleep problems (McGreavy, Donnan, Pagliari, & Sullivan, 2005; Mindell, 2004). Predictors of behavioral sleep disorders in children are numerous and multifactorial (Reid, Hong, & Wade, 2009), but environmental factors like noise, temperature, and light can be modified by parents. An environment that is conducive to sleep goes beyond a child's bedroom, however; exposure to too-bright light *before* bedtime can also interfere with sleep (Duffy & Wright, 2005; Khalsa, Jewett, Cajochen, & Czeisler, 2003). Published studies, however, do not provide these guidelines for parents. Two key questions remain: 1) When, before bedtime, should parents begin to limit their preschool-aged child's light exposure? And 2) At what brightness does pre-bedtime light begin to interfere with a preschool-aged child's sleep?

Sleep Regulation

The body governs sleep through two distinct processes: homeostasis and the circadian rhythm. In homeostasis, the body seeks to balance sleeping and waking states by initiating sleep after periods of wake, and wake after periods of sleep (Bathroy & Tomopoulos, 2017). The circadian rhythm, via timed hormone release, can dictate when we tend to feel alert or tired during each 24-hour cycle, and it takes its cues from the circadian “clock” in the hypothalamus. While circadian timing is set by unmodifiable factors like genetics, external cues like sound, light, and meal timing can exert an influence, too (Wams et al., 2017).

Light and the Circadian Rhythm

Of all extrinsic factors that can modify the circadian rhythm, light has the strongest influence (Duffy & Wright, 2005; Khalsa, Jewett, Cajochen, & Czeisler, 2003). When light hits the retina, photoreceptors activate, signaling the pineal gland to stop production of melatonin, the main sleep hormone (Akacem, Wright, & LeBourgeois, 2016b; Batheroy & Tomopoulos, 2017). The circadian rhythm resumes its morning processes, such as increases in metabolism, body temperature, and alertness, when, after a dark night, the retina senses light.

Light exposure in the hours before bedtime can postpone melatonin onset and suppress melatonin, even after light exposure has stopped (Wams et al., 2017). This tends to result in “phase delay,” or later circadian-regulated sleep onset (Gooley et al., 2011), and explains why adults exposed to bright versus dim at-home lighting in the evenings feel less sleepy (Molina & Burgess, 2014). Because they have larger pupils and more transparent lenses than adolescents and adults, young children absorb more retinal light and are more prone to its phase-shifting effects (Batheroy & Tomopoulos, 2017). In one study, school-aged children (mean age: 9 years) experienced almost twice as much melatonin

suppression than their parents after exposure to bright light in the evening (Higuchi, Nagafuchi, Lee, & Harada, 2014).

The timing, intensity, and duration of light exposure determine the extent of phase delay (Akacem et al., 2016a). Humans are most susceptible to light-induced circadian shifting during the biological night, which begins when the body's melatonin levels begin to rise (Zeitzer, Dijk, Kronauer, Brown, & Czeisler, 2000). In dim light conditions, this can begin 2 to 4 hours before habitual bedtime (Gooley et al., 2011; Santhi et al., 2012). Light exposure during this period can significantly delay melatonin onset.

The intensity of light as it hits a surface—in sleep studies, this tends to be the human eye—is measured in lux. Very dim at-home lighting is between <1 to 10 lux, while dim to moderately bright is 10 to 100 lux (Scheuermaier et al., 2010) and very bright at-home light is 200 to 300 lux (Molina & Burgess, 2014). In field studies, adults sustained an average of 40 lux or less while at home in the evening hours, with a range of 28 to 200 lux (Burgess & Eastman, 2004; Santhi et al. 2012; Scheuermaier, Laffan & Duffy, 2010). Bright at-home light (100 to 200 lux) and moderately bright at-home light (65 lux) can delay dim-light melatonin in adults. This lengthens sleep onset latency, or the time it takes to initiate sleep after lights-out. (Molina & Burgess, 2014). In 2011, Gooley et al. found that adults who spent the evening hours in bright room lighting (~200 lux) did not experience melatonin onset until 23 minutes before habitual bedtime, on average.

The relationship between light and phase delay is dose-dependent (Zeitzer et al., 2000). Relatively short exposure to bright light can result in phase delay, while it takes longer for dim lighting to have an equivalent effect. A study of preschool-aged children found that kids exposed to bright light (~1,000 lux) had suppressed melatonin levels after 10 minutes compared to kids in dim-light conditions (Gooley et al., 2011), while the circadian timing of adults has been delayed by prolonged (6.5 hours pre-bedtime) exposure to dim lighting (~12 lux) (Shanahan, Zeitzer, & Czeisler, 2007). Pulses of light may

have the same effect as constant light. Adults exposed to cyclic periods of 15 minutes of extremely bright light (~9,500 lux) followed by 60 minutes of very dim light over the course of 6.5 hours experienced similar phase shifts to adults exposed to 9,500 lux of light for a constant, 6.5-hour period (Zeitzer et al., 2000).

