Sleep Medicine 54 (2019) 121–125

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Pre-awake light exposure and sleep disturbances: findings from the HEIJO-KYO cohort

Kenji Obayashi*, Yuki Yamagami, Norio Kurumatani, Keigo Saeki

Department of Epidemiology, Nara Medical University School of Medicine, Nara, Japan

ARTICLE INFO

Article history:
Received 18 July 2018
Received in revised form
6 September 2018
Accepted 17 October 2018
Available online 14 November 2018

Keywords:
Sleep
Actigraphy
Pre-awake
Light at night
Circadian rhythm

ABSTRACT

Objectives: To evaluate the associations between pre-awake light (PAL) exposure and subjective and objective measures of sleep quality.
Methods: In this cross-sectional study of 1108 elderly participants (mean age, 71.9 years), we measured bedroom light intensity using a bedside light meter for two nights and sleep quality using actigraphy and a questionnaire. PAL was determined as the 2h-average light intensity before rise time, and sleep disturbance was defined as the Pittsburgh sleep quality index score ≥6.
Results: Sleep disturbance prevalence increased with increased PAL exposure ($P = 0.002$). In multivariable models adjusted for potential confounders, the odds ratio (OR) for sleep disturbances was significantly higher in the highest quartile PAL group (Q4) than in the lowest quartile group (Q1) (OR, 1.65; 95% CI, 1.16–2.34). This association occurred independent of post-bedtime light exposure; and was stronger in the later chronotype group (n = 556) (OR, 1.80; 95% CI, 1.05–3.09) than in the earlier chronotype group (n = 552) (OR, 1.64; 95% CI, 1.01–2.64). Actigraphic sleep efficiency in the Q4 group was significantly lower by 2.6% (95% CI, 1.3–3.8) than that in the Q1 group. Moreover, longer wake after sleep onset by 7.5 min (95% CI, 2.8–12.2) and sleep onset latency by 0.2 log min (95% CI, 0.1–0.4) were observed in the Q4 group than the Q1 group.
Conclusions: Higher PAL exposure was significantly associated with a higher prevalence of sleep disturbances, independent of post-bedtime light exposure. Consistent results were observed in the actigraphy analysis.

1. Introduction

Sleep disturbances are common in elderly and young populations, with prevalence estimates as high as 40% in recent years [1–4]. Poor sleep is associated with increased risk of a variety of diseases, such as depression, dementia, and cardiovascular diseases [5–7]. Several factors increase the risk of sleep disturbances; and circadian misalignment between internal biological and external environments is an important risk factor [8]. Chronic circadian misalignment (typically related to night-shift work or clock-gene variants), is associated with disrupted function of suprachiasmatic nucleus (SCN), the master biological clock, and increased risk of sleep disturbances [9–11].

Light exposure at an inappropriate timing against the sleep–wake cycles can lead to the circadian misalignment [12,13]. Numerous experimental studies suggested that nocturnal inputs of light information into the SCN increase core body temperature, suppress melatonin secretion, and stimulate brain activity under controlled laboratory conditions [14,15]. In epidemiological studies, sleep quality was affected by the light exposure at night [16,17]. To date, most studies reporting the effects of nighttime light exposure on sleep quality focused on light exposure around bedtime. To our knowledge, no studies have evaluated the effects of light exposure during the pre-awake period, which is several hours before waking up.

In our previous publication, we reported that humans are commonly exposed to pre-awake light (PAL), the main source of which is morning sunlight through the bedroom window [18]. Morning sunlight was believed to possess health benefits; however, human lifestyle in modern society often forces individuals to stay up late, resulting in late chronotype [19]. Therefore, PAL exposure may disturb circadian physiology and sleep in humans. In this

* Corresponding author. Department of Epidemiology, Nara Medical University School of Medicine, 840 Shijocho, Kashiharashi, Nara, 634-8521, Japan. Fax: +81 744 29 0673.
E-mail address: obayashi@naramed-u.ac.jp (K. Obayashi).

https://doi.org/10.1016/j.sleep.2018.10.027
1389-9457/

cross-sectional study of 1108 elderly individuals, we evaluated the associations of PAL exposure measured using a bedside light meter with subjective and actigraphic measures of sleep quality.

