Impact of Noise and Light Pollution on Sleep in Preschoolers

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Abstract

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Sleep is an important predictor of health and wellbeing. In preschool aged children, poor sleep is associated with poorer emotional regulation, future attentional problems, impaired growth, and higher risk of injury. Sleep problems in preschool aged children are also associated with sleep problems in parents, which in turn impact parent functioning and wellbeing. Noise pollution and light pollution are associated with altered circadian rhythms through increased stress hormone production and depressed melatonin; noise also interrupts sleep directly and causes annoyance, further impacting parental functioning. In this study, we examined the association of environmental noise and light with sleep outcomes in a sample of preschool aged children with sleep problems. Linear regression was performed. We found that levels of artificial light at night >16.76 nanoWatts/cm^2/sr*10^9 were associated with increased sleep disruption at baseline: 1.29 (95% CI: 0.02, 2.51) additional percentage points wake after sleep onset compared to artificial light at night ≤9.52 nanoWatts/cm^2/sr*10^9. This finding supports the need for management of light pollution to limit impacts on sleep. No other sleep outcomes at baseline or three months follow-up were associated with artificial light at night or transportation noise.

Introduction

Importance of sleep

Sleep is vital to human health and wellbeing. Restricted or insufficient sleep leads to a host of negative health consequences for adults, including neurobehavioral deficits, compromised metabolic, immune, and hormone function, decreased cardiovascular health, increased risk of depression, and increased mortality risk.(1-16)

Short sleep duration is associated with poorer emotional regulation, impaired growth, and higher risk of injuries in children 0-4 years old.(17) Sleep-deprivation in both adults and children lowers the psychological threshold for stress, and may elevate subjective stress, anxiety and anger.(18-20) In preschool children, some elements of sleep (insomnia, sleep problems, and bedtime) are associated with aggressiveness; in one study of children 48 months of age, those with sleep duration <9.44 hours had 1.81 times greater odds of being frequently aggressive.(19, 20) Internalizing problems are also linked to sleep: 24 month old children who slept less than 12.5 hours per day had 1.47 times greater odds of anxiety or depressive symptoms at age 3, when compared to those who slept more than 13.5 hours.(19) Neurobehavioral and cognitive function are impaired by sleep deprivation in both children and adults; sleep deprivation in children may also impact healthy neurodevelopment.(21-23)

Sleep quality influences academic success: in school aged children, insufficient sleep manifests in ADHD-like symptoms, such as poor attention and behavior regulation.(21, 23, 24) Daytime sleepiness likewise impacts their ability to function in the classroom.(23) In a meta-analysis of cross-sectional studies, sleep quality ($r = 0.10$), sleep duration ($r = 0.07$), and sleepiness ($r = -0.13$) were associated with school performance, with larger effect sizes in younger participants.(25) In one study where 4th and 6th grade students were randomly assigned to go to

sleep one hour earlier or later, this modest change in sleep duration led to substantially decreased performance on various neurobehavioral functioning tests that correlate with classroom behaviors and achievement test scores.(26) In experimental studies of children 6-16 years old, sleep-deprived children fell asleep more easily during the day and were less attentive; in these studies, there was also evidence of impaired higher level cognitive skills like creativity, reasoning and executive function.(23) Bedtimes and sleep problems are positively but weakly (r = 0.10) associated with attention problems in preschool aged children.(27) Evidence that sleep problems have lasting attentional impacts is more worrisome, however: children with sleep problems that occur "often" between the ages of 2 and 4 years had substantially elevated risk of persistent attention problems at ages 5 and 14: 3.84 times greater odds for boys and 4.42 times greater odds for girls.(19, 27) In short, sleep is important in the lives of children, with potentially long-lasting health and academic impacts.

Sleep problems in children negatively impact the sleep, physical and mental health of parents.(28-30) An Australian study of child sleep problems and parental wellbeing provides weak evidence of association between sleep problems in preschoolers and paternal mental, maternal mental and maternal general health (p = 0.06, 0.06 and 0.05, respectively). (29)A Swedish study found that mothers of children with frequent night wakings had elevated rates of sleep problems, while fathers had elevated rates of sleep problems when their children had sleep disordered breathing or difficulty falling asleep.(31) Additional evidence suggests that the impact of child sleep problems extends to parent functioning. One study found that parent daytime sleepiness was associated with child sleep disorders, while another found that child sleep quality predicted the quality of maternal sleep, which in turn predicted maternal mood, stress, and

fatigue.(32, 33) It is plausible that child sleep problems on parents may diminish parents' capacity for positive parenting and behavioral intervention.

Environmental impacts

Environmental and neighborhood characteristics play a role in sleep problems, although the majority of evidence for associations between sleep and environmental exposure to light and noise is observational.(34)

Sleep is governed by the circadian system, which synchronizes sleep with the 24-hour day through cycles of hormone secretion and blood pressure regulation.(22) The circadian rhythm is driven by the zeitgebers—external cues for sleep and wakefulness—such as light. Artificial light at night (ALAN) disrupts circadian rhythms, including secretion of melatonin, prolonging sleep latency.(34) ALAN is associated with increased cancer risk and impacted metabolic processes.(34, 35) Circadian rhythms are more strongly suppressed by higher temperature, shorter wavelength (bluer) light; circadian disruption increases with the duration of light exposure and exposure later in the evening.(35) Even exposure to the longest wavelengths of light and intermittent light can induce circadian responses, as can exposure to low light levels (5-10 lux) while sleeping.(36) However, recovery of melatonin after light exposure is rapid (within 15 minutes).(36) Living in areas with outdoor nighttime light is associated with delayed bedtime, delayed waking, decreased sleep duration, increased reports of insufficient sleep, and increased daytime sleepiness in adults, however, effect sizes are small.(37, 38) In one study using BRFSS data, a 10-unit increase in nighttime light (in nW/[cm^2 sr]) was associated with a 5.6 minute decline in self-reported sleep duration and 13.8% higher odds of reporting insufficient sleep in adults.(38) In a logistic regression of outdoor nighttime light (ONL), self-reported bedtime and other factors, ONL explained 13.7% of variance in late bedtime (after midnight) ($p<0.0001$).(37)

Very little research has addressed the impact of light pollution on child sleep. In one field study, light exposure in preschool aged children in the two hours preceding bedtime predicted 13.3% of variance in circadian phase, while bedtime itself predicted 37.3% of variance.(39) The same lab found that one hour of 1000 lux exposure before bedtime induced a 87% suppression of melatonin, which remained attenuated 50 minutes after the bright light exposure, suggesting that preschool aged children may be more sensitive to light exposure than adults.(40) This is supported by another study which found 88% melatonin suppression in 9 year old children but 46% suppression in adults in response to moderately bright light (580 lux).(41)

Sleep is likewise vulnerable to noise, which disrupts and decreases sleep in a dose-response fashion.(42, 43) Nighttime traffic noise, including aircraft and road, is associated with increased sleep disturbance and insomnia symptom in adults: for a 10 db increase in night noise level (L_{night}), the odds ratios of percent highly sleep disturbed were 1.94, 2.13, and 3.06 for aircraft, road and rail noise, respectively.(44, 45) Evidence for the effect of environmental noise in children is sparse and slightly mixed. One study found a small, significant decrease in self-reported sleep quality and an increase in daytime sleepiness associated with increased outdoor noise among 9-12 year olds.(46) Actigraphy-measured sleep quality tended to decrease alongside increasing noise; however, there was an indication of better sleep at the highest outdoor noise levels (>64 dB $L_{Aeq,24h}$). Another found a significant association (OR=1.79) between L_{night} at the children's home's least exposed façade and problems falling asleep or sleeping through the night among 10 year olds.(44) Among children 7-13, increases in maximum sound (L_{max}) were significantly associated with cortisol and other stress hormone excretion in the first half of the night, causing disturbances in circadian rhythm.(47) These studies suggest that environmental noise may negatively impact children's sleep, however, no studies address the impact of

environmental noise on the sleep of preschool aged children. It is worth noting that noise, particularly traffic noise, is spatially correlated with air pollutants such as nitrous oxide.(48) Noise and air pollution are associated with many similar health outcomes and may even act synergistically to cause them.(48) Air pollution may affect sleep through other pathways—obstructive sleep apnea and asthma.(49-51) Few if any studies on traffic noise and sleep control for air pollution.

Noise impacts children through pathways other than sleep disturbance. Noise exposure leads to annoyance, including reactions such as irritation, dissatisfaction, or anger.(52, 53) Annoyance substantially impacts mental and cognitive wellbeing in adults. It is associated with depression and anxiety: prevalence ratios of depression and anxiety for moderate annoyance are 1.2 and 1.4, for extreme annoyance, they are 1.97 and 2.14, compared to no annoyance. Noise exposure reduces cognitive performance and short-term memory performance in adults; in children, annoyance is related to impaired cognitive and executive function.(54, 55) The response to noise is moderated by its features, including intermittentness, intensity and noise type, with intermittent, intense noise being the most disturbing.(55) While the impact of environmental noise on parenting has not been studied directly, depression and reduced executive function are known to negatively impact parenting. Depression is associated with more hostile, negative, and disengaged parenting; in contrast, greater inhibitory control (executive function) is associated with responsive, positive and collaborative parenting behaviors.(56, 57) Therefore, we can reasonably expect that a parent hampered by noise annoyance would have difficulty implementing behavioral interventions. Likewise, the impact of parent sleep deprivation, whether due to noise, light, or their children's sleep, on decision making and cognitive ability is

likely to negatively impact the parent's capacity to successfully implement behavioral interventions.(6)

The majority of sleep problems in children are behavioral.(58) Accordingly, many evidence-based interventions for sleep problems are behavioral, centering on sleep hygiene, including routines and behavior management strategies.(22, 59) The provision of dark, quiet sleeping environments is one feature of good sleep hygiene, as is limiting exposure to light and other external stimulation preceding bedtime.(22) The vast majority of evidence concerning interventions to decrease noise and/or light and improve sleep concern adults in inpatient hospital settings, which are excessively noisy and disruptive.(60) These interventions typically consist of eye mask and earplug provision, and/or measures to implement quiet hours in the ICU; these interventions improve sleep across several dimensions, including night wakings, sleep latency, and overall sleep quality.(60) Interventions that use white noise or music also improve sleep for ICU patients, as well as colicky babies; however, white noise is not known to improve sleep in healthy young adults.(60-62)

This Study

In this study, we investigate the association of environmental light and noise with sleep outcomes including parent-reported and actigraphic sleep duration, sleep onset latency, and sleep disturbance.