Laboratory vs. At-Home Lighting Conditions

To date, most sleep studies have been conducted in laboratory settings. In the laboratory, lighting is administered at a constant lux, the angle of light on subjects' eyes is maintained, and subjects are still for prolonged periods. These studies provide valuable information on how controlled light can affect sleep; however, lighting conditions in the real world are quite different. At home, the light we are exposed to varies constantly as we move, even if our movements are small. When we walk from room to room, lighting conditions change, and when we go to the mailbox or take out the garbage, we're exposed to brief, bright outdoor lighting before we return to the dimmer lights of our homes.

Laboratory studies do not mimic these frequent changes in environmental lighting. Furthermore, they do not tend to focus on the narrow range of light (<1 to 200 lux) found in typical homes in the evening hours. The ecological validity, or extent to which laboratory light studies can be generalized to true-life environmental lighting conditions, has not been determined. As such, it is not well understood how environmental light affects sleep, or whether environmental lighting exerts acute shifts (i.e., delaying the ability to fall asleep) or longer-lasting shifts (i.e., decreasing sleep quality and total sleep time). Despite the importance of early-childhood sleep and the high sensitivity of children to light-induced phase shifting, few light-and-sleep studies have examined preschool-aged children.

Our study aims to fill these gaps by examining how environmental lighting, when sustained in the evening, affects the sleep of preschool-aged children with behavioral sleep problems. Specifically, our aims are as follows:

1. Primarily, we aim to determine whether lighting intensity in the 15-minute and 2-hour periods before bedtime predicted sleep onset latency (SOL), total sleep time (TST), and sleep efficiency (SE) in our study subjects.
2. If, in our primary analysis, lighting intensity predicts SOL, TST, and/or SE, we will conduct a secondary, exploratory analysis to determine 1) the “critical period”, or time point up to which pre-bedtime light exposure had an effect on the sleep outcome and 2) the extent (i.e., added minutes to SOL, minutes lost from TST, or percent loss of SE) to which light sustained during the critical period affected subsequent sleep outcomes.

Methods

Study Setting and Design

The Sleep Health in Preschoolers (SHIP) study was developed by the Seattle Children’s Research Institute. The SHIP study was reviewed and approved by the Seattle Children’s Research Institute Institutional Review Board. SHIP is a longitudinal study to test the effects of an at-home sleep intervention. At baseline, SHIP subjects were randomized to control and intervention arms, and data were collected for both groups. There was no difference in collection for the actigraphy or survey data between arms, so we combined both arms’ baseline data for the present analysis. Although follow-up data collection is ongoing as of 2018, the present analysis is limited to baseline data, which were collected between fall 2014 and summer 2017.

Recruitment

Children were recruited via advertisements in print and social media, and flyers in pediatric clinics, schools, childcares, community centers, and churches.

Child Sleep Habits Questionnaire

Interested parents completed the Child Sleep Habits Questionnaire (CSHQ) online, at a link provided on the flyer, or contacted the study team by phone. The CSHQ consists of 56 questions to detect symptoms of behavioral sleep disorders in the International Classification of Sleep Disorders Diagnostic and Classification Manual (AASM, 2005). Although it was developed for 4-to-10 year-olds (Owens, Spirito, & McGuinn, 2000), the CSHQ has been tested and validated for use in 2-to-5 year-olds (Goodline-Jones, Sitnick, Tange, Liu, & Anders, 2008). A cut-off score of 41 maximizes sensitivity (ability to detect true positives) and specificity (ability to detect true negatives) for behavioral sleep problems (Owens et al., 2000), while a cutoff of 50 increases specificity at the cost of some sensitivity.

Sample

Children were eligible for inclusion in the SHIP study if their CSHQ score was ≥ 50 or if they scored ≥ 41 and slept ≤ 9 hours, on average, each day. Children were excluded from study consideration if they had sleep-disordered breathing or cancer, diabetes, or other serious comorbid conditions that could affect sleep, or if they were on stimulant medication (Galland, Tripp, & Taylor, 2010). Because the SHIP study is testing a behavioral intervention, children with significant developmental disabilities that could interfere with intervention's fidelity, including pervasive developmental disorder and autism, were excluded. The parent data-collection materials used for SHIP were only available in English, so children whose parents did not read in English were also excluded. If parents returned CSHQs for more than one child, the child with the lower score was excluded from study participation.

The number of subjects needed to detect an intervention effect in the SHIP study, estimated by SHIP researchers, was 500. Of the 897 children screened, 459 met eligibility criteria and obtained parental consent. Twenty-six children withdrew their children prior to randomization, and 433 children were

randomized to the control or intervention arm. Twelve of these children withdrew prior to the baseline home visit; in the end, 424 children underwent baseline data collection.

Consent and Incentives

At the baseline home visit, study personnel discussed protocol. Parents had the opportunity to ask questions before they gave written consent for their children to participate. Participating families were paid up to \$80 for baseline data collection: \$20 each for completing the in-home survey, sleep diary, and actigraphy, and an additional \$20 for returning all three components to the SHIP team on time.

Data Collection

The AW-2 actigraphy watch (Actiwatch™, Phillips Respironics, Bend, OR) was used to collect data. The AW-2 collects lux, movement, and sleep data in minute-by-minute epochs.

