Dietary Patterns Only Partially Explain the Effect of Short Sleep Duration on the Incidence of Obesity

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Study Objectives: To investigate whether dietary patterns explain the possible association between short sleep duration and obesity.
Design: Longitudinal study.
Setting: Annual health checkup at a Japanese workplace over a 4-year period from 1994-1995 (baseline) to 1998-1999 (follow-up).
Participants: Nonobese Japanese male workers aged 40 to 59 years (n = 2632)
Measurements and Results: Trained health professionals conducted a questionnaire-based survey. Preference for fatty food, skipping breakfast, and eating out were significantly associated with short sleep duration. Snacking and preference for fatty food significantly predicted the incidence of obesity, which was defined as a body mass index of at least 25 kg/m^2. Hierarchic logistic regression analyses were conducted to test the significance of the association between sleep duration and the incidence of obesity, before and after controlling for covariates, including dietary patterns (preference for fatty food, skipping breakfast, snacking, and eating out). Participants who slept less than 6 hours were compared with those who slept 7.0 to 7.9 hours. The odds ratio for the incidence of obesity was 2.55 (95% confidence interval [CI] 1.48, 4.42; trend P = 0.007) with covariate adjustment, except for dietary patterns, and 2.46 (95% CI 1.41, 4.31; trend P = 0.011) with complete adjustment, including dietary patterns.
Conclusions: Preference for fatty food, skipping breakfast, snacking, and eating out only partially explained the effects of short sleep duration on the incidence of obesity, suggesting that other factors, including physiologic mechanisms, may largely explain the sleep-obesity association.
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WHETHER SHORT SLEEP DURATION IS ASSOCIATED WITH A HIGHER RISK OF OBESITY HAS BEEN A TOPIC OF DEBATE.[1-5] RECENT REVIEW ARTICLES[6-8] HAVE concluded that the results of previous studies do not lead to a consensus and that the results might have been affected by confounding behavior factors such as diet, smoking, and exercise. Since lack of sleep and the associated psychological stress likely lead to overeating, a preference for fatty food, and a subsequent increase in total energy intake,[9] it is assumed that the diet factor is the most influential intermediary variable in the association between sleep duration and obesity.

In a recent study, Patel et al.[3] observed the independent association of short sleep duration with obesity after adjusting for total energy intake, which was estimated using a self-administered food frequency questionnaire. They concluded that the prospective relationship of short sleep duration with obesity is independent of total energy intake. Although the association between increased total energy intake and obesity seems quite obvious in theory, studies on nutrition epidemiology have determined that energy intake estimated using food frequency questionnaires often fails to predict obesity status. The estimated energy intake is likely biased, presumably because obese subjects tend to underreport their food intake.[10] In fact, Patel et al. failed to show a relationship between the estimated total energy intake and body mass index (BMI).[3] Thus, the independent association between short sleep duration and obesity observed by Patel et al. might have been influenced by uncontrolled residual dietary factors.

Several studies have reported that dietary habits, such as preference for fatty food, frequency of snacking, and eating out, better reflect obesogenic dietary behaviors and significantly predict the risk of obesity, as compared with the estimated total energy intake.[9,11-15] In another study with Japanese workers, a sleep duration of less than 6 hours was found to be associated with a higher frequency of eating out, snacking, and irregular meal patterns.[16] Thus, controlling for the 4 dietary patterns mentioned above rather than using the estimated total energy intake would better explain the association between sleep duration and obesity.

In this study, we tested whether the association between short sleep duration and the incidence of obesity could be explained by the 4 dietary patterns, in addition to other behavior factors, such as smoking and exercise.

