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ORIGINAL ARTICLE

Inverse relationship of food and alcohol intake to sleep measures in obesity

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BACKGROUND: Short sleep and weight gain are inversely related. Sleep deprivation acutely increases food intake but little is known about eating behavior in chronically sleep-deprived, obese individuals.
OBJECTIVE: To characterize the relationship between sleep, food intake and alcohol consumption under free-living conditions in obese, chronically sleep-deprived individuals.
DESIGN: Cross-sectional study of a cohort of obese men and premenopausal women.
SUBJECTS: A total of 118 obese subjects (age: 40.3 ± 6.7 years; 91 females/27 males; body mass index 38.7 ± 6.4 kg m^{-2}).
MEASUREMENTS: Energy, macronutrient, alcohol and caffeine intake assessed by 3-day food records. Sleep duration estimated by actigraphy. Respiratory disturbance index assessed by a portable device.
RESULTS: Subjects slept 360.7 ± 50.2 min per night and had a total energy intake of 2279.1 ± 689 kcal per day. Sleep duration and energy intake were inversely related ($r = -0.230$, $P = 0.015$). By extrapolation, each 30-min deficit per day in sleep duration would translate to an ~83 kcal per day increase in energy intake. In addition, sleep apnea was associated with a shift from carbohydrate to fat intake. Alcohol intake in subjects consuming >3.5 g of alcohol per day ($N = 41$) was inversely related to sleep duration ($r = -0.472$, $P = 0.002$).
CONCLUSIONS: Shorter sleep duration and obstructive sleep apnea are associated with higher energy, fat and alcohol intakes in obese individuals. The importance of this study relies on the population studied, obese subjects with chronic sleep deprivation. These novel findings apply to the large segment of the US population who are obese and sleep-deprived.

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INTRODUCTION

Based on the thermodynamic law, obesity results from a state of positive energy balance in which energy intake exceeds energy expenditure over a prolonged period of time. In the past decades, there has been a dramatic increase in the prevalence of obesity.[1] The reasons for such epidemic are unknown and are the subject of an ongoing debate.[2,3] In the search for hidden causative factors, it was noted that the obesity epidemic has been paralleled by a concomitant shortening in self-reported sleep and by an increase in the prevalence of obstructive sleep apnea (OSA).[2] Thus, we and others have hypothesized that these pervasive contemporaneous epidemics, sleep deprivation and obesity, may be linked together in causal, reverberating circuits.[2]

Most of the information available for the effects of sleep deprivation on food intake is derived from acute studies conducted in healthy lean male volunteers. Experimentally induced acute sleep deprivation increases appetite in healthy lean humans.[4] In rodents, sleep deprivation changes the hypothalamic expression of several neuropeptides, including the orexogenic neuropeptide Y and the anorexogenic proopiomelanocortin.[5] Less is known about food intake in chronically sleep-deprived human subjects, especially in those with obesity. The information about long-term association between sleep duration and body weight stems from large, epidemiological studies, mostly designed for other purposes.[6–8] Short sleep increases the risk of obesity, with a 0.35 kg m^{-2} change in body mass index (BMI) per hour of sleep change, as indicated by a meta-analysis.[9] Another recent meta-analysis quantified the impact of the drive to eat on alcohol intake, sleep deprivation and television watching;[10] all three factors had an impact, with large size effects for alcohol intake (1.02) and sleep deprivation (0.49), and a smaller effect (0.2) for television watching. Data from the Quebec Family Study, a cohort study for the etiology of obesity, indicated that short sleep duration, together with low dietary calcium intake and high disinhibited eating behavior is associated with a higher BMI.[11] Excessive fat intake and irregular meal patterns relate to both short sleep duration and OSA.[12–17] Of note, acute experiments reveal little about long-term adaptations, and epidemiological studies lack mechanistic information and cannot establish causality.[18]

Ethanol is a sleep-promoting gamma-amino-butyric acid receptor agonist.[19] Ethanol relaxes the upper airway dilator muscles, increases upper airway resistance and may promote OSA in susceptible subjects.[20] Heavy drinking is associated with short sleep duration, as indicated by the 2004–2006 National Health

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Institute Interview.[21] Whether moderate alcohol consumption is related to sleep alterations is not well established. This is an important question, if one keeps in mind that moderate alcohol consumers comprise a large proportion of the American population.[22]

Our aim was to characterize the relationships between sleep characteristics, nutritional habits and alcohol intake in a sample of chronically sleep-deprived obese men and women. The importance of our study resides in the fact that for the first time the relationship between sleep and dietary intake was studied in such a population.