At the baseline home visit, study personnel demonstrated use of the actigraphy watches, and parents were also given written and illustrated instructions. Parents were directed to attach the watch to the non-dominant wrist of their child for seven consecutive days. If handedness had not been established, children wore it on the left wrist. To prevent children from removing them, the watches were attached using hospital bands, rather than typical watch bands. The AW-2 did not need to be removed during baths and showers, although it could not be worn while swimming.

Study personnel provided parents with sleep diaries. Parents were asked to complete the sleep diary for each of the 7 days of actigraphy data collection. In the diaries, parents completed questionnaires about their child's sleep, including bedtime. Bedtime was defined as the time the parent said "good-night" to their child and expected their child to start trying to fall asleep for the night. During the home visit, parents completed additional surveys on their child's habitual sleep tendencies.

Data Cleaning

Each time a parent returned a watch, study personnel downloaded the actigraphy data and used Actiware-Sleep software (Version 5.0, Mini-Mitter & Respironics, Bend, OR) to code sleep and wake patterns. In the diaries, parents noted any periods where the watch was off-wrist; during these stretches, watch data was coded as “missing.” If diary data were unclear, study personnel contacted parents within 2 days of data collection. If confusion remained, or if the subject contributed less than 8 hours of valid actigraphy data in a given day, the entire 24 hours of actigraphy recording was excluded. Some children did not achieve 7 days of actigraphy recording due to non-compliance or missing data. If there were under 4 days of actigraphy recording for a subject, that subject’s data was removed from the study.

At each minute, the actigraphy watch coded either “S” for sleep or “W” for wake, based on the child’s movements. We used data from the actigraphy watches and sleep diaries to create our sleep variables.

- *Sleep onset* began at the first minute of the first 10-minute stretch of consistent sleep epochs. (One wake epoch was allowed between minutes 1 and 10.)
- *Sleep onset latency (SOL)* was the time between bedtime and sleep onset.
- *Sleep offset* began at the first minute of the first 10-minute stretch of consistent wake epochs; again, one sleep epoch was allowed.
- *Total time in bed* was the total time between sleep onset at night and permanent sleep offset in the morning.
- *Total sleep time (TST)* was the total number of “S” epochs during total time in bed.
- *Sleep efficiency (SE)* was the percent of time sleeping (“S” epochs) given total time in bed.

At each minute, the actigraphy watch recorded mean lux. We used the upper quartile (75th percentile) of lux values to indicate environmental lighting in a given window of time. For example, in the 2 hours before bedtime, actigraphy recorded 120 mean lux values (one value per minute.) The upper quartile of lux exposure during this two-hour stretch, then, was the 30th-largest out of all 120 values. Means and maximums can be misleading due to outliers, and values above the upper quartile don't always represent sustained lighting conditions (that is, they represent only brief exposure). For our purposes, the upper quartile was the best indicator of the brightest sustained light a subject was exposed to.

After initial data cleaning, data were moved to STATA[®] Statistics/Data Analysis version 14.2 for analysis. Our dataset started with 2,610 nights of data from 431 subjects. For 227 nights of data, SOL was 0 due to the child falling asleep outside of the bedroom before bedtime; these nights were removed. We eliminated 14 nights of data where the child slept less than 60 minutes and one night with unrealistically high (>90,000) levels of sustained lux at 15 minutes and 2 hours pre-bedtime. We were left with 2,368 nights of data from 411 children.

Statistical Models

To assess the relationship between 75th percentile lighting intensity in the 15-minute and 2-hour periods before bedtime and subsequent sleep outcomes, we ran six multilevel models: lighting in the 15 minutes pre-bedtime as a predictor of SOL, TST, and SE; and lighting in the 2 hours pre-bedtime for each of these 3 sleep outcomes. We used multilevel modeling to account for the non-independence of our observations; each child contributed a mean of 5.8 nights of data. Therefore, child was the random-effects variable in our model, while our covariates were fixed effects. In all analyses, we considered low p-values (<0.05) to indicate statistical significance, while narrow confidence intervals and sufficient observations were indicators of statistical validity.

In each model, we included the following covariates: month of data collection, bedtime, child age, and child sex. Seasonality can confound the relationship between sleep and light. Humans stay up later, sleep less, and are exposed to more light as days get longer (Allebrandt et al., 2014). Age, too, is a confounder. As children get older, they become less sensitive to the circadian-shifting effects of light and require less sleep (Bathroy & Tomopoulos, 2017). Bedtime, which varied by day of data collection for each child, was also included as a covariate, as it is associated with the sleep outcomes we tested (Asarnow, McGlinchey, & Harvey, 2014) and with light exposure (i.e., children who go to bed while it is still light out may be exposed to brighter light before bedtime).

For part 2 of our analysis, we conducted an exploratory analysis to determine 1) the time point up to which pre-bedtime light exposure significantly predicted SOL, and 2) minutes added to SOL for each increasing category of lux exposure. We ran multilevel, multivariate models (as in part 1 of analysis) with lux levels at 30, 35, 45, 60, 75, 90, and 105 minutes pre-bedtime as predictors.

