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Actigraphic sleep fragmentation, efficiency, and duration associate with dietary intake in the Rotterdam Study

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Abstract

Background—Short self-reported sleep duration is associated with dietary intake and this association may partly mediate the link between short sleep and metabolic abnormalities. Subjective sleep measures, however, may be inaccurate and biased.

Objective—To evaluate the associations between actigraphic measures of sleep fragmentation, efficiency and duration and energy and macronutrient intakes.

Methods—We used data from a subgroup of 439 participants of the population-based cohort, Rotterdam Study. Sleep was assessed using 7-d actigraphy and sleep diaries, and dietary data with a validated food frequency questionnaire. We assessed the associations of actigraphic sleep parameters with dietary intake using multivariable linear regression models.

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Conflict of interest: Hassan S Dashti, Lisette A Zuurbier, Ester de Jonge, Trudy Voortman, Paul F Jacques, Stefania Lamon-Fava, Frank AJL Scheer, Jessica C Kiefte-de Jong, Albert Hofman, José M Ordovás, Oscar H Franco, and Henning Tiemeier declare no conflicts of interest.

Results—Higher sleep fragmentation was associated with 4.19 g lower carbohydrate intake per standard deviation of fragmentation [β (95%CI) =-4.19 (-8.0, -0.3); P=0.03]. Each additional % increase in sleep efficiency was associated with 11.1 kcal lower energy intake [β (95%CI) =-11.1 (-20.6, -1.7); P=0.02]. Furthermore, very short sleep duration (<5.5h) was associated with 218.1 kcal higher energy intake [β (95%CI) =218.06 (33.3, 402.8), P=0.02], relative to the reference group (≥ 6.5 to <7.5h).

Conclusions—We observed associations between higher sleep fragmentation with lower carbohydrate intake, and both lower sleep efficiency and very short sleep duration (<5h) with higher energy intake. The association between short sleep duration and higher energy intake could in part mediate the link between short sleep or sleep fragmentation index and metabolic abnormalities.

Keywords

Sleep; actigraphy; dietary intake; macronutrients; sleep duration; fragmentation

Introduction

Habitual sleep duration among US adults falls short of the recommended hours of sleep by the National Sleep Foundation of 7-9 h for adults (26-64 y) and 7-8 h for older adults (≥65 y) to maintain health (Buman et al. 2014; Hirshkowitz et al. 2015). Short self-reported sleep duration has been associated with obesity (Cappuccio et al. 2008), diabetes (Ayas et al. 2003), cardiovascular disease (Cappuccio et al. 2011), and all-cause mortality (Cappuccio et al. 2010). Cardiometabolic diseases associated with short sleep are possibly mediated in part through differences in dietary intake (Dashti et al. 2015). Indeed, large cross-sectional studies have observed associations between short sleep and higher total energy (Stern et al. 2014; Grandner et al. 2010) and altered macronutrient (Dashti et al. 2015; Grandner et al. 2010; Kant and Graubard 2014) intakes. In addition, sleep extension trials have identified overall reduced food intake, particularly lower fat and carbohydrate intakes, and lower overall appetite for sweet and salty foods following extended sleep hours (Markwald et al. 2013; Tasali et al. 2014), suggesting that achieving recommended habitual sleep durations may ameliorate risk for cardiometabolic diseases via a more favorable dietary profile (Dashti et al. 2015).

Research has focused primarily on self-reported sleep duration, although it is well recognized that subjective sleep measures may be inaccurate and biased (Lauderdale et al. 2009). The concordance between subjective and actigraphic sleep measures is generally low (van den Berg et al. 2008b; Silva et al. 2007). Disagreements between subjective and actigraphic sleep measures may contribute to the observed differences in the associations between sleep duration and dietary intake depending on the method for sleep assessment (Grandner et al. 2010). Therefore, more accurate assessment of sleep through polysomnography or actigraphy might better capture the true associations between various sleep parameters and dietary intake (Zhang and Zhao 2007).

In addition to sleep duration, recent reports have suggested that other sleep measures such as fragmentation and efficiency are important predictors of metabolic disorders (Sawamoto et

al. 2014; Gonnissen et al. 2013). For example, greater sleep fragmentation has been associated with higher BMI (Lauderdale et al. 2009; van den Berg et al. 2008a) and lower success at weight-loss (Sawamoto et al. 2014). However, whether these associations may be explained by differences in dietary intake remains unknown. The aim of the present study was to evaluate the associations between sleep parameters measured with actigraph (sleep fragmentation, efficiency and duration) and dietary intake (energy and macronutrient intakes).