Sleep is an important predictor of health and wellbeing. In many populations, including adults and school aged children, noise pollution is known to impact sleep, however little evidence exists for children under the age of seven. Using a national transportation noise dataset, this study seeks to determine whether noise pollution is associated with sleep outcomes in preschool aged

children. Noise may directly interrupt sleep, or it may decrease the quantity and quality of sleep by increasing cortisol levels, thereby disrupting ciradian rhythms (see Figure 1). We hypothesize that noise will be positively associated with sleep disruption and sleep latency, and negatively associated with sleep duration at baseline. We predict the association will be strongest between noise and sleep disruption.

ALAN is associated with poor sleep in adults and with altered circadian rhythms in both adults and children. Existing evidence suggests that children are more sensitive to ALAN than adults, however studies on children are limited to circadian rhythms and hormone levels, and do not address sleep outcomes themselves. As with noise, ALAN impacts sleep through disrupted circadian rhythms. This study aims to determine whether ALAN is associated with sleep outcomes in this population. We hypothesize that ALAN will be positively associated with sleep disruption, sleep latency, and negatively associated with sleep duration, the strongest association being with sleep latency.

Another aim of this study is to understand how and if noise and ALAN impact the effectiveness of behavioral parenting interventions. One effect of noise is annoyance. As shown in Figure 1, parent functioning is affected by annoyance as well as through disruption of parent sleep. If parents are able to substantially decrease the noise or ALAN a child is exposed to, higher levels of noise or ALAN could represent an opportunity for improvement. The intervention group is likely to take steps to decrease exposure. However, environmental noise may be difficult to mitigate depending on the resources of the family, and thus prove to be a stumbling block. Very high levels of noise may be impossible to mitigate and obviate any effect of an intervention. We hypothesize that noise will interact negatively with the intervention; that is, higher noise levels mediated through multiple pathways including through annoyance and parent functioning will

make the intervention less effective. As with noise, the impacts of ALAN on parent sleep may subsequently impact parent wellbeing and parent capacity to implement an intervention. We hypothesize that ALAN will interact negatively with the intervention.

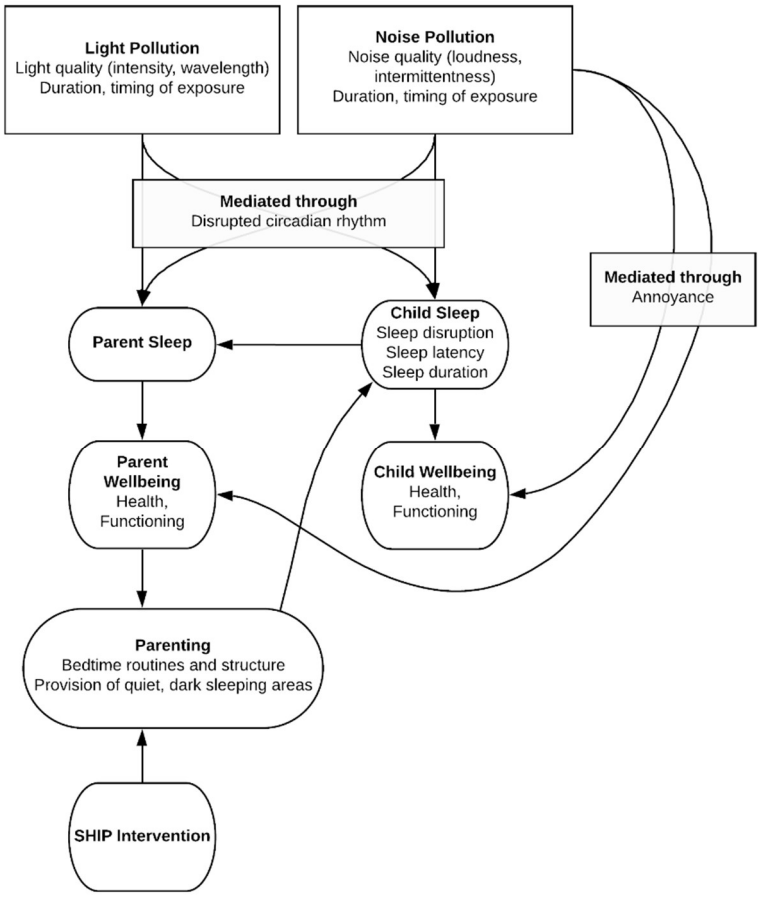

Figure 1. Conceptual model.

Methods

Data Sources

Sleep outcome data and other co-variates are sourced from the Sleep Health in Preschoolers (SHIP) study. SHIP is a randomized behavioral intervention that focuses on establishing routines and boundaries to develop good sleep hygiene. SHIP study subjects are preschoolers (30 to 71 months of age at baseline) with sleep problems, as screened by the Children's Sleep Habits Questionnaire. Of the 343 subjects, 179 were randomized to the intervention arm, 164 to the control arm. Subjects lived in the Puget Sound area of Western Washington state. They were recruited over a period of years and began participation in the study between October 2014 and October 2017.

VIIRS DNB data were used to estimate nighttime light at subjects' residences.(63) The *vcm-orn-ntl* data, which "contains cloud-free average radiance values that have undergone an outlier removal process to filter out fires and other ephemeral lights with background (non-lights) set to zero" were selected, to best approximate artificial light at night (ALAN). The average of annual values for 2015 and 2016 were used, which coincides with majority of the time period under study. These data provide average radiance values in nanoWatts/cm^2/sr *10^9.

Noise data were sourced from the US Department of Transportation Noise Map.(64) The USDOT Noise Map acoustically sums modeled aviation and road noise from the year 2014, producing 24-hour average transportation noise values ranging from 35 to 125 dB.

Variables

Variable name	Definition	Type
WASO(T0)	Wake after sleep onset is the percent of total sleep duration the individual was awake in percentage points at baseline.	Continuous
WASO(T3)	Wake after sleep onset is the percent of total sleep duration the individual was awake in percentage points at three months follow up.	Continuous
SOL(T0)	Sleep onset latency is the time measured in minutes between going to bed and falling asleep at baseline.	Continuous
SOL(T3)	Sleep onset latency in minutes at three months follow up.	Continuous
TST(T0)	Total sleep time is the total sleep duration minus waking minutes at baseline.	Continuous
TST(T3)	Total sleep time is the total sleep duration minus waking minutes at three months follow up.	Continuous
Noise < 40 dB	Transportation noise level (A-weighted 24-hour equivalent) at study subject residence below 40 dB	Categorical
Noise ≥ 40 dB	Transportation noise level (A-weighted 24-hour equivalent) at study subject residence equal to or greater than 40 dB	Categorical
Age	Subject age in months	Continuous
Sex	Subject sex at birth, male = 1, female = 0.	Binary
SES_1	Household annual income > $100,000	Categorical
SES_2	Parent education at or beyond undergraduate degree and household income ≤ $100,000	Categorical
SES_3	Parent education less than an undergraduate degree and household income ≤ $100,000	Categorical
CF	Circadian fragility score	Continuous
Bedtime_1	Bedtime 7:45 PM or earlier	Categorical
Bedtime_2	Bedtime from 7:46 PM to 8:15 PM	Categorical
Bedtime_3	Bedtime 8:16 PM or later	Categorical
Bedtime_none	No regular bedtime	Categorical
CSHS	Child Sleep Hygiene Scale score	Continuous
Light_q1	Light radiance at night in the first quartile (≤9.52 nanoWatts/cm^2/sr)	Categorical
Light_q2	Light radiance at night in the second quartile (9.52 nanoWatts/cm^2/sr <x≤16.76 nanoWatts/cm^2/sr)	Categorical
Light_q34	Light radiance at night in the third and fourth quartiles (above the median) (>16.76 nanoWatts/cm^2/sr)	Categorical
Arm	Intervention arm, control = 0, intervention = 1.	Binary

Table 1. Variables and definitions.

Variables and their definitions are listed in Table 1. The primary outcomes of interest, *wake after sleep onset* (WASO), *sleep onset latency* (SOL), and *total sleep time* (TST), were measured using both actigraphy and sleep diaries. WASO is defined as the percent of total sleep duration

the individual was awake, as detected by actigraphy. SOL is the time in minutes between going to bed as noted in the sleep diary and falling asleep. Total sleep time is the total sleep duration minus waking minutes. These sleep outcomes were measured over the course of one week, at baseline (T0) and at three months follow-up (T3). The nightly values were averaged to find T0 and T3 values. In their sleep diaries, most subjects have seven consecutive nights of recorded data, although they recorded as few as five or as many as ten, sometimes inconsecutively.