We binned 75th percentile lux-exposure values into ranges: <1, 1 to <10, 10 to <20, 20 to <30, 30 to <50, 50 to <75, and ≥ 75 lux. We chose ranges of different widths for several reasons. In our lux data (see Figure 1 for a histogram), there were few values at 30 and 15 minutes pre-bedtime that were over 50 lux; thus, because our data was right-skewed, larger values were grouped into wider bins. Furthermore, the relationship between light exposure and melatonin suppression is not linear; a constant increase in lux does not result in a constant increase in melatonin suppression. For example, a predictive model has estimated that exposure to 10,000 lux does not suppress melatonin much more than 1,000 lux, whereas the increase in suppression associated with a move from 50 lux to 100 lux is large (Zeitzer et al., 2000). Finally, the lux ranges we created are approximations of indoor situations. For example, 1-10 lux approximates being near a nightlight in a dark bedroom, while 10-20 lux approximates a dark bedroom with a door open to a lit hallway. We tested home lighting situations using a lux meter

(Digital Illuminance/Light Meter LX1330B; Dr. Meter, Union City, CA) to confirm that the lux levels of each situation fell into the appropriate range.

Results

Sleep Characteristics

The 411 subjects contributed 2,368 nights of data. Mean bedtime for all nights was 20:24 (SD = 53 minutes), mean SOL was 44 minutes (SD = 35 minutes), mean TST was 9 hours and 44 minutes (SD = 1.1 hours), and mean SE was 85% (SD = 7%). For each night, we calculated the 75th percentile of lux readings in the 2 hours, 30 minutes, and 15 minutes pre-bedtime. The median 75th percentile reading was 10 lux in the 2 hours pre-bedtime, 5 lux at 30 minutes pre-bedtime, and 3 lux at 15 minutes pre-bedtime.

Lighting Intensity as a Predictor of SOL, TST, and SE

The first aim of our analysis was to determine whether lighting intensity in the 15-minute and 2-hour periods before bedtime predicted subsequent SOL, TST, and SE. When we fitted multilevel models, lux levels in the 2 hours pre-bedtime did not significantly predict SOL, TST, or SE (see Table 3 for coefficients and p-values). Lux levels in the 15 minutes before bedtime did not predict TST or SE; however, it predicted SOL (coefficient = 0.005, 95% CI = 0.002 to 0.008, $p < 0.01$). This result could indicate that the light sustained by preschool-aged children prior to bedtime, if too bright, results in a phase delay that is relatively mild: it postpones sleep onset but does not affect TST or SE.

Light Timing as a Predictor of SOL

The second aim of our analysis was to explore the relationship between pre-bedtime light and SOL. Lux levels predicted sleep onset at 30 minutes (coefficient = 0.012, 95% CI = 0.005 to 0.019, $p < 0.01$) and 35 minutes (coefficient = 0.013, 95% CI = 0.005 to 0.021, $p < 0.01$) pre-bedtime, but not 40 minutes pre-bedtime. See Table 4 for extended results. This finding could indicate that the critical period during which too-bright light could delay SOL in preschool-aged children is between 35 and 40 minutes pre-bedtime, whereas environmental light sustained at ≥ 40 minutes pre-bedtime does not affect SOL in this population.

Lighting Intensity and Added Minutes to SOL

At 15 minutes pre-bedtime, compared to 75th percentile readings that were < 1 lux, 1 to < 10 lux added 6 minutes to SOL (95% CI = 3 to 9, $p < 0.01$), 10 to < 20 lux added 6 minutes (2 to 3, $p < 0.01$), 20 to < 30 lux added 9 minutes (3 to 16, $p < 0.01$), 30 to < 50 lux added 12 minutes (5 to 19, $p < 0.01$), 50 to < 75 lux added 11 minutes (1 to 9, $p < 0.05$), and 75 lux or more added 15 minutes (7 to 24, $p < 0.01$). See Table 5 for all results from this model.

At 30 minutes pre-bedtime, compared to 75th percentile readings that were < 1 lux, 1 to < 10 lux added 6 minutes to SOL (95% CI = 3 to 9, $p < 0.001$), 10 to < 20 lux added 6 minutes (95% CI = 2 to 11, $p < 0.05$), 20 to < 30 lux added 9 minutes (95% CI = 3 to 15, $p < 0.01$), 30 to < 50 lux added 11 minutes (95% CI = 4-18, $p < 0.01$), and 75 lux or more added 15 minutes (7 to 24, $p < 0.01$). In our data, 50 to < 75 lux was not significantly associated with SOL.

These findings could indicate that, in the 30 minutes pre-bedtime, there is a positive correlation between sustained environmental light levels and time to sleep onset in preschool-aged children, although this effect may be more pronounced 15 minutes pre-bedtime than it is 30 minutes pre-bedtime.