Methods

Study population

The described analyses were conducted in a non-shift work cohort from the Rotterdam Study, a population-based cohort started in early 2006 onwards of middle-aged adults of ages 45 and over residing in Rotterdam, the Netherlands (Hofman et al. 2015). The study was conducted in accordance with the guideline proposed in the World Medical Association Declaration of Helsinki and approved by the medical ethics committee according to the Wet Bevolkingsonderzoek ERGO (Population Study Act Rotterdam Study), executed by the Ministry of Health, Welfare and Sports of the Netherlands. Written informed consent was obtained from all participants. At the time of recruitment, among 1,515 individuals from the Rotterdam Study invited to participate in the actigraphy study, a total of 627 individuals had dietary data of which 188 were further excluded (van den Berg et al. 2008b). Of these who were excluded, 43 refused participation, 17 did not have actigraphic recordings of 4 consecutive days and nights, 16 had recordings collected within a week of daylight saving time to eliminate discrepant sleep data alignment between actigraphs and sleep diaries and acute changes in sleep patterns, 58 had actiwatches malfunctioning, 31 had missing dietary data, and 23 frequently used sleep medication/sedative. Thus, 439 individuals were included in the current analysis.

Assessment of actigraphic sleep parameters

Actigraphy was used to estimate sleep parameters objectively (Kushida et al. 2001; Ancoli-Israel et al. 2003). All participants wore an actigraph around the non-dominant wrist (Actiwatch model AW4, Cambridge Neurotechnology Ltd) continuously for 7 consecutive days and nights, and the actigraph was only to be removed while bathing. Actigraphs measured in 30-s epochs (Kushida et al. 2001). Recordings had to consist of at least 96 h. All 24-h periods with more than three continuous hours missing were excluded from the analyses to prevent a time-of-day effect. The average duration of the actigraphy recordings was 138 h [standard deviation (SD): 14 h]. A validated algorithm was used to calculate sleep duration (calculated as the total time of the epochs classified as sleep between sleep start and sleep end, according to sleep diaries), fragmentation index (the number of interruptions of sleep by physical movement calculated as 100 × the number of groups of consecutive mobile 30-s epochs/by the total number of immobile epochs) and sleep efficiency (100% × sleep duration/the time between bed time and get up time) using actigraphy data as reported previously (van den Berg et al. 2008a; Luik et al. 2013; Cambridge Neurotechnology Ltd.)

Assessment of subjective sleep parameters

A 7-d sleep diary completed during the same week of the actigraphy was used to estimate subjective sleep parameters. The sleep diary included questions on sleep characteristics, sleep medication (frequency and kind), and alcohol and coffee intakes. Self-reported sleep duration was averaged over the 7-d period (Luik et al. 2013). Daytime napping was evaluated by questions regarding napping before 1800 hours and estimated as the total number of days during which the participant took a nap, adjusted for the total number of days for which the participant contributed data to the current analysis.

Assessment of dietary intake

Dietary data were collected via an interviewer-administered, 389-item semi-quantitative validated food frequency questionnaire (FFQ) at the time of participants' recruitment. The FFQ is based on an existing and validated FFQ developed for Dutch adults (Goldbohm et al. 1994). For each food item, the frequency (in times per day, week, or month), serving sizes per frequency (expressed in natural units, household measures, or grams), and preparation methods were included. Food and dietary supplement data were then converted into energy and nutrient intakes per day using the Dutch Food Composition Table of 1993 (RIVM, 1993). The present analysis quantified energy intake in kcal/day and macronutrient intakes [carbohydrate [simple (monosacchride and disaccharide) and complex carbohydrate (polysaccharide)], fat, and protein intakes] as absolute intakes in grams/day. Evening alcohol and coffee consumption were self-reported during the week of actigraphy and expressed as the average number of drinks/cups per week after 1800 hours over the 7-d period of sleep assessment.

