Chronobiology International, 2014; 31(9): 976–982

ISSN: 0742-0528 print / 1525-6073 online
DOI: 10.3109/07420528.2014.937491

informa
healthcare

ORIGINAL ARTICLE

Association between light exposure at night and insomnia in the general elderly population: The HEIJO-KYO cohort

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Chronic circadian misalignment between the internal and environmental rhythms, which is typically related to night-shift work and clock-gene variants, is associated with disruption of suprachiasmatic nucleus function and increased risk of insomnia. Under controlled laboratory conditions, light at night (LAN) suppresses melatonin secretion, delays the internal biological rhythm, and reduces sleepiness. Therefore, LAN exposure may cause circadian misalignment and insomnia, though it remains unclear in real-life situations whether LAN exposure is associated with insomnia. To evaluate an association between LAN exposure and sleep quality in home settings, we conducted a cross-sectional community-based study in 857 elderly individuals (mean age, 72.2 years). We evaluated bedroom light intensity using a light meter and subjectively and objectively measured sleep quality using the Pittsburgh Sleep Quality Index and an actigraph, respectively, along with urinary 6-sulfatoxymelatonin excretion. Compared with the lowest quartile group of LAN intensity, the highest quartile group revealed a significantly higher odds ratio (OR) for subjective insomnia in a multivariate model adjusted for age, gender, body mass index, daytime physical activity, urinary 6-sulfatoxymelatonin excretion, bedtime, rising time, and day length (adjusted OR, 1.61, 95% confidence interval, 1.05–2.45, $p = 0.029$). In addition, higher OR for subjective insomnia was significantly associated with the increase in quartiles of LAN intensity ($p_{trend} = 0.043$). Consistently, we observed significant association trends between the increase in quartiles of LAN intensity and poorer actigraphic sleep quality, including decreased sleep efficiency, prolonged sleep-onset latency, increased wake-after-sleep onset, shortened total sleep time, and delayed sleep-mid time in multivariate models adjusted for the covariates mentioned above (all $p_{trend} < 0.001$). In conclusion, we demonstrated that LAN exposure in home settings is significantly associated with both subjectively and objectively measured sleep quality in a community-based elderly population.

Keywords: Actigraphy, circadian rhythms, insomnia, light at night, melatonin, sleep quality

INTRODUCTION

Epidemiological studies have demonstrated a higher prevalence of insomnia among the elderly than the younger population as well as a steady increase in the prevalence of insomnia in recent decades (Calem et al., 2012; Ford & Kamerow, 1989; Prinz et al., 1990). Previous studies involving a large elderly population showed that approximately 40% of elderly individuals reported any type of insomnia such as difficulty initiating sleep, difficulty maintaining sleep, early morning awakening, and non-restorative sleep (Calem et al., 2012; Walsh et al., 2011). Insomnia among the elderly is one of the most important public health issues because of its high prevalence and its association with increased risk of psychiatric and neurodegenerative disorders such as depression and dementia, cardiovascular diseases, and mortality (Cricco et al., 2001; Dew et al., 2003; Eaker et al., 1992; Yokoyama et al., 2010).

The solar 24 h cycle has led to the evolution of the human circadian rhythms. The suprachiasmatic nucleus (SCN) of the hypothalamus, which is an essential component of the master biological clock, synchronizes the internal biological rhythm to the environmental rhythm. Physiologically, light exposure is the most important environmental entraining cue for SCN function. Chronic circadian misalignment between the internal and environmental rhythms, which is typically related to night-shift work and clock-gene variants, is associated with disruption of SCN function and increased risk of insomnia (Allebrandt et al., 2010; Boudreau et al., 2013; Wyatt et al., 1999). Melatonin, a pineal gland hormone, is hypothesized to be a major

Submitted January 19, 2014, Returned for revision May 31, 2014, Accepted June 18, 2014

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internal contributor to the association between circadian misalignment and insomnia because endogenous melatonin levels are closely associated with both light exposure and sleepiness (Dijk & Cajochen, 1997; Zeitzer et al., 2000).