Figure 1: Participants' observed 75th percentile lux in the 15 minutes, 30 minutes, and 2 hours pre-bedtime

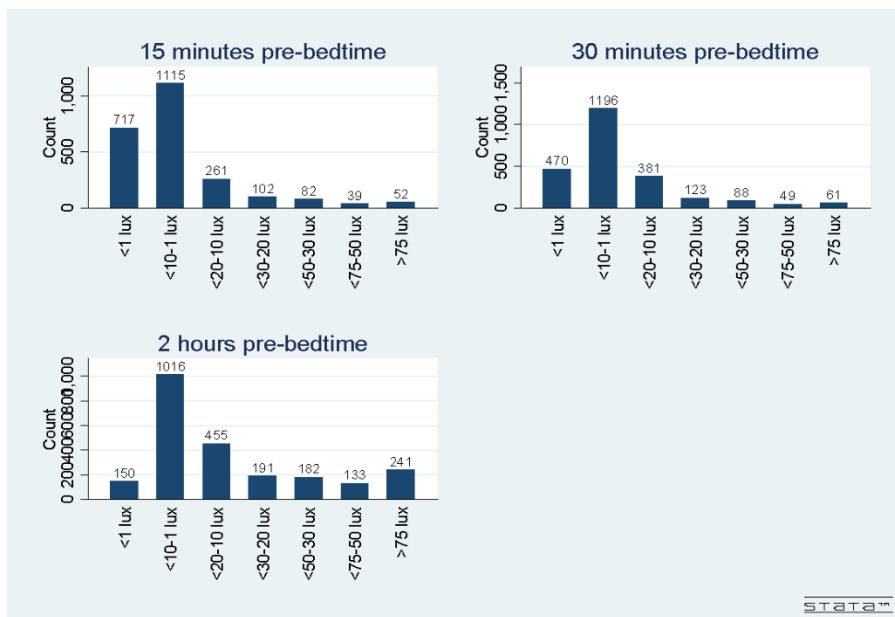


Table 1: Characteristics of study participants (n=411)

Race/ethnicity (%)¹	
American Indian or Alaska Native	2.7%
Asian	16.5%
Black or African American	3.2%
Hispanic or Latino	9.4%
Pacific Islander, including Native Hawaiian	1.7%
White	88.0%
Prefer not to answer/missing	2.9%
Sex (% female)	47.0%
Age (mean months) (SD) (min to max)	44.5 (10.3) (30 to 71)
Shares bedroom (% yes)	69.2%

¹Due to the multiple response format, total for race categories exceeds 100%.

Table 2: Sleep and light exposure characteristics of nights (n=2,368)

Nights	Mean (SD)	Median (IQR)
Nights of actigraphy data	5.8 (1.4)	4 (2.0 to 6.0)
Bedtime	20:24 (53 min)	20:20 (19:48 to 21:00)
Sleep		
Onset latency (minutes)	43.7 (34.9)	35.0 (17.0 to 61.0)
Total sleep time (hours)	9.7 (1.1)	9.8 (9.1 to 10.5)
Efficiency ¹ (%)	84.5 (6.7)	85.5 (81.5 to 89.0)
Upper quartile lux exposure		
2 hrs before bedtime	64.8 (271.4)	10.3 (4.7 to 28.1)
30 mins before bedtime	4.9 (178.9)	4.9 (1.5 to 11.8)
15 mins before bedtime	22.0 (440.7)	3.2 (0.6 to 8.9)

¹Total sleep time/time in bed × 100%

Table 3: Results of multi-level regression model: SOL, TST, and SE as predicted by 75th percentile lux in the 15 minutes and 2 hours pre-bedtime¹

Lux in the 15 minutes pre-bedtime		
	Coefficient (95% CI)	p-value
SOL	0.005(0.002 to 0.008)	< 0.01
TST	0.004(-0.005 to 0.013)	0.41
SE	0.0(0.00 to 0.001)	0.53
Lux in the 2 hours pre-bedtime		
	Coefficient (95% CI)	p-value
SOL	0.0(-0.005 to 0.005)	0.92
TST	-0.01(-0.006 to 0.004)	0.59
SE	0.0(-0.001 to 0.001)	0.67

¹Models corrected for child sex, child age, bedtime, habitually sharing a bedroom, and month of data collection; child was the random effect in the model, to account for the non-independence of multiple nights of data from the same child.

Table 4: Timing of 75th percentile lux exposure as a predictor of SOL¹

	Coefficient (95% CI)	p-value
15 minutes pre-bedtime	0.005 (0.002 to 0.008)	< 0.001
30 minutes pre-bedtime	0.012 (0.005 to 0.019)	< 0.01
35 minutes pre-bedtime	0.013 (0.005 to 0.021)	< 0.01
40 minutes pre-bedtime	0.001 (0.00 to 0.018)	0.10

¹Models corrected for child sex, child age, bedtime, habitually sharing a bedroom, and month of data collection; child was the random effect in the model, to account for the non-independence of multiple nights of data from the same child.