Assessment of confounders

Along with dietary intake, demographics and lifestyle information were collected at in-home interviews in this study. Depressive symptoms were assessed with the Center for Epidemiologic Studies Depression scale (Radloff 1977). The Pittsburgh Sleep Quality Index (PSQI) (Buysse et al. 1989) was used to determine possible sleep apnea by responses to the following two questions: (1) loud snoring at least two nights per week and at least occasional respiratory pauses, or (2) respiratory pauses during sleep with a frequency of at least 1-2 nights weekly. Height and weight were measured without shoes and heavy clothing during a center visit and body mass index (BMI) was calculated as weight (kg) divided by the square of height (m^2). Physical activity was assessed by means of an adapted version of the Zutphen Physical Activity Questionnaire and the LASA Physical Activity Questionnaire (Hofman et al. 2015) and information on education level (low/intermediate/high), smoking status (never/former/current), and type 2 diabetes status were also collected.

Statistical analysis

Dietary intake outcome variables (energy, carbohydrate, fat, and protein intakes), sleep fragmentation index, and sleep efficiency were expressed continuously. Fragmentation index was expressed as standard deviation units to facilitate interpretation. Actigraphic and subjective sleep durations were first analyzed continuously, then divided into 4 categories [*actigraphic*: <5.5 h, ≥ 5.5 to < 6.5 h, ≥ 6.5 to < 7.5 h (reference), and ≥ 7.5 h; *subjective*: <6

h, ≥ 6 to < 7 h, ≥ 7 to < 8 h (reference), and ≥ 8 h] (van den Berg et al. 2008a) to achieve groups of adequate sample sizes necessary to reflect the possible non-linear (U- or J-shaped) associations between sleep duration and dietary intake outcomes. Missing values [*n*=6 (education level); *n*=3 (BMI, sleep apnea); *n*=2 (alcohol, coffee intakes); *n*=52 (physical activity)] were imputed as follows: missing continuous variables were replaced with the median, and missing values for categorical variables were placed in a separate category. Individuals with missing sleep fragmentation index data (*n* =50) and improbable caloric (<500 kcal/day and >5000 kcal/day) and nutrient intakes (*n*=1; fat) were excluded from their respective analyses. The degree of agreement between actigraphic and subjective sleep duration estimates was quantified by computing a concordance correlation coefficient.

We assessed the associations between actigraphic sleep parameters [fragmentation index, sleep efficiency, and sleep duration (continuous and categorical)] or subjective sleep duration (continuous and categorical) on dietary intake [total energy intake (kcal/day), total carbohydrate (g/day), total fat (g/day), and total protein intakes (g/day)] using multivariable linear regression models in successive models. Model 1 was adjusted for sex, age, BMI, and total energy intake (except for when assessing energy intake as an outcome). Model 2 further accounted for lifestyle factors by adjusting for smoking status, education level, alcohol intake, coffee intake, daytime napping, depressive symptoms, physical activity, sleep apnea, and type-2 diabetes status. We presented results from Model 2 only, except when the results differed between the two models.

In *post hoc* analysis, based on findings from the models described above, we further investigated the associations between sleep fragmentation index on simple and complex carbohydrate intakes using linear regression models adjusted for the aforementioned covariates. Furthermore, in sensitivity analyses, we removed BMI from our Model 1 for all described analyses, and also excluded participants with sleep apnea from our significant findings. All statistical analyses were performed using R statistical software (v3.1.0, Vienna, Austria). As our dietary outcome variables were highly correlated, Bonferroni correction was regarded as too conservative, and instead a two-tailed p value of < 0.05 was considered statistically significant.

Results

General characteristics

The general characteristics of the participants (*n*=439) are presented in Table 1. The mean age of the population was 55.9 yrs (SD: 5.4) and 59.2% was female. Mean fragmentation index was 6.1 (SD: 2.1), mean sleep efficiency was 79.9% (SD: 6.1), mean sleep duration was 6.4 h (SD: 0.8) and 6.9 h (SD: 0.8) as measured by 7-d actigraphy and self-report, respectively. The coefficient of concordance between actigraphic and self-reported sleep duration was 0.40 [95% confidence interval (CI): 0.33, 0.46; $P < 0.001$], whereby self-reported sleep duration overestimated actigraphic sleep duration on average by 24 min (95% CI: 20, 28). Average energy intake was 2239 kcal per day (SD: 636), and macronutrient intake distribution was as follows: 254.0 (SD: 78.9), 79.9 (SD: 29.6), and 83.5 (SD: 24.9) g per day from carbohydrate, fat, and protein, respectively.