2. Participants and methods

2.1. Participants

A total of 1127 community-based elderly subjects (≥60 years) were voluntarily enrolled in a study entitled "Housing Environments and Health Investigation among Japanese Older People in Nara, Kansai Region: a prospective community-based cohort (HEIJO-KYO) study" between September and March in 2010, 2011, 2012, 2013, and 2014 [20]. Among these, 1108 home-dwelling participants of the HEIJO-KYO cohort with complete measurement sets of bedroom light intensities and sleep questionnaires were included in this cross-sectional analysis. The study protocol was approved by the medical ethics committee of Nara Medical University.

2.2. Measurement of PAL exposure

Bedroom nighttime light intensity (self-reported bedtime to rising time) was measured for two consecutive nights at 1-min intervals using a portable light meter (LX-28SD; Sato Shouji Inc., Kanagawa, Japan), which was placed facing the ceiling at the head of the participant's bed 60 cm above the floor [illuminance sensitivity, 0–100,000 lux; resolution, 1 lux (under 2000 lux); and accuracy, ± (4% reading + 2 digits, under 2000 lux)]. PAL exposure was treated as the 2 h-average light intensity before rising time; post-bedtime light exposure was the 4 h-average light intensity after bedtime.

2.3. Measurements of sleep quality

Subjective sleep quality was measured using the Pittsburgh sleep quality index (PSQI) questionnaire [21]. Specifically, sleep quality over the previous month was determined using seven subscales that measured different components of sleep: sleep quality, latency, duration, efficiency, disturbances, sleep medication use, and daytime dysfunction. Each component was scored from 0 to 3, with 3 indicating the worst sleep quality. Sleep disturbances were determined as a global score ≥6 and/or a previous diagnosis of insomnia and current sleep medication use.

Objective sleep quality was measured using an actigraph (Actiwatch 2; Respironics Inc., PA, USA), worn on the non-dominant wrist, at 1-min intervals on two consecutive nights. The sleep statuses at each epoch, sleep onset, and sleep termination were determined by Actiware version 5.5 (Respironics Inc.) using the default algorithm. Epochs with higher-than-moderate-activity counts (40 counts/min) were treated as awake. Sleep onset was defined as the first minute followed by a 10-min period of immobility, comprising ≥4 counts/min. Sleep termination was determined as the last minute following a 10-min period of immobility. Four actigraphic sleep parameters were determined using the following objective data (sleep status and sleep onset and termination) and self-reported data (bedtime and rising time): sleep efficiency (SE) [percentage calculated from time spent sleeping (below the activity threshold of 40 counts/min) between sleep onset and sleep offset divided by the time between bedtime and rising time]; wake after sleep onset (WASO) [time spent awake (activity above the threshold of 40 counts/min) between sleep onset and rising time]; sleep onset latency (SOL) [time between bedtime and sleep onset]; and total sleep time (TST) [sleep duration (time between bedtime and rising time) multiplied by SE].

2.4. Measurement of covariates

Body mass index (BMI) was calculated as body weight divided by the square of body height (kg/m^2). Smoking and drinking habits and medication use were evaluated using a self-administered questionnaire. The presence of hypertension was determined on the basis of medical history and current use of antihypertensive drugs. Diabetes mellitus was determined on the basis of medical history, current use of anti-diabetic therapy, and glycated hemoglobin levels ≥6.5% as per the National Glycohemoglobin Standardization Program value. Daytime physical activity was measured at 1-min intervals using an actigraph (Actiwatch 2) between rise time and bedtime. As an index of melatonin secretion, urinary 6-sulfatoxymelatonin excretion was measured from urine samples collected overnight as described in our previous publication [20]. Day lengths (sunrise to sunset), based on the measurement days in Nara, Japan, were extracted from the website of the National Astronomical Observatory of Japan.