SUBJECTS AND METHODS

Study cohort

The analyses reported here are based on the baseline evaluation of a cohort of obese men and premenopausal women enrolled in a sleep extension study.[23] In this randomized, controlled trial, subjects were randomized in a 2:1 ratio to an Intervention Group and to a Comparison Group and were followed for 1 year (efficacy phase). Subjects in the Intervention Group were encouraged to progressively extend sleep duration and to improve sleep quality in a non-pharmacological fashion by lifestyle modifications. An individualized plan was developed at the randomization visit by the study participant with the help of the study team and progresses were followed with short outpatient visits at monthly intervals. The Comparison Group was seen at the same time points and received information about the standard of care. At the end of 1 year, each subject, including those originally randomized to the Comparison Group, was invited to extend sleep duration and subjects were continued to be followed with less frequent visits for up to additional 3 years (effectiveness phase). Inclusion criteria included BMI of 29–55 kg m^{-2} and ages of 18–50 years. We advertised for subjects sleeping less than 6.5 h per night by self-assessment. Subjects were excluded if they were postmenopausal, regularly used sleep medications, consumed more than 500 mg of caffeine per day and/or had poorly controlled depression or anxiety, diabetes, hypertension or other systemic disease. Additional details of this study have been reported elsewhere.[23] The study was conducted at the NIH Clinical Center in Bethesda under NIDDK protocol 06-DK-0036, is listed in ClinicalTrials.gov (identifier: NCT00261898), and was approved by the Scientific Review Board and the Institutional Review Board of the NIDDK.

Assessments

Energy and alcohol intake. Subjects were given verbal and written instructions to record their food and beverage intake for 3 consecutive days (2 weekdays and 1 weekend day, preferably Thursday, Friday and Saturday). Food records were reviewed by registered dietitians and health technicians using three-dimensional food models and other visual aids. Dietary intake data were analyzed using the Nutrition Data System for Research software versions 2007 through 2010, developed by the Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN.[24] Daily macronutrient, caffeine, alcohol and energy intakes were determined. Eating occasions were defined as eating episodes, which contained at least 20 kcal, and were consumed at least 30 min apart or in a different location.

Sleep measures. Habitual sleep was estimated subjectively by sleep diary and objectively by wrist actigraphy (Actiwatch-64, Mini Mitter/Respironics/Philips, Bend, OR, USA). A device similar to a wristwatch was worn continuously for 14 days. The Actiwatch-64 has been validated against polysomnography with excellent correlations in various patient populations.[25] We based our analyses on two measures: night-time sleep and sleep efficiency (percentage of time in bed spent sleeping).

Sleep apnea was assessed using a portable screening device (Apnea Risk Evaluation System, Advanced Brain Monitoring, Inc., Carlsbad, CA, USA). This device estimates the respiratory disturbance index (RDI), which is the number of apneas and hypopneas per hour of sleep. This device has been validated with high sensitivity and specificity against polysomnography.[26] Participants completed the Pittsburgh Sleep Quality Index, a validated 21-items questionnaire with inquiries about sleep, including perceived quality over the past month.[27] Pittsburgh Sleep Quality Index scores range from 0 to 21, with higher scores indicating worse sleep quality. Scores were dichotomized at $\leqslant 5$ or >5, which conventionally indicates poor sleep quality. Subjective sleep duration was derived from Question Four: how many hours of sleep the participant usually gets per night. Daytime sleepiness was assessed by the Epworth Sleepiness Scale (ESS), a validated eight-item questionnaire.[28] ESS scores range from 0 to 24, higher scores representing increased daytime sleepiness, while scores >10 indicate an excessive sleepiness.

Anthropometrics. Height was measured to the nearest centimeter using a wall mounted stadiometer (SECA 242, SECA North America East, Hanover, MD, USA) and weight was measured using a stand-on-scale in a hospital gown to the nearest 1/10th of a kg (SR555 SR Scales, SR Instruments, Inc, Tonawanda, NY, USA). Circumference measurements were done using a non-stretch measuring tape in triplicate to the nearest 1 mm. Waist circumference was measured at the uppermost lateral border of iliac crest at the end of a normal expiration. If this site could not be determined, the maximum circumference was measured at or near the level of the umbilicus. Neck circumference was measured at the minimal circumference with the head in the Frankfort Horizontal Plane.

Body composition. Body composition was measured by dual energy X-ray absorptiometry (Lunar iDXA, General Electric, Chicago, IL, USA).

Statistical analysis

Descriptive statistics were calculated first for the cohort as a whole and then separately by gender. Tests used to compare men and women included Student's *t*-test for difference in mean values, Mann–Whitney U-test for skewed variables, Fisher exact test and Pearson Chi-square test for difference in counts and frequency. The Kolmogorov–Smirnov test was used to assess normality; logarithmic transformations were employed for skewed variables (e.g. RDI values, alcohol and caffeine intakes). Pearson (r) and Spearman (rho) correlation coefficients were employed for Gaussian and skewed variables, respectively. The degree of agreement between subjective and objective sleep duration estimates was quantified by computing a concordance correlation coefficient.

