



## Original Contribution

# Environmental Influences on Sleep in the California Teachers Study Cohort

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Only two-thirds of Americans meet the recommended 7 hours of sleep nightly. Insufficient sleep and circadian disruption have been associated with adverse health outcomes, including diabetes and cardiovascular disease. Several environmental disruptors of sleep have been reported, such as artificial light at night (ALAN) and noise. These studies tended to evaluate exposures individually. We evaluated several spatially derived environmental exposures (ALAN, noise, green space, and air pollution) and self-reported sleep outcomes obtained in 2012–2015 in a large cohort of 51,562 women in the California Teachers Study. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated for sleep duration and latency. After adjusting for age, race/ethnicity, chronotype, use of sleep medication, and self-reported trouble sleeping, ALAN (per 5 millicandela (mcd)/m<sup>2</sup> luminance, OR = 1.13, 95% CI: 1.07, 1.20) and air pollution (per 5 µg/m<sup>3</sup> PM<sub>2.5</sub>, OR = 1.06, 95% CI: 1.04, 1.09) were associated with shorter sleep duration (<7 hours), and noise was associated with longer latency (>15 minutes) (per 10 decibels, OR = 1.05, 95% CI: 1.01, 1.10). Green space was associated with increased duration (per 0.1 units, OR = 0.41, 95% CI: 0.28, 0.60) and decreased latency (per 0.1 units, OR = 0.55, 95% CI: 0.39, 0.78). Further research is necessary to understand how these and other exposures (e.g., diet) perturb an individuals' inherited sleep patterns and contribute to downstream health outcomes.

air pollution; artificial light at night; circadian rhythm; cohort study; epidemiology; green space; noise; sleep disruption

Abbreviations: ALAN, artificial light at night; CI, confidence interval; CTS, California Teachers Study; dB, decibel; EVI, enhanced vegetation index; mcd, millicandela; OR, odds ratio; PM<sub>2.5</sub>, particulate matter with an aerodynamic diameter ≤2.5 µm.

**Editor's note:** An invited commentary on this article appears on page 1540, and the authors' response appears on page 1544.

Disruptions in sleep have been associated with increased risk of several health conditions, including cardiovascular disease, diabetes, and cancer (1). In 2015, the American Academy of Sleep Medicine and Sleep Research Society published a joint consensus statement recommending that adults get at least 7 hours of sleep each night (2); however, 35% of American adults fall short of this recommendation (3).

Light is one of the main environmental factors affecting the sleep-wake cycle. Photosensitive cells in our retina

respond to light and trigger the suprachiasmatic nucleus in the hypothalamus. The suprachiasmatic nucleus then modulates body temperature and regulates levels of cortisol and melatonin in response to the amount of light we perceive. The reduction of exposure to light at night triggers the increase in melatonin, leading to the feeling of sleepiness. Exposure to bright lights before sleep can delay the release of melatonin, therefore increasing sleep latency and disrupting circadian rhythm (4). While the direct stimuli of light indoors prior to sleep has been shown to delay sleep onset and reduce sleep quality (5), so has artificial light at night (ALAN) from the outdoor environment (6, 7). ALAN has been increasing several percent yearly over the past decades due to increasing human development and urbanization and may be playing a role in disrupting circadian rhythm (8, 9).

In addition to ALAN, another large component to sleep disruption is noise. In 2011, the WHO published a report on the “burden of disease from environmental noise,” estimating almost a million disability-adjusted life-years of sleep lost to noise (10). Like ALAN, noise may contribute to a delay in sleep latency and has been associated with nocturnal awakenings. As we progress into the later and lighter stages of sleep, external noise stimuli contribute to the possibility of waking earlier (11). In addition to noise, our increasing urbanization has also disrupted green space and increased air pollution; these additional environmental factors may be linked to sleep disruption. Higher levels of green space have been directly and indirectly associated with improved sleep outcomes. The beneficial associations with green space were seen not only in studies that assessed use of green space (gardening or walking), but were also present in studies that only assessed residential surroundings (12). It is not clear the exact mechanisms of green space on health, but it is believed that mimicking the natural environment helps improve mental health through the reduction of stress and pain (13). Last, air pollution has been associated with poorer sleep (14), potentially due to inflammation and irritation of breathing airways and not limited to individuals with asthma and sleep apnea (15, 16).