Sleep fragmentation and efficiency associations with dietary intake

Associations between actigraphic sleep fragmentation and efficiency with dietary intake are presented in Table 2. A negative association was identified between sleep fragmentation and carbohydrate intake: higher fragmentation was associated with 4.19 g lower carbohydrate intake per standard deviation of fragmentation [β (95%CI) =-4.19 (-8.0, -0.3); P =0.03], after adjusting for confounders (Model 2). In *post hoc* analysis, we identified associations between sleep fragmentation and types of carbohydrate: higher fragmentation was associated with 4.27 g lower simple carbohydrate intake per standard deviation of fragmentation [β (95%CI) =-4.27 (-8.2, -0.4); P =0.03], but not complex carbohydrate intake [β (95%CI) =0.54 (-0.7, 1.8); P =0.40] after adjusting for sex, age, BMI, and total energy intake (Model 1) only.

Furthermore, we identified an association between sleep efficiency and energy intake (Table 2). Each additional % increase in sleep efficiency was associated with 11.1 kcal lower energy intake [β (95%CI) =-11.1 (-20.6, -1.7); P =0.02] after adjusting for sex, age, and BMI (Model 1). However, the association was attenuated when adjusting for lifestyle factors (Model 2) [β (95%CI) =-8.75 (-18.4, 0.9); P =0.08]. No associations were identified between sleep efficiency and macronutrient intakes.

Sleep duration associations with dietary intake

Associations between actigraphic and self-reported sleep durations (assessed continuously and categorically) and dietary intake are presented in Table 3. An association was evident between short actigraphic sleep duration and energy intake: sleep < 5.5 h was associated with 218.1 kcal higher energy intake [β (95%CI) = 218.06 (33.3, 402.8), P =0.02], relative to the reference sleep duration group (≥ 6.5 to < 7.5 h). No associations were evident between actigraphic sleep duration and macronutrient intake, or self-reported sleep duration and dietary intake. Lastly, the removal of BMI from our Model 1 and exclusion of participants with sleep apnea in sensitivity analyses did not influence the identified findings (results not shown).

Discussion

Overall, we found associations between higher sleep fragmentation and lower carbohydrate intake, particularly simple carbohydrate. We also identified that lower sleep efficiency and very short sleep duration (< 5.5 h) as assessed by actigraphy were associated with higher energy intake. Although our association effect sizes tended to be small, it's possible that these have accumulative influences over a lifespan contributing to increased risk of obesity and metabolic abnormalities.

Emerging studies investigating sleep parameters as assessed by actigraphy have identified associations between fragmented sleep and higher BMI and risk of obesity (van den Berg et al. 2008a), elevated cortisol and cholesterol levels (Ekstedt et al. 2004), and lower weight reduction among obese women in a weight-loss trial (Sawamoto et al. 2014). Meanwhile, induced sleep fragmentation in experimental studies observed weight gain and changes in plasma leptin concentrations in mice (Wang et al. 2014), but no changes in appetite-related

hormones leptin and ghrelin in humans (Gonnissen et al. 2013). We observed associations between higher sleep fragmentation and lower carbohydrate intake, primarily of simple carbohydrate.

As our present investigation cannot inform us of the temporal relation or the causal pathways linking sleep and diet, association in the reverse direction (i.e., dietary intake on sleep) is plausible (Lauderdale et al. 2009). For example, it is also possible that higher carbohydrate intake is associated with less fragmented sleep (Hartmann and Spinweber 1979, Wurtman et al. 2003), as supported by earlier observations that a carbohydrate-based high glycemic index (GI) meal administered 4-h before bedtime shortened sleep onset latency in healthy sleepers relative to a low GI meal (Afaghi et al. 2007), and reports that indicate that fruits, which contribute to simple carbohydrates, may promote sleep (Peuhkuri et al. 2012). One potential mechanism is through brain tryptophan (Trp), a precursor for brain sleep-inducing agent serotonin (Hartmann and Spinweber 1979), however the exact mechanism remains unclear.