Established methods for measuring sleep include polysomnography, videosomnography, actigraphy, sleep diaries and questionnaires. While polysomnography may be considered the gold standard for measuring sleep, including duration and night wakings, it is laboratory-based and therefore limited for understanding sleep of children over long periods in their homes.(42) In contrast, actigraphs, which are accelerometers that detect motion, can be worn for several days and nights at a time unsupervised. In validation studies of actigraphy for pediatric sleep, actigraphy has high sensitivity and low specificity compared to polysomnography, direct observation, and videosomnography—that is, it has a low ability to detect night wakings and may overestimate sleep duration and underestimate sleep fragmentation and sleep latency.(65, 66) Parent report of sleep onset time, end time, and sleep period correlates well with actigraphic measurements in children, but consistently overestimates sleep duration and underestimates night wakings compared to actigraphy.(66-69) It is likely that parent report of sleep in children contains substantial wake time. However, parent report has unique value in that it provides insight into the impact of child sleep problems on the family.

The primary predictors of interest are artificial light at night (ALAN) and transportation noise. To obtain these values, subject addresses were geocoded using the US Census Geocoder. Addresses that were not matched to a geolocation were inspected for errors (e.g., a zip code with

transposed digits inconsistent with the rest of the address) and re-entered to find a geolocation. Remaining addresses were searched in Google Maps and the resulting geolocation was used. A small number of addresses (n=5) were too vague to locate or otherwise impossible to geocode; these were excluded. Light and noise values were assigned to subjects' residence geolocations using ESRI ArcGIS Desktop Suite (v10.4). Geolocations that did not return a noise value (as they were <35 dB, the lower limit provided by the USDOT Noise Map) were assigned the value $\frac{35}{\sqrt{2}}$, as done by Sears et al. in a study using this dataset.(70) Noise was subsequently divided into two categories: <40 dB, ≥40 dB. These cutoffs are based on WHO recommendations (no more than 40 dB at night).(71) ALAN values were separated into first quartile, second quartile, and above median, at ≤9.52, 9.52<x≤16.76, and >16.76 nanoWatts/cm^2/sr *10^9. ALAN values were divided as such as it is plausible that above some threshold of nighttime light, it is impossible to sleep, and additional light has no additional impact.

Socioeconomic status was determined using both family income and completed parent education, which were categorized into three groups: higher income (>$100,000), lower income/higher education (≤$100,000 and at least an undergraduate degree), and finally lower income/lower education (≤$100,000 and associate-level or less). The categories were defined based on their distribution in this dataset and on income and education distributions in the region: in King County, WA the median income is $95,000 and 53% of adults hold at least a Bachelor's degree.(72, 73) Socioeconomic status might be related to light and noise exposure as well as a family's ability to modify the child's sleeping environment. The Child Sleep Hygiene Scale (CSHS), a 23-item scale that measures sleep hygiene in six subscales: physiological, cognitive, emotional, environmental, bedtime routine, and sleep stability, was also included.(74) CSHS score, the presence of a regular bedtime and the bedtime itself were included to control for

behavioral sleep problems that might drown out the effects of noise and light on sleep. Furthermore, exposure to light and noise pollution and subsequent disruption or decrease in sleep may vary based on the child's bedtime. Bedtimes were converted to 24-hour decimal values (e.g., 8:30 PM became 20.50) for ease of use in statistical models. Obvious errors, such as bedtimes recorded as AM, were corrected. Bedtimes were subsequently sorted into tertiles (7:45 pm or earlier, 7:46 PM to 8:15 PM, and 8:16 PM or later); a categorical bedtime variable was created with four categories (the above tertiles and no bedtime). Circadian fragility (the degree to which changes in sleep schedule disrupt functioning and to which the child is flexible in their sleep schedule) was measured using a questionnaire created for this study (see Appendix). This questionnaire used a 1-3 scale for each question; higher scores indicate greater fragility. Mean response values were calculated for participants who completed the questionnaire with one or zero nonresponses out of five.

Analysis

Linear regression was performed for three sleep outcome variables, WASO), SOL, and TST, as measured over one week at baseline. The threshold for statistical significance was set at $\alpha < 0.05$. P values and confidence intervals were calculated using robust standard errors. See Table 2 for regression equations. Light and noise were the predictors of interest. We controlled for child age, sex at birth, socioeconomic status, circadian fragility, and in the case of SOL and TST, bedtime and CSHS score. Regressions examining the interaction of the SHIP intervention and light/noise pollution were performed for SOL and TST at three months, controlling for the sleep outcome at baseline, child age, sex at birth, socioeconomic status, circadian fragility, bedtime and CSHS score. These analyses were completed in *R* version 4.0.0.

Baseline noise models
$WASO(T0) = \beta_0 + \beta_1 X_{Noise \geq 40\ dB}$
$WASO(T0) = \beta_0 + \beta_1 X_{Noise \geq 40\ dB} + \beta_2 X_{Age} + \beta_3 X_{Sex} + \beta_4 X_{SES\_2} + \beta_5 X_{SES\_3} + \beta_6 X_{CF}$
$SOL(T0) = \beta_0 + \beta_1 X_{Noise \geq 40\ dB}$
$SOL(T0) = \beta_0 + \beta_1 X_{Noise \geq 40\ dB} + \beta_2 X_{Age} + \beta_3 X_{Sex} + \beta_4 X_{SES\_2} + \beta_5 X_{SES\_3} + \beta_6 X_{CF} + \beta_7 X_{Bedtime\_none} + \beta_8 X_{Bedtime\_2} + \beta_9 X_{Bedtime\_3} + \beta_{10} X_{CSHS}$
$TST(T0) = \beta_0 + \beta_1 X_{Noise \geq 40\ dB}$
$TST(T0) = \beta_0 + \beta_1 X_{Noise \geq 40\ dB} + \beta_2 X_{Age} + \beta_3 X_{Sex} + \beta_4 X_{SES\_2} + \beta_5 X_{SES\_3} + \beta_6 X_{CF} + \beta_7 X_{Bedtime\_none} + \beta_8 X_{Bedtime\_2} + \beta_9 X_{Bedtime\_3} + \beta_{10} X_{CSHS}$
Baseline light models
$WASO(T0) = \beta_0 + \beta_1 X_{Light\_q2} + \beta_2 X_{Light\_q34}$
$WASO(T0) = \beta_0 + \beta_1 X_{Light\_q2} + \beta_2 X_{Light\_q34} + \beta_3 X_{Age} + \beta_4 X_{Sex} + \beta_5 X_{SES\_2} + \beta_6 X_{SES\_3} + \beta_7 X_{CF}$
$SOL(T0) = \beta_0 + \beta_1 X_{Light\_q2} + \beta_2 X_{Light\_q34}$
$SOL(T0) = \beta_0 + \beta_1 X_{Light\_q2} + \beta_2 X_{Light\_q34} + \beta_3 X_{Age} + \beta_4 X_{Sex} + \beta_5 X_{SES\_2} + \beta_6 X_{SES\_3} + \beta_7 X_{CF} + \beta_8 X_{Bedtime\_none} + \beta_9 X_{Bedtime\_2} + \beta_{10} X_{Bedtime\_3} + \beta_{11} X_{CSHS}$
$TST(T0) = \beta_0 + \beta_1 X_{Light\_q2} + \beta_2 X_{Light\_q34}$
$TST(T0) = \beta_0 + \beta_1 X_{Light\_q2} + \beta_2 X_{Light\_q34} + \beta_3 X_{Age} + \beta_4 X_{Sex} + \beta_5 X_{SES\_2} + \beta_6 X_{SES\_3} + \beta_7 X_{CF} + \beta_8 X_{Bedtime\_none} + \beta_9 X_{Bedtime\_2} + \beta_{10} X_{Bedtime\_3} + \beta_{11} X_{CSHS}$
Noise models at three months follow up
$SOL(T3) = \beta_0 + \beta_1 X_{Noise \geq 40\ dB} + \beta_2 X_{Arm} + \beta_3 X_{Arm} * X_{Arm} + \beta_4 X_{SOL(T0)}$
$SOL(T3) = \beta_0 + \beta_1 X_{Noise \geq 40\ dB} + \beta_2 X_{Arm} + \beta_3 X_{Noise \geq 40\ dB} * X_{Arm} + \beta_4 X_{Age} + \beta_5 X_{Sex} + \beta_6 X_{SES\_2} + \beta_7 X_{SES\_3} + \beta_8 X_{CF} + \beta_9 X_{Bedtime\_none} + \beta_{10} X_{Bedtime\_2} + \beta_{11} X_{Bedtime\_3} + \beta_{12} X_{CSHS} + \beta_{13} X_{SOL(T0)}$
$TST(T3) = \beta_0 + \beta_1 X_{Noise \geq 40\ dB} + \beta_2 X_{Arm} + \beta_3 X_{Noise \geq 40\ dB} * X_{Arm} + \beta_4 X_{TST(T0)}$
$TST(T3) = \beta_0 + \beta_1 X_{Noise \geq 40\ dB} + \beta_2 X_{Arm} + \beta_3 X_{Noise \geq 40\ dB} * X_{Arm} + \beta_4 X_{Age} + \beta_5 X_{Sex} + \beta_6 X_{SES\_2} + \beta_7 X_{SES\_3} + \beta_8 X_{CF} + \beta_9 X_{Bedtime\_none} + \beta_{10} X_{Bedtime\_2} + \beta_{11} X_{Bedtime\_3} + \beta_{12} X_{CSHS} + \beta_{13} X_{TST(T0)}$
Light models at three months follow up
$SOL(T3) = \beta_0 + \beta_1 X_{Light\_q2} + \beta_2 X_{Light\_q34} + \beta_3 X_{Arm} + \beta_4 X_{Light\_q2} * X_{Arm} + \beta_5 X_{Light\_q34} * X_{Arm} + \beta_6 X_{SOL(T0)}$
$SOL(T3) = \beta_0 + \beta_1 X_{Light\_q2} + \beta_2 X_{Light\_q34} + \beta_3 X_{Arm} + \beta_4 X_{Light\_q2} * X_{Arm} + \beta_5 X_{Light\_q34} * X_{Arm} + \beta_6 X_{Age} + \beta_6 X_{Sex} + \beta_7 X_{SES\_2} + \beta_8 X_{SES\_3} + \beta_9 X_{CF} + \beta_{10} X_{Bedtime\_none} + \beta_{11} X_{Bedtime\_2} + \beta_{12} X_{Bedtime\_3} + \beta_{13} X_{CSHS} + \beta_{14} X_{SOL(T0)}$
$TST(T3) = \beta_0 + \beta_1 X_{Light\_q2} + \beta_2 X_{Light\_q34} + \beta_3 X_{Arm} + \beta_4 X_{Light\_q2} * X_{Arm} + \beta_5 X_{Light\_q34} * X_{Arm} + \beta_5 X_{TST(T0)}$
$TST(T3) = \beta_0 + \beta_1 X_{Light\_q2} + \beta_2 X_{Light\_q34} + \beta_3 X_{Arm} + \beta_4 X_{Light\_q2} * X_{Arm} + \beta_5 X_{Light\_q34} * X_{Arm} + \beta_5 X_{Age} + \beta_6 X_{Sex} + \beta_7 X_{SES\_2} + \beta_8 X_{SES\_3} + \beta_9 X_{CF} + \beta_{10} X_{Bedtime\_none} + \beta_{11} X_{Bedtime\_2} + \beta_{12} X_{Bedtime\_3} + \beta_{13} X_{CSHS} + \beta_{14} X_{TST(T0)}$

Table 2. Linear model equations.