The implication of such disruptions extends beyond sleep to other detrimental health outcomes. Mounting evidence of a link between breast cancer and circadian disruption due to night-shift work led the World Health Organization’s International Agency for Research on Cancer to classify night-shift work as a probable human carcinogen (17). We previously reported associations of ALAN with breast cancer and lymphoma in our cohort (18, 19). Air pollution has been associated with a host of adverse health outcomes, including but not limited to cancer risk, cardiovascular disease risk, and impairment of lung function and growth (20). After adjusting for near-roadway air pollution, noise pollution has also been associated with increased morbidity and mortality. A 2018 meta-analysis found 7% increased risk of diabetes mellitus per 5-decibel (dB) increase in road traffic noise (21), while a 2021 meta-analysis found no increased risk of death from road traffic noise but did see increased risk from aircraft noise (22). Finally, higher levels of green space are also associated with improvements in mental health and reduced mortality (23), with 2 studies reporting up to a 10% reduction in mortality for those living in greener areas (24, 25).

Many studies of environmental sleep disruption examined a single environmental exposure at a time. In this work, we assessed the association of several geospatially derived environmental exposures concurrently on self-reported sleep outcomes in the California Teachers Study (CTS), a large cohort of women residing in California.

## METHODS

### Study population

The CTS is a large cohort of 133,477 women recruited in 1995–1996 from the California State Teachers’ Retirement System. Women returned a baseline questionnaire and have since participated in ongoing follow-up activities. Addi-

tional details of the cohort have been previously published (26). A total of 56,114 women responded to a follow-up questionnaire, conducted from 2012–2015, that included self-reported questions of sleep history. Of the respondents, 53,426 answered all questions regarding their sleep habits. We were able to geocode exposure data for 51,562 participants who were residents in the state of California in 2014, when our environmental exposures were assessed.

### Environmental exposures

We assessed 2 primary environmental factors that may contribute to sleep disruption, outdoor artificial light at night and environmental noise, as well as 2 secondary exposures, green space and air pollution. We used the New World Atlas of Artificial Night Sky Brightness as our measure of ALAN (27). The World Atlas provides a global 750-m gridded measure of estimated light at zenith (the sky directly overhead) in millicandela (mcd) per meters squared based on the Visible Infrared Imaging Radiometer Suite satellite and thousands of handheld sky quality measurements. This measurement has shown to be a better estimate of ground-level light exposure than use of satellite data alone (28), and its higher resolution makes it superior to older, coarser measurements and less likely to be highly correlated with air and noise pollution (29).

Environmental noise was assessed with the National Park Service sound map (30). The sound model used to produce the map uses collected data coupled with geospatial prediction to calculate the L50 (sound level exceeded half the time) sound pressure–level metric, which is a weighted measure that considers how the human ear perceives sound. The median L50 sound level in dB is produced over a 270-m grid representing natural sound (e.g., wind and animals) as well as for sound from human sources (e.g., traffic). The model provides data on longitude, latitude, weather, land cover, distance to bodies of water/streams, distance to roads, and air traffic, and it was validated with thousands of measurements taken from 479 sites across the contiguous United States.

To assess green space, we used the enhanced vegetation index (EVI). EVI is derived from the normalized difference vegetation index, which is a calculation of the ratio of near infrared to visible light observed by the Moderate Resolution Imaging Spectroradiometer instrument onboard NASA’s Terra and Aqua Satellites. The EVI improves upon the normalized difference vegetation index by incorporating blue light to correct for atmospheric conditions and dense vegetation (31). EVI is unitless ranging from  $-1$  to  $1$ ; where  $-1$  represents completely barren land and  $1$  represents areas of high-density vegetation. The 2014 annual, cloud-free, average EVI was calculated in 250-m grids.

Last, air pollution was assessed utilizing an ensemble-based machine-learning model developed by Di et al. (32). The model augmented the GEOS-Chem chemical transport model output with land-use variables including elevation, road density, the Environmental Protection Agency’s National Emissions Inventory, population density, urbanization, normalized difference vegetation index, weather patterns, and Moderate Resolution Imaging Spectroradiometer satellite–derived aerosol optical depth. The model estimates

concentrations of particulate matter with an aerodynamic diameter  $\leq 2.5 \mu\text{m}$  ( $\text{PM}_{2.5}$ ) in a 1-km grid. Participants were assigned the average level of  $\text{PM}_{2.5}$  for 2014.