Table 5: Added minutes to SOL by 75th percentile lux category (fitted values), 15 minutes and 30 minutes pre-bedtime¹

Lux range	Example lighting condition at this range	15 minutes pre-bedtime: added minutes SOL as compared to <1 lux (95% CI), p-value	30 minutes pre-bedtime: added minutes SOL as compared to <1 lux (95% CI), p-value
<1 lux	Dark room (no lights, closed shades)	-	-
1 to <10 lux	Dark room, child is within 2 feet of nightlight	5.5 (2.5 to 8.6), p <0.001	4.1 (0.7 to 7.5), p <0.05
10 to <20 lux	Room with dim to moderately bright lamp (several feet from child), or dark room with door open to brightly-lit hallway	6.2 (1.7 to 10.8), p <0.01	5.1 (0.7 to 9.6), p <0.05
20 to <30 lux	Room with moderately bright lamp (child 1-2 feet from lamp)	9.4 (2.8 to 15.9), p <0.01	8.8 (2.5 to 15.1), p <0.01
30 to <50 lux	Room with built-in overhead lighting on (standard 30-100 watt bulbs)	12.0 (4.9 to 19.2), p <0.01	11.1 (3.9 to 18.2), p <0.01
50 to <75 lux	Room with multiple light sources (overhead lighting is on plus child is 1-2 feet from lamp)	10.5 (0.73 to 20.3), p <0.05	0.9 (-8.1 to 10.0), p = 0.84
≥75 lux	Child is directly under very bright reading light, or child is by window in afternoon/evening light	15.4 (6.5 to 24.2), p <0.01	15.2 (6.5 to 23.9), p <0.01

¹Models corrected for child sex, child age, bedtime, habitually sharing a bedroom, and month of data collection; child was the random effect in the model, to account for the non-independence of multiple nights of data from the same child.

Figure 2: Minutes added to SOL associated with upper quartile of lux exposure during the 15 minutes pre-bedtime

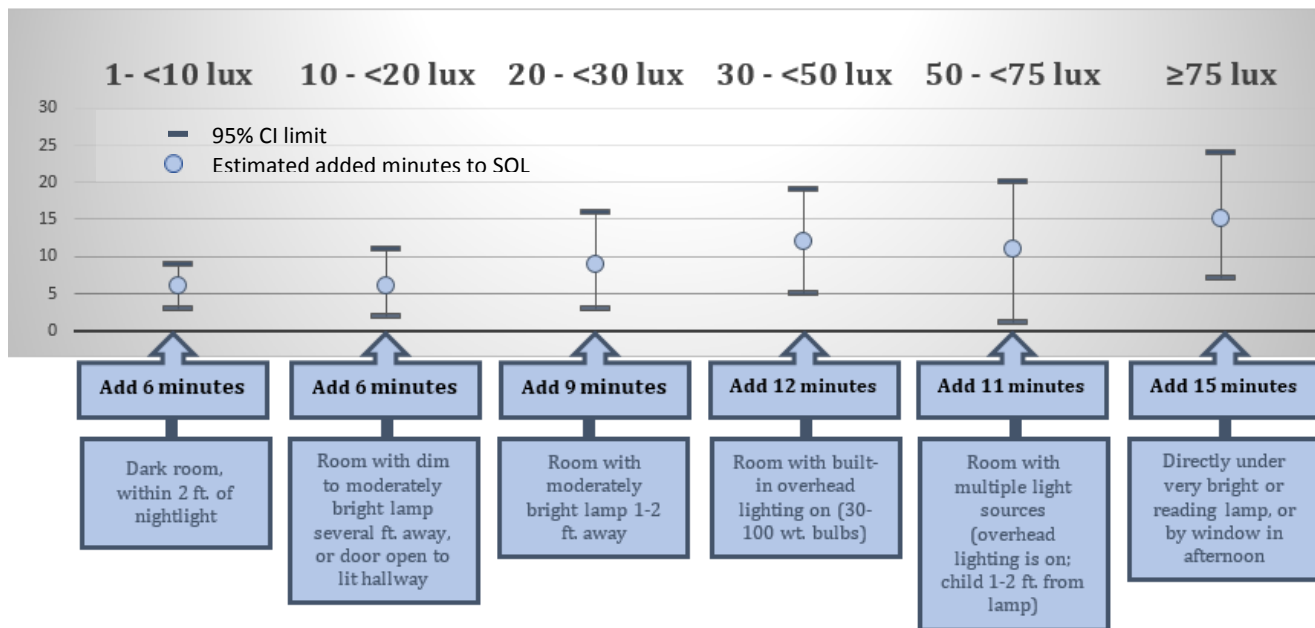
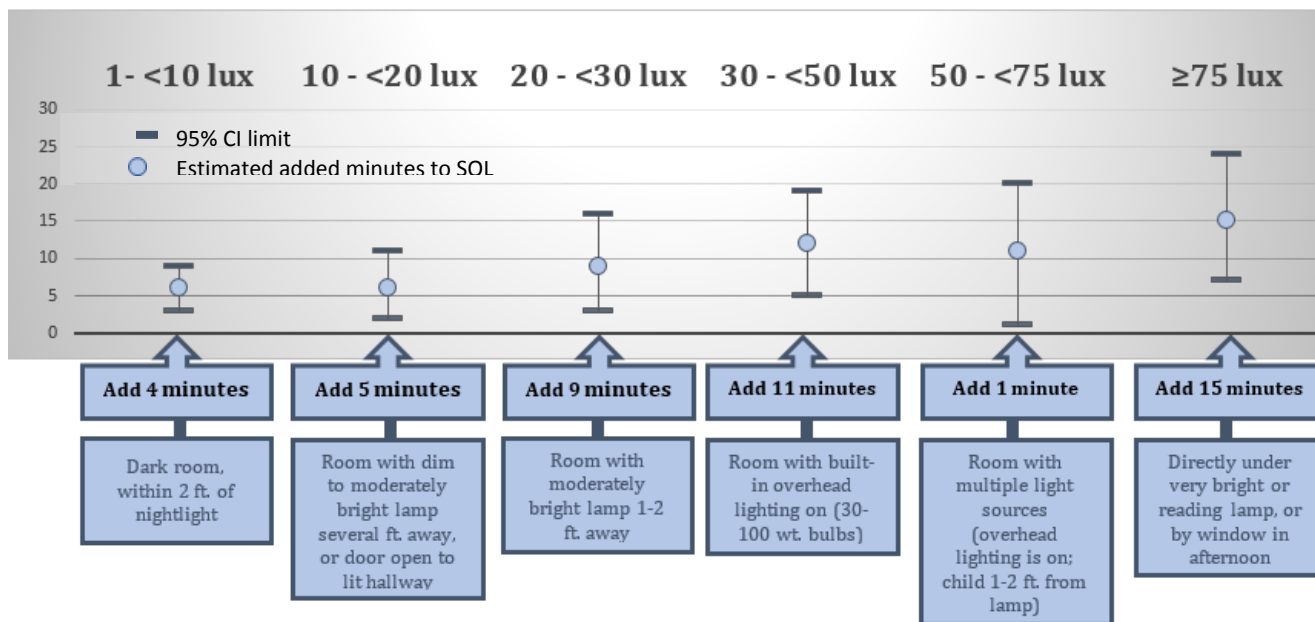