METHODS

Data Source

The data presented herein were derived from annual health checkups that were conducted at a gas company in Japan from October 1994 to September 1995 (the year of the baseline survey) and from October 1998 to September 1999 (the year of the follow-up survey) over a 4-year period. Almost all employees of this company were white-collar workers. Since the Japanese domestic law mandates that all employers provide their employees with an annual health checkup, the participation rate

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for health checkups was as high as 98.1% at baseline and 97.8% at follow-up. At the checkup in the year of the baseline survey, the company offered employees between the ages 40 and 59 years a lifestyle questionnaire, including questions on sleep duration and dietary patterns, which formed the data source of our current study. The available data were limited to men because the company authorities did not provide us with data on its female employees due to their small numbers and confidentiality concerns. The total number of male workers aged 40 to 59 years was 4826 at baseline. After excluding those who had missed check-up at baseline (n = 142) and not responded to key variables (n = 240), we arrived at a total of 4444 respondents who provided complete data at baseline. Of these, 3803 respondents (response rate, 79% of the 4826 subjects) were available after excluding those lost at follow-up (n = 641). As compared with the remaining 3803 subjects included in the analysis, the 240 subjects with missing questionnaire responses were significantly older (mean 49.3 years vs. 47.8 years) but did not differ in BMI. As compared with the subjects included in the analysis, those lost to follow-up were significantly older (mean 53.6 years vs. 47.8 years), less likely to be smokers (51.0% vs. 56.3%), show a preference for fatty food (14.8% vs. 18.3%), have a family history of diabetes (11.9% vs. 15.0%), and more likely to take medication for hypertension (13.1% vs. 8.5%), but they did not differ in any other variables, including BMI and sleep duration. Since our main concern was the incidence of obesity, we limited the subjects to nonobese (BMI < 25 kg/m^2) respondents at baseline for subsequent analyses (n = 2632). The definition of obesity complied with the criteria of the Japan Society for the Study of Obesity.[17] The company's health authority approved the secondary use of the health checkup data for research purposes, and verbal consent for the generic use of the health check-up data for research purposes was obtained from all participants at checkup. All data were provided anonymously.

Study Measures

Dependent variable

The target outcome was the incidence of obesity, which was defined as a BMI of at least 25 kg/m^2.[17] Height and weight were objectively measured.

Independent variables

The question for the data on sleep duration was "How many hours, on average, do you sleep during the night?" The data were further categorized into 4 groups: less than 6 hours of sleep, 6 to 6.9 hours of sleep, 7 to 7.9 hours of sleep, and more than or equal to 8 hours of sleep. The range of 7 to 7.9 hours of sleep was set as a reference for comparison with previous studies. Data on dietary patterns (preference for fatty food, skipping breakfast, snacking, and eating out) were collected as dichotomous variables (*Yes* or *No*). In the case of preference for fatty food, for example, the question asked was "Do you mostly prefer for fatty foods?" The self-administered questionnaire also inquired about current medication (none, 1 or more medications for diabetes, dyslipidemia, hypertension, or psychiatric disorders); family history of disease (none, 1 or more of family history of diabetes, dyslipidemia, or hypertension); smoking status (current smoker or nonsmoker); habitual drinking (defined as consuming alcohol once or more per week); and habitual exercise (defined as exercising once or more per week). Age data were obtained from the company's administrative records.

Statistical Analysis

Baseline differences among the sleep-duration categories were assessed by analysis of variance for continuous variables and by χ^2 tests for categorical variables. Crude associations of dietary patterns with the sleep category were tested. We then carried out univariate logistic regression analyses to test the association between self-reported dietary patterns and subsequent obesity to determine the pattern of influence. Finally, we conducted hierarchic logistic regression analyses to determine the impact of dietary patterns on the prospective relationship between self-reported sleep duration and the incidence of obesity. More specifically, we conducted logistic regression modeling to estimate the relationship between self-reported sleep duration and the incidence of obesity with hierarchically adding adjustments for covariates to the models: age and baseline BMI (model 1), model 1 plus individual behaviors (smoking, drinking, and exercise), and medical history (family history of a disease and current medication) (model 2). Thereafter, we performed further adjustments for the possible effects of dietary patterns by adding preference for fatty food to model 2 (model 3) and skipping breakfast, snacking, and eating out to model 3 (model 4). Tests for linear trends were computed using sleep-duration categories as an ordinary variable. These analyses were performed using SPSS version 15.0J (SPSS, Inc. Chicago, IL). A value of 5% was set as the conventional significance level.