Results

Characteristics of the study population are shown in Table 3. On average, this population experienced 36.55 minutes of SOL and 15.49% WASO at baseline. Mean TST at baseline was 600.86 minutes (10.0 hours) measured by sleep diary, and 493.44 minutes (8.2 hours) measured by actigraphy; it is typical for sleep diaries to overestimate TST compared to actigraphy.(68, 69) Transportation noise at subjects' residents is fairly low, with the mean at 37.60 dB (comparable to whispering or the hum of a refrigerator) and maximum of 62.10 dB (comparable to the noise level of a normal conversation).(75) The transportation noise distribution of this sample is shown in Figure 2.

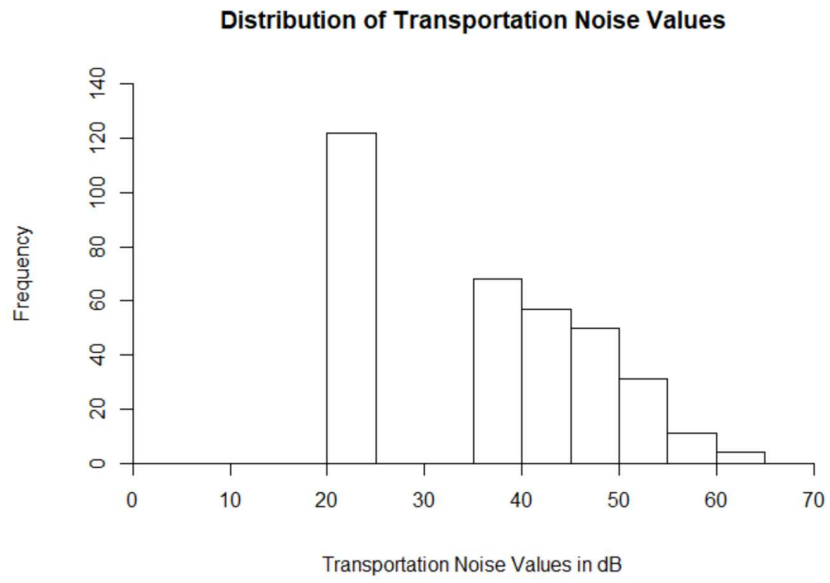

Figure 2. Sample distribution of transportation noise values.

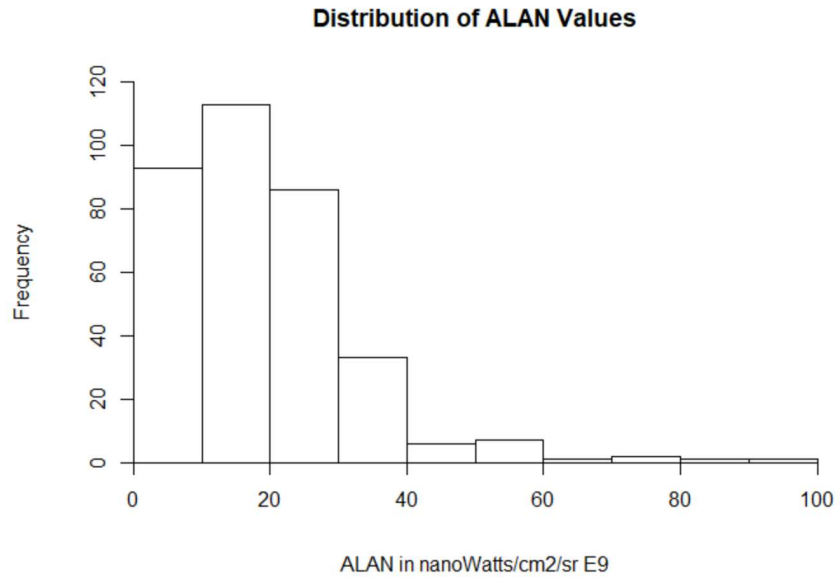

Figure 3. Sample distribution of ALAN values.

The ALAN distribution of this sample is right-skewed (see Figure 3). Mean ALAN level in this sample is 18.72 nanoWatts/cm^2/sr *10^9, and the maximum value was 90.81 nanoWatts/cm^2/sr *10^9. For context, Times Square has an ALAN value of 260.14 nanoWatts/cm^2/sr *10^9; Westlake Park in downtown Seattle is 222.60 nanoWatts/cm^2/sr *10^9; downtown Tacoma, 125.00 nanoWatts/cm^2/sr *10^9; a quiet street in Kirkland, 28.92 nanoWatts/cm^2/sr *10^9; and the center of Carnation, a small town in northeast King County, 13.62 nanoWatts/cm^2/sr *10^9. Many rural or wild areas have ALAN values of 0 nanoWatts/cm^2/sr *10^9.

	Entire sample. n = 343.			Intervention arm. n= 179.			Control arm. n = 164.		
	total n	n, percentage of total	Mean, standard deviation	total n	n, percentage of total	Mean, standard deviation	total n	n, percentage of total	Mean, standard deviation
Male sex	343	184, 54%		179	95, 53%		164	89, 54%	
Age in months	342		44.77, 10.34	179		44.69, 10.46	163		44.85, 10.23
Higher income	340	212, 62%		178	116, 65%		162	96, 59%	
Lower income, higher education	340	90, 26%		178	41, 23%		162	49, 30%	
Lower income, lower education	340	38, 11%		178	17, 12%		162	21, 10%	
Bedtime	293		20.01, 0.65	151	20.01, 0.61		142		20.01, 0.70
Bedtime 7:45 pm or earlier	337	114, 34%		174	52, 36%		163	62, 32%	
Bedtime 7:46 pm-8:15 pm	337	92, 27%		174	51, 24%		163	41, 31%	
Bedtime 8:16 pm or later	337	87, 26%		174	39, 28%		163	48, 24%	
No regular bedtime	340	44, 13%		177	21, 13%		163	23, 13%	
Mean circadian fragility	340		1.75, 0.44	177		1.73, 0.43	163		1.76, 0.46
Mean CSHS score	340		2.20, 0.40	177		2.17, 0.39	163		2.22, 0.41
WASO at T0 in percentage points (actigraphy)	342		15.49, 4.72	179		15.59, 4.97	163		15.37, 4.43
TST at T0 in minutes (actigraphy)	343		493.44, 47.30	179		490.43, 47.44	163		496.74, 47.08
TST at T0 in minutes (diary)	339		600.86, 44.66	176		596.96, 43.62	163		605.07, 45.53
SOL at T0 in minutes (diary)	339		36.55, 23.64	176		36.30, 24.75	163		36.81, 22.46
Artificial light at night in nanoWatts/cm^2/sr *10^9	343		18.72, 13.29, median: 16.75, IQR: [9.52, 24.79]	179		18.77, 13.22, median: 16.79, IQR: [9.15, 24.86]	164		18.67, 13.40, median: 16.63, IQR: [10.01, 24.60]
Artificial light at night ≤ 9.52 nanoWatts/cm^2/sr *10^9	343	86, 25%		179	49, 27%		164	37, 23%	
Artificial light at night > 9.52 and ≤ 16.76 nanoWatts/cm^2/sr *10^9	343	86, 25%		179	40, 22%		164	46, 28%	
Artificial light at night > 16.76 nanoWatts/cm^2/sr *10^9	343	171, 50%		179	90, 50%		164	81, 49%	
Transportation noise, 24-hour mean in dB	343		37.60, 10.80, median: 38.97, IQR: [24.75, 46.09]	179		37.65, 10.77, median: 38.79, IQR: [24.75, 38.79]	164		37.54, 10.87, median: 39.22, IQR: [24.75, 46.64]
Transportation noise, 24-hour mean < 40 dB	343	55%		179	100, 56%		164	90, 55%	
Transportation noise, 24-hour mean ≥ 40 dB	343	45%		179	79, 44%		164	74, 45%	

Table 3. Descriptive data.

No baseline sleep outcomes were significantly associated ($\alpha < 0.05$) with transportation noise (see Table 4). Subjects in the higher transportation noise group (≥ 40 dB) have 0.86 (95% CI: -0.15, 1.85; p = 0.09) percentage points higher WASO, compared to the lower noise group, in the unadjusted model. The results for the adjusted model are almost identical: the higher transportation noise group has 0.85 (95% CI: -0.15, 1.85; p = 0.097) percentage points higher WASO compared to the lower noise group. In the unadjusted model, the higher noise group is estimated to have 1.61 fewer (95% CI: 6.72 fewer to 3.29 more) minutes of diary-measured sleep onset latency compared to the lower noise group; the estimate in the adjusted model was higher and more precise (1.82 fewer minutes; 95% CI: -6.93, 3.29). The higher noise group was estimated to sleep 6.60 fewer (95% CI: -16.83 fewer to 3.64 more) minutes per night than the lower noise group in the adjusted model. In the adjusted model, the estimate is 6.13 fewer minutes (95% CI: -16.17, 3.29).