2.5. Statistical analyses

Participants were divided into quartiles according to their PAL intensity (lowest–highest, Q1–Q4) and we analyzed association trends for the variables using linear and logistic regression models or the Jonckheere–Terpstra test, as appropriate. In the logistic regression analysis and analysis of covariance, odds ratios (OR) for sleep disturbances and mean values of sleep quality in relation to quartiles of PAL intensities were adjusted for age (≥70 years), gender (male vs. female), BMI (≥25 kg/m^2), current smoking status (yes vs. no), alcohol consumption (≥30 g/day), hypertension (yes vs. no), diabetes (yes vs. no), daytime physical activity (per 100 counts/min), melatonin secretion (per log μg), bedtime (per 1 h delay), rising time (per 1 h delay), day length (per quartile), and post-bedtime light exposure (per quartile). A stratified analysis was performed using the median value of sleep-mid time (earlier vs. later chronotypes). All analyses were performed using SPSS version 23.0 for Windows (IBM-SPSS, Inc., Chicago, IL, USA). A two-sided P value < 0.05 was considered to be statistically significant.

3. Results

The mean age of the 1108 participants was 71.9 (SD, 7.1) years, and 521 (47.0%) individuals were male. Time-dependent changes in light intensity from bedtime to rise time between the lowest quartile group (Q1) and highest quartile group (Q4) of PAL exposure are shown in Fig. 1. The mean bedroom light intensity of the Q4 group increased from 2-h before rise time; at 30 min before rising time it was over 50 lux. The overall median PAL intensity was 0.7 lux (interquartile range, 0–5.1); the median PAL intensity in Q1 was 0 lux (range, <0.004); Q2, 0.2 lux (0.004–0.8); Q3, 1.9 lux (0.8–5.1); and Q4, 11.7 lux (>5.1). Higher PAL intensities were significantly associated with older age, lower daytime physical activity, later rising time, and longer day length (Table 1).

The overall prevalence of sleep disturbances was 37.2%; the prevalence of sleep disturbances increased with increasing quartiles of PAL exposure (Q1, 32.2%; Q2, 33.7%, Q3, 37.7%, and Q4, 45.4%; P for trend = 0.002; Table 2). The unadjusted OR for sleep disturbances was significantly higher in Q4 than in Q1 [OR, 1.75; 95% confidence interval (CI), 1.25–2.47; $P = 0.001$]. This was consistent in the analysis where sleep disturbances were determined using the PSQI score only (prevalence, 35.3%) (OR, 1.71; 95% CI, 1.21–2.42; $P = 0.002$). In addition, this association between PAL exposure and sleep disturbances was consistent in the age- and gender-adjusted model (model 1: OR, 1.66; 95% CI, 1.17–2.36; $P = 0.004$) and multivariable models (model 2: OR, 1.65; 95% CI, 1.16–2.34;

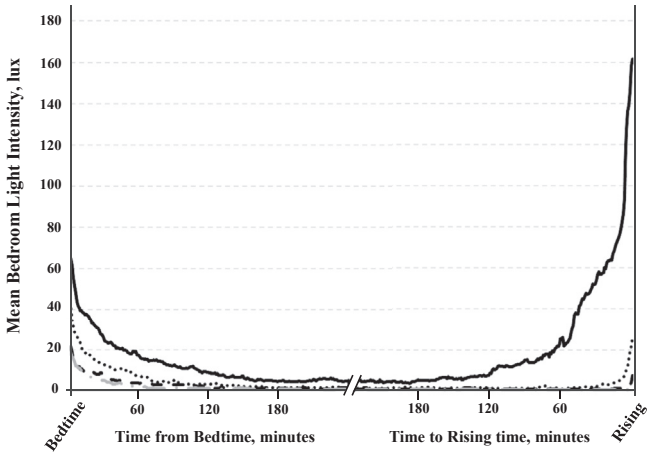

Fig. 1. Time-dependent changes in light intensity from bedtime to rising time. The solid line indicates the average pre-awake light intensity in the highest quartile group (Q4); the short-dotted line indicates the average pre-awake light intensity in the second highest quartile group (Q3); the long-dotted line indicates the average pre-awake light intensity in the third highest quartile group (Q2); and the grey-dotted line indicates the average pre-awake light intensity in the lowest quartile group (Q1).