RESULTS

Table 1 displays the baseline characteristics of the 4 sleep-duration groups. In baseline cross-sectional observation, shorter sleepers tended to weigh more than those who slept longer. The individuals who slept less than 6 hours were less likely to exercise, compared with those in the other groups, although the difference was marginally significant. Those who slept 8 hours or more were, on average, older, more likely to smoke, and to take more medications for hypertension and psychiatric disease. The proportions of preference for fatty food, skipping breakfast, and eating out significantly increased with a decrease in sleep duration. Snacking showed a similar trend, although the difference was not statistically significant.

Table 2 shows the results of a univariate logistic regression analysis to predict the prospective incidence of obesity by dietary patterns. The variables associated with a higher risk of obesity were preference for fatty food (odds ratio [OR] = 2.01, 95% confidence interval [CI] 1.34, 3.00) and snacking (OR = 2.10, 95% CI 1.21, 3.65). Skipping breakfast and eating out did not significantly predict the incidence of obesity in our study.

Table 3 demonstrates the longitudinal association between short sleep duration and the incidence of obesity before and after accounting for the effect of self-reported dietary patterns. Compared with model 1, or the model adjusted for age and baseline BMI, model 2, incorporating all covariates other than dietary patterns, showed slightly larger OR for sleep duration for the group of individuals who slept less than 6 hours (OR = 2.55,

95% CI 1.48, 4.42). In either model, short sleep duration significantly predicted the subsequent incidence of obesity. Then, we evaluated the impact of dietary patterns on the association between short sleep duration and the incidence of obesity. Adjustment for preference for fatty food slightly attenuated the point estimation of sleep duration on the incidence of obesity and narrowed the 95% CI (OR = 2.50, 95% CI 1.44, 4.33) (model 3). Finally, we adjusted for the 3 remaining dietary patterns and observed an additional slight attenuation of the OR, yet sleep duration remained significant (model 4). P values for a trend test were 0.008 for model 1, 0.007 for model 2, 0.010 for model 3, and 0.011 for model 4.

Table 1—Baseline characteristics[a] of nonobese respondents[b] by sleep duration (n = 2,632)

	Self-reported sleep duration (h)				
	less than 6 (n = 228)	6–6.9 (n = 1,079)	7–7.9 (n = 1,073)	8 or more (n = 252)	P Value[c]
Age (years)	47.8 (5.6)	47.5 (5.1)	47.9 (5.3)	48.6 (5.6)	0.021
Height (cm)	168.6 (5.4)	168.7 (5.5)	168.6 (5.6)	168.4 (5.6)	0.872
Weight (kg)	64.6 (6.0)	64.2 (6.3)	63.9 (6.3)	63.2 (6.5)	0.033
BMI (kg/m^2)	22.7 (1.6)	22.5 (1.7)	22.5 (1.8)	22.3 (1.9)	0.015
Current medication (%)					
Diabetes	2.6	2.3	1.4	1.6	0.355
Dyslipidemia	1.3	0.9	1.5	2.0	0.492
Hypertension	5.7	5.6	6.3	9.1	0.206
Psychiatric disease	0.0	0.3	0.2	1.2	0.052
1 or more of the above	8.8	8.5	8.9	13.5	0.099
Family history of disease (%)					
Diabetes	15.8	13.3	12.6	15.9	0.386
Dyslipidemia	0.4	0.4	0.9	0.4	0.367
Hypertension	31.6	36.1	31.6	36.1	0.118
1 or more of the above	39.5	43.5	40.3	44.4	0.322
Smoking habit (%)	55.3	56.3	57.4	62.7	0.285
Drinking habit (%)	81.6	82.7	84.5	83.7	0.578
Exercise habit (%)	32.5	39.1	35.6	34.5	0.136
Preference for fatty food (%)	19.3	16.0	11.3	14.3	0.002
Skipping breakfast (%)	22.8	15.8	14.4	13.9	0.013
Snacking (%)	6.1	6.7	4.8	5.6	0.289
Eating out (%)	25.0	19.6	10.9	7.9	< 0.001

[a]Data are presented as means (standard deviations) or percentages; [b]Respondents with BMI < 25 kg/m^2 at baseline; [c]Statistical significance was assessed by analysis of variance with continuous variables and by a chi-square test with categorical variables.