Simple regression analyses of sleep and diet components selected *a priori* were conducted based on pre-defined hypotheses. Specific predictors from these regression analyses showing a P-value less than 0.10 in univariate analysis were then included into multiple linear regression models. Age, gender, race and BMI were used as covariates.

Data are presented as a mean value ± s.d. Analyses were performed using SAS (version 9.1.3, SAS Institute Inc., Cary, NC, USA), JMP (version 8.0, SAS Institute) and SPSS (version 19 IBM SPSS North America, Chicago, IL, USA).

RESULTS

Demographic, anthropometric and sleep characteristics

A total of 118 individuals out of the 126 subjects randomized between 22 January 2007 and 28 June 2011 for whom dietary information was available were the object of this analysis. Age was 40.3 ± 6.7 years and BMI was 38.7 ± 6.4 kg m^{-2} (Table 1). Three quarters of the participants were women and two-thirds were self-identified as non-Hispanic blacks. Men had a larger waist and neck circumference, a larger fat-free mass and a lower percentage of body fat than women. On average, our sample had short sleep duration and decreased sleep efficiency, as indicated by both total sleep time, (~6 h) and sleep efficiency (~80%). Sleep apnea was highly prevalent: only 42% of the participants had a normal (<5) RDI score. We noted the following gender differences: sleep apnea (RDI >5) was more prevalent, 80% vs 50%, and more severe in men than women. By actigraphy, women slept on average 13 min longer than men. The coefficient of concordance between sleep duration by actigraphy and sleep diary was significant, 0.413 (95% confidence interval: 0.244–0.557, $P<0.001$, $N=90$) (Figure 1). Nevertheless, sleep duration by sleep diary and sleep duration by actigraphy were different (sleep diary: +23 min 95% confidence interval: 12–33 min, $P<0.001$) (Figure 1).

The average total energy intake was ~2280 kcal/day; 48% of calories derived from carbohydrates, 14% from proteins and 36% from fat. On average, there were four eating occasions per day (Table 2). Alcohol intake had a non-Gaussian distribution with a mean of 43 kcal per day. Of the 118 subjects, 77 reported consuming less than 3.5 g of alcohol per day, roughly the equivalent of $\frac{1}{4}$ of an alcoholic beverage per day. The remaining

Table 1. Demographic, anthropometric and sleep characteristics of the study population (mean ± s.d.)

	All (N = *118*)	*Men* (N = *27*)	*Women* (N = *91*)	P-*value*[a]
Age (years)	40.3 ± 6.7	39.8 ± 7.2	40.4 ± 6.6	0.695
BMI (kg m^{-2})	38.7 ± 6.4	37.2 ± 6.2	39.1 ± 6.5	0.171
Waist circumference (cm)	113.9 ± 13.3	119.5 ± 14.0	112.2 ± 12.6	**0.012**
Neck circumference (cm)	39.2 ± 3.9	44.2 ± 3.5	37.7 ± 2.7	**<0.001**
% Body fat	41.5 ± 6.6	34.1 ± 5.3	44.0 ± 5.0	**<0.001**
Lean body mass (kg)	60.6 ± 12.0	74.6 ± 12.8	55.9 ± 7.1	**<0.001**
Race N (%)				
Non-Hispanic black	69 (58.5%)	8 (29.6%)	61 (67.0%)	**0.002**
Non-Hispanic white	43 (36.4%)	17 (63.0%)	26 (28.6%)	
Other	6 (5.1%)	2 (7.4%)	4 (4.4%)	
Actigraphy sleep duration (min per night)	360.7 ± 50.2	343.7 ± 53.7	366.1 ± 48.1	**0.043**
Actigraphy sleep efficiency (%)	79.5 ± 7.5	78.1 ± 7.8	80.0 ± 7.4	0.276
RDI score (events per hour)	12.2 ± 15.6 (*N* = 98)	21.9 ± 24.9 (*N* = 20)	9.7 ± 11.1 (*N* = 78)	**0.019**
RDI by score category (events per hour)				
⩽5	41 (41.8%)	4 (20.0%)	37 (47.4%)	0.083
6–15	36 (36.7%)	9 (45.0%)	27 (34.6%)	
16–30	12 (12.2%)	3 (15.0%)	9 (11.5%)	
>30	9 (9.1%)	4 (20.0%)	5 (6.4%)	
PSQI Global Score (optimal: 0–5)	8.0 ± 2.7	7.7 ± 3.0	8.1 ± 2.6	0.512
Self-reported sleep duration (min per night)	387.8 ± 48.1	375.4 ± 43.7	390.9 ± 49.0	0.221
Epworth sleepiness score (optimal: 0–10)	8.2 ± 4.4	8.8 ± 4.6	8.1 ± 4.3	0.440

Abbreviations: PSQI, Pittsburgh Sleep Quality Index; RDI, respiratory disturbance index. [a]Men vs women. Bold characters indicate significant values.