Exposure to light at night (LAN) is increasing globally, not only among night-shift workers but also among the general population because of the increased use of artificial lighting in modern society (Navara & Nelson, 2007). The nocturnal input of non-visual light information into the SCN through the photosensitive retinal ganglion cells (pRGCs) is associated with a range of neurobiological effects, including increased core body temperature, suppression of melatonin secretion, and stimulation of brain activity. Under controlled laboratory conditions, LAN suppresses melatonin secretion, delays the internal biological rhythm, and reduces sleepiness (Cajochen et al., 2000, 2005; Czeisler et al., 1986; Lockley et al., 2006). Thus, LAN exposure may cause circadian misalignment and insomnia, though it remains unclear in real-life situations whether LAN exposure is associated with insomnia.

In this cross-sectional study on 857 elderly individuals, we examined the association between LAN in home settings and sleep quality. We evaluated subjectively and objectively measured sleep quality using the Pittsburgh Sleep Quality Index (PSQI) and an actigraph, respectively, which are the two principal methods used to measure sleep quality in field studies. We also measured bedroom light intensity at night using light meters as well as overnight urinary 6-sulfatoxymelatonin excretion (UME), the major melatonin metabolite, as an index of melatonin secretion.

METHODS

Participants and study protocol

Between September 2010 and March 2013, 880 community-based elderly subjects voluntarily enrolled in a study titled ''Housing Environments and Health Investigation among Japanese Older People in Nara, Kansai Region: a prospective community-based cohort (HEIJO-KYO) study.'' Of these, 857 home-dwelling participants met the inclusion criteria of age $\geq$60 years and completion of the PSQI questionnaire. All participants provided written informed consent, and the study protocol was performed in accordance with the ethics committee of Nara Medical University and the ethical standards of the Journal (Portaluppi et al., 2010).

The protocols were described in our previous study (Obayashi et al., 2012). In brief, after collecting demographic and medical information using a standardized questionnaire, we initiated measurements of LAN exposure and actigraphic parameters in two consecutive days. Subsequently, we instructed the participants to collect their urine the following night and to maintain a standardized sleep diary by logging their bedtime and rising time.

Measurements of subjective and objective sleep quality

Subjective sleep quality was measured using the PSQI questionnaire, in which sleep quality over the previous month was asked using seven subscales measuring different components of sleep, including sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbance, use of sleep medication, and daytime dysfunction. The score of each component ranges from 0 to 3, with 3 indicating the worst sleep quality (Buysse et al., 1989). A cut-off value of 6 was used for the detection of sleep disturbance. Insomnia was defined as individuals who were previously diagnosed of insomnia and currently received a sleep medication and/or individuals who had PSQI score of $\geq$6.

Objective sleep quality was measured at 1 min intervals using an actigraph (Actiwatch 2; Respironics Inc., Murrysville, PA) that was worn on the non-dominant wrist. Sleep/awake status at each epoch, sleep onset, and sleep offset were automatically detected by the Actiware version 5.5 (Respironics Inc.) with the default algorithm (Philips Respironics Actiware Tutorials, 2013). Epochs with higher activity counts than moderate threshold (40 counts/min) were treated as awake. Sleep onset was the first minute followed by 10-min immobility period comprising not more than one epoch with any motion count, and sleep offset was the last minute following the 10 min period of immobility.

Five actigraphic sleep parameters were determined using objective data (awake/sleep status and sleep onset/offset) and self-reported data (bedtime and rising time) as follows: (1) total sleep time (TST), the time spent with sleep (below the activity threshold of 40 counts/min) between sleep onset and sleep offset; (2) sleep efficiency (SE), the percentage calculated from TST divided by the time between bedtime and rising time; (3) wake after sleep onset (WASO), the time spent with awake (above the activity threshold of 40 counts/min) between sleep onset and rising time; (4) sleep onset latency (SOL), the time between bedtime and sleep onset; and (5) sleep-mid time, the mid-time between sleep onset and sleep offset.