Outcome	WASO_act(T0)	WASO_act(T0)	SOL_diary(T0)	SOL_diary(T0)	TST_act(T0)	TST_act(T0)
Coefficient†	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**
Noise < 40 dB	REF	REF	REF	REF	REF	REF
Noise ≥ 40 dB	0.86 (-0.14, 1.87) (p = 0.09)	0.85 (-0.15, 1.85) (p =	-1.61 (-6.72, 3.49)	-1.82 (-6.93, 3.29)	-6.60 (-16.83, 3.64)	-6.13 (-16.17, 3.90)
Age		-0.10*** (-0.14, -0.05)		-0.26 (-0.54, 0.03) (p = 0.08)		1.21*** (0.77, 1.65)
Female sex		REF		REF		REF
Male sex		0.62 (-0.38, 1.62)		-0.34 (-5.46, 4.77)		-16.73*** (-26.57, -6.89)
SES_1		REF		REF		REF
SES_2		-0.41 (-1.52, 0.70)		0.08 (-5.31, 5.46)		3.12 (-8.09, 14.32)
SES_3		0.31 (-1.48, 2.10)		1.78 (-9.20, 12.76)		-12.61 (-28.92, 3.70)
Circadian fragility		-0.09 (-1.36, 1.19)		7.42* (0.46, 14.39)		2.57 (-8.86, 13.99)
Bedtime_1				REF		REF
Bedtime_2				-1.69 (-7.96, 4.58)		-7.89 (-19.74, 3.96)
Bedtime_3				-4.69 (-10.83, 1.45)		-22.38*** (-35.62, -9.14)
Bedtime_none				-3.62 (-12.88, 5.65)		-13.28 (-31.69, 4.60)
CSHS				18.17*** (9.95, 26.39)		-3.91 (-17.23, 9.42)

Table 4. Sleep outcomes and transportation noise. These models use baseline (T0) outcomes. Variable definitions can be found in Table 1. Reference group is indicated by "REF". † * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. $p < 0.1$ indicated.

Table 5 shows the regression results for baseline sleep outcomes and ALAN. WASO was significantly associated with above median values of ALAN (>16.76 nanoWatts/cm^2/sr) in the unadjusted and adjusted models compared to ALAN in the first quartile (≤9.52 nanoWatts/cm^2/sr). Figure 4 displays WASO and ALAN values at baseline. Subjects with highest ALAN levels save 1.41 (95% CI: 0.20, 2.62) greater percentage points of sleep disruption in the unadjusted model compared to subjects with lowest ALAN levels. In the adjusted model, this result is attenuated slightly to 1.29 (95% CI: 0.02, 2.51) greater percentage points of WASO. Highest ALAN level subjects had 1.35 (95% CI: -4.44, 7.14) more minutes of

SOL compared to lowest ALAN level subjects in the unadjusted model; in the adjusted model, their SOL is 0.73 (95% CI: -4.87, 6.32) minutes greater. TST was borderline associated with highest ALAN levels (p = 0.07) in the unadjusted model: highest ALAN subjects slept 11.33 fewer (95% CI: 23.46 fewer, 0.80 more) minutes compared to lowest ALAN level subjects. However, this result was not significant in the adjusted model, where highest ALAN subjects slept 7.15 fewer (95% CI: 18.95 fewer, 4.65 more) minutes than lowest ALAN subjects.

Outcome	**WASO_act(T0)**	**WASO_act(T0)**	**SOL_diary(T0)**	**SOL_diary(T0)**	**TST_act(T0)**	**TST_act(T0)**
Coefficient†, (95% CI)	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**
Light_q1	REF	REF	REF	REF	REF	REF
Light_q2	0.83 (-0.54, 2.21)	0.82 (-0.53, 2.17)	3.13 (-3.28, 9.55)	3.60 (-2.78, 9.98)	-0.70 (-14.97, 13.56)	1.43 (-12.33, 15.19)
Light_q34	1.41* (0.20, 2.62)	1.29* (0.02, 2.51)	1.35 (-4.44, 7.14)	0.73 (-4.87, 6.32)	-11.33 (-23.46, 0.80)	-7.15 (-18.95, 4.65)
Age		-0.09*** (-0.05, 0.14)		-0.24 (-0.52, 0.04) (p = 0.09)		1.20*** (0.76, 1.64)
Female sex		REF		REF		REF
Male sex		0.66 (-0.33, 1.65)		-0.20 (-5.31, 4.90)		-16.81*** (-26.60, -7.03)
SES_1		REF		REF		REF
SES_2		-0.52 (-1.67, 0.62)		0.01 (-5.39, 5.41)		3.65 (-7.68, 14.98)
SES_3		0.23 (-1.59, 2.05)		2.35 (-8.54, 13.24)		-12.32 (-28.06, 4.59)
Circadian fragility		0.02 (-1.25, 1.28)		7.12* (0.12, 14.12)		1.72 (-9.65, 13.08)
Bedtime_1				REF		REF
Bedtime_2				-1.82 (-8.12, 4.47)		-8.23 (-20.10, 3.65)
Bedtime_3				-4.97 (-11.19, 1.24)		-22.08*** (-35.33, -8.83)
Bedtime_none				-3.36 (-12.63, 5.91)		-12.32 (-30.43, 5.78)
CSHS				18.21*** (10.08, 26.35)		-3.60 (-16.96, 9.76)

Table 5. Sleep outcomes and ALAN. These models use baseline (T0) outcomes. Variable definitions can be found in Table 1. Reference group is indicated by "REF". † * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. $p < 0.1$ indicated.

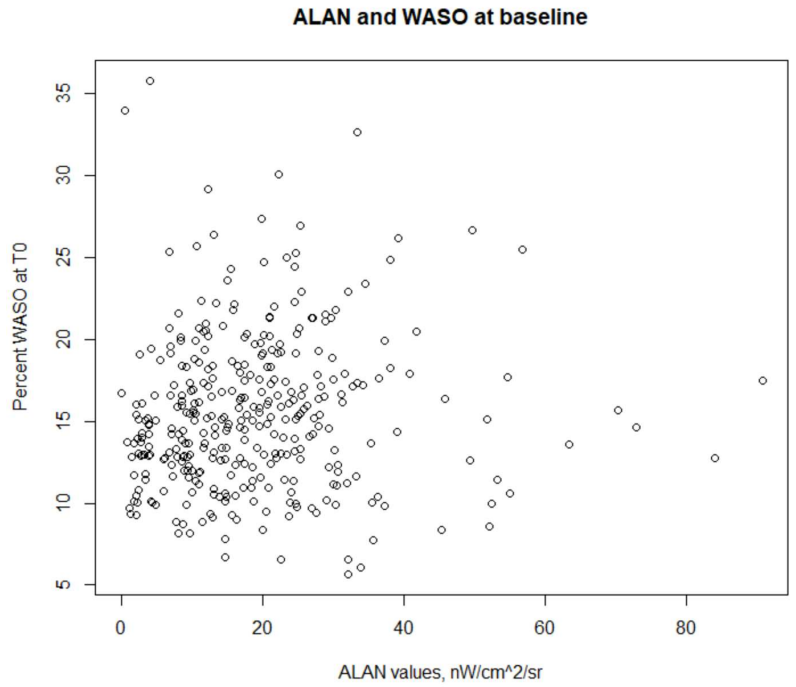

Figure 4. Plot of WASO(T0) and ALAN.

Transportation noise was not significantly associated with sleep outcomes at three months into intervention, nor were there statistically significant interactions between noise and intervention arm (see Table 6). Higher transportation noise appeared to hamper the effect of the intervention; in any case, confidence intervals were broad and p values were high (above 0.3) for the intervention term. Higher transportation noise appeared to decrease the beneficial impact of the intervention on SOL by 2.70 (95% CI: -4.03, 9.43) minutes in the unadjusted model and by 1.86 (95% CI: -5.03, 8.75) minutes in the adjusted model. Higher transportation noise appeared to decrease the beneficial impact of the intervention on TST by 5.64 (95% CI: -10.30, 21.58) minutes TST in the unadjusted model and by 8.17 (95% CI: -8.54, 24.89) minutes TST in the adjusted model.

Outcome	**SOL_diary(T3)**	**SOL_diary(T3)**	**TST_diary(T3)**	**TST_diary(T3)**
Coefficient†	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**
Noise < 40 dB	REF	REF	REF	REF
Noise ≥ 40 dB	-0.97 (-6.12, 4.19)	-0.90 (-6.06, 4.25)	1.65 (-10.80, 14.10)	3.04 (-9.68, 15.77)
Control group	REF	REF	REF	REF
Intervention arm	-8.72*** (-13.31, 4.13)	-8.29 (-13.00, -3.59)	8.05 (-2.71, 18.81)	9.48 (-1.84, 20.80)
Noise ≥ 40 dB *Intervention arm	2.70 (-4.03, 9.43)	1.86 (-5.03, 8.75)	-5.64 (-21.58, 10.30)	-8.17 (-24.89, 8.54)
SOL_diary(T0)	0.38*** (0.28, 0.48)	0.35*** (0.23, 0.47)		
TST_diary(T0)			0.67*** (0.57, 0.76)	0.63*** (0.52, 0.74)
Age		-0.19* (-0.36, -0.02)		0.11 (-0.28, 0.50)
Female sex		REF		REF
Male sex		-2.71 (-6.18, 0.77)		3.52 (-4.66, 11.71)
SES_1		REF		REF
SES_2		1.88 (-2.16, 5.92)		-2.17 (-10.74, 6.40)
SES_3		-0.86 (-7.20, 5.47)		3.40
Circadian fragility		-0.55 (-4.79, 3.68)		8.22 (-1.16, 17.61) (p = 0.09)
Bedtime_1		REF		REF
Bedtime_2		-0.53 (-5.06, 4.01)		-1.74 (-17.17, 23.97)
Bedtime_3		3.53 (-0.93, 8.00)		-14.58** (-25.16, -4.00)
Bedtime_none		2.89 (-3.11, 8.89)		-2.54 (-16.81, 11.72)
CSHS		4.49* (0.10, 8.88)		0.64 (-10.43, 11.70)

Table 6. Transportation noise and sleep outcomes at 3 months. These models use sleep outcomes at 3 months into the intervention (T3). Variable definitions can be found in Table 1. Reference group is indicated by "REF". † * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. $p < 0.1$ indicated.