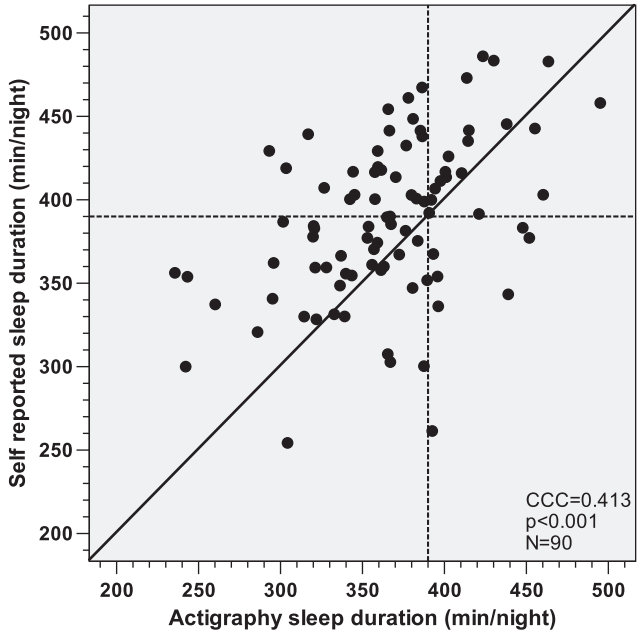

Figure 1. Concordance between sleep duration per night as assessed subjectively by sleep diary and objectively by actigraphy. Dotted lines indicate 390 min (that is, 6.5 h) sleep per night; a marker of sleep deprivation.

41 alcohol drinkers, had a consumption of less than two drinks per day, with only 7 subjects consuming more than four drinks per day (Figure 2). Total energy intake was higher in men compared with women, with no differences in macronutrient composition. Caffeine intake was ~40% higher in men than women ($P = 0.013$; Table 2).

Relationship between sleep and total energy intake

Sleep duration by actigraphy was inversely related to total energy intake, whereas severity of sleep apnea, as indicated by RDI scores, was directly related to total energy intake (Figures 3a and b). Thus, sleeping less and having more episodes of sleep apnea was associated with a higher total energy intake. Furthermore, RDI was inversely related to carbohydrate intake ($r = -0.309$; $P = 0.001$; $N = 98$) and directly related to fat intake ($r = 0.239$; $P = 0.017$; $N = 98$), both expressed as percent of total energy intake. When assessed by gender, these relationships persisted only in women: the carbohydrate intake was inversely ($r = -0.396$; $P < 0.001$; $N = 78$), while the fat intake was directly ($r = 0.325$; $P = 0.017$; $N = 78$) related to RDI. Neither caffeine intake nor the number of eating occasions were related to any of the sleep variables ($P > 0.05$).

To further explore the relationship between sleep and dietary intake, we constructed several multivariate models (Table 3). Sleep duration by actigraphy and RDI accounted for ~9% of the variability in total energy intake (Model 1). After adjustment for age and gender, the variability rose to 21% (Model 2). Results remained similar after adding BMI to the model (Model 3). When race/ethnicity was included (Model 4), the variability in total energy intake accounted for by the model became 26%. Based on Model 4, a 30-min decrease in sleep would correspond to 83-kcal increase ($30 \times 2.76 = 82.8$) in energy intake. Age was inversely related to total energy intake, so that a 10-year increase in age would correspond to ~236 kcal decrease in energy intake. In each one of the four models, sleep apnea, as characterized by RDI scores, was inversely related to carbohydrate intake and directly related to fat intake.

Relationships between sleep and alcohol intake

Alcohol intake and sleep parameters were correlated in the 41 subjects consuming more than $\frac{1}{4}$ alcoholic beverage per day (3.5 g per day) (Table 4). Alcohol intake was inversely related to total sleep time (Figure 3c). In the multivariate analyses, alcohol intake explained ~22% of the variability in sleep duration (Model 1) (Table 4). Incremental adjustments for age and gender (Model 2), plus BMI (Model 3), plus race/ethnicity (Model 4), increased the variability accounted for by the models to 33%.

Table 2. Energy, macronutrient and alcohol intake of the study population (mean ± s.d.)

