

Ambient Light Exposure and Changes in Obesity Parameters: A Longitudinal Study of the HEIJO-KYO Cohort

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Context: Previous epidemiological studies have suggested an association between nighttime light levels and the prevalence of obesity, although evidence is limited to cross-sectional studies.

Objective: To evaluate the longitudinal association between ambient light exposure and the subsequent changes in obesity parameters.

Design and Participants: Data from 1110 elderly participants at baseline (mean age, 71.9 years) and data from 766 at follow-up (median, 21 months) were included in this prospective population-based study.

Measures: Time-dependent ambient light exposure based on objective measurements and changes in the waist-to-height ratio (WHtR) and body mass index (BMI) were measured.

Results: Multivariable mixed-effect linear regression models showed a significant association between light exposure and the %WHtR gain; this was independent of potential confounders (eg, caloric intake, physical activity, and sleep/wake parameters). Nighttime or evening exposure to higher light intensity was significantly associated with subsequent %WHtR gain. Morning exposure to a longer time ≥ 500 lux or nighttime exposure to a longer time < 3 lux was significantly associated with subsequent %WHtR loss. These association trends were nearly consistent when the BMI was used as an obesity parameter. Increased nighttime light exposure (mean ≥ 3 vs < 3 lux) was estimated to correspond to a 10.2% WHtR gain and a 10.0% increase in BMI over 10 years.

Conclusions: Ambient light exposure, such as increased nighttime or evening light exposure and decreased morning light exposure, was independently associated with subsequent increases in obesity parameters. Further interventional studies are warranted to establish an optimal controlled lighting environment as a preventive option against obesity. (*J Clin Endocrinol Metab* 101: 3539–3547, 2016)

The prevalence of obesity has increased over time in most countries and has now reached epidemic proportions (1–4). Obesity is widely recognized as one of the leading health threats worldwide and a major risk factor for hypertension, diabetes, cardiovascular diseases, cancer, and early mortality (5–9). Although obesity is primarily caused by overeating and physical inactivity, existing diet and physical exercise programs have failed to

prevent this epidemic (10), warranting novel innovative approaches.

Energy homeostasis is fundamentally maintained by the endogenous circadian timing system under the control of the suprachiasmatic nucleus of the hypothalamus (the master biological clock) (11). Clock gene mutations induced obesity in mice (12), and circadian misalignment between the internal and environmental rhythms de-

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Abbreviations: BMI, body mass index; CI, confidence interval; UME, urinary 6-sulfatoxymelatonin excretion; WHtR, waist-to-height ratio.

creased daily energy expenditure and leptin levels in human experimental studies (13, 14). These findings agree with previous epidemiological findings of obesity being induced by shiftwork, which is associated with chronic circadian misalignment (15).

The solar 24-hour cycle has led to the evolution of the human circadian timing system, which is crucially influenced by light information. Light exposure at an inappropriate timing against the solar cycle can desynchronize the internal rhythms from the environmental rhythms, resulting in circadian misalignment (16). Modern societies have developed with the widespread use of electrical lighting at night (17). Recent experimental studies using mice (18, 19) demonstrated that an 8-week exposure to nighttime light significantly increased body mass, even at a low intensity (5 lux), and 5-week exposure to a shorter nighttime dark period dose-dependently increased fat mass. In epidemiological studies (20, 21), a higher prevalence of obesity was observed in individuals with a higher nighttime bedroom light intensity measured objectively and subjectively; however, evidence is limited to cross-sectional studies (22). The purpose of this study is to evaluate the hypothesis about whether objectively measured ambient light exposure is longitudinally associated with the subsequent changes in obesity parameters.

Subjects and Methods

Study participants and design

Baseline data of the HEIJO-KYO cohort was collected between September and April, 2010–2014, and included 1127 community-dwelling elderly individuals aged ≥ 60 years. Of these, we excluded participants who did not complete measurements of light exposure and obesity parameters. As a result, we evaluated the primary outcomes from 1110 participants at baseline and from 766 at follow-up (median duration, 21 months). Follow-up was conducted between August and September. All participants provided written informed consent. The study protocol was approved by the Nara Medical University ethics committee.