ALAN was not significantly associated with sleep outcomes at three months; no statistically significant interactions between ALAN and intervention arm were found (see Table 7). As with noise, higher ALAN appeared to decrease the effectiveness of the intervention: it increased SOL and decreased TST. Second quartile ALAN appeared to decrease the beneficial impact of the intervention on SOL by 5.30 (95% CI: -4.15, 14.73) minutes in the unadjusted model and by 4.12 (95% CI: -5.43, 13.67) minutes in the adjusted model. Above median ALAN appeared to decrease the beneficial impact of the intervention on SOL by 2.16 (95% CI: -5.32, 9.63) minutes in the unadjusted model and by 1.55 (95% CI: -5.99, 9.08) minutes in the adjusted model.

Second quartile ALAN appeared to decrease the beneficial impact of the intervention on TST by 4.21 (95% CI: -25.50, 17.08) minutes in the unadjusted model and by 3.53 (95% CI: -26.43, 19.37) minutes in the adjusted model. Above median ALAN appeared to decrease the beneficial impact of the intervention on TST by 1.98 (95% CI: -20.29, 16.33) minutes in the unadjusted model and by 2.85 (95% CI: -22.01, 16.32) minutes in the adjusted model.

Outcome	**SOL_diary(T3)**	**SOL_diary(T3)**	**TST_diary(T3)**	**TST_diary(T3)**
Coefficient†	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**	**Estimate (95% CI)**
Light_q1	REF	REF	REF	REF
Light_q2	-2.37 (-9.58, 4.83)	-3.07 (-10.49, 4.36)	11.06 (-5.48, 27.6)	14.28 (-3.60, 32.15)
Light_q34	-2.33 (-8.28, 3.62)	-3.45 (-9.33, 2.43)	2.72 (-10.80, 16.23)	6.40 (-7.50, 20.30)
Control group	REF	REF	REF	REF
Intervention arm	-9.92*** (-15.62, -4.22)	-9.32** (-15.01, -3.63)	8.01 (-6.33, 22.35)	8.77 (-6.48, 24.02)
Light_q2 *Intervention arm	5.30 (-4.13, 14.73)	4.12 (-5.43, 13.67)	-4.21 (-25.50, 17.08)	-3.53 (-26.43, 19.37)
Light_q34 *Intervention arm	2.16 (-5.32, 9.63)	1.55 (-5.99, 9.08)	-1.98 (-20.29, 16.33)	-2.85 (-22.01, 16.32)
SOL_diary(T0)	0.38*** (0.28, 0.48)	0.35*** (0.23, 0.47)		
TST_diary(T0)			0.66*** (0.56, 0.76)	0.62*** (0.52, 0.73)
Age		-0.20* (-0.37, -0.03)		0.16 (-0.23, -0.54)
Female sex		REF		REF
Male sex		-2.83 (-6.28, 0.62)		3.85 (-4.28, 11.99)
SES_1		REF		REF
SES_2		2.23 (-1.82, 6.27)		-2.93 (-11.40, 5.54)
SES_3		-0.87 (-7.49, 5.75)		5.24 (-15.25, 25.73)
Circadian fragility		-0.61 (-4.84, 3.63)		7.73 (-1.53, 16.98)
Bedtime_1		REF		REF
Bedtime_2		-0.44 (-4.93, 4.04)		-2.04 (-12.09, 8.02)
Bedtime_3		3.73 (-0.80, 8.26)		-15.55** (-26.59, -4.50)
Bedtime_none		3.07 (-2.92, 9.05)		-1.87 (-16.07, 12.33)
CSHS		4.86* (0.47, 9.25)		-0.03 (-11.27, 11.21)

Table 7. ALAN and sleep outcomes at 3 months. These models use sleep outcomes at 3 months into the intervention (T3). Reference group is indicated by "REF". † * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. $p < 0.1$ indicated.

Discussion

Findings

There were no significant ($\alpha < 0.05$) associations between transportation noise and sleep outcomes, although transportation noise ≥ 40dB was borderline associated with WASO at baseline ($p = 0.097$). Subjects in the higher transportation noise group (≥ 40 dB) had 0.86 (95% CI: -0.15, 1.85) percentage points higher WASO compared to the lower noise group. Transportation noise was not related to TST or SOL. This suggests that transportation noise at the levels present in this study might directly disrupt sleep, but do not affect sleep through disrupted circadian rhythm pathway (see Figure 1). ALAN was associated with WASO at baseline but no other sleep outcomes. We predicted that light would be most strongly associated with SOL, making this a surprising outcome. Subjects with ALAN above sample median (>16.76 nanoWatts/cm^2/sr *10^9) had 1.29 (95% CI: 0.02, 2.51) greater percentage points of sleep disruption; with mean WASO in this sample at 15.49%, this is a considerable increase. There were no statistically significant interactions between transportation noise or ALAN and the intervention at three months follow-up in adjusted models, nor were there statistically significant associations between the exposures of interest and sleep outcomes.

Implications

These results—the only significant or borderline significant associations being with WASO—suggests that sleep fragmentation may be the first sleep outcome to be disrupted by light and noise pollution (vulnerable even to the mild levels present in this sample), while higher levels of noise and light would increase sleep latency or decrease sleep quantity. This study supports the

need for policies that limit ALAN (to 16 nanoWatts/cm^2/sr *10^9) where children live to limit sleep disruption and fragmentation induced by ALAN.

Limitations & Future Research

A major limitation of this study is that individual exposure to light and noise pollution is unknown. While most noise pollution is likely from transportation, the data from the US DOT Noise Map leaves many unknowns regarding noise exposure. A child might have noisy neighbors or siblings, or live close to banging trash cans. We utilized addresses that families provided for follow-up to find noise and ALAN values, but it is unknown how long they had lived there, or whether they moved after the study began. ALAN values averaged over 2015 and 2016 were used, and therefore it is possible that variation (including seasonal variation) in ALAN values and exposure occurred. Furthermore, many intervening variables might modify a child's individual exposure to light or transportation noise: presence, size, and proximity of windows to street lights, proximity of the child's bedroom to the building façade nearest the road, how sound-insulated the residence is, and any measures the family may have taken to decrease exposure. Future studies should elucidate the connections between ecological ALAN/noise, individual exposure, and sleep outcomes. Furthermore, studies should collect information regarding the quality of these exposures: light color, light and noise intermittentness, timing of exposure to light and noise, and light and noise maximums.

This sample had very mild transportation noise and ALAN levels, which likely contributed to the many null results. Of the 343 subjects, 122 live at locations with transportation noise < 35 dB, while only 15 subjects live at locations with transportation noise > 55 dB. The maximum value is 62.10 dB, which is comparable to the noise level of a normal conversation. The mean ALAN value in this sample was 18.72 nanoWatts/cm^2/sr *10^9, which is comparable to ALAN levels in

suburban residential or rural center settings. Maximum ALAN was 90.81 nanoWatts/cm^2/sr *10^9, which is somewhat darker than small urban centers (downtown Tacoma, 125.00 nanoWatts/cm^2/sr *10^9), but far darker than large urban centers (Westlake Park in downtown Seattle is 222.60 nanoWatts/cm^2/sr *10^9).Future studies should recruit a study sample with a wide range of ecological ALAN and noise values.

Another major limitation of this study is generalizability. This study used a sample of preschool aged children with sleep problems. The families enrolled in this study tended to be higher income and more educated (62% with > $100,000 annual family income, 84% with at least a bachelor's degree) than the general US population. Future research should investigate associations between noise/light and sleep outcomes using a more representative sample for more generalizable results.

Conclusion

In this study, we found that higher levels of ALAN (> 16.76 nanoWatts/cm^2/sr *10^9) were associated with increased sleep disruption (1.29 (95% CI: 0.02, 2.51) additional percentage points WASO). This finding supports the needs for limitations on ALAN to preserve sleep quality and therefore health and wellbeing. Future studies on environmental noise and light exposure and sleep in should recruit representative samples of young children with a wide range of environmental noise and light levels; they should take individual exposure and the qualities of noise and light into account.