	All (N = *118*)	*Men* (N = *27*)	*Women* (N = *91*)	P-*value*[a]
Total energy intake (kcal per day)	2279.1 ± 688.6	2758.5 ± 707.9	2136.9 ± 618.2	**<0.001**
Carbohydrate intake (% of total energy intake)	48.3 ± 7.8	47.5 ± 9.0	48.6 ± 7.4	0.548
Protein intake (% of total energy intake)	14.2 ± 3.1	14.6 ± 3.2	14.0 ± 3.1	0.453
Fat intake (% of total energy intake)	35.8 ± 6.3	35.5 ± 6.5	35.9 ± 6.3	0.775
Eating occasions (number per day)	4.4 ± 1.2	4.1 ± 1.0	4.5 ± 1.3	0.215
Alcohol intake (kcal per day)	43.2 ± 84.6	68.8 ± 122.3	35.6 ± 68.7	0.507
(% of total energy intake)	(1.7 ± 3.3%)	(2.4 ± 4.6%)	(1.5 ± 2.8%)	(0.773)
Caffeine (mg per day)	101.4 ± 97.8	144.6 ± 118.2	89.1 ± 88.1	**0.013**

[a]Men vs women. Bold characters indicate significant values.

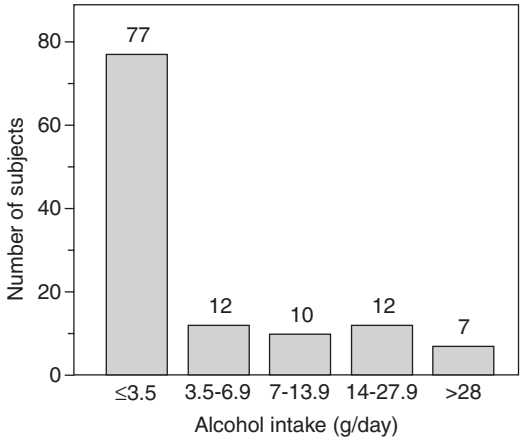

Figure 2. Distribution of alcohol intake (g per day) in total population of 118 subjects. One drink corresponds to ~14 g of alcohol.

DISCUSSION

To our knowledge, this is the first report on the association between sleep and dietary/alcohol intake in a real life setting in obese, chronically sleep-deprived subjects. We assessed sleep duration subjectively and objectively, determined the presence and severity of sleep apnea, and estimated dietary intake using food records for 3 consecutive days. Energy intake in women was ~15% higher than that of an average American woman of comparable age, whereas in men the reported intake was similar to the national average.[29] Diet composition in both genders was similar to the pattern reported for a typical American adult.[29]

Together, short sleep duration and sleep apnea accounted for a substantial amount of the variability in energy intake, a parameter notoriously influenced by a variety of different factors.[30] In our sample, a 30-min decrease in sleep duration would correspond to an increase of 83 kcal per day. Sleeping less than 6.5 h per night, instead of the recommended 7.5 h per night would translate into an excess consumption of 1200 kcal per week, and, if no compensation occurs, a weight gain of ~8 kg in 1 year. Because of the cross-sectional nature of our report, we neither can claim causality, nor can we account for its direction. In addition, given the known presence of long-term adaptive mechanisms, our extrapolation should be taken with caution and verified in prospective studies.

In healthy lean humans, acute sleep restriction induces food intake[31–33] and stimulates hunger via increasing plasma ghrelin (a 'hunger' hormone) levels and decreasing plasma leptin (a 'satiety' hormone).[4] We have previously reported that leptin concentrations were not related to sleep measurements in our study.[8] Our findings were in agreement with another short-term study of sleep deprivation,[34] observing no changes in leptin levels, but they were at variance with another short-term study conducted in healthy lean male subjects[4]

Sleep curtailment and the modern environment, which is characterized by endless opportunities to eat, potentiate each other in a vicious circle.[35] Excessive food intake, especially consumed late in the day, could be responsible for digestive difficulties and consequent sleep-time reduction.[36–38] Furthermore, several studies have identified single nutrients (that is, glucose, amino acids), capable of resetting or phase-shifting circadian rhythms of the internal clock.[39] Finally, inadequate sleep, excessive energy intake and obesity are associated with lower socioeconomic status.[40,41] A cross-sectional survey of 2670 women, ages 18–55 years, reported that several socioeconomic factors including age, education, depressive symptoms, marital and employment status were associated with sleep characteristics such as short sleep and inadequate sleep[42]

Severity of sleep apnea was associated with a shift in macronutrient consumption. Total energy intake and fat intake are strong determinants of energy balance and predictors of weight gain. High-fat diets are more likely than high carbohydrates and protein diets to lead to weight gain.[43,44] We observed a direct relationship between RDI and carbohydrate and fat intake only in women, possibly because of the smaller number of males in our sample. These findings of greater carbohydrate intake in women with sleep apnea are similar to a cross-sectional study of teenage Saudi girls reporting greater ~16% greater carbohydrate intake in subjects sleeping less than 5 h vs those sleeping more than 7 h.[45]