Additional covariates on the built environment were obtained from 2015 5-year average American Community Survey data at the census-tract level (33). We estimated tract-level socioeconomic status by creating a *z* score of homeowner percentage, college education percentage, home value, and income. We also assessed whether participants resided in a rural or urban location based on the United States Department of Agriculture 2010 Rural-Urban Commuting Area Codes (34).

## Outcomes

Sleep outcomes were ascertained by self-reported questionnaire between 2012–2015. Chronotype was assessed with a single question based on the Horne-Ostberg Morningness-Eveningness Questionnaire (35). While the full Horne-Ostberg Morningness-Eveningness Questionnaire contains 19 questions, it has been shown that a single question predicts overall chronotype nearly as well as the full questionnaire (36). An adapted version of the Pittsburgh Sleep Quality Index was used to ask participants about their sleep habits over the past month, including duration, latency, trouble sleeping, and any use of sleep-aid medication (37). Sleep duration is the amount of time slept each night and sleep latency is the time it takes for an individual to transition from wakefulness to sleep. We dichotomized our main outcomes, sleep duration ( $\geq 7$  hours vs.  $< 7$  hours) and sleep latency ( $\leq 15$  minutes vs.  $> 15$  minutes), based on the recommendations and consensus of the American Academy of Sleep Medicine, Sleep Research Society, and National Sleep Foundation (2, 38); This and all past CTS questionnaires are available online at <https://www.calteachersstudy.org/>.

## Statistical methods

Pearson correlation coefficients were calculated between the light-at-night, noise, green space, and air pollution exposures. We evaluated the association of the exposures with sleep duration and sleep latency using logistic regression, calculating age-adjusted odds ratios (ORs) and 95% confidence intervals (CIs). In multipollutant models, we also adjusted for race and chronotype. All analyses were conducted in SAS, version 9.4 (SAS Institute, Inc., Cary, North Carolina).

## RESULTS

The average age of participants who responded to questionnaire 5 was 66.9 years (range, 39.6–106.8), and a majority were non-Hispanic White (88%) (Table 1). Most women reported having an early or more-early-than-late chronotype (59%). The most common length of sleep was 7 hours (42%), followed by 8 or more hours (31%). Only 2% of women reported getting less than 5 hours of sleep. Most women reported low sleep latency, 47%  $< 15$  minutes and 36% 15–30 minutes. Only 22% reported not having any

trouble sleeping in the past month, with 20% reporting having trouble more than 3 times/week; however, only 30% reported use of any sleep medication. The vast majority (92%) of participants resided in an urban setting (Rural-Urban Commuting Area Codes 1–3).

The average ALAN among the sleep respondents was 2.81 (standard deviation, 2.09)  $\text{mcd}/\text{m}^2$  and ranged from a minimum of almost no ALAN to 13.1  $\text{mcd}/\text{m}^2$ . The average sound level was 47.37 (standard deviation, 4.07) dB and ranged from 24.9 to 59.1 dB. Participants resided in moderately green areas with an average EVI of 0.21 (standard deviation, 0.06), ranging from  $-0.51$  to 0.59. The average  $\text{PM}_{2.5}$  was 10.43 (standard deviation, 4.04)  $\mu\text{g}/\text{m}^3$  and ranged from 1.71 to 34.2  $\mu\text{g}/\text{m}^3$ . The distributions of these environmental factors across the state of California are available in Web Figure 1 (available at <https://doi.org/10.1093/aje/kwab246>).

ALAN was somewhat correlated with air pollution ( $r = 0.40$ ), and green space was negatively correlated with ALAN ( $r = -0.46$ ) and air pollution ( $r = -0.22$ ) (Web Table 1). There was a modest correlation between self-reported sleep disturbance with latency ( $r = 0.46$ ) and duration ( $r = 0.41$ ) (Web Table 2).