$P = 0.006$; model 3: OR, 1.56; 95% CI, 1.07–2.25; $P = 0.019$). Furthermore, after adjusting for post-bedtime light exposure, the results remained significant (model 4: OR, 1.63; 95% CI, 1.10–2.42; $P = 0.016$). When divided into two groups using the median value of sleep-mid time, this association between PAL exposure and sleep disturbance was stronger in the later chronotype group (n = 556) (model 2: OR, 1.80; 95% CI, 1.05–3.09; $P = 0.033$) than in the earlier chronotype group (n = 552) (model 2: OR, 1.64; 95% CI, 1.01–2.64; $P = 0.044$).

Regarding objective sleep quality, the overall mean SE was 84.7% (SD, 7.6); WASO, 49.6 min (SD, 29.0); SOL, 3.0 log min (SD, 1.0); and TST, 420.1 min (SD, 69.0). Multivariable analysis adjusted for age, gender, BMI, smoking and drinking habits, and sleep medication use, revealed significantly lower SE in Q4 than in Q1 (mean difference: 2.6%; 95% CI, 1.3–3.8; $P < 0.001$) (Table 3). WASO and SOL were significantly longer in Q4 than in Q1 (mean difference: WASO, 7.5 min; 95% CI, 2.8–12.2; $P = 0.002$; SOL, 0.2 log min; 95% CI, 0.1–0.4; $P = 0.002$). In contrast, TST was significantly longer in Q4 than in Q1 (mean difference: 15.1 min; 95% CI, 3.8–26.3; $P = 0.009$).

4. Discussion

The results of the present study suggest that higher PAL exposure was significantly associated with a higher prevalence of sleep disturbances in the general elderly population, independent of post-bedtime light exposure and other several potential confounding factors. Quantitative associations between PAL exposure and sleep quality were suggested in the analysis using actigraphy data. Furthermore, the association between PAL exposure and sleep disturbances may be stronger in the later chronotype group than in the earlier chronotype group. This is, to the best of our knowledge, the first report of the association between PAL exposure and sleep quality in a large population.

PAL exposure is common and its intensity seems to be high to disturb sleep. Bedroom light exposure patterns at night include three phenotypes 'the first-sided', 'constant', and 'the last-sided' patterns [22]. 'The last-sided' pattern was the PAL exposure in the present study, and humans are exposed to PAL, mainly caused by morning sunlight through the bedroom window. As shown in the figure, the mean bedroom light intensity of the highest quartile PAL group increased beginning 2-h before rising time, reaching over 50 lux 30 min before rising time. These light levels may affect sleep physiology and daytime brain function [12,15]. In addition, PAL exposure may be reduced by blackout curtains, window shutters, or eye masks.

Our data suggested morning sunlight in the bedroom was a potential risk factor for sleep disturbances; however, for a long time, morning sunlight was thought to be beneficial for health [23]. The human circadian timing system developed based on the solar 24-h cycle. However, in modern society, human sleep–wake timing has shifted away from the solar cycle [19]. Therefore, it is possible that morning sunlight can disturb circadian physiology and behaviors, resulting in sleep disturbances. The present study suggested that higher PAL was significantly associated with a higher prevalence of sleep disturbances in our study cohort. Although PAL intensity correlated with post-bedtime light intensity as shown in the figure, the association between PAL exposure and sleep disturbances was independent of post-bedtime light exposure. Notably, in our subgroup analysis, the later chronotype group exhibited a stronger association between PAL exposure and sleep disturbances than the earlier chronotype group. This suggest that PAL exposure may partly explain the underlying mechanism of the previous knowledge of the high prevalence of sleep disturbances in

Table 1
Basic and clinical characteristics according to PAL exposure (n = 1108).