In agreement with our findings, the APPLE Study showed that fat consumption was higher in women with severe sleep apnea.[46] Analyzing food intake in subjects before and after treatment with OSA would be helpful in understanding the cause–effect link between OSA and eating habits and assist in implementing therapy.[47]

Alcohol intake and total sleep time were inversely related in our subjects consuming at least $\frac{1}{4}$ serving of alcohol per day, the equivalent of 1–2 alcoholic beverages per week. Daily alcohol intake accounted for ~1/5 of the variability in sleep duration. Each increase of one drink (~14 g of alcohol) would result in a 30-min decrease in sleep duration. Thus, alcohol intake, even in small quantities may interfere with sleep. Up to two or three standard drinks before bedtime favor sleep initiation, an effect that vanishes after a few days.[48] In a large clinical series of 2933 healthy Japanese individuals, aged 46–60 years, drinkers were less likely to maintain sleep than nondrinkers.[49] Chronic alcohol abuse disrupts sleep even in abstinent alcoholics.[50–53] Insufficient and poor quality sleep induce chronic stress[54] and subjects with chronic anxiety and post-traumatic stress disorder often use alcohol to relieve stress.[55] Similarly, chronically sleep-deprived subjects may attempt to self-medicate themselves with alcohol. Additional factors of the relationship of alcohol and sleep include alcohol doses, the times of consumption relative to sleep, and the

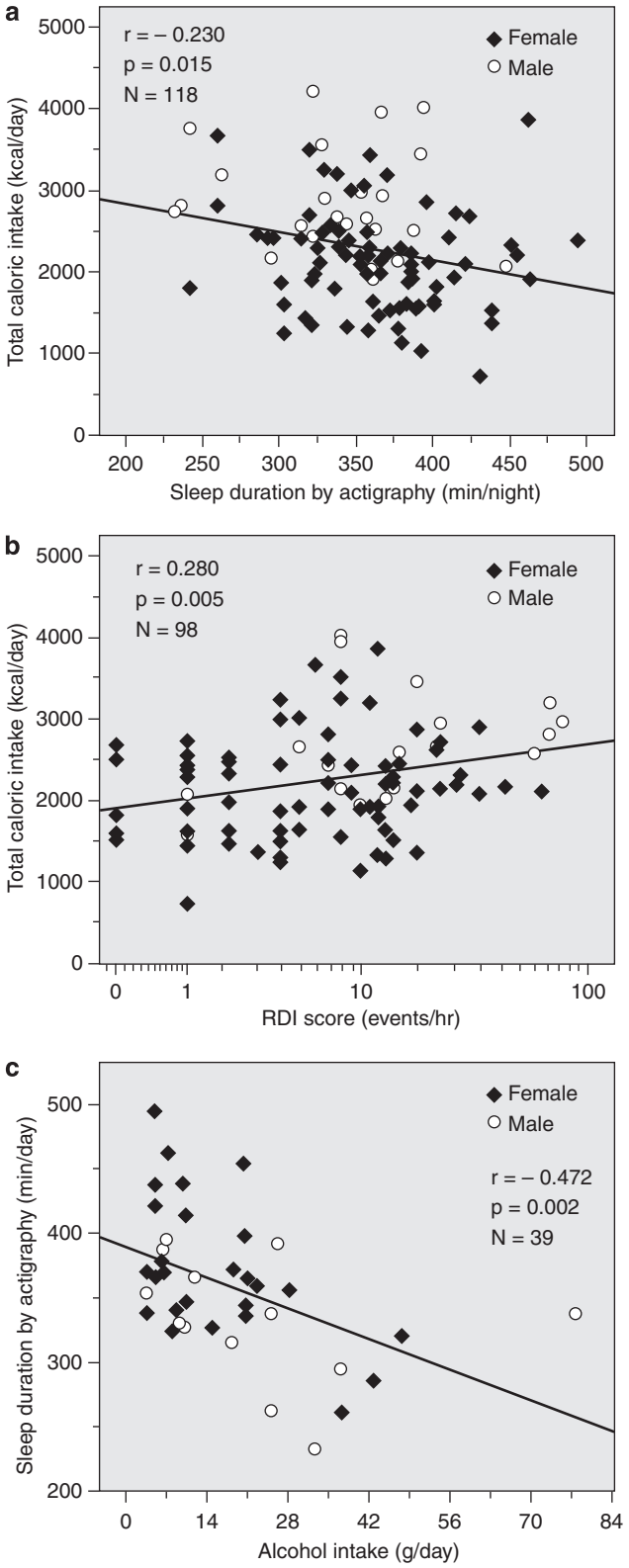

Figure 3. Relationships between total energy intake and sleep duration by actigraphy (**a**) and RDI score (**b**) and relationship between sleep duration by actigraphy and alcohol intake in the subsample of subjects consuming more than $\frac{1}{4}$ alcoholic beverage per day (**c**). Note use of 'Safe Log' (that is, $\log_{10}(1 + \text{RDI})$) for the logarithmic transformation of RDI scores in the analyses and graph (**b**).

time of the day. Alcohol consumption increases upper airway resistance,[20] especially during the first 2 h after ingestion.