The baseline study protocol was reported in our previous study (23). In brief, the homes of participants were visited predominantly on weekdays and demographic and medical information was collected during face-to-face interviews by trained interviewers using a standardized questionnaire. Waist circumference, body weights, and heights were measured and venous blood samples were collected. Then, light exposure measurements were performed for 48-hour from noon of the first day to noon of the third day and participants were instructed to maintain a standardized sleep diary for the first and second night. Overnight urine for the second night was collected. Finally, the instruments, sleep diaries, and urine samples were collected from the homes of the participants. All participants did not receive any instruction on their eating, activity or sleep behavior, except for

drinking. They were instructed not to consume alcohol during the designated monitoring period.

Measurements of light exposure

Morning (4 hours after rising time), evening (4 hours before bedtime), daytime (rising time to bedtime), and nighttime (bedtime to rising time) were defined using data from the sleep diary.

Light exposures in the morning, evening, and daytime, were measured for two consecutive days at 1-minute intervals using a wrist light meter (Actiwatch 2; Respironics Inc) worn on the nondominant wrist. The device has a photodiode with a spectral sensitivity approximating that of the human eye (illuminance sensitivity, 0–150 000 lux). All participants were given special rubber bands to prevent their shirtsleeves from covering the device. Daytime values of <1 lux were considered artifact data because of the sensor coverage and were not considered for calculating mean values (24). If light data for more than half of the daytime period were interpreted as artifacts, the data were considered missing. Mean light intensities were used as parameters of exposed light amplitude. Times above the threshold intensities were the total minutes above the thresholds, which were based on the values of 500 and 1000 lux during daytime used in the previous observational studies (23–25). The thresholds of 50 and 100 lux on evening light exposure were based on our previous data showing that median intensity of evening light exposure was approximately 1/10 of that of daytime (26). Data were analyzed after base 10 logarithmic transformation.

Light exposures during the nighttime and in the first (1 hour after bedtime) and last hour of the night (1 hour before rising time) were measured for two consecutive nights at 1-minute intervals using a portable light meter (LX-28SD; Sato Shouji Inc) placed facing the ceiling by the head of the subject's bed at 60 cm above the floor (illuminance sensitivity, 0–100 000 lux). Light meter measurements were synchronized to those of the wrist light meter. Mean light intensities were used as parameters of exposed light amplitude. Times below the threshold intensities (3 and 10 lux) were the total minutes below the thresholds. Since normalizing the distribution of nighttime light was difficult, we conducted categorical analysis by tertile value or the predefined threshold (3 and 10 lux) which were based on the values that have minimal effects on human physiology (16).

Measurements of the obesity parameters

Waist circumference at the umbilicus level, height, and body weight were measured in the standing position. The waist-to-height ratio (WHtR) was calculated as the waist circumference (in meters) divided by height (in meters), and the body mass index (BMI) was calculated as body weight (in kilograms) divided by height (in meters²). WHtR is more accurate than BMI or waist circumference for evaluating fat distribution and accumulation and for determining cardiometabolic risk (27, 28). Abdominal obesity was defined as WHtR ≥ 0.6 because clinical significance increases beyond this value in the elderly (29).

Measurements of the covariates

Current smoking, drinking, and socioeconomic statuses were surveyed using a questionnaire. Caloric intake was assessed by a validated food frequency questionnaire containing 29 food groups and 10 kinds of cookery; the weekly intake of each food

group was estimated by commonly used units or portion sizes (30). Physical activity was assessed using the International Physical Activity Questionnaire (Japanese version) containing questions on the amount of time spent performing moderate and vigorous activities and walking per week (31). A standardized sleep diary was used to estimate bedtimes and rising times. Subjective sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI) questionnaire, and sleep disturbances were defined as a PSQI score ≥ 6 (32). As an index of melatonin secretion, urinary 6-sulfatoxymelatonin excretion (UME) was measured from urine samples collected overnight as described in our previous publication (23). Day length (sunrise to sunset), based on the measurement days in Nara, Japan (latitude 34°N), was extracted from the website of the National Astronomical Observatory of Japan (33).