In addition, we observed an association between lower sleep efficiency and higher energy intake. This suggests that inefficient sleep, i.e., spending a longer time awake in bed, may induce increased energy intake. Crispim et al. (Crispim et al. 2011) observed an association between lower sleep efficiency and higher nighttime energy, carbohydrate, and fat intakes among women in a small observational study using polysomnography and 3-d food diary. Therefore it is conceivable that lower sleep efficiency may present additional opportunities for food intake, possibly in the form of nighttime, energy-dense snacks, contributing to higher energy intakes (Kant and Graubard 2014). As the FFQ used in the present study did not assess time-specific meals, i.e., daytime and nighttime, we were unable to investigate the influence of sleep measures on time-specific meals, which may provide a more comprehensive understanding of the relationships between sleep and dietary intake (Crispim et al. 2011). Nonetheless, if unmet by an equivalent increase in energy expenditure, chronic inefficient sleep may mediate increases in BMI through higher energy intake (Markwald et al. 2013).

Our analyses of sleep duration and dietary intake revealed associations between very short sleep duration (< 5.5 h) and higher energy intake. Higher energy intake with short sleep duration (5-6 h) has been reported previously in the Women's Health Initiative (Grandner et al. 2010). Our present findings are also in line with reports of higher BMI with actigraphic short sleep duration (< 6 h) in the Rotterdam Study (van den Berg et al. 2008a), suggesting that these associations may be mediated in part by higher energy intakes. Furthermore, as no associations were evident between self-reported sleep duration and dietary intake, our findings provide additional evidence of the differences in the associations between actigraphic and subjective sleep durations and dietary intake as has been previously observed (Grandner et al. 2010). The low concordance between actigraphic and self-reported sleep duration support the need for objective measures of sleep duration in unraveling the true associations between sleep duration and various traits.

Previously investigated mechanisms linking short sleep duration and higher energy intake may also explain the associations observed between sleep fragmentation and efficiency with

dietary intake in the present study. These mechanisms include differences in appetite-related hormones leptin and ghrelin (Stern et al. 2014; Spiegel et al. 2004; Taheri et al. 2004); changes in hedonic pathways related to enhanced activity of the brain reward and food-sensitive centers in response to unhealthy foods (Chaput 2014; St-Onge et al. 2014); increases in snacking behavior typically in the form of convenient, carbohydrate-rich and energy-dense snacks (Kant and Graubard 2014; Weiss et al. 2010); and late-time eating behavior, particularly after dinner and before breakfast (Kant & Graubard 2014; Nedeltcheva et al. 2009). Considering the small overlap in associations observed for sleep fragmentation, efficiency, and duration with dietary intakes in the present study, whether these obesogenic tendencies also exist in the context of sleep fragmentation and inefficiency requires further investigation.

Our study has several strengths and limitations. While most reports of sleep have relied on self-report, our study used more accurate measures of sleep through 7-d actigraphy. Furthermore, our use of 7-d sleep diaries concurrently with actigraphy allowed us to assess various confounders at the time of sleep data collection. However, our use of self-reported dietary intake via a validated FFQ was susceptible to reporting bias, including overestimation of various nutrients, which may increase measurement error, and bias our results toward the null (Neuhouser et al. 2008). Therefore, to account for some of the systematic measurement error, we have adjusted by energy when assessing macronutrients as outcomes Goldbohm et al. 1994). In addition, statistically controlling for lifestyle factors may be considered overadjustment and may have resulted in fewer observable associations. While our investigation focused on single nutrients, additional associations may be evident between sleep and dietary patterns. Whereas our cohort benefitted from homogeneity among the participants recruited including a narrow age group, the associations observed in this population may not be generalizable to other populations, including those of younger age groups, as previous findings have reported age-specific associations between sleep and dietary intake (Dashti et al. 2015). Lastly, further longitudinal and experimental investigations are imperative to establish the causal relation and mechanistic link between objective sleep measures and dietary intake.

In conclusion, our findings support a link between sleep and dietary intake and appear to suggest that sleep fragmentation, efficiency, and duration vary in their associations with dietary intake. We observed associations between higher sleep fragmentation with lower carbohydrate intake, particularly simple carbohydrate, and both lower sleep efficiency and very short sleep duration (<5 h) with higher energy intake. The influence of sleep on higher energy intake could in part mediate the link between short sleep or sleep fragmentation and metabolic abnormalities (Nedeltcheva et al. 2014). These results are inline with other studies that emphasize the importance of adequate and improved sleep for health and weight management. Future longitudinal studies with follow-up sleep and dietary intake assessment and experimental studies including sleep extension trials are imperative to further elucidate the causal and mechanistic links between sleep and dietary intake.