It is well known that the accuracy of dietary intake data is limited and depends on many factors, including the rigor of the dietary data collection procedures as well as personal characteristics of the subjects, including gender and BMI. Obese women tend to under-report energy intake compared with predicted energy intake estimated from predictive equations.[56] The extent to which obese subjects under-report their dietary intake vs actually restricting their intake when asked to record their food intake, remains to be determined. We recently reported[55] that the subjects in this study had a measured average resting energy expenditure of 1700 kcal per day. An empirical rule calls for multiplying the measured resting energy expenditure by a factor of 1.4 in sedentary individuals to estimate the total daily energy expenditure. Thus, the mean energy intake reported in this study, ~2300 kcal per day was very close to the theoretical was very close to the theoretical energy expenditure reported previously.[56]

Our study merits include a prospectively assembled, and well-characterized sample. Consistent with the epidemiology of sleep deprivation, our sample included a large proportion of minorities and women. Sleep was characterized by an array of objective measures and standardized questionnaires. Our analyses were, however, cross-sectional and the study design did not include a group of obese, non-sleep-deprived subjects. Dietary intake was assessed via 3-day food record collection, a method known to be associated with under-reporting in obese, female populations. However, the current food records were in agreement with previously reported data of predicted energy needs in this group of subjects as assessed using measured resting energy expenditure, indicating that the rigorous methods for collection of this data may have improved the quality of this data. Unfortunately, validation of this data with doubly labeled water, the gold standard for assessing dietary intake was impractical for this study. More than two-thirds of the subjects in our sample did not consume alcohol in a regular fashion, and the amount of alcohol intake was assessed together with food intake, rather than by a specific questionnairie. Therefore, the relatively small number of subjects consuming alcohol and the method of estimation of alcohol intake limited the extension of our findings to the general population. Finally, our sample size of 118 subjects may have had a limited statistical power to detect some of the more subtle associations.

Approximately 36%, roughly 80 millions, of adult Americans, are obese[1] and 29% get insufficient rest or sleep.[57] Based on the 2005–2008 NHANES data, the prevalence of self-reported sleep apnea is ~9%.[58] It is estimated that up to 80% of individuals with moderate or severe sleep apnea, a treatable condition by continuous positive airway pressure (CPAP) or similar devices, currently go undiagnosed.[59] A conservative estimate calls for at least 18 million of adult Americans with sleep apnea, most of them undiagnosed and untreated. Long-term compliance with CPAP is known to be poor, due to its limited tolerability. In summary, this study provides quantitative estimates of the associations between sleep and food and alcohol intake in obese subjects in free-living conditions. Even small amounts of alcohol intake can disrupt sleep duration and quality. Sleep duration and OSA are related to total energy intake and severity of OSA is associated with a shift from carbohydrate to fat intake.

Our findings have important bearings on long-term weight regulation for the increasing number of the US population, who are obese, chronically sleep deprived, and suffer from sleep apnea. Clinical approaches to this population should be individualized and informed by determination of dietary intake and assessment of sleep apnea. Sleep apnea, a major determinant of food intake in these subjects, should be diagnosed and treated. Novel devices with a better tolerability profile should be developed.

Table 3. Multivariate statistical models of sleep duration and RDI predicting total energy intake