Measurement of LAN exposure

LAN exposure was measured at 1-min intervals using a light meter (LX-28SD; Sato Shouji Inc., Kanagawa, Japan) with the sensor fixed at 60 cm above the floor, near the head of the bed, and facing the ceiling. The optical sensor had spectral sensitivity that approximated that of the human eye, the illuminance sensitivity that ranged from 0 to 100 000 lux. The internal clocks of the light meter and the actigraph were synchronized. We used the average light intensity between bedtime and rising time for a parameter of LAN exposure. We divided the participants into quartile groups according to the intensity of LAN exposure. Among the 841 participants, the day-to-day reproducibility of average LAN intensity

in two consecutive days was moderately high (the Spearman rank correlation coefficient = 0.66).

UME measurement

The overnight urine collection protocol involved discarding the last void at bedtime and collecting each subsequent void, including the first morning void. Samples were stored in a dark bottle with a cold pack at room temperature, the total volume was measured, and the samples were stored at −20 °C until the assay. Urinary 6-sulfatoxymelatonin concentrations were measured using a highly sensitive enzyme-linked immunosorbent assay kit (RE54031; IBL International, Hamburg, Germany). UME was calculated as follows: UME (μg) = 6-sulfatoxymelatonin concentration (μg/mL) × total overnight urine volume (mL). UME was used as an index of melatonin secretion because there is evidence that UME correlates closely with the secreted levels of this hormone (Baskett et al., 1998).

Other measurements

Body mass index (BMI) was calculated as weight (kg)/height (m^2). Current smoking status, habitual alcohol consumption, and sleep medication use were evaluated by administering a questionnaire to participants. The estimated glomerular filtration rate (eGFR) was calculated using the formula from the Japanese Society of Nephrology-Chronic Kidney Disease Practice Guide: eGFR (mL/min/1.73 m^2) = 194 × [serum creatinine (mg/dl)]$^{-1.094}$ × [age (years)]$^{-0.287}$. The result was multiplied by a correction factor of 0.739 for female. Diabetes mellitus was diagnosed on the basis of the following assessments: medical history, current antidiabetic therapy, fasting plasma glucose levels ≥ 7.0 mmol/L, and glycated hemoglobin levels ≥ 6.5% of the National Glycohemoglobin Standardization Program value. Daytime physical activity was the average of all valid physical activity counts evaluated using the actigraph (Actiwatch 2) between rising time and bedtime. Data regarding the day length from sunrise to sunset in Nara (latitude 34°N) on the measurement days were obtained from the webpage of the National Astronomical Observatory of Japan (National Astronomical Observatory of Japan, 2013).

Statistical analyses

Variables with a normal distribution were expressed as the mean and standard deviation (SD), whereas asymmetrically distributed variables were reported as the median and interquartile range (IQR). Means and medians were compared between the subjective insomnia and non-insomnia groups using the unpaired *t*-test and the Mann–Whitney *U* test, respectively. The chi-square test was used for comparing categorical data. Comparisons of adjusted means were conducted using analysis of covariance (ANCOVA). Variables including age, gender, BMI, current smoking status, alcohol consumption habit, diabetes, eGFR, daytime physical activity, UME, bedtime, rising time, day length, and actigraphic sleep parameters were compared between the subjective insomnia and non-insomnia groups. The average values of LAN exposure, daytime physical activity, UME, bedtime, rising time, day length, and actigraphic sleep parameters on two consecutive days were used for further analyses. UME and SOL with a skewed distribution were naturally log-transformed for analyses. Odds ratios (ORs) for subjective insomnia and means of actigraphic sleep parameters were simultaneously adjusted for variables that were marginally to significantly associated with subjective insomnia ($p < 0.20$) in the univariate comparisons (Table 1). Trends in the association of quartiles of LAN intensity with ORs for subjective insomnia and means of actigraphic sleep parameters were evaluated using linear regression models for trends. Statistical analyses were performed using SPSS version 19.0 for Windows (IBM SPSS Inc., Chicago, IL). A two-sided p value < 0.05 was considered significant.

RESULTS

The mean age of the 857 participants was 72.2 ± 7.1 years, and 432 (50.4%) individuals were female. The median intensity of LAN was 0.8 lux (IQR, 0.1–3.4 lux). There were 310 individuals with subjective insomnia. Of these, 84 participants were diagnosed of insomnia and currently received a sleep medication, and 294 participants had PSQI score of ≥ 6. The subjective insomnia group ($n = 310$) showed significantly higher age, more female, less daytime physical activity, earlier bedtime, and later rising time than the non-insomnia group ($n = 547$, Table 1). The subjective insomnia group showed marginally but insignificantly lower BMI ($p = 0.10$) and UME ($p = 0.11$) and shorter day length on measurement day ($p = 0.09$) than the non-insomnia group.