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Table 1

General characteristics of Rotterdam Study (*n* =439) cohort[1].

Characteristics	All
General Characteristics	
Age (yrs)[2]	55.9 (5.4) [45.7-79.6]
Sex (% female)	59.2
BMI (kg/m^2) [2]	27.5 (3.9) [18.2-41.3]
Smoking Status	
Never, *n* (%)	95 (21.6)
Former, *n* (%)	203 (46.2)
Current, *n* (%)	141 (32.1)
Education Status	
Low, *n* (%)	45 (10.3)
Intermediate, *n* (%)	285 (64.9)
High, *n* (%)	104 (23.9)
Missing, *n* (%)	6 (1.4)
Depressive Symptoms (CES-D score)	5.0 (6.2)
Leisure time physical activity, MET-hours	57.7 (46.1)
Probable Sleep Apnea, *n* (%)	101 (23)
Type 2 Diabetes, *n* (%)	37 (8.4)
Dietary Intake	
Energy (kcal/day)	2239 (636)
Carbohydrate (g/d)	254.0 (78.9)
Simple Carbohydrate (g/d)	124.1 (50.3)
Complex Carbohydrate (g/d)	130.0 (43.6)
Fat (g/d)	79.9 (29.6)
Protein (g/d)	83.5 (24.9)
Alcohol (drinks per week)	5.9 (6.8)
Coffee (drinks per week)	7.0 (5.8)
Multivitamin/Multimineral Supplements, n (%)	108 (24.6)
Actigraphic Sleep Parameters	
Sleep Duration, *h*	6.4 (0.8)
< 5.5 h, *n* (%)	60 (13.7)
≥ 5.5 to < 6.5 h, *n* (%)	178 (40.5)
≥ 6.5 to < 7.5 h, *n* (%)	175 (39.9)
≥ 7.5 h, *n* (%)	26 (5.9)
Fragmentation Index	6.1 (2.1)
Sleep Efficiency, *%*	79.9 (6.1)
Self-reported (subjective) Sleep Parameters	
Sleep Duration, *h*	6.9 (0.8)
< 6 h, *n* (%)	51 (11.6)
≥ 6 to < 7 h, *n* (%)	157 (35.8)

Characteristics	All
≥ 7 to < 8 h, *n* (%)	195 (44.4)
≥ 8 h, *n* (%)	36 (8.2)
Global PSQI score	3.3 (3.0)
Napping before 1800 hours, *n* (%)	128 (29.2)

[1]Values expressed as mean (standard deviation), unless stated otherwise. Abbreviations: BMI, body mass index; CES-D, Center for Epidemiologic Studies Depression; *d*, day; *h*, hours; *n*, number of observations; PSQI, Pittsburgh Sleep Quality Index.

[2]Range also presented.

Table 2

Associations between sleep fragmentation and efficiency on dietary intake[1].

	Energy, *kcal*		Fat, *g*		Protein, *g*		Carbohydrate, *g*	
Dietary Intake	**β (95% CI)**	***P* value**	**β (95% CI)**	***P* value**	**β (95% CI)**	***P* value**	**β (95% CI)**	***P* value**
Sleep Fragmentation, *SD*								
Model 1[2]	25.32 (-37.3, 87.9)	0.43	1.00 (-0.5, 2.5)	0.18	0.42 (-1.0, 1.8)	0.55	-5.38 (-9.4, -1.4)	**0.01**[4]
Model 2[3]	0.08 (-64.0, 64.0)	1.00	1.28 (-0.2, 2.8)	0.10	0.46 (-1.0, 1.9)	0.54	-4.19 (-8.0, -0.3)	**0.03**[5]
Sleep Efficiency, *%*								
Model 1[2]	-11.10 (-20.6, -1.7)	**0.02**	-0.02 (-0.3, 0.2)	0.85	-0.04 (-0.3, 0.2)	0.75	0.13 (-0.5, 0.7)	0.67
Model 2[3]	-8.75 (-18.4, 0.9)	0.08	-0.09 (-0.3, 0.1)	0.47	0.01 (-0.2, 0.2)	0.91	0.15 (-0.4, 0.7)	0.62

[1] Association coefficients are shown as *β*s (95% CIs). *β* represents the change in total energy (in kcal per day) macronutrient intake (in g per day) per each additional change in sleep fragmentation or sleep efficiency. Bold type indicates significant values. Abbreviations: CI, confidence interval.