Statistical analysis

Averages of the data for light exposure, sleep diary, and day length on two measurement days were used for analysis. Means were compared between the dichotomous groups using the unpaired *t* test, and the χ^2 test was used to compare categorical data. The mean, median, or proportion was substituted for missing data of independent variables. Data of total participants (*n* = 1110) were used in cross-sectional analysis of the association between light exposure and abdominal obesity. In a longitudinal analysis using data of 1110 participants at baseline and 766 participants at follow-up, the association between light exposure and changes in the obesity parameters was evaluated by mixed-effect linear regression analysis consisting of the individual-level variables (light exposure, age [≥ 70 vs < 70 years], sex [male vs female], follow-up duration [≥ 2 vs < 2 years], current smoking status [yes vs no], alcohol consumption [≥ 30 vs < 30 g/d], house-

hold income [≥ 4 vs < 4 million Japanese yen per year], education [≥ 13 vs < 13 years], caloric intake [per 100 kcal/d], physical activity [per log metabolic equivalent hours per week], sleep disturbances [yes vs no], bedtime [per 1 hour delay], duration in bed [per 1 hour], UME [per log μg], and day length [per quartile] and the day-level variables (WHtR and BMI). Regression coefficients were estimated by the maximum likelihood method. All analyses were performed using SPSS, version 19.0 for Windows (IBM SPSS Inc). Statistical significance was set at a two-sided *P* value $< .05$. Time-dependent changes in exposed light intensity were smoothed by the generalized additive model, minimizing the generalized cross-validation score (34) using the R statistics package (35).

Results

Baseline characteristics

The mean age of participants at baseline was 71.9 ± 7.1 years (Table 1). The mean WHtR was 0.536 ± 0.057 . WHtR correlated better with waist circumference than height ($r = 0.85$ vs -0.31 , respectively). The median of mean light intensities in the morning, evening, daytime, and nighttime were 345.9, 24.0, 337.2, and 0.7 lux, respectively (Table 2). The median time ≥ 500 lux in the morning, ≥ 100 lux in the evening, ≥ 500 lux during daytime, and < 3 lux during nighttime were 24, 6, 74, and 467 minutes. These baseline characteristics stratified by the follow-up status are summarized in tables.

Table 1. Baseline Characteristics

Variables	All	Follow-Up Status		<i>P</i> ^a
		Yes	No	
No. of participants	1110	766	344	
Basic parameters				
Age, mean (SD), y	71.9 (7.1)	71.0 (6.7)	73.7 (7.6)	$< .001$
Male gender	520 (46.8)	355 (46.3)	165 (48.0)	.62
Current smoker	56 (5.0)	40 (5.2)	16 (4.7)	.96
Alcohol consumption ≥ 30 g/d	159 (14.3)	113 (14.8)	46 (13.4)	.58
Household income ≥ 4 million Japanese yen/y	440 (42.9)	319 (44.3)	121 (39.5)	.59
Education ≥ 13 y	295 (26.6)	228 (29.8)	67 (19.5)	.014
Caloric intake, mean (SD), kcal/d	1991.1 (484.6)	1997.4 (534.1)	1973.8 (534.1)	.28
Physical activity, median (IQR), MET-h/wk	22.3 (7.7, 46.2)	23.1 (8.8, 46.7)	18.8 (5.1, 40.9)	.007 ^b
Sleep disturbances (PSQI ≥ 6)	388 (35.2)	260 (33.9)	128 (37.3)	.17
Obesity parameters				
WHtR, mean (SD)	0.536 (0.057)	0.534 (0.057)	0.541 (0.057)	.28
BMI, mean (SD), kg/m ²	23.1 (3.0)	23.1 (3.0)	23.1 (3.2)	.43
Circadian rhythm parameters				
Bedtime, mean (SD), clock time	22:29 (1:10)	22:34 (1:08)	22:17 (1:14)	.050
Duration in bed, mean (SD), min	497 (76.8)	491.2 (74.7)	511.5 (79.7)	.030
UME, median (IQR), μg	6.8 (4.0, 10.6)	7.0 (4.2, 11.2)	5.9 (3.7, 9.1)	.003 ^b
Day length, median (IQR), min	654 (623, 682)	649 (614, 682)	657 (634, 682)	.053 ^c

Abbreviations: IQR, interquartile range; MET, metabolic equivalent; PSQI, Pittsburgh Sleep Quality Index. Data are expressed as number (percentage), unless specified otherwise.