Comparisons of actigraphic sleep parameters after adjustment for age and gender (Table 2) revealed significantly lower SE and longer SOL, WASO, and TST in the subjective insomnia group than the non-insomnia group (SE, 83.6 versus 85.0%, $p = 0.019$; log-transformed SOL, 3.1 versus 2.9 log min, $p = 0.035$; WASO, 85.6 versus 74.8 min, $p = 0.001$; TST, 429.2 versus 417.9 min, $p = 0.020$).

Compared with the lowest quartile group of LAN intensity (median, 0 lux), the unadjusted OR for subjective insomnia in the highest quartile group (median, 9.7 lux) was 1.82 [95% confidence interval (CI), 1.22–2.72, $p = 0.004$; Table 3]. Consistently, in the multivariate logistic model, the adjusted OR for subjective insomnia in the highest quartile group was significantly higher than that in the lowest quartile group even after adjusting for age, gender, BMI, daytime physical activity, log-transformed UME, bedtime, rising time, and day length (adjusted OR, 1.61; 95% CI, 1.05–2.45; $p = 0.029$). In addition, the higher adjusted ORs for subjective

TABLE 1. Basic and clinical characteristics between subjective insomnia and non-insomnia.

Characteristics	Insomnia ($n=310$)	Non-insomnia ($n=547$)	p
Basic parameters			
Age, mean (SD), years	73.6 (6.7)	71.4 (7.2)	<0.001
Gender, female, number (%)	180 (58.1)	252 (46.1)	0.001
Body mass index, mean (SD), kg/m^2	22.9 (3.0)	23.2 (3.0)	0.10
Current smoker, number (%)	15 (4.8)	29 (5.3)	0.77
Alcohol consumption (>30 g/day), number (%)	39 (12.6)	78 (14.3)	0.49
Clinical parameters			
Diabetes, number (%)	33 (10.6)	65 (11.9)	0.58
eGFR, mean (SD), mL/min/1.73m^2	71.1 (16.0)	72.5 (14.1)	0.23
Daytime physical activity, mean (SD), count/min	286.7 (107.8)	305.4 (105.1)	0.014
UME, median (IQR), (μg)	6.2 (3.8–9.1)	6.9 (4.1–10.3)	0.11
Bedtime, mean (SD), clock time	22:16 (1:14)	22:33 (1:06)	0.001
Rising time, mean (SD), clock time	6:55 (0:52)	6:42 (0:58)	0.003
Day length, median (IQR), min	642 (606–681)	650 (610–690)	0.09

eGFR, estimated glomerular filtration rate; UME, urinary 6-sulfatoxymelatonin excretion; IQR, interquartile range.

insomnia were significantly associated with the increase in quartiles of LAN intensity ($p_{trend}=0.043$). Consistently, after adjustment for age and gender, the higher adjusted ORs for subjective insomnia defined only by PSQI score were significantly associated with the increase in quartiles of LAN intensity ($p_{trend}=0.016$). In contrast, UME did not significantly differ across quartiles of LAN intensity ($p_{trend}=0.60$).

Regarding the actigraphic sleep parameters, the increase in quartiles of LAN intensity were significantly associated with poorer sleep quality, including decreased SE (both $p_{trend}<0.001$), prolonged SOL (both $p_{trend}<0.001$) and WASO (both $p_{trend}<0.001$), and delayed sleep-mid time (unadjusted $p_{trend}=0.003$, adjusted $p_{trend}<0.001$) in both unadjusted and adjusted models (Table 4). Although prolonged TST was marginally associated with the increase in quartiles of LAN intensity in an unadjusted model ($p_{trend}=0.07$), this parameter was significantly shortened with quartiles of LAN intensity increasing in an adjusted model ($p_{trend}<0.001$).