Figure 3: Minutes added to SOL associated with upper quartile of lux exposure during the 15 minutes pre-bedtime



Discussion

This study was one of the first to examine the relationship between environmental lighting conditions and sleep in young children. We found that slight differences in environmental lighting levels are associated with longer SOL. Phase shifts caused by dim environmental light in the evening (<1 to 200 lux) may be minimal, as SOL was delayed by light sustained up to, but not beyond, 35 minutes before habitual bedtime. Furthermore, light exposure in the 2 hours before bedtime did not predict TST or SE, which suggests that the melatonin suppression induced by environmental light did not last beyond sleep onset.

This study adds to the results of laboratory experiments that have measured melatonin suppression in response to light exposure. Because circadian response to light is dependent upon the timing, duration, and intensity of light exposure (Akacem et al., 2016b), the inconsistent methodologies of published sleep studies (i.e., pulses of light vs. constant light, intensity and timing of light, characteristics of subjects) make it difficult to compare findings between studies. With this caveat, we compare our results to other studies below.

Timing of Light Exposure

In our study, environmental lux levels up to 35 minutes before habitual bedtime prolonged SOL. Other studies have suggested that the critical period during which lighting exposure pre-bedtime can affect sleep is longer than what we observed. However, these studies have tended to test brighter lighting levels than what our subjects were exposed to, on average. In one laboratory study of preschool-aged children, for example, the melatonin levels of kids who sustained 60 minutes of bright light during the hour before habitual bedtime was still significantly lower than the control group 50 minutes after light exposure ended (Akacem et al., 2016b). Light has a dose-response effect on sleep,

with lower levels resulting in less melatonin suppression (Zeitser, 2000). This may explain why, in Akacem's study, bright light in the hour before bed had a lasting suppression effect, while the dimmer environmental light the children in our study were exposed to did not appear to have a prolonged effect.

Intensity of Light Exposure

Experiments on melatonin suppression and phase shifting have not produced, with consistency, a minimal value of evening lux exposure at which sleep can be delayed in humans. This value varies by person and by day, as it is determined by multiple factors, such as age and a person's exposure to lighting over the previous 24 hours (Akacem et al., 2016b). Laboratory experiments have reported that exposure to as little as 12 lux of prolonged (6.5 hours) light prior to bedtime could result in phase delay; this is about the intensity of candlelight (Shanahan et al., 1997; Wright & Czeisler, 2002). A near-dark 1.5 lux was sufficient to entrain the circadian clock of adults who had been living in very dark conditions for a prolonged time (Duffy & Wright, 2005). However, this result is not consistent across other settings: another laboratory study of adults suggested that prolonged exposure to dim (<15 lux) light during biological night did not alter sleep timing, while melatonin suppression began at about 80 lux of prolonged exposure, maximal melatonin suppression occurred at about 200 lux, and maximum phase delay occurred at about 500 lux (Zeitser et al., 2000). As we expected, based on our knowledge of young children's heightened sensitivity to the effects of light (Akacem et al., 2016b), our findings indicate that children may experience phase shifts at lower levels of lux exposure than older children and adults. We observed that kids who, in the 15 minutes before bedtime, sustained just 1 to 10 lux of environmental lighting (upper quartile of exposure) took a significantly longer time to fall into sleep as compared to kids who sustained under 1 lux. This may indicate that, in preschool-aged children, the phase-shifting effects of environmental light can occur at relatively dim light levels.