^a Test (yes vs no) after adjustment for age and gender.

^b Test after log-transformation.

^c Test after quartile transformation.

Table 2. Baseline Light Exposure Characteristics

Variables	All	Follow-Up Status		<i>P</i> ^a
		Yes	No	
No. of participants	1110	766	344	
Mean light intensity, lux				
Morning (4 h)	345.9 (121.5, 796.3)	352.9 (123.9, 828.3)	315.2 (110.5, 771.1)	.40 ^b
Evening (4 h)	24.0 (15.9, 37.0)	23.8 (15.9, 37.1)	24.6 (16.0, 35.4)	.32 ^b
Daytime (all)	337.2 (165.4, 719.2)	336.9 (167.2, 702.9)	348.2 (156.9, 784.7)	.91 ^b
Nighttime (all)	0.7 (0.1, 3.3)	0.6 (0.1, 2.9)	1.0 (0.2, 4.4)	.017 ^c
First night (1 h)	1.3 (0, 9.5)	1.2 (0, 8.3)	1.7 (0, 13.6)	.46 ^c
Last night (1 h)	1.1 (0, 8.0)	0.8 (0, 6.7)	1.6 (0.02, 11.0)	.007 ^c
Time below/above threshold, min				
Morning (4 h)				
≥500 lux	24 (9, 46)	24 (9, 46)	22 (8, 45)	.65 ^b
≥1000 lux	15 (5, 34)	16 (5, 35)	13 (5, 33)	.58 ^b
Evening (4 h)				
≥50 lux	28 (12, 53)	29 (12, 54)	28 (12, 51)	.97 ^b
≥100 lux	6 (1, 19)	6 (1, 19)	6 (1, 18)	.83 ^b
Daytime (all)				
≥500 lux	74 (40, 129)	76 (43, 128)	70 (34, 132)	.35 ^b
≥1000 lux	50 (25, 96)	50 (27, 97)	51 (22, 94)	.55 ^b
Nighttime (all)				
<3 lux	467 (420, 516)	465 (417, 509)	473 (430, 526)	.13
<10 lux	473 (427, 521)	470 (421, 515)	481 (437, 537)	.18

Data are expressed as median (interquartile range).

^a Test (yes vs no) after adjustment for age and gender.

^b Test after log-transformation.

^c Test after tertile transformation.

Figure 1 shows time-dependent changes in exposed light intensity between the groups with (*n* = 138) and without (*n* = 972) abdominal obesity at baseline. The abdominal obesity group was exposed to a lower light intensity from rising time to near the beginning of the evening than the group with no abdominal obesity. There-