Characteristics	Quartiles of PAL intensity (lux) [median, range]				
	Q1	Q2	Q3	Q4	P_{trend}
	0 [<0.004]	0.2 [0.004–0.8]	1.9 [0.8–5.1]	11.7 [>5.1]	
No. of participants	289	273	273	273	
Basic parameters					
Age, mean (SD), years	70.9 (7.1)	71.2 (7.1)	72.5 (6.9)	72.9 (7.1)	<0.001
Gender, male, number	137 (47.4%)	115 (42.1%)	137 (50.2%)	132 (48.4%)	0.43
Body mass index, mean (SD), kg/m^2	22.8 (2.9)	23.4 (3.2)	23.0 (3.0)	23.2 (3.1)	0.28
Current smoker, number	12 (4.5%)	15 (5.5%)	13 (4.8%)	15 (5.5%)	0.56
Alcohol consumption (≥30 g/day), number	47 (16.3%)	32 (11.7%)	38 (13.9%)	43 (15.8%)	0.97
Clinical parameters					
Hypertension, number	128 (44.3%)	117 (42.9%)	120 (44.0%)	127 (46.5%)	0.57
Diabetes, number	36 (14.3%)	33 (12.1%)	43 (15.8%)	42 (15.4%)	0.18
Daytime physical activity, mean (SD), count/min	312.2 (105.6)	291.2 (99.8)	299.8 (102.1)	279.0 (105.3)	<0.001
Melatonin secretion, median (IQR), μg/night	6.7 (3.8, 10.5)	6.4 (4.2, 10.0)	7.1 (4.1, 10.9)	6.8 (4.1, 10.9)	0.31
Bedtime, mean (SD), clock time	22:25 (1:06)	22:36 (1:07)	22:30 (1:11)	22:24 (1:17)	0.73
Rising time, mean (SD), clock time	6:23 (1:01)	6:45 (0:47)	6:55 (0:49)	7:03 (0:57)	<0.001
Day length, median (IQR), min	648 (613, 676)	645 (619, 676)	658 (628, 692)	662 (625, 700)	<0.001

PAL, pre-awake light; SD, standard deviation; IQR, interquartile range.

Table 2
Odds ratio for sleep disturbances in relation to PAL exposure.

	Quartiles of PAL intensity				
	Q1	Q2	Q3	Q4	P_{trend}
No. of participants	289	273	273	273	
No. of cases, prevalence	93 (32.2%)	92 (33.7%)	103 (37.7%)	124 (45.4%)	
Unadjusted OR (95% CI)	1.00 (ref)	1.07 (0.75–1.52)	1.28 (0.90–1.81)	1.75 (1.25–2.47)	0.002
P		0.70	0.17	0.001	
Model 1					
Adjusted OR (95% CI)	1.00 (ref)	1.03 (0.72–1.47)	1.21 (0.85–1.72)	1.66 (1.17–2.36)	0.003
P		0.88	0.30	0.004	
Model 2					
Adjusted OR (95% CI)	1.00 (ref)	1.02 (0.71–1.47)	1.21 (0.85–1.72)	1.65 (1.16–2.34)	0.003
P		0.90	0.30	0.006	
Model 3					
Adjusted OR (95% CI)	1.00 (ref)	1.00 (0.69–1.45)	1.19 (0.82–1.72)	1.56 (1.07–2.25)	0.012
P		0.998	0.35	0.019	
Model 4					
Adjusted OR (95% CI)	1.00 (ref)	1.01 (0.70–1.46)	1.23 (0.84–1.80)	1.63 (1.10–2.42)	0.010
P		0.96	0.29	0.016	

PAL, pre-awake light; IQR, interquartile range; OR, odds ratio; CI, confidence interval.
Model 1: adjusted for age and gender.
Model 2: adjusted for variables in model 1 plus body mass index, smoking, and drinking.
Model 3: adjusted for variables in model 2 plus hypertension, diabetes, daytime physical activity, melatonin secretion, bedtime, rising time, and day length.
Model 4: adjusted for variables in model 3 plus post-bedtime light exposure.

the late chronotype. Moreover, our result suggests the effects of PAL exposure on sleep quality may be stronger among younger populations whose chronotypes are later than those of our study participants aged older than 60 years. Further epidemiological studies evaluating PAL exposure and sleep quality in younger populations are needed.