In the single pollutant models, for each 5- $\text{mcd}/\text{m}^2$  increase in light at night, there was a 1.35 (95% CI: 1.29, 1.41) times greater risk of getting less than 7 hours of sleep (Table 2). A 0.1-unit increase in EVI was associated with 0.16 (95% CI: 0.11, 0.22) decreased risk of sleeping less than 7 hours, while each 5- $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$  was associated with a 1.13 (95% CI: 1.10, 1.16) times greater risk of less sleep. A 10-dB increase in sound was associated with 1.05 (95% CI: 1.01, 1.10) increased risk of longer sleep latency (taking  $> 15$  minutes to fall asleep). A 0.1-unit increase in green space was associated with improved sleep latency (OR = 0.57, 95% CI: 0.43, 0.76), and a 5- $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$  was associated with longer sleep latency (OR = 1.04, 95% CI: 1.01, 1.06). Compared with those that reported early chronotype, those that reported the late chronotype had worse sleep duration (OR = 1.22, 95% CI: 1.15, 1.29). The more intermediate chronotypes, however, appeared to have a 15% lower risk of having a shorter sleep duration. There was a consistent, increasing risk of longer sleep latency with later chronotypes ( $P$  for trend  $< 0.001$ ).

In the multipollutant model, adjusting for age, race, chronotype, and other self-reported sleep factors, each 5- $\text{mcd}/\text{m}^2$  increase in light at night was associated with a 1.13 (95% CI: 1.07, 1.20) increased risk of getting less than 7 hours of sleep (Table 3). A 10 dB-increase in sound was not associated with sleep duration but was associated with a 1.05 (95% CI: 1.01, 1.10) increased risk of longer sleep latency (taking  $> 15$  minutes to fall asleep). Increasing EVI was associated with decreased risk of poor sleep latency (OR = 0.55, 95% CI: 0.39, 0.78). Increased air pollution was associated with lower sleep duration (OR = 1.06, 95% CI: 1.04, 1.09) but not sleep latency (OR = 1.02, 85% CI: 1.00, 1.05). Results were similar when stratified to only urban participants (Web Table 3). When we considered only the natural sound model, green space was still associated with better sleep latency (OR = 0.59, 95% CI: 0.41, 0.84), but the association with natural sound intensity and sleep latency was not significant (OR = 1.09, 95% CI: 0.99, 1.20,

**Table 1.** Self-Reported Sleep Outcomes and Built Environment ( $n = 51,562$ ), California Teachers Study Cohort, California, United States, 2012–2015

Characteristic	No.	%
Age, years <sup>a</sup>	66.9 (10.9)	(39.6, 106.8)
Race/ethnicity		
Non-Hispanic White	45,351	88
Other <sup>b</sup>	6,211	12
Light at night, mcd/m <sup>2a</sup>	2.81 (2.09)	(0.01, 13.1)
Noise, dB <sup>a</sup>	47.37 (4.07)	(24.9, 59.1)
Green space <sup>a</sup>	0.21 (0.06)	(−0.51, 0.59)
PM <sub>2.5</sub> air pollution, $\mu\text{g}/\text{m}^3\text{a}$	10.43 (4.04)	(1.71, 34.2)
Chronotype		
Early	22,131	43
More early than late	7,970	15
Neither	6,969	14
More late than early	6,795	13
Late	7,697	15
Sleep duration, hours		
$\geq 8$	16,167	31
7	21,748	42
5–6	12,417	24
$< 5$	1,230	2
Sleep latency, minutes		
$< 15$	24,359	47
16–30	18,776	36
31–60	6,494	13
$> 60$	1,933	4
Sleep medication use, times per week		
Not in past month	36,158	70
$< 1$	5,567	11
1–2	3,020	6
$\geq 3$	6,817	13
Sleep trouble, times per week		
Not in past month	11,506	22
$< 1$	17,066	33
1–2	12,864	25
$\geq 3$	10,126	20

Abbreviations: dB, decibel; mcd, millicandela; PM<sub>2.5</sub>, particulate matter with an aerodynamic diameter  $\leq 2.5 \mu\text{m}$ .  
<sup>a</sup> Values are expressed as mean (standard deviation) and range.

<sup>b</sup> Other races/ethnicities included Black, Hispanic, Native American, Asian/Pacific Islander, and other/mixed.

Web Table 4). Adjusting for our neighborhood-level  $z$  score of socioeconomic status did not alter the associations of the environmental exposures with sleep duration but did attenuate the associations with sleep latency and were no longer statistically significant (Web Table 5).