Furthermore, we have conducted additional analyses with regard to the association between LAN and actigraphic sleep parameters in the actual sleep period defined by actigraphy. Consistently, after adjustment for age, gender, bedtime, and rising time, an increase in quartiles of LAN intensity was significantly associated with poorer sleep quality, including decreased SE (Q1, 88.8%; Q2, 88.6%; Q3, 88.4%; Q4, 87.4%; $p_{trend}=0.041$),

TABLE 2. Comparisons of actigraphic sleep parameters between subjective insomnia and non-insomnia.

	Adjusted mean* (95% CI)		
Characteristics	Insomnia ($n=310$)	Non-insomnia ($n=547$)	p
PSQI score	8.3 (8.1–8.5)	3.0 (2.8–3.1)	<0.001
Actigraphic sleep parameters			
SE, %	83.6 (82.7–84.5)	85.0 (84.3–85.6)	0.019
Log-transformed SOL, log min	3.1 (3.0–3.2)	2.9 (2.8–3.0)	0.035
WASO, min	85.6 (80.7–90.5)	74.8 (71.1–78.5)	0.001
TST, min	429.2 (421.6–436.8)	417.9 (412.2–423.5)	0.020
Sleep-mid time, clock time	02:37 (02:31–02:43)	02:38 (02:33–02:43)	0.85

PSQI, Pittsburgh Sleep Quality Index; SE, sleep efficiency; SOL, sleep-onset latency; WASO, wake after sleep-onset; TST, total sleep time; CI, confidence interval. *Adjusted for age and gender using analysis of covariance.

TABLE 3. Logistic regression analysis for the associations of exposure to LAN with subjective insomnia.

	Quartiles of intensity of LAN (lux) [median, range]				
	0 [<0.1]	0.4 [0.1–0.8]	1.7 [0.8–3.4]	9.7 [>3.5]	p_{trend}
No. of participants (missing)	205 (5)	208 (2)	207 (4)	208 (2)	
No. of cases	63	74	76	92	
Unadjusted OR (95% CI)	1.00 (ref)	1.27 (0.84–1.91)	1.31 (0.87–1.98)	1.82 (1.22–2.72)	0.005
Age/gender-adjusted OR (95% CI)	1.00 (ref)	1.26 (0.83–1.92)	1.23 (0.81–1.87)	1.86 (1.23–2.82)	0.005
Fully-adjusted OR* (95% CI)	1.00 (ref)	1.25 (0.81–1.91)	1.19 (0.77–1.83)	1.61 (1.05–2.45)	0.043

LAN, light at night; OR, odds ratio; CI, confidence interval; UME, urinary 6-sulfatoxymelatonin excretion.
*Adjusted for age, gender, body mass index, daytime physical activity, log-transformed UME, bedtime, rising time, and day length (per quartile increment).

TABLE 4. Actigraphic sleep parameters stratified by LAN exposure.

	Quartiles of intensity of LAN (lux) [median, range]				
	0 [<0.1]	0.4 [0.1–0.8]	1.7 [0.8–3.4]	9.7 [>3.5]	
No. of participants (missing)	205 (5)	208 (2)	207 (4)	208 (2)	
Unadjusted	Mean (5%–95% range)				p_{trend}
SE, %	86.3 (75.5–93.7)	85.3 (71.8–93.1)	84.5 (72.4–93.1)	81.8 (65.3–91.5)	<0.001
Log-ransformed SOL, log min	2.6 (0.8–4.1)	2.8 (1.4–4.4)	3.1 (1.3–4.3)	3.4 (1.8–4.7)	<0.001
WASO, min	66.0 (27.4–128.5)	73.0 (30.5–144.3)	79.5 (29.0–155.8)	96.4 (40.8–201.0)	<0.001
TST, min	416.5 (312.0–530.8)	417.1 (314.9–523.3)	428.3 (313.0–556.5)	425.7 (314.3–542.0)	0.07
Sleep-mid time, clock time	02:26 (0:37–03:46)	02:40 (01:14–04:00)	02:43 (01:24–03:58)	02:42 (00:57–04:12)	0.003
Adjusted*	Mean (95% CI)				p_{trend}
SE, %	85.9 (84.9–87.0)	85.2 (84.1–86.2)	84.7 (83.7–85.7)	82.0 (81.0–83.0)	<0.001
Log-transformed SOL, log min	2.7 (2.6–2.8)	2.8 (2.7–3.0)	3.0 (2.9–3.2)	3.3 (3.2–3.4)	<0.001
WASO, min	71.1 (65.7–76.5)	75.5 (70.2–80.8)	77.3 (71.9–82.6)	92.1 (86.7–97.4)	<0.001
TST, min	430.0 (424.6–435.4)	425.8 (420.5–431.1)	423.9 (418.6–429.3)	409.0 (403.7–414.3)	<0.001
Sleep-mid time, clock time	02:34 (02:31–02:37)	02:34 (02:31–02:37)	02:39 (02:36–02:42)	02:43 (02:40–02:46)	<0.001