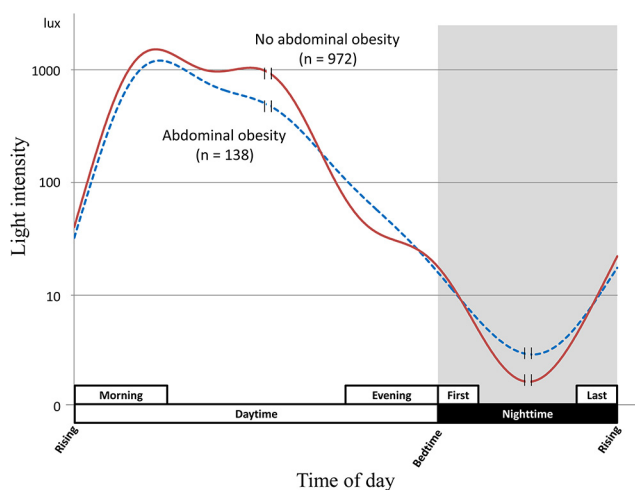


Figure 1. Cross-sectional associations between time-dependent changes in exposed light intensities and abdominal obesity at baseline. Solid line, No abdominal obesity group; dotted line, abdominal obesity group. Abdominal obesity, WHtR ≥ 0.6 ; morning, 4 hours after rising; evening, 4 hours before bedtime; the first hour of the night, 1 hour after bedtime; the last hour of the night, 1 hour before rising.

after, the former was mostly exposed to higher light intensity throughout the night.

Longitudinal association between light exposure and obesity parameters

The mean follow-up WHtR was 0.550 ± 0.059 , and the mean WHtR difference (follow-up minus baseline) was 0.016 (95% confidence interval [CI], 0.014 to 0.019), respectively.

Morning exposure to higher light intensity was significantly associated with subsequent %WHtR loss in the unadjusted model over a median follow-up of 21 months ($P = .002$; Table 3), but this was insignificant in the minimally adjusted model ($P = .07$; model 1). A longer time ≥ 500 lux in the morning was significantly associated with subsequent %WHtR loss in the unadjusted model; the association remained significant in the fully adjusted model ($P = .022$; model 2), whereas there was a marginal association between a longer time ≥ 1000 lux in the morning and %WHtR loss ($P = .06$; model 2).

Regarding evening light, no parameter was significantly associated with a change in %WHtR in the unadjusted models. After adjusting for confounders, evening exposure to higher light intensity and a longer time ≥ 100 lux were significantly associated with subsequent %WHtR gain ($P = .045$ and $P = .032$, respectively; model 2).

Table 3. Longitudinal Association Between Light Exposure and Obesity Risk Using the Mixed-Effect Linear Regression Analysis