Duration of Light Exposure

Preschool-aged kids who were exposed to very bright (~1,000 lux) light for 10 minutes in the 1 hour before habitual bedtime exhibited about 88% (+/-10%) melatonin suppression in a laboratory-based study (Akacem et al., 2016b). On average, subjects in our study were exposed to much dimmer light (median 3 to 5 lux in the 15 minutes and 30 minutes pre-bedtime, respectively), yet our data suggest that even brief (15 to 35 minutes pre-bedtime) exposure to these environmental lux levels may be enough to delay sleep onset in preschool-aged children.

Total Sleep Time and Sleep Efficiency

Zeitzer et al. estimate that it would take about 6.5 hours of constant exposure to very bright (~10,000 lux) light to induce a 3-hour phase shift in adults, and that just 1/10th of this amount (80 to 160 lux) can result in a half-maximal shift of 1.5 hours (Zeitzer et al., 2000). Since the environmental light our subjects were exposed to was, on average, dimmer (and we examined shorter time periods of exposure), it would make sense that the effects of light on phase delay were acute. In our study, we observed no association between lighting levels and subsequent TST or SE. After sleep onset, there was no difference in the sleep quality or quantity of kids who sustained higher versus lower lighting levels prior to bedtime. This may indicate that the levels of environmental light kids in our sample were exposed to pre-bedtime were too dim to induce major phase shifts. In a crossover study, Burgess & Molina (2014) found that adults who were exposed to bright at-home lighting (~65 lux) in the four hours before usual bedtime went to bed slightly later than when they were exposed to dim (~3 lux) levels, but that pre-bedtime light did not affect TST or SE. This corroborates our findings, as it may indicate that typical environmental light levels can cause acute phase shifts, but are not strong enough to sustain a lasting phase shift.

Strengths and Limitations

Our study had several strengths. It is one of the only studies that has been conducted on light and sleep in preschool-aged children. Among the published studies on light and sleep, this is one of the few to use ecologically valid data; because most light studies take place in highly-controlled laboratories, they may not apply to real-life conditions. While laboratory studies tend to test exposure to very high versus very low amounts of light (Duffy & Wright, 2005), our study tested more levels of light in home environments. Data were also collected from a demographically-diverse sample of children, and our large dataset (n=411 children and 2,368 nights of data) resulted in high statistical validity.

Our study had weaknesses, too. Our analysis was not based on randomized, controlled data, so we cannot conclude causal effects. Levels of environmental light exposure were not randomly allocated to subjects, so patterns of pre-bedtime light exposure are likely correlated with other pre-bedtime behaviors. For example, we did not capture data on parenting style, nor did we take pre-bedtime physical activity into account. Our multi-level models corrected for confounding variables, but we did not account for previous light exposure during the day, which may determine the extent to which evening light suppresses melatonin (Duffy & Wright, 2005; Scheuermaier et al., 2010). Our results could be biased by unmeasured confounders, such as ambient noise. Ours was not a laboratory-controlled study, so fidelity (i.e., leaving the actigraphy wrist uncovered by clothing or blankets) could not be monitored, and we cannot be sure that the lux recorded by the actigraphy watches matched the light absorbed by participants' eyes. Because our analysis was designed to observe and explore data rather than make future predictions, findings may not apply to children outside of our study. In the models in which we treated lux ranges as a categorical variable, at least one range (50 to 75 lux) was underpowered to detect a significant effect on SOL. Finally, the actigraphy watches we used to collect light data did not measure light wavelength. Blue light, the short-wavelength light emitted by

smartphones and computers, may have a stronger melatonin-suppressing effect than other wavelengths of light (Wright & Lack, 2011). However, one study found that phase shift did not differ between subjects who did and did not wear blue light-blocking goggles indoors in the evening (Burgess & Molina, 2014). Furthermore, in-home lighting is usually polychromatic (Santhi et al., 2012).

In our data, the distribution of lux levels pre-bedtime was right-skewed; thus, future research should make a concerted effort to collect more observations in the moderate-to-bright at-home light range (e.g., ~30 to 200 lux). Future in-home studies should also assess to what extent lux levels sustained over the day can confound the effect of evening light on subsequent sleep and determine how light exposure during sleep can affect sleep quality.

Study Implications

These results corroborate existing clinical recommendations to achieve sleep onset in problem preschool-aged sleepers by limiting bright light in the evening. Our suggestion that even slight differences in the relatively narrow range of at-home lighting (<1 to 200 lux) up to 35 minutes before habitual bedtime can make a significant impact the SOL of preschool-aged children, but that it may not be necessary for parents to restrict lighting >35 minutes before habitual bedtime, adds new knowledge to the body of sleep research.

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