LAN, light at night; SE, sleep efficiency; SOL, sleep-onset latency; WASO, wake after sleep-onset; TST, total sleep time; CI, confidence interval; UME, urinary 6-sufatoxymelatonin excretion.

*Adjusted for age, gender, body mass index, daytime physical activity, log-transformed UME, bedtime, rising time, daylength (per quartile increment) using ANCOVA.

prolonged WASO (Q1, 46.8 min; Q2, 49.4 min; Q3, 51.5 min; Q4, 56.4 min; $p_{trend} < 0.001$), and delayed sleep-mid time (Q1, 02:34; Q2, 02:35; Q3, 02:38; Q4, 02:43; $p_{trend} < 0.001$). In contrast, there was no significant association trend between LAN intensity and TST ($p_{trend} = 0.79$).

DISCUSSION

The present study demonstrated that LAN exposure in home settings was significantly associated with both subjective and objective sleep quality in a community-based elderly population. The findings were evidenced by the significant association trends of an increase in LAN intensity with higher prevalence of subjective insomnia and poorer actigraphic sleep measures, independent of several potential confounding factors ($p_{trend} = 0.043$ in subjective measurements and $p_{trend} < 0.001$ in all actigraphic measurements). Furthermore, the OR for subjective insomnia was 61% higher in the highest quartile group of LAN intensity than that in the lowest quartile group (adjusted OR, 1.61; 95% CI, 1.05–2.45; $p = 0.029$).

The present study, to the best of our knowledge, provided the first human evidence regarding the association between very low average intensity of LAN exposure in home settings and risk of insomnia. A previous well-controlled experimental study indicated that LAN exposure can exert a dose-dependent alerting effect, as assessed by subjective ratings, slow eye movements, and electroencephalographic activity (Cajochen et al., 2000); whereas, the effective LAN intensity was higher than that of the highest LAN quartile group in the present study (median, 9.7 lux). However, LAN intensity in the present study can include LAN with high intensity but short duration, because the LAN intensity was the average bedroom light intensity during the in-bed period. A recent experimental study indicated that light exposure with high intensity but short duration may effect on human circadian physiology using the duration-response curve for the association between light exposure and melatonin suppression (Chang et al., 2012). In addition, human circadian physiology is more closely correlated to shorter wave length rather than intensity (Cajochen et al., 2005). Thus, additional experimental researches are needed to better understand the effect of LAN with high intensity but short duration, or low intensity but short wave length as well as very low intensity on sleep quality in humans.

The clinical implications of sleep disturbances measured in the present study could be interpreted by comparing with previously reported data of actigraphic sleep measures in elderly individuals. A previous well-designed study (Levenson et al., 2013), where participants' age and actigraphic sleep measurement methods were similar to those in our study, reported that SE was the best predictor for insomnia because SE takes into account TST, SOL, and WASO in its measurement, and that SE was 81.3% and 83.7% in elderly individuals with and without insomnia, respectively. These results were also similar to our current data, although SE was 1–2% higher and difference of SE between insomniacs and non-insomniacs was 1% smaller in our study than that in the previous study. On the other hand, in our study, SE was 3.9% lower in the highest LAN quartile group than that in the lowest quartile group (82.0% versus 85.9%, respectively). This difference of actigraphic SE was larger than those observed between elderly individuals with and without depressed mood in large-scale

community-based studies (Maglione et al., 2012; Paudel et al., 2008).