Light Exposure Parameters	% WHtR Change									BMI Change		
	Unadjusted			Adjusted Model 1			Adjusted Model 2			Adjusted Model 2		
	β	95% CI	<i>P</i>	β	95% CI	<i>P</i>	β	95% CI	<i>P</i>	β	95% CI	<i>P</i>
Morning (4 h)												
Mean light intensity												
Continuous (log lux)	-0.898	-1.472, -0.325	.002	-0.534	-1.106, 0.039	.07	-0.393	-0.972, 0.186	.18	-0.143	-0.456, 0.170	.37
Time above ≥ 500 lux												
Continuous (log min)	-1.275	-1.918, -0.631	<.001	-0.897	-1.536, -0.258	.006	-0.970	-1.798, -0.143	.022	-0.257	-0.605, 0.091	.15
Time above ≥ 1000 lux												
Continuous (log min)	-1.003	-1.607, -0.398	.001	-0.644	-1.244, -0.044	.035	-0.699	-1.428, 0.030	.06	-0.148	-0.474, 0.178	.37
Evening (4 h)												
Mean light intensity												
Continuous (log lux)	0.325	-0.851, 1.501	.59	1.202	0.038, 2.365	.043	1.209	0.026, 2.392	.045	1.046	0.410, 1.682	.001
Time above ≥ 50 lux												
Continuous (log min)	-0.034	-0.741, 0.673	.93	0.436	-0.262, 1.134	.22	0.506	-0.195, 1.207	.16	0.467	0.089, 0.846	.015
Time above ≥ 100 lux												
Continuous (log min)	0.253	-0.327, 0.833	.39	0.598	0.028, 1.167	.040	0.626	0.055, 1.196	.032	0.452	0.145, 0.760	.004
Daytime (all)												
Mean light intensity												
Continuous (log lux)	-1.176	-1.902, -0.450	.002	-0.451	-1.120, 0.293	.23	-0.350	-1.107, 0.408	.37	-0.274	-0.683, 0.135	.19
Time above ≥ 500 lux ^a												
Continuous (log min)	-1.800	-2.597, -1.003	<.001	-1.067	-1.881, -0.252	.010	-0.980	-1.810, -0.151	.021	-0.540	-0.988, -0.093	.018
Time above ≥ 1000 lux ^a												
Continuous (log min)	-1.469	-2.169, -0.769	<.001	-0.813	-1.528, -0.098	.026	-0.706	-1.436, 0.024	.06	-0.432	-0.826, -0.038	.032
Nighttime (all)												
Mean light intensity												
≥ 3 vs < 3 lux	0.783	0.033, 1.533	.041	0.919	0.190, 1.649	.014	0.959	0.230, 1.687	.010	0.405	0.011, 0.799	.044
≥ 10 vs < 10 lux	1.091	0.066, 2.116	.037	1.073	0.076, 2.070	.035	0.999	0.004, 1.993	.049	0.391	-0.143, 0.932	.15
Time below < 3 lux ^b												
Continuous (per min)	-0.006	-0.012, -0.001	.029	-0.006	-0.012, -0.0005	.034	-0.006	-0.012, -0.001	.023	-0.003	-0.006, -0.00002	.048
Time below < 10 lux ^b												
Continuous (per min)	-0.008	-0.014, -0.001	.018	-0.007	-0.014, -0.001	.023	-0.007	-0.013, -0.001	.021	-0.003	-0.007, 0.00004	.053
First night (1 h)												
Mean light intensity												
T1 (low)	ref			ref			ref			ref		
T2 (intermediate)	0.151	-0.660, 0.963	.71	0.270	-0.521, 1.062	.50	0.219	-0.564, 1.002	.58	-0.048	-0.472, 0.375	.82
T3 (high)	0.375	-0.437, 1.187	.37	0.408	-0.382, 1.199	.31	0.219	-0.576, 1.013	.59	-0.067	-0.497, 0.362	.76
Last night (1 h)												
Mean light intensity												
T1 (low)	ref			ref			ref			ref		
T2 (intermediate)	0.534	-0.275, 1.344	.20	0.411	-0.379, 1.200	.31	0.423	-0.368, 1.215	.29	0.288	-0.140, 0.717	.19
T3 (high)	0.935	0.125, 1.746	.024	0.842	0.050, 1.635	.037	0.898	0.089, 1.707	.030	0.367	-0.071, 0.804	.10

Model 1 is adjusted for age, gender, and follow-up duration. Model 2 is adjusted for age, gender, follow-up duration, smoking and drinking status, household income, education, caloric intake, physical activity, bedtime, duration in bed, and day length.

^a Adjusted for duration out of bed (continuous).

^b Adjusted for duration in bed (continuous).

Daytime light, including morning and evening light, was significantly associated with subsequent %WHtR loss in the unadjusted models, and the only association between a longer time ≥ 500 lux during the daytime and subsequent %WHtR loss remained significant in the adjusted model ($P = .021$; model 2).

Nighttime exposure to higher light intensity (mean ≥ 3 lux) was significantly associated with subsequent %WHtR gain in each statistical model ($P = .010$, model 2). A longer time < 3 lux during the nighttime was significantly associated with subsequent %WHtR loss in each statistical model ($P = .021$; model 2). These results were consistent when a higher cutoff intensity of 10 lux was used. Regarding light intensity in the first hour of the nighttime period, no significant association with %WHtR change

was observed in any statistical model; however, higher light intensity in the last hour of the night (the highest vs lowest tertiles) was significantly associated with subsequent %WHtR gain in each statistical model ($P = .030$; model 2).

When using BMI as the obesity parameter, the association trends between light exposure and change in BMI were mostly consistent. In the fully adjusted models, BMI gain was significantly associated with evening or nighttime exposure to higher light intensity and evening exposure to a longer time above the thresholds. BMI loss was significantly associated with daytime exposure to a longer time ≥ 500 lux and nighttime exposure to a longer time below the thresholds. Additionally, daytime exposure to a longer time ≥ 500 lux and nighttime exposure to higher

light intensity (≥ 3 lux) were independently and significantly associated with both WHtR and BMI changes in the same model (model 2; data not shown).