References

1. Banks S, Dinges DF. Behavioral and physiological consequences of sleep restriction. Journal of clinical sleep medicine : JCSM : official publication of the American Academy of Sleep Medicine. 2007;3(5):519.
2. Watson NF, Badr MS, Belenky G, Bliwise DL, Buxton OM, Buysse D, Dinges DF, Gangwisch J, Grandner MA, Kushida C, Malhotra RK, Martin JL, Patel SR, Quan SF, Tasali E. Joint Consensus Statement of the American Academy of Sleep Medicine and Sleep Research Society on the Recommended Amount of Sleep for a Healthy Adult: Methodology and Discussion. Journal of clinical sleep medicine : JCSM : official publication of the American Academy of Sleep Medicine. 2015;11(8):931. doi: 10.5664/jcsm.4950.
3. Xie L, Kang H, Xu Q, Chen MJ, Liao Y, Thiyagarajan M, O'Donnell J, Christensen DJ, Nicholson C, Iliff JJ, Takano T, Deane R, Nedergaard M. Sleep drives metabolite clearance from the adult brain. Science (New York, NY). 2013;342(6156):373. doi: 10.1126/science.1241224.
4. Wang G, Grone B, Colas D, Appelbaum L, Mourrain P. Synaptic plasticity in sleep: learning, homeostasis and disease. Trends in Neurosciences. 2011:452.
5. Goel N, Rao H, Durmer J, Dinges D. Neurocognitive Consequences of Sleep Deprivation. Semin Neurol. 2009;29(4):320-39. doi: 10.1055/s-0029-1237117.
6. Harrison Y, Horne JA. The Impact of Sleep Deprivation on Decision Making: A Review. Journal of Experimental Psychology: Applied. 2000;6(3):236-49. doi: 10.1037/1076-898X.6.3.236.
7. Van Dongen HPA, Maislin G, Mullington JM, Dinges DF. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. Sleep. 2003;26(2):117. doi: 10.1093/sleep/26.2.117.
8. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. The Lancet. 1999;354(9188):1435-9. doi: 10.1016/S0140-6736(99)01376-8.
9. Irwin MR, Olmstead R, Carroll JE. Sleep Disturbance, Sleep Duration, and Inflammation: A Systematic Review and Meta-Analysis of Cohort Studies and Experimental Sleep Deprivation. Biological Psychiatry. 2016;80(1):40-52. doi: 10.1016/j.biopsych.2015.05.014.
10. Kato M, Phillips BG, Sigurdsson G, Narkiewicz K, Pesek CA, Somers VK. Effects of sleep deprivation on neural circulatory control. Hypertension (Dallas, Tex : 1979). 2000;35(5):1173. doi: 10.1161/01.HYP.35.5.1173.
11. Tochikubo O, Ikeda A, Miyajima E, Ishii M. Effects of insufficient sleep on bled pressure monitored by a new multibiomedical recorder. Hypertension. 1996;27(6):1318-24. doi: 10.1161/01.HYP.27.6.1318.
12. Lange T, Perras B, Fehm HL, Born J. Sleep enhances the human antibody response to hepatitis A vaccination. Psychosomatic medicine. 2003;65(5):831. doi: 10.1097/01.PSY.0000091382.61178.F1.
13. Spiegel K, Sheridan JF, Van Cauter E. Effect of Sleep Deprivation on Response to Immunizaton. JAMA. 2002;288(12):1471-2. doi: 10.1001/jama.288.12.1469.
14. Lange T, Dimitrov S, Bollinger T, Diekelmann S, Born J. Sleep after vaccination boosts immunological memory. Journal of immunology (Baltimore, Md : 1950). 2011;187(1):283. doi: 10.4049/jimmunol.1100015.

15. Dew MA, Hoch CC, Buysse DJ, Monk TH, Begley AE, Houck PR, Hall M, Kupfer DJ, Reynolds CF. Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. Psychosomatic medicine. 2003;65(1):63. doi: 10.1097/01.PSY.0000039756.23250.7C.
16. Gallicchio L, Kalesan B. Sleep duration and mortality: a systematic review and meta-analysis. Journal of Sleep Research. 2009;18(2):148-58. doi: 10.1111/j.1365-2869.2008.00732.x.
17. Chaput JP, Gray CE, Poitras V, Carson V, Gruber R, Birken CS, Maclean J, Aubert S, Sampson M, Tremblay MS. Systematic review of the relationships between sleep duration and health indicators in the early years (0-4 years). BMC Public Health2017.
18. Minkel JD, Banks S, Htaik O, Moreta MC, Jones CW, McGlinchey EL, Simpson NS, Dinges DF. Sleep Deprivation and Stressors: Evidence for Elevated Negative Affect in Response to Mild Stressors When Sleep Deprived. Emotion. 2012;12(5):1015-20. doi: 10.1037/a0026871.
19. Reynaud E, Vecchierini M-F, Heude B, Charles M-A, Plancoulaine S. Sleep and its relation to cognition and behaviour in preschool-aged children of the general population: a systematic review. Journal of sleep research. 2018;27(3):e12636. doi: 10.1111/jsr.12636.
20. Scharf RJ, Demmer RT, Silver EJ, Stein REK. Nighttime sleep duration and externalizing behaviors of preschool children. Journal of developmental and behavioral pediatrics : JDBP. 2013;34(6):384. doi: 10.1097/DBP.0b013e31829a7a0d.
21. Dahl RE. The impact of inadequate sleep on children's daytime cognitive function. Seminars in Pediatric Neurology. 1996;3(1):44-50. doi: 10.1016/S1071-9091(96)80028-3.
22. Bathory E, Tomopoulos S. Sleep Regulation, Physiology and Development, Sleep Duration and Patterns, and Sleep Hygiene in Infants, Toddlers, and Preschool-Age Children. Current Problems in Pediatric and Adolescent Health Care. 2017;47(2):29-42. doi: 10.1016/j.cppeds.2016.12.001.
23. Beebe DW. Cognitive, Behavioral, and Functional Consequences of Inadequate Sleep in Children and Adolescents. The Pediatric Clinics of North America. 2011;58(3):649-65. doi: 10.1016/j.pcl.2011.03.002.
24. Paavonen EJ, Räikkönen K, Lahti J, Komsi N, Heinonen K, Pesonen A-K, Järvenpää A-L, Strandberg T, Kajantie E, Porkka-Heiskanen T. Short sleep duration and behavioral symptoms of attention-deficit/hyperactivity disorder in healthy 7- to 8-year-old children. Pediatrics. 2009;123(5):e857. doi: 10.1542/peds.2008-2164.
25. Dewald JF, Meijer AM, Oort FJ, Kerkhof GA, Bögels SM. The influence of sleep quality, sleep duration and sleepiness on school performance in children and adolescents: A meta-analytic review. Sleep Medicine Reviews. 2010;14(3):179-89. doi: 10.1016/j.smrv.2009.10.004.
26. Sadeh A. The Effects of Sleep Restriction and Extension on School-Age Children: What a Difference an Hour Makes. Child development. 2004;74(2):444-55.
27. O'Callaghan FV, Al Mamun A, O'Callaghan M, Clavarino A, Williams GM, Bor W, Heussler H, Najman JM. The link between sleep problems in infancy and early childhood and attention problems at 5 and 14years: Evidence from a birth cohort study. Early Human Development. 2010;86(7):419-24. doi: 10.1016/j.earlhumdev.2010.05.020.
28. Lam P, Hiscock H, Wake M. Outcomes of infant sleep problems: a longitudinal study of sleep, behavior, and maternal well-being. Pediatrics. 2003;111(3):e203-7. doi: 10.1542/peds.111.3.e203.
29. Martin J, Hiscock H, Hardy P, Davey B, Wake M. Adverse associations of infant and child sleep problems and parent health: An Australian population study. Pediatrics. 2007;119(5):947-55. doi: 10.1542/peds.2006-2569.

30. Meltzer LJ, Montgomery-Downs HE. Sleep in the Family. The Pediatric Clinics of North America. 2011;58(3):765-74. doi: 10.1016/j.pcl.2011.03.010.
31. Smedje H, Broman J-E, Hetta J. Sleep disturbances in Swedish pre-school children and their parents. Nordic journal of psychiatry. 1998;52:59-67.
32. Boergers J, Hart C, Owens JA, Streisand R, Spirito A. Child Sleep Disorders: Associations With Parental Sleep Duration and Daytime Sleepiness. Journal of Family Psychology. 2007;21(1):88-94. doi: 10.1037/0893-3200.21.1.88.
33. Meltzer LJ, Mindell JA. Relationship Between Child Sleep Disturbances and Maternal Sleep, Mood, and Parenting Stress: A Pilot Study. Journal of Family Psychology. 2007;21(1):67-73. doi: 10.1037/0893-3200.21.1.67.
34. Johnson D, Billings M, Hale L. Environmental Determinants of Insufficient Sleep and Sleep Disorders: Implications for Population Health. Current Epidemiology Reports. 2018;5(2):61-9. doi: 10.1007/s40471-018-0139-y.
35. Cho Y, Ryu S-H, Lee BR, Kim KH, Lee E, Choi J. Effects of artificial light at night on human health: A literature review of observational and experimental studies applied to exposure assessment. Chronobiology International. 2015;32(9):1294-310. doi: 10.3109/07420528.2015.1073158.
36. Tähkämö L, Partonen T, Pesonen A-K. Systematic review of light exposure impact on human circadian rhythm. Chronobiology international. 2019;36(2):151-70. doi: 10.1080/07420528.2018.1527773.
37. Ohayon MM, Milesi C. Artificial Outdoor Nighttime Lights Associate with Altered Sleep Behavior in the American General Population. Sleep. 2016;39(6):1311-20. doi: 10.5665/sleep.5860.
38. Patel PC. Light pollution and insufficient sleep: Evidence from the United States. American Journal of Human Biology. 2019. doi: 10.1002/ajhb.23300.
39. Akacem LD, Wright KP, Lebourgeois MK. Bedtime and evening light exposure influence circadian timing in preschool-age children: A field study. Neurobiology of Sleep and Circadian Rhythms. 2016;1(2):27-31. doi: 10.1016/j.nbscr.2016.11.002.
40. Akacem LD, Wright KP, Lebourgeois MK. Sensitivity of the circadian system to evening bright light in preschool-age children. Physiological Reports. 2018;6(5):n/a-n/a. doi: 10.14814/phy2.13617.
41. Higuchi S, Nagafuchi Y, Lee S-I, Harada T. Influence of light at night on melatonin suppression in children. The Journal of clinical endocrinology and metabolism. 2014;99(9):3298. doi: 10.1210/jc.2014-1629.
42. Basner M, Brink M, Elmenhorst E-M. Critical appraisal of methods for the assessment of noise effects on sleep. Noise & Health. 2012;14(61):321-9. doi: 10.4103/1463-1741.104902.
43. Perron S, Plante C, Ragettli MS, Kaiser DJ, Goudreau S, Smargiassi A. Sleep disturbance from road traffic, railways, airplanes and from total environmental noise levels in montreal. International Journal of Environmental Research and Public Health. 2016;13(8). doi: 10.3390/ijerph13080809.
44. Basner M, McGuire S. WHO Environmental Noise Guidelines for the European Region: A Systematic Review on Environmental Noise and Effects on Sleep. Int J Environ Res Public Health. 2018;15(3). doi: 10.3390/ijerph15030519.
45. Halonen JI, Vahtera J, Stansfeld S, Yli-Tuomi T, Salo P, Pentti J, Kivimäki M, Lanki T. Associations between nighttime traffic noise and sleep: The Finnish public sector study. Environmental Health Perspectives. 2012;120(10):1391-6. doi: 10.1289/ehp.1205026.