Causality of LAN exposure with regard to insomnia could not be ascertained in the present study because it was a cross-sectional design. Some participants with poor sleep may turn on light in the bedrooms. In addition, epidemiological data in elderly individuals have demonstrated that poor sleep quality causes psychiatric and neurodegenerative disorders such as depression and dementia (Cricco et al., 2001; Yokoyama et al., 2010), and recent experimental studies conducted in mice showed that chronic exposure to dim LAN (5 lux) causes depressed mood and impaired cognition compared with complete darkness at night (Bedrosian et al., 2013; Fonken et al., 2012). When considering together, the present study may indicate that LAN exposure possibly and partly explains the risk for psychiatric and neurodegenerative disorders in poor sleepers. Further research using a longitudinal design is required to improve our understanding of the association between LAN, sleep quality, and psychiatric and neurodegenerative disorders.

Although the neurophysiologic mechanisms that mediate LAN-induced insomnia are not fully understood, possible mechanisms are suggested by data regarding circadian phase delay. LAN is a well-established suppressor of melatonin secretion, and endogenous melatonin levels are closely associated with sleepiness (Dijk & Cajochen, 1997; Zeitzer et al., 2000). Therefore, melatonin is hypothesized to be one of the major internal contributors to the association between LAN exposure and insomnia, although the present study showed no significant associations between LAN exposure and UME in home settings. In addition, we did not measure potential melatonin suppression by LAN exposure. However, the present study indicated a significant association between LAN exposure and sleep disturbances even after adjusting for UME, an index of endogenous melatonin amplitude. Whereas, changes in circadian biological phase remain to be a potential mediator to the association between LAN exposure and insomnia. According to the circadian phase–response curve to light (Khalsa et al., 2003), LAN delays the subsequent circadian phase, which may be a potential risk for circadian misalignment between the internal and environmental rhythms. Chronic circadian misalignment is associated with the disruption of SCN function and increased risk of insomnia (Boudreau et al., 2013; Wyatt et al., 1999). In the present study, a modest but significant association trend was observed between an increase in LAN intensity and a delay in sleep-mid time, which is correlated with a marker of circadian phase (Burgess et al., 2003).

The strength of the present study included a large study sample size. This advantage enabled us to analysis the associations between LAN intensity and outcomes for actigraphic measurements, although our previous study was limited to report the association between LAN and SOL (Obayashi et al., 2014). In addition to its cross-sectional design, the present study has three limitations. The first was that the light meters used in our study were not ambulatory devices; thus, LAN intensity may have been underestimated because the light meters did not record light exposure in rooms other than the bedroom (e.g. during nocturia). Further research using ambulatory eye-level light meters is required to assess the association between LAN exposure in home settings and sleep quality. However, compared with a wrist light meter, the fixed bedroom light meter used in the present study presents the great advantage of never being covered by bed linen or night clothes. Second, lighting sources could not be distinguished, e.g. bedroom light or morning sun light entering the bedroom, and we have no information related to the location of the windows in the bedroom. Therefore, interpretation of our results would be limited to the association between total amounts of LAN intensity and sleep quality. The third limitation was non-random sampling because participants were recruited with the cooperation of local resident associations and elderly-resident clubs, which may have led to a selection bias. However, the generalizability of our findings may be acceptable because some basic data (e.g. BMI and eGFR) were consistent with those of the National Health and Nutrition Survey in Japan in 2010 (The National Health and Nutrition Survey Japan, 2010).

In conclusion, the present study demonstrated that LAN exposure in home settings was significantly associated with both subjectively and actigraphically measured sleep quality in a general elderly population.

ACKNOWLEDGMENTS

We are indebted to all participants of this study. We would also like to thank Sachiko Uemura and Naomi Takenaka for their valuable support during the data collection.

DECLARATION OF INTEREST

All authors report no conflicts of interest. This work was supported by Grants from the Department of Indoor Environmental Medicine, Nara Medical University; Scientific Research from the Ministry of Education, Culture, Sports, Science and Technology; Mitsui Sumitomo Insurance Welfare Foundation; Meiji Yasuda Life Foundation of Health and Welfare; Osaka Gas Group Welfare Foundation; Japan Diabetes Foundation; and the Japan Science and Technology Agency.

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