Additional analysis

Sensitivity analysis using light data during the actual sleep period, as defined by actigraphy (moderate threshold, 40 counts/min), showed consistently significant associations of nighttime exposure to a longer time < 3 lux (per minute) with subsequent losses in WHtR and BMI (β , -0.009 ; 95% CI, -0.014 to -0.005 ; $P < .001$; and β , -0.005 ; 95% CI, -0.007 to -0.002 ; $P < .001$; model 2; respectively). In addition, similar results were observed in the sensitivity analysis excluding the participants with previous history of shiftwork ($n = 74$) (data not shown). Furthermore, in the additional analysis adjusted for sleep disturbances or UME, results were consistent with those in Table 3 (data not shown).

Independent variables, eg, the duration in bed and bedtime (Pearson's coefficient, 0.69), duration in bed and time < 3 lux (0.72), and duration in bed and time < 10 lux (0.77) were moderately correlated. The categorical data of duration in bed (≥ 8 or < 8 hours) had lower correlations with nighttime times < 3 and < 10 lux than the continuous data (Pearson's coefficient, 0.57 and 0.62, respectively). A significant association between WHtR loss and nighttime exposure to a longer dark exposure was observed in the models adjusted for the categorical data of duration in bed (β , -0.007 ; 95% CI, -0.012 to -0.002 ; $P = .007$; and β , -0.007 ; 95% CI, -0.013 to -0.002 ; $P = .006$; model 2; respectively).

Discussion

We found independent associations between ambient light exposure, such as increased nighttime or evening light exposure and decreased morning or daytime light exposure, and subsequent increases in obesity parameters. To our knowledge, this is the first longitudinal study reporting a significant association between time-dependent ambient light levels and obesity risk in a large general population.

Nighttime exposure to higher light intensity was significantly associated with WHtR and BMI gain; conversely, a longer dark exposure during the nighttime was significantly associated with WHtR and BMI loss. Although the nighttime light levels we observed were low, our findings were consistent with previous experimental studies. Fonken et al (18) showed that exposure to nighttime light for 8 weeks significantly increased the body mass of mice, even at a low intensity (5 lux), independently of caloric intake and physical activity. Kooijman et al (19)

suggested that exposure to a shorter nighttime dark period for 5 weeks increased the fat mass of mice dose-dependently through a decrease in energy expenditure by the inactivation of brown adipose tissue. Additionally, minimizing light exposure just before rising may be important to prevent obesity. Although median light intensity and variation were similar between the first and last hour of the night, the obesity parameter changes were more strongly associated with light exposure during the last hour of the night than the first hour of the night. According to the phase-response curve to light (36), early morning light advances the internal circadian phase, suggesting that light exposure during the last part of the night may cause circadian misalignment between the internal and environmental rhythms.

In the evening, increased light exposure was significantly associated with both WHtR and BMI gain, which may have resulted from the circadian phase delay or the shortened biological night. Although this association was insignificant in the unadjusted models, after adjusting for important confounders (ie, age and gender), this association became significant. The phase-response curve indicated that the magnitude of phase delay is greater in the evening than in the other period (36). Moreover, previous studies suggested that evening exposure to room light < 200 lux suppressed the onset of melatonin synthesis (37) and prolonged sleep initiation (38), indicating that evening light shortened the melatonin-organized biological night and sleep duration. In our present study, the associations between light exposure and obesity parameters were independent of the total amount of nighttime melatonin secretion and subjective sleep quality.

Morning or daytime exposure to a longer time ≥ 500 lux significantly reduced the obesity parameters, which was possibly associated with the circadian alignment and enhanced sleep quality and melatonin levels. This moderate light intensity measured using wrist light meters generally originates in sunlight and would contribute to synchronization of the internal rhythms to the environmental rhythms. A previous cross-sectional study suggested that early daytime exposure to moderate light intensity ≥ 500 lux was significantly associated with lower BMI (26). Midday bright light intervention of 2500 lux enhanced nocturnal melatonin secretion and improved sleep quality among elderly patients with insomnia (39). In our previous study (22), higher daytime ambient light exposure was associated with greater nocturnal melatonin levels, which relate to circadian alignment and may regulate energy expenditure through growing and activating brown adipose tissue (40, 41).