Table 2—Univariate analysis of dietary patterns on the incidence of obesity[a] (n = 2,632)

Preference for fatty food	No. of Respondents	No. of Obesity	Odds Ratio	95% CI
Yes	374	35	2.01	(1.34, 3.00)
No	2,258	107	1.00	
Skipping breakfast				
Yes	411	26	1.17	(0.75, 1.83)
No	2,221	116	1.00	
Snacking				
Yes	151	16	2.10	(1.21, 3.65)
No	2,481	126	1.00	
Eating out				
Yes	406	25	1.09	(0.70, 1.72)
No	2,226	117	1.00	

[a]Obesity was defined as a BMI ≥ 25 kg/m^2 according to the criteria determined by the Japan Society for the Study of Obesity.

DISCUSSION

In this study, we demonstrated that the longitudinal association between short sleep duration and the incidence of obesity was not susceptible to the effect of a preference for fatty food, skipping breakfast, snacking, and eating out.

Diet seems to naturally be the most likely mediator in sleep-obesity association. Patel et al. explored the impact of diet using total energy intake, estimated by a food frequency questionnaire,[3] although the estimated total energy intake was not associated with BMI, presumably due to reporting bias.[10] We examined the impact of diet by including questions on dietary patterns, which have been reported to better reflect the association between dietary habit and obesity.[9,11-15] As expected, self-reporting on a preference for fatty food and snacking habits successfully predicted the incidence of obesity (Table 2), and the preference for fatty food was significantly associated with sleep duration, although these dietary patterns did not fully cancel the direct association between short sleep duration and obesity. As previously reported, short sleepers may suffer from chronic stress[5] and show increased preferences for eating energy-dense foods such as fatty food or sweets.[9] Thus, there would be no surprise if the preference for energy-dense foods mediates the sleep-obesity association. If so, adjusting for the preference would remarkably attenuate the statistical association between sleep duration and the incidence obesity. In our results, however, such an attenuation by food preference was limited, which strongly suggests an alternative pathway between sleep duration and obesity.

In contrast with our findings, some previous cross-sectional studies have reported that long sleepers tend to be obese.[7] In our study, those with the longest sleep duration were the oldest and the most unhealthy, suggesting a reverse causality among long sleepers for obesity because of their age and poor health conditions, which are both related to obesity.

A sleep-obesity mechanism mediated by dietary behavior has often been discussed in previous studies, e.g., overeating

Table 3—The effect of dietary patterns on the longitudinal association of sleep duration with the incidence of obesity[a] (n = 2,632)

	Self-reported sleep duration (h)						
	less than 6 (n = 228)		6–6.9 (n = 1,079)		7–7.9 (n = 1,073)	8 or more (n = 252)	
	Odds Ratio	95% CI	Odds Ratio	95% CI	Reference	Odds Ratio	95% CI
Model 1[b]	2.53	(1.47, 4.37)	1.08	(0.71, 1.64)	1.00	0.78	(0.36, 1.67)
Model 2[c]	2.55	(1.48, 4.42)	1.07	(0.71, 1.63)	1.00	0.76	(0.36, 1.65)
Model 3[d]	2.50	(1.44, 4.33)	1.06	(0.70, 1.61)	1.00	0.76	(0.35, 1.63)
Model 4[e]	2.46	(1.41, 4.31)	1.03	(0.68, 1.58)	1.00	0.75	(0.35, 1.62)

[a]Obesity was defined as a BMI ≥ 25 kg/m^2 according to the criteria determined by the Japan Society for the Study of Obesity; [b]Model 1, age and baseline BMI; [c]Model 2, Model 1 + current medication, family history of disease, smoking, drinking, and exercise; [d]Model 3, Model 2 + preference for fatty food; [e]Model 4, Model 3 + skipping breakfast, snacking, and eating out.