The present study included important strengths in that we used objective measures in a large study cohort. First, PAL intensity was measured using a bedside light meter. Most previous studies evaluating the association between nighttime indoor light levels and health outcomes assessed light levels using self-reported questionnaires; however, self-reported indoor light levels have not been validated [24,25]. Second, our results suggest consistent associations of PAL exposure with both subjective and objective sleep measures. These associations may improve the reliability of our results because some inconsistencies between subjective and objective sleep measures were reported [26]. In addition, our results suggested quantitative associations between PAL exposure and actigraphic measures of sleep quality, specifically an association between PAL exposure and longer sleep durations. This requires further investigation

Table 3
Actigraphic sleep parameters in relation to PAL exposure.

	Quartiles of PAL intensity				
	Q1	Q2	Q3	Q4	
Unadjusted	Mean (5%–95% range)				P_{trend}
SE, %	86.2 (85.5, 86.9)	84.9 (83.8, 85.9)	84.3 (83.4, 85.1)	83.4 (82.4, 84.3)	<0.001
Mean difference, % (95% CI)	reference	−1.3 (−2.6, −0.1)	−1.9 (−3.2, −0.7)	−2.8 (−4.1, −1.6)	
P		0.037	0.003	<0.001	
WASO, min	45.1 (42.1, 48.2)	48.2 (44.8, 51.7)	51.2 (47.5, 54.9)	54.0 (50.4, 57.5)	<0.001
Mean difference, % (95% CI)	reference	3.1 (−1.7, 7.9)	6.0 (1.2, 10.8)	8.8 (4.0, 13.6)	
P		0.21	0.014	<0.001	
SOL, log min	2.8 (2.7, 2.9)	2.9 (2.7, 3.0)	3.1 (2.9, 3.2)	3.1 (3.0, 3.2)	<0.001
Mean difference, % (95% CI)	reference	0.4 (−0.1, 0.2)	0.2 (0.1, 0.4)	0.3 (0.1, 0.4)	
P		0.62	0.003	0.001	
TST, min	411.8 (404.0, 419.6)	413.8 (405.5, 422.1)	424.1 (415.9, 432.3)	430.9 (422.6, 439.3)	<0.001
Mean difference, % (95% CI)	reference	2.0 (−9.5, 13.4)	12.3 (0.9, 23.7)	19.1 (7.7, 30.5)	
P		0.74	0.035	0.001	
Adjusted[a]	Mean (95% CI)				P_{trend}
SE, %	86.1 (85.2, 86.9)	84.7 (83.8, 85.6)	84.4 (83.5, 85.3)	83.5 (82.6, 84.4)	<0.001
Mean difference, % (95% CI)	reference	−1.3 (−2.6, −0.1)	−1.7 (−2.9, −0.4)	−2.6 (−3.8, −1.3)	
P		0.035	0.009	<0.001	
WASO, min	45.8 (42.6, 49.1)	48.9 (45.6, 52.3)	50.4 (47.1, 53.7)	53.3 (50.0, 56.6)	0.002
Mean difference, % (95% CI)	reference	3.1 (−1.5, 7.8)	4.6 (−0.1, 9.3)	7.5 (2.8, 12.2)	
P		0.19	0.054	0.002	
SOL, log min	2.8 (2.7, 2.9)	2.9 (2.8, 3.0)	3.0 (2.9, 3.2)	3.1 (3.0, 3.2)	0.001
Mean difference, % (95% CI)	reference	0.03 (−0.1, 0.2)	0.2 (0.1, 0.4)	0.2 (0.1, 0.4)	
P		0.57	0.010	0.003	
TST, min	413.8 (406.1, 421.6)	415.5 (407.4, 423.5)	422.3 (414.3, 430.3)	428.9 (420.9, 437.0)	0.004
Mean difference, % (95% CI)	reference	1.6 (−9.6, 12.8)	8.5 (−2.7, 19.7)	15.1 (3.8, 26.3)	
P		0.78	0.14	0.009	

PAL, pre-awake light; SE, sleep efficiency; SOL, sleep-onset latency; WASO, wake after sleep-onset; TST, total sleep time; CI, confidence interval.
[a] Adjusted for age, gender, body mass index, smoking, drinking, and sleep medication.

because long sleep in the elderly may affect health outcomes [27]. Third, the large sample size allowed multivariable statistical models to be constructed that included important confounding factors and enabled stratified analyses based on chronotypes.