Cumulative effects of light exposure on the obesity risk can be estimated from our results. Higher nighttime light

intensity (≥ 3 vs < 3 lux) and increased evening light intensity from the 25th to 75th percentiles (15.9–37.0 lux) corresponded to 10.2 and 4.5% for WHtR gain and 10.0 and 9.1% for BMI gain, respectively, over 10 years. Conversely, increased daytime exposure to time ≥ 500 lux and increased nighttime exposure to time < 3 lux from the 25th to 75th percentiles (40–129 and 420–516 minutes, respectively) corresponded to 5.3 and 6.1% for WHtR loss, and 6.7% and 7.1% for BMI loss, respectively, over 10 years. In our study, nighttime light intensity and daytime light exposure had mutually independent effects on the obesity parameters. Additionally, increased nighttime light and decreased daytime light are common exposures in modern society, suggesting high population attributable risks. Thus, changing daily light exposure patterns may be clinically significant for reducing the obesity risk.

Previous epidemiological studies have indicated that short sleep duration increases the risk of obesity (42). In fact, inadequate sleep decreases leptin levels and increases ghrelin levels, resulting in increased appetite and obesity risk (43); however, our main results were independent of baseline caloric intake. Also, nighttime light is a potential sleep disruptor even at low light intensity (44). Additionally, previous population-based studies suggested that the late chronotype was associated with obesity in adult and children (45, 46) and showed the late chronotype was exposed to a higher light intensity in evening and short sleep duration (47, 48). Thus, light exposure may be the potential upstream factor of this previous epidemiological evidence. Longitudinal studies investigating the associations of light exposure with sleep duration and chronotypes are needed.

This study has several limitations. First, participants were not randomly selected, possibly leading to selection bias. However, the parameters of BMI and the estimated glomerular filtration rate were similar to the corresponding national data for Japan (49). Second, the follow-up data on obesity parameters were only available from 766 participants (69%), possibly affecting the validity of the statistical results. As shown in Tables 1 and 2, participants lost to follow-up were significantly older, had lower education levels, engaged in less physical activity, spent longer durations in bed, had lower UME levels, and were exposed to higher nighttime light levels. These factors may lead to an underestimation of the detected influence because of exposure to higher nighttime light intensity and poor lifestyles leading to obesity. Third, the present cohort only included elderly participants; therefore, the generalizability of our findings to a younger population remains undetermined. Age-related cloudiness of the crystalline lens causes decreased light reception to the retina, even before cataract diagnosis, and the capacity for light recep-

tion in a 70-year-old is one-fifth of that in a teenager (50), possibly implying that light exposure has greater effects in younger individuals. Studies on the younger population are warranted. In addition, we had no information related to the incidence of chronic diseases associated with a loss of WHtR and BMI in our elderly population, so some residual confounding effect of the chronic diseases may exist. Fourth, light levels were measured over only 2 days without eye-level light meters, possibly leading to misclassification of the ambient light status. A previous study reported significant correlations between illumination at the cornea level and the wrist level ($r = 0.76$) (51). The day-to-day reproducibility of light exposure parameters was reported in our previous study where both daytime and nighttime light intensities were moderately correlated ($r = 0.61$ – 0.70) (22). The interseasonal reproducibility was fair ($r = 0.45$ – 0.47) (39); however, the present statistical models were adjusted for day length. Furthermore, future studies measuring wave lengths would reveal more appropriate associations because shorter wave lengths are mostly sensitive to alignment of circadian biological rhythms (50).

In conclusion, ambient light exposure, such as increased nighttime or evening light exposure and decreased morning or daytime light exposure, was independently associated with subsequent increases in obesity parameters in a general elderly population. Returning to a natural light exposure pattern may help prevent obesity. Further interventional studies are warranted to establish an optimal controlled lighting environment as a preventive option against obesity.

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