[2] Model 1: adjusted for sex, age, BMI, and total energy intake (except for when assessing total energy intake as an outcome).

[3] Model 2: adjusted for Model 1 covariates and smoking status, education level, alcohol intake, coffee intake, daytime napping, depressive symptoms, physical activity, sleep apnea, and type 2 diabetes status.

[4] Simple Carbohydrate [*β* (95% CI) =-4.27 (-8.2, -0.4); *P* =0.03]; Complex Carbohydrate [*β* (95% CI) =0.54 (-0.7, 1.8); *P* =0.40].

[5] Simple Carbohydrate [*β* (95% CI) =-3.30 (-7.3, 0.7); *P* =0.10]; Complex Carbohydrate [*β* (95% CI) =-0.89 (-3.6, 1.8); *P* =0.51].

Table 3

Associations between sleep duration (continuous and categorical) and dietary intake[1].

	Energy, *kcal*		Fat, *g*		Protein, *g*		Carbohydrate, *g*	
Dietary Intake	**β (95% CI)**	***P* value**	**β (95% CI)**	***P* value**	**β (95% CI)**	***P* value**	**β (95% CI)**	***P* value**
Sleep duration, actigraphic								
Continuous, *h*	-53.3 (-131.8, 25.4)	0.18	-0.18 (-2.1, 1.7)	0.85	-0.83 (-2.6, 0.9)	0.36	-1.03 (-5.7, 3.7)	0.67
Categorical								
< 5.5 h	218.06 (33.3, 402.8)	**0.02**	3.65 (-0.8, 8.1)	0.11	1.91 (-2.3, 6.1)	0.37	-3.55 (-14.7, 7.6)	0.53
≥ 5.5 to < 6.5 h	47.05 (-79.4, 173.5)	0.47	-0.11 (-3.1, 2.9)	0.94	1.93 (-0.9, 4.8)	0.19	2.04 (-5.6, 9.6)	0.60
≥ 6.5 to < 7.5 h	*Ref*	-	*Ref*	-	*Ref*	-	*Ref*	-
≥ 7.5 h	105.5 (-140.9, 351.9)	0.40	1.86 (-4.0, 7.8)	0.54	1.59 (-4.0, 7.1)	0.58	-2.26 (-17.1, 12.6)	0.77
Sleep duration, self- reported								
Continuous, *h*	-36.07 (-104.8, 32.7)	0.30	0.24 (-1.4, 1.9)	0.78	0.26 (-1.3, 1.8)	0.74	-1.77 (-5.9, 2.3)	0.40
Categorical								
< 6.0 h	88.22 (-104.3, 280.7)	0.37	0.04 (-4.6, 4.6)	0.99	0.27 (-4.0, 4.6)	0.90	1.95 (-9.6, 13.5)	0.74
≥ 6.0 to < 7.0 h	81.41 (-45.4, 208.3)	0.21	0.79 (-2.2, 3.8)	0.61	-2.34 (-5.2, 0.5)	0.11	4.53 (-3.1, 12.1)	0.24
≥ 7.0 to < 8.0 h	*Ref*	-	*Ref*	-	*Ref*	-	*Ref*	-
≥ 8.0 h	-0.49 (-219.0, 220.0)	1.00	2.56 (-2.7, 7.9)	0.34	-3.11 (-8.0, 1.8)	0.22	-2.53 (-15.7, 10.6)	0.71

[1] Association coefficients are shown as *βs* (95% CIs). *β* represents 1) continuous: the change in energy (in kcal per day) or macronutrient intake (in g per day) per each additional hour of sleep; or 2) categorical: the difference in energy (in kcal per day) or macronutrient intake (in g per day) from reference sleep group (≥7 to < 8 h); adjusted for sex, age, BMI, and total energy intake (except for when assessing energy intake as an outcome), smoking status, education level, alcohol intake, coffee intake, daytime napping, depressive symptoms, physical activity, sleep apnea, and type 2 diabetes status (Model 2). Bold type indicates significant values. Abbreviations: CI, confidence interval. Actigraphic and subjective sleep durations were divided into 4 categories using previously determined cutoffs (van den Berg et al. 2008a) to achieve groups of adequate sample sizes necessary to reflect the possible U- or J- shaped association between sleep duration and dietary intake outcomes.