Our study has several potential limitations. First, the cross-sectional design limited causality inference for association between PAL exposure and sleep disturbances. Furthermore, prospective studies investigating the influence of PAL exposure on sleep quality are warranted, although a longitudinal association between PAL exposure and metabolic parameters were reported in our previous study [28]. Second, participants were not randomly selected, possibly leading to selection bias. However, body mass index and estimated glomerular filtration rate were similar to the corresponding national data for the elderly Japanese population. Third, bedroom light intensity was measured only over two nights, possibly leading to misclassification of PAL exposure status. Yet, our previous studies reported moderate night-to-night reproducibility of bedroom light intensity [20].

In conclusion, our study suggested that higher PAL exposure was significantly associated with a higher prevalence of sleep disturbances in the general elderly population. This association occurred independent of post-bedtime light exposure and several potential confounding factors, where the association may be stronger in the later chronotype group than in the earlier chronotype group. This suggest that PAL exposure may partly explain the underlying mechanism of the previous knowledge of the high prevalence of sleep disturbances in the late chronotype. Further prospective studies investigating the influence of PAL exposure on sleep quality in younger populations are warranted.

Acknowledgments

We would like to thank Naomi Takenaka, Sachiko Sogahara, Keiko Nakajima, Yumi Takeda, and Megumi Natsuaki for their valuable help with data collection and analysis. This work was supported by research funding from Department of Indoor Environmental Medicine, Nara Medical University; JSPS KAKENHI (grant numbers: 24790774, 22790567, 25860447, 25461393, 15H04776, and 10124877); Mitsui Sumitomo Insurance Welfare Foundation; Meiji Yasuda Life Foundation of Health and Welfare; Osaka Gas Group Welfare Foundation; Japan Diabetes Foundation; Daiwa Securities Health Foundation; Japan Science and Technology Agency; YKK AP Inc.; Ushio Inc.; Nara Prefecture Health Promotion Foundation; Nara Medical University Grant-in-Aid for Collaborative Research Projects; Tokyo Electric Power Company; EnviroLife Research Institute Co., Ltd.; and Sekisui Chemical Co., Ltd. The funders had no role in the study design, data collection and analysis, decision to publish, and preparation of the manuscript.

Conflict of interest

KO and KS received research grant from YKK AP Inc., Ushio Inc.; Tokyo Electric Power Company; EnviroLife Research Institute Co., Ltd.; and Sekisui Chemical Co., Ltd. YY and NK reported no conflicts of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2018.10.027.