Dependent variable	*Model 1 unadjusted*	*Model 2 adjusted for age and gender*	*Model 3 adjusted for age, gender and BMI*	*Model 4 adjusted for age, gender and ethnicity/race*
Total energy intake (kcal per day)				
Predictors	$R^2 = 0.088$, $P = 0.017$	$R^2 = 0.212$, $P < 0.001$	$R^2 = 0.217$, $P = 0.001$	$R^2 = 0.261$, $P < 0.001$
Sleep duration by actigraphy (min per night)	− 2.37 (− 5.25–0.51)	− 2.01 (− 4.73–0.72)	− 2.01 (− 4.74–0.73)	− 2.76 (− 5.63–0.12)
RDI (events per hour)	8.58 (− 0.66–17.83)	5.18 (− 3.88–14.25)	6.75 (− 3.24–16.74)	3.42 (− 5.81–12.65)
Age (years)	NA	− 18.37 (− 37.74–0.99)	**− 21.60 (− 42.83– − 0.37)**	**− 23.67 (− 43.95– − 3.38)**
Gender (female = 1, male = 2)	NA	**505.72 (177.79–833.66)**[a]	**467.01 (123.24–811.79)**[a]	**446.96 (83.16–809.77)**[a]
BMI (kg m^{-2})	NA	NA	− 8.98 (− 32.85–14.90)	NA
Race (black = 1, white = 2)	NA	NA	NA	288 (− 18–595)[b]
Constant	3029 (1943–4115)	3059 (1649–4468)	3565 (1614–5516)	3238 (1776–4700)
Carbohydrate intake (% of total energy intake)				
Predictors	$R^2 = 0.096$, $P = 0.002$	$R^2 = 0.104$, $P = 0.006$	$R^2 = 0.108$, $P = 0.029$	$R^2 = 0.105$, $P = 0.043$
RDI (events per hour)	**− 0.15 (− 0.25– − 0.06)**	**− 0.16 (− 0.26– − 0.06)**	**− 0.17 (− 0.29– − 0.06)**	**− 0.16 (− 0.27– − 0.06)**
Age (years)	NA	− 0.09 (− 0.31–0.14)	− 0.05 (− 0.30–0.19)	− 0.09 (− 0.33–0.15)
Gender (female = 1, male = 2)	NA	0.76 (− 3.03–4.55)	1.16 (− 2.82–5.13)	0.81 (− 3.46–5.08)
BMI (kg m^{-2})	NA	NA	0.09 (− 0.18–0.37)	NA
Race (black = 1, white = 2)	NA	NA	NA	0.57 (− 3.02–4.16)
Constant	50 (48–52)	52 (42–63)	48 (29–66)	50 (48–52)
Fat intake (% of total energy intake)				
Predictors	$R^2 = 0.057$, $P = 0.018$	$R^2 = 0.082$, $P = 0.044$	$R^2 = 0.094$, $P = 0.056$	$R^2 = 0.083$, $P = 0.102$
RDI (events per hour)	**0.09 (0.02–0.18)**	**0.11 (0.02–0.19)**	**0.13 (0.04–0.22)**	**0.11 (0.02–0.19)**
Age (years)	NA	0.11 (− 0.08–0.29)	0.06 (− 0.14–0.27)	0.12 (− 0.08–0.32)
Gender (female = 1, male = 2)	NA	− 1.60 (− 4.73–1.54)	− 2.12 (− 5.40–1.15)	− 1.35 (− 4.48–2.17)
BMI (kg m^{-2})	NA	NA	− 0.13 (− 0.35–0.10)	NA
Race (black = 1, white = 2)	NA	NA	NA	− 1.06 (− 4.03–1.90)
Constant	34 (33–36)	32 (23–40)	39 (24–55)	32 (23–41)

Abbreviations: BMI, body mass index; NA, not available; RDI, respiratory disturbance index. Beta coefficients in each cell are reported as mean values with 95% confidence interval. Bold values indicate significance with $P < 0.05$. [a]Men show on average a higher energy intake compared with women. [b]White individuals tend to have a higher energy intake compared with black individuals.

Table 4. Multivariate model of alcohol intake in the subsample of 41 subjects consuming more than $\frac{1}{4}$ of alcoholic beverage per day predicting total sleep

Dependent variable: total sleep time (min per 24 h)	*Model 1 unadjusted*	*Model 2 adjusted for age and gender*	*Model 3 adjusted for age, gender and BMI*	*Model 4 adjusted for age, gender and ethnicity/race*
Predictors	$R^2 = 0.222$, $P = 0.002$	$R^2 = 0.320$, $P = 0.003$	$R^2 = 0.320$, $P = 0.009$	$R^2 = 0.335$, $P = 0.006$
Alcohol intake >3.5 g per day (g per day)	**− 1.75 (− 2.84– − 0.66)**	**− 1.49 (− 2.57– − 0.41)**	**− 1. 50 (− 2.64– − 0.37)**	**− 1.39 (− 2.50– − 0.27)**
Age (years)	NA	− 1.68 (− 4.17–0.81)	− 1.70 (− 4.32–0.92)	− 1.85 (− 4.38–0.68)
Gender (female = 1, male = 2)	NA	− 23.45 (− 59.93–13.04)	− 23.46 (− 60.52–13.59)	− 30.36 (− 70.35–9.62)
BMI (kg m^{-2})	NA	NA	− 0.07 (− 2.45–2.31)	NA
Race (black = 1, white = 2)	NA	NA	NA	15.36 (− 20.18–50.92)
Constant	391 (366–416)	482 (388–577)	486 (326–646)	473 (375–571)

Abbreviations: BMI, body mass index; NA, not available. Beta coefficients in each cell are reported as mean values with 95% confidence interval. Bold values indicate significance with $P < 0.05$.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

GCi and GCs designed research, MSM, ABC, GCi, conducted research, GG, PP and LDJ analyzed data or performed statistical analysis, GG, PP, LDJ, AP, FS, GCi and GCs wrote paper (only authors who made a major contribution); GG, GCi and GCs had primary responsibility for final content.

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