## DISCUSSION

Self-reported chronotype tended to fall into the earlier (59% “early” or “more early than late”) categories in our

cohort. Those reporting the late chronotype had shorter sleep duration and increased sleep latency. Studies of shift-work show disruptions in sleep for individuals with later chronotypes who have earlier shift schedules (39), a pattern that would be typical in our cohort of school educators.

Light exposure meeting certain thresholds suppresses melatonin, thereby delaying the onset of sleep (6, 7, 40, 41); however, we did not see an association between ALAN and sleep latency. We did see an association with lower sleep duration and increased ALAN. Data on sleep timing

**Table 2.** Characteristics and Environmental Exposures Associated With Sleep ( $n = 51,562$ ), California Teachers Study Cohort, California, United States, 2012–2015

Characteristic	Sleep Duration <7 Hours		Sleep Latency >15 Minutes	
	Age-Adjusted OR	95% CI	Age-Adjusted OR	95% CI
Light at night, 5 mcd/m <sup>2</sup>	1.35	1.29, 1.41	1.02	0.98, 1.07
Noise, 10 dB	1.03	0.98, 1.08	1.05	1.01, 1.10
Green space, 0.1 EVI units	0.16	0.11, 0.22	0.57	0.43, 0.76
PM <sub>2.5</sub> air pollution, 5 µg/m <sup>3</sup>	1.13	1.10, 1.16	1.04	1.01, 1.06
Chronotype				
Early	1.00	Referent	1.00	Referent
More early	0.79	0.74, 0.84	1.13	1.07, 1.19
Neither	0.87	0.82, 0.93	1.24	1.17, 1.31
More late	0.84	0.79, 0.90	1.48	1.40, 1.56
Late	1.22	1.15, 1.29	1.65	1.56, 1.74
Race/ethnicity, non-Hispanic White vs. other	2.08	1.96, 2.19	1.15	1.09, 1.21
Self-reported use of sleep medication, never vs. ever	1.55	1.59, 1.62	2.82	2.71, 2.94
Self-reported trouble sleeping, never vs. ever	2.79	2.64, 2.96	4.25	4.06, 4.46

Abbreviations: CI, confidence interval; dB, decibel; EVI, enhanced vegetation index; mcd, millicandela; OR, odds ratio; PM<sub>2.5</sub>, particulate matter with an aerodynamic diameter  $\leq 2.5$  µm.

were not collected for the cohort, so we were unable to assess the association between the environment and when participants went to sleep. Beyond the associations seen with sleep disruption and disease risk (7), studies have also shown an association with ALAN and tumor progression (42, 43). In contrast to ALAN, noise pollution was not significantly associated with sleep duration but was associated with sleep latency. This is consistent with several studies that have shown an association with sound and increase in sleep latency (44–46).

Green space was significantly associated with longer sleep duration and shorter sleep latency. While we have no data on participant use of green space, in a review

by Shin et al. (12), the associations of green space with improved sleep outcomes were shown to not only be limited to studies in which participant use of green space was assessed, but they were seen in studies that only evaluated study participant surroundings. Green space is believed to help improve mood and mental health as well as mitigate noise pollution, air pollution, and ALAN, leading to reduced morbidity and mortality (13, 23, 47, 48). Some studies have reported potential gene-environment interaction, indicating some individuals may have differential benefit from residing in green spaces (25, 49). We saw an inverse association with EVI and ALAN ( $r = -0.46$ ) and air pollution ( $r = -0.22$ ). The associations of green space do not seem to be

**Table 3.** Multipollutant Model of Environmental Exposures and Sleep ( $n = 51,562$ ), California Teachers Study Cohort, California, United States, 2012–2015

Environmental Exposure	Sleep Duration <7 Hours <sup>a</sup>		Sleep Latency >15 Minutes <sup>b</sup>	
	OR	95% CI	OR	95% CI
Light at night, 5 mcd/m <sup>2</sup>	1.13	1.07, 1.20	0.97	0.91, 1.02
Noise, 10 dB	1.02	0.97, 1.08	1.05	1.01, 1.10
Green space, 0.1 EVI units	0.41	0.28, 0.60	0.55	0.39, 0.78
PM <sub>2.5</sub> air pollution, 5 µg/m <sup>3</sup>	1.06	1.04, 1.09	1.02	1.00, 1.05

Abbreviations: CI, confidence interval; dB, decibel; EVI, enhanced vegetation index; mcd, millicandela; OR, odds ratio; PM<sub>2.5</sub>, particulate matter with an aerodynamic diameter  $\leq 2.5$  µm.