References

[1] Calem M, Bisla J, Begum A, et al. Increased prevalence of insomnia and changes in hypnotics use in England over 15 years: analysis of the 1993, 2000, and 2007 National Psychiatric Morbidity Surveys. Sleep 2012;35:377–84.
[2] Walsh JK, Coulouvrat C, Hajak G, et al. Nighttime insomnia symptoms and perceived health in the America Insomnia Survey (AIS). Sleep 2011;34:997–1011.
[3] Kim K, Uchiyama M, Okawa M, et al. Sleep 2000;23:41–7.
[4] Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? JAMA 1989;262:1479–84.
[5] Yokoyama E, Kaneita Y, Saito Y, et al. Association between depression and insomnia subtypes: a longitudinal study on the elderly in Japan. Sleep 2010;33:1693–702.
[6] Cricco M, Simonsick EM, Foley DJ. The impact of insomnia on cognitive functioning in older adults. J Am Geriatr Soc 2001;49:1185–9.
[7] Eaker ED, Pinsky J, Castelli WP. Myocardial infarction and coronary death among women: psychosocial predictors from a 20-year follow-up of women in the Framingham Study. Am J Epidemiol 1992;135:854–64.
[8] Arendt J. Melatonin, circadian rhythms, and sleep. N Engl J Med 2000;343:1114–6.
[9] Wyatt JK, Ritz-De Cecco A, Czeisler CA, et al. Circadian temperature and melatonin rhythms, sleep, and neurobehavioral function in humans living on a 20-h day. Am J Physiol 1999;277:R1152–63.
[10] Boudreau P, Dumont GA, Boivin DB. Circadian adaptation to night shift work influences sleep, performance, mood and the autonomic modulation of the heart. PLoS One 2013;8, e70813.
[11] Allebrandt KV, Teder-Laving M, Akyol M, et al. CLOCK gene variants associate with sleep duration in two independent populations. Biol Psychiatry 2010;67:1040–7.
[12] Zeitzer JM, Dijk DJ, Kronauer R, et al. Sensitivity of the human circadian pacemaker to nocturnal light: melatonin phase resetting and suppression. J Physiol 2000;526:695–702.
[13] Khalsa SB, Jewett ME, Cajochen C, et al. A phase response curve to single bright light pulses in human subjects. J Physiol 2003;549:945–52.
[14] Cajochen C, Münch M, Kobialka S, et al. High sensitivity of human melatonin, alertness, thermoregulation, and heart rate to short wavelength light. J Clin Endocrinol Metab 2005;90:1311–6.
[15] Cajochen C, Zeitzer JM, Czeisler CA, et al. Dose-response relationship for light intensity and ocular and electroencephalographic correlates of human alertness. Behav Brain Res 2000;115:75–83.
[16] Obayashi K, Saeki K, Iwamoto J, et al. Effect of exposure to evening light on sleep initiation in the elderly: a longitudinal analysis for repeated measurements in home settings. Chronobiol Int 2014;31:461–7.
[17] Koo YS, Song JY, Joo EY, et al. Outdoor artificial light at night, obesity, and sleep health: cross-sectional analysis in the KoGES study. Chronobiol Int 2016;33:301–14.
[18] Obayashi K, Saeki K, Kurumatani N. Bedroom light exposure at night and the incidence of depressive symptoms: a longitudinal study of the HEIJO-KYO cohort. Am J Epidemiol 2018;187:427–34.
[19] Roenneberg T, Allebrandt KV, Merrow M, et al. Social jetlag and obesity. Curr Biol 2012;22:939–43.
[20] Obayashi K, Saeki K, Iwamoto J, et al. Positive effect of daylight exposure on nocturnal urinary melatonin excretion in the elderly: a cross-sectional analysis of the HEIJO-KYO study. J Clin Endocrinol Metab 2012;97:4166–73.
[21] Buysse DJ, Reynolds 3rd CF, Monk TH, et al. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatr Res 1989;28:193–213.
[22] Obayashi K, Saeki K, Iwamoto J, et al. Exposure to light at night, nocturnal urinary melatonin excretion, and obesity/dyslipidemia in the elderly: a cross-sectional analysis of the HEIJO-KYO study. J Clin Endocrinol Metab 2013;98:337–44.
[23] Mead MN. Benefits of sunlight: a bright spot for human health. Environ Health Perspect 2008;116:A160–7.
[24] Davis S, Mirick DK, Stevens RG. Night shift work, light at night, and risk of breast cancer. J Natl Cancer Inst 2001;93:1557–62.
[25] McFadden E, Jones ME, Schoemaker MJ, et al. The relationship between obesity and exposure to light at night: cross-sectional analyses of over 100,000 women in the Breakthrough Generations Study. Am J Epidemiol 2014;180:245–50.
[26] Mehra R, Stone KL, Ancoli-Israel S, et al., Study of Osteoporotic Fractures Research Group. Interpreting wrist actigraphic indices of sleep in epidemiologic studies of the elderly: the Study of Osteoporotic Fractures. Sleep 2008;31:1569–76.
[27] Hall MH, Smagula SF, Boudreau RM, et al. Association between sleep duration and mortality is mediated by markers of inflammation and health in older adults: the Health, Aging and Body Composition Study. Sleep 2015;38:189–95.
[28] Obayashi K, Saeki K, Kurumatani N. Ambient light exposure and changes in obesity parameters: a longitudinal study of the HEIJO-KYO cohort. J Clin Endocrinol Metab 2016;101:3539–47.