46. Öhrström E, Hadzibajramovic E, Holmes M, Svensson H. Effects of road traffic noise on sleep: Studies on children and adults. Journal of Environmental Psychology. 2006;26(2):116-26. doi: 10.1016/j.jenvp.2006.06.004.
47. Ising H, Ising M. Chronic cortisol increases in the first half of the night caused by road traffic noise. Noise and Health. 2002;4(16):13-21.
48. Davies HW, Vlaanderen JJ, Henderson SB, Brauer M. Correlation between co-exposures to noise and air pollution from traffic sources. Occupational and Environmental Medicine. 2009;66(5):347. doi: 10.1136/oem.2008.041764.
49. Billings ME, Gold D, Szpiro A, Aaron CP, Jorgensen N, Gassett A, Leary PJ, Kaufman JD, Redline SR. The Association of Ambient Air Pollution with Sleep Apnea: The Multi-Ethnic Study of Atherosclerosis. Annals of the American Thoracic Society. 2019;16(3):363. doi: 10.1513/AnnalsATS.201804-248OC.
50. Cukic V, Lovre V, Dragisic D. Sleep Disorders in Patients with Bronchial Asthma. Materia Socio-Medica. 2011;23(4):235-7. doi: 10.5455/msm.2011.23.235-237.
51. Hansel NN, Romero KM, Pollard SL, Bose S, Psoter KJ, Underhill L, Johnson C, Williams DA, Curriero FC, Breysse P, Koehler K, Checkley W. Ambient Air Pollution Adversely Impacts Various Domains of Asthma Morbidity among Peruvian Children. Annals of the American Thoracic Society. 2018;16(3):448OC. doi: 10.1513/AnnalsATS.201807-448OC.
52. Berglund B, Lindvall T, Schwela DH, World Health Organization Occupational and Environmental Health Team. Guidelines for community noise. 1999. doi: https://apps.who.int/iris/handle/10665/66217.
53. Basner M, Babisch W, Davis A, Brink M, Clark C, Janssen S, Stansfeld S. Auditory and non-auditory effects of noise on health. The Lancet. 2014;383(9925):1325-32. doi: 10.1016/S0140-6736(13)61613-X.
54. van Kempen EEMM, van Kamp I, Stellato RK, Lopez-Barrio I, Haines MM, Nilsson ME, Clark C, Houthuijs D, Brunekreef B, Berglund B, Stansfeld SA. Children's annoyance reactions to aircraft and road traffic noise. The Journal of the Acoustical Society of America. 2009;125(2):895-904. doi: 10.1121/1.3058635.
55. Szalma JL, Hancock PA. Noise Effects on Human Performance: A Meta-Analytic Synthesis. Psychological Bulletin. 2011;137(4):682-707. doi: 10.1037/a0023987.
56. National Research Council (US) and Institute of Medicine (US) Committee on Depression, Parenting Practices, and the Healthy Development of Children. Depression in parents, parenting, and children : opportunities to improve identification, treatment, and prevention. Washington, DC: Washington, DC : National Academies Press; 2009.
57. Shaffer A, Obradović J. Unique Contributions of Emotion Regulation and Executive Functions in Predicting the Quality of Parent–Child Interaction Behaviors. Journal of Family Psychology. 2017;31(2):150-9. doi: 10.1037/fam0000269.
58. Owens JA, Spirito A, McGuinn M, Nobile C. Sleep habits and sleep disturbance in elementary school-aged children. Journal of developmental and behavioral pediatrics : JDBP. 2000;21(1):27. doi: 10.1097/00004703-200002000-00005.
59. Hannan K, Hiscock H. Sleep problems in children. Australian family physician. 2015;44(12):880.
60. Bion V, Lowe AS, Puthucheary Z, Montgomery H. Reducing sound and light exposure to improve sleep on the adult intensive care unit: An inclusive narrative review. Journal of the Intensive Care Society. 2018;19(2):138-46. doi: 10.1177/1751143717740803.

61. Alkahtani M, Alshathri N, Aldraiweesh N, Aljurf L, Aldaej L, Olaish A, Nashwan S, Almeneessier A, BaHammam A. The effect of air conditioner sound on sleep latency, duration, and efficiency in young adults. Annals of Thoracic Medicine. 2019;14(1):69-74. doi: 10.4103/atm.ATM_195_18.
62. Sezici E, Yigit D. Comparison between swinging and playing of white noise among colicky babies: A paired randomised controlled trial. Journal of Clinical Nursing. 2018;27(3-4):593-600. doi: 10.1111/jocn.13928.
63. Earth Observation Group, NOAA National Centers for Environmental Information (NCEI). Version 1 VIIRS Day/Night Band Nighttime Lights. Available from: https://ngdc.noaa.gov/eog/viirs/download_dnb_composites.html.
64. Bureau of Transportation Statistics (BTS), US Department of Transportation. National Transportation Noise Map. Available from https://maps.bts.dot.gov/services/rest/services/Noise/CONUS_ROAD_AND_AVIATION_NOISE_IS_Apr_2018/ImageServer .
65. Meltzer LJ, Montgomery-Downs HE, Insana SP, Walsh CM. Use of actigraphy for assessment in pediatric sleep research. Sleep Medicine Reviews. 2012;16(5):463-75. doi: 10.1016/j.smrv.2011.10.002.
66. Sadeh A. The role and validity of actigraphy in sleep medicine: An update. Sleep Medicine Reviews. 2011;15(4):259-67. doi: 10.1016/j.smrv.2010.10.001.
67. Iwasaki M, Iwata S, Iemura A, Yamashita N, Tomino Y, Anme T, Yamagata Z, Iwata O, Matsuishi T. Utility of Subjective Sleep Assessment Tools for Healthy Preschool Children: A Comparative Study Between Sleep Logs, Questionnaires, and Actigraphy. Journal of Epidemiology. 2010;20(2):143-9. doi: 10.2188/jea.JE20090054.
68. Werner H, Molinari L, Guyer C, Jenni OG. Agreement Rates Between Actigraphy, Diary, and Questionnaire for Children's Sleep Patterns. Archives of Pediatrics & Adolescent Medicine. 2008;162(4):350-8. doi: 10.1001/archpedi.162.4.350.
69. Nelson TD, Lundahl A, Molfese DL, Waford RN, Roman A, Gozal D, Molfese VJ, Ferguson MC. Estimating Child Sleep From Parent Report of Time in Bed: Development and Evaluation of Adjustment Approaches. Journal of Pediatric Psychology. 2014;39(6):624-32. doi: 10.1093/jpepsy/jsu020.
70. Sears CG, Braun JM, Ryan PH, Xu Y, Werner EF, Lanphear BP, Wellenius GA. The association of traffic-related air and noise pollution with maternal blood pressure and hypertensive disorders of pregnancy in the HOME study cohort. Environment International. 2018;121(Pt 1):574-81. doi: 10.1016/j.envint.2018.09.049.
71. Hurtley C, World Health Organization. Regional Office for E. Night noise guidelines for Europe. Copenhagen, Denmark: Copenhagen, Denmark : World Health Organization Europe; 2009.
72. The Office of Economic and Financial Analysis. King County Educational Attainment 2018 [cited 2020]. Available from: https://www.kingcounty.gov/independent/forecasting/King%20County%20Economic%20Indicators/KC%20Educational%20Attainment.aspx.
73. The Office of Economic and Financial Analysis. Household Income in King County 2017 [cited 2020]. Available from: https://www.kingcounty.gov/independent/forecasting/King%20County%20Economic%20Indicators/Household%20Income.aspx.

74. Harsh, Jr., Easley A, Lebourgeois M. A measure of children's sleep hygiene. Sleep. 2002;25:A316-A7.
75. National Center for Environmental Health, CDC. What Noises Cause Hearing Loss? 2019. Available from: https://www.cdc.gov/nceh/hearing_loss/what_noises_cause_hearing_loss.html#.

Appendix

Think back to how your child has responded to changes in schedule over the last year:	About the same as usual	A little worse than usual	Much worse than usual
When your child has a late night (i.e., bedtime is 2+ hours later than usual) for a holiday, party, or family event, what impact does that have the next day on your child:			
Waking up at their usual time the next morning			
Having a hard time dealing with emotions the next day			
Having difficult falling asleep at the usual bedtime the next night			
How long does it take your child to become adjusted to the change in bedtime when your regular routine is shifted…	Within a day or two	Several days	More than a week
An hour LATER than usual for awhile, such as with the Fall daylight savings time change, or traveling to a later time zone			
An hour EARLIER than usual for awhile, such as with the Spring daylight savings time change, or traveling to an earlier time zone			

Circadian Fragility questionnaire.