as a stress-coping behavior[18] and appetite increase as a result of subsequent hormone responses induced by leptin and ghrelin.[19] Residual dietary patterns, such as preference for sweet foods, may mediate the sleep-obesity association. Several physiologic mechanisms might also be involved in the association between short sleep duration and obesity. First, body thermoregulation is affected by neuropeptides. One of the functions of leptin is to increase energy expenditure by activating proopiomelanocortin neurons as well as appetite control.[20] Short sleep duration is associated with reduced leptin levels.[19] Hence, short sleep duration might lead to decreased energy expenditure. Second, the sleep quality of short sleepers might be poorer,[21] and this might cause obesity via altered glucose metabolism.[22] Individuals who sleep less (e.g., by working for long hours[23]) might be under greater stress than those who sleep more; this might also affect metabolism by activating the corticoadrenal pathway.[5] Third, short sleep duration might activate inflammatory pathways, leading to obesity.[24] Since we do not have data on the biomarkers mentioned above, we could not determine the most plausible mechanism. We have to await the results of future studies that measure related biomarkers to detect the underlying physiologic mechanism that links sleep to obesity.

This study has several additional limitations that require discussion. First, sleep duration was dependent on self-reporting, as in previous studies. A misclassification of sleep duration would attenuate any association between short sleep duration and obesity if the misclassification were nondifferential. If misclassification were related to baseline BMI, this would not be the case. Lauderdale et al.[25] reported that obese subjects systematically report a shorter sleep duration than do the nonobese at the same level of objectively measured sleep duration. Thus, in cross-sectional observation, our results might have been affected by differential misclassification. If differential misclassification arises in longitudinal observation, however, the longitudinal trend between sleep duration and changes in BMI should vary across baseline BMI levels. We conducted further analysis stratified by baseline BMI and observed the same trend independent of baseline BMI levels (data not shown). For this reason, we believe that our self-reported measure of sleep duration did not affect our conclusions. Second, this study also relied on self-reporting of 4 dietary patterns, which might suffer from low reliability due to misclassification, and the effect by dietary patterns could be underevaluated. As shown in Table 2, a relationship of dietary patterns with obesity was significantly apparent. Despite this, inclusion of dietary patterns only partially explains the significance of sleep duration on the incidence of obesity. Although the possibility of uncontrolled residual effect due to dietary behavior remains, the point estimates virtually unaffected by dietary behaviors surveyed suggests that the intermediary effect of the presented dietary factors may be limited in the prospective relationship between sleep duration and obesity. Since we did not include questions on appetite change and regular portion size, however, there is another possibility that increased appetite could lead to increased caloric intake without affecting the dietary behaviors. Third, our study population was restricted to Japanese male workers aged 40 to 59 years, which may limit the generalizability of our results. Compared with our samples, younger people or women could have different dietary patterns and sleep habits because of their different socioeconomic status, such as marital status, families living together, and their work. Additional studies with a more diverse population might be required to extend the range of application of the findings. Fourth, our sleep questionnaire lacked a definition of timeframe. Nevertheless, it is unlikely that this causes systematic bias in the results. Fifth, the available data on sleep were limited to sleep duration. Since quality of sleep and sleep disorders might affect obesity, additional study examining a wide variety of sleep parameters would be necessary to confirm our results. Finally, we did not consider the timing of meal intake as a possible risk for obesity. Oscillations in plasma glucose levels and insulin secretion rate are known to increase during sleep in humans under continuous enteral nutrition.[26] Thus, the timing of the last meal intake, which should be associated with shorter sleep duration, might be worth future investigation.

In conclusion, our prospective analysis showed that a preference for fatty food, skipping breakfast, snacking, and eating out only partially explained the effects of short sleep duration on the incidence of obesity. This suggests that other factors, including physiologic mechanisms, may largely explain the sleep-obesity association. From a practical aspect, these results may also imply that modification of the 4 presented dietary patterns through health education may not be enough to prevent obesity among individuals who have inadequate sleep. In addition to individual education, modification of environmental conditions, such as work hours, to allow for an adequate duration of sleep might be necessary for obesity prevention.

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DISCLOSURE STATEMENT

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