<sup>a</sup> Adjusted for age, race, chronotype, use of sleep medication, trouble sleeping, and sleep latency.

<sup>b</sup> Adjusted for age, race, chronotype, use of sleep medication, trouble sleeping, and sleep duration.

limited entirely to the surrounding environment, as studies have shown that viewing simulated imagery of green space can also improve both mood and sleep (50–52). While we did control for self-reported trouble sleeping, this may not fully capture psychosocial stressors of the built environment (53) that can contribute to poor sleep (54). Results were consistent when restricted to participants residing in urban Rural-Urban Commuting Area Codes (Web Table 3). When we adjusted for other features of the built environment, with census tract-level socioeconomic status as a proxy, the associations between exposures and sleep latency were similar but no longer significant (Web Table 5). However, the size of the census tracts, as they are based on population, compared with the resolution of other data sources, may obscure real patterns. A recent study by McIsaac et al. (29) demonstrated the potential to misclassify exposure as spatial resolution decreased.

Air pollution was significantly associated with reduced sleep duration and was marginally significant for increased sleep latency. There was a modest inverse correlation between air pollution and green space, and the association with air pollution was attenuated in the multipollutant model while the association of sound on sleep was not. Air pollution's detrimental association with sleep may not be limited to individuals with asthma or other respiratory conditions that are exacerbated by poor air quality (14, 15). Gene-environment interactions have also been observed for light at night (55) and air pollution (56). Interestingly, there was no correlation between sound levels and any of the other exposures. Unlike ALAN and air pollution, which are largely human-made, the noise model includes ambient sound (nature) as well as human-made sound. When we evaluated natural sound only, it was no longer significantly associated with sleep latency (per 5 dB, OR = 1.09, 95% CI: 0.99, 1.20; Web Table 4), indicating that the association may be attributed more heavily to anthropogenic sound. In the multipollutant model, associations with sleep duration were attenuated substantially but less so for sleep latency. These factors likely point to shared attributes of the built environment that may work synergistically to disrupt sleep.

Strengths of this study include a large sample size, comprising 50,000 California teachers and school administrators. Our continued follow-up provided high-quality residential address history of participants and allowed us to assess multiple environmental exposures. Our measure of ALAN, the New World Atlas of Artificial Night Sky Brightness, better captures ground-level exposure to light at night than raw values of upward radiance recorded by satellites (28). Additionally, our air pollution model provides a highly accurate and validated measure of PM<sub>2.5</sub> at a 1-km resolution compared with use of sparsely located air-quality monitoring stations (32).

Study limitations included the self-reported nature of the sleep variables. We also did not have information on the timing of sleep, nor could we assess a direct correlation between indoor exposure to light and exposure to outdoor light. One small study did not see a correlation between outdoor light and indoor light exposure in the bedroom (57); however, light exposure and disruptions to sleep have

been found to persist for hours (40, 41). In a recent Dutch study of children, individual-level light exposure showed the influence of outdoor light on indoor light during the darkest time period with a correlation of 0.31 (58), even when nearly all bedrooms had blackout curtains. In a survey of lighting designers using their own light meters, Miller and Kinzey (59) reported measurements in a number of different contexts within homes. At windows without drapes, a maximum of 20 lux was reported, with a mean of 5 lux and median of 0.5 lux. We also did not have information on any use of handheld devices before bedtime. The US Parks noise model provided daytime levels incorporating ambient sound, unlike many noise models that are largely focused on traffic and not as accurate on smaller roadways in less dense urban areas (60). Similar to ALAN, it may be the exposure prior to sleep and waking that are most associated with sleep disruption, and not necessarily continued exposure during the nighttime (44, 45).

In summary, higher levels of light at night and air pollution were associated with less sleep duration, and higher sound levels were associated increased sleep latency. There was a significant positive association with sleep duration and latency from green space. In the multipollutant model, the detrimental association of light at night and air pollution with sleep duration were attenuated, but this was not so for sleep latency. As our understanding of sleep improves, additional studies need to be done to understand the mechanisms by which the environment affects sleep.

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members is available at <https://www.calteachersstudy.org/team>.

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