

The Association Between Sleep Duration and Weight Gain in Adults: A 6-Year Prospective Study from the Quebec Family Study

Jean-Philippe Chaput, MSc¹; Jean-Pierre Després, PhD²; Claude Bouchard, PhD³; Angelo Tremblay, PhD^{1,4}

¹Division of Kinesiology, Department of Social and Preventive Medicine, Faculty of Medicine, Laval University, Quebec City, Quebec, Canada;

²Quebec Heart Institute, Hôpital Laval Research Center, Hôpital Laval, Quebec City, Quebec, Canada; ³Human Genomics Laboratory, Pennington Biomedical Research Center, Baton Rouge, LA

Study Objective: To investigate the relationship between sleep duration and subsequent body weight and fat gain.

Design: Six-year longitudinal study.

Setting: Community setting.

Participants: Two hundred seventy-six adults aged 21 to 64 years from the Quebec Family Study. More than half of the sample is drawn from families with at least 1 parent and 1 offspring with a body mass index of 32 kg/m² or higher.

Measurements and Results: Body composition measurements and self-reported sleep duration were determined. Changes in adiposity indices were compared between short- (5-6 hours), average- (7-8 hours), and long- (9-10 hours) duration sleeper groups. After adjustment for age, sex, and baseline body mass index, short-duration sleepers gained 1.98 kg (95% confidence interval: 1.16-2.82) more and long-duration sleepers gained 1.58 kg (95% CI: 1.02-2.56) more than did average-duration sleepers over 6 years. Short- and long-duration sleepers were 35% and 25% more likely to experience a 5-kg weight gain, respec-

tively, as compared with average-duration sleepers over 6 years. The risk of developing obesity was elevated for short- and long-duration sleepers as compared with average-duration sleepers, with 27% and 21% increases in risk, respectively. These associations remained significant after inclusion of important covariates and were not affected by adjustment for energy intake and physical activity participation.

Conclusions: This study provides evidence that both short and long sleeping times predict an increased risk of future body weight and fat gain in adults. Hence, these results emphasize the need to add sleep duration to the panel of determinants that contribute to weight gain and obesity.

Keywords: adiposity, body mass index, body weight, sleep loss, sleep deprivation, waist circumference

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OBESITY IS ONE OF THE MOST IMPORTANT PUBLIC HEALTH PROBLEMS TODAY, AND, ALTHOUGH MUCH HAS BEEN LEARNED REGARDING THE REGULATION of body weight, the prevalence of obesity continues to rise. Individual and environmental factors that have an influence on energy balance are not fully understood. Current treatments for obesity have been largely unsuccessful in maintaining long-term weight loss, suggesting the need for new insight into the mechanisms that result in altered metabolism and behavior and may lead to obesity. Parallel to an increase in body weight, one has also observed a reduction in sleep times. Indeed, over the past 40 years, daily sleep duration in the United States popu-

lation has decreased by 1.5 to 2 hours, and the proportion of young adults sleeping less than 7 hours per night has more than doubled between 1960 and 2001-2002 (from 15.6% to 37.1%).¹⁻⁴ Thus, lack of sleep has become a widespread habit driven by the demands and opportunities of our modern “24-hour” lifestyle. Nearly one third of adults report sleeping less than 6 hours per night, leading some to suggest that we live in a sleep-deprived society.⁵ Not surprisingly, reports of fatigue and tiredness are more frequent today than a few decades ago.⁶

Cross-sectional studies in adults have repeatedly found an association between reduced sleep and increased weight.⁷⁻⁹ Similar findings have been observed in cross-sectional studies of children, suggesting that short sleep duration correlates with an increased risk of being overweight or obese.¹⁰⁻¹² The most plausible explanation to date of the sleep duration–body weight relationship is an alteration of the neuroendocrine control of appetite characterized by a decrease in the levels of the anorexigenic hormone leptin and an increase in the levels of the orexigenic factor ghrelin.^{7,9,13} Thus, these neuroendocrine changes have the potential to favor a positive caloric balance and weight gain over time.¹³

However, these studies cannot differentiate cause from effect. It may, therefore, be argued that the association between short sleep duration with higher body weight is bidirectional and results from other factors that impact on body weight regulation. To our knowledge, only 3 studies have examined associations between sleep duration and obesity in a longitudinal design in adults. The first study from Hasler et al (2004) reported a relationship between sleep time and future weight.¹⁴ However, the generalizability of the results is uncertain, as

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Address correspondence to: Angelo Tremblay, PhD, Division of Kinesiology (PEPS), Department of Social and Preventive Medicine, Laval University, Quebec City, Quebec, Canada, G1K 7P4; Tel: (418) 656-7294; Fax: (418) 656-3044; E-mail: angelo.tremblay@kin.msp.ulaval.ca

the study was based on subjects with a high risk of psychiatric disorders. The second study from Gangwisch et al (2005) showed that sleep duration is associated with obesity in a large longitudinally monitored United States sample.⁸ However, the study lacked repeated measures of sleep duration and used self-reported weights. Finally, the third study from Patel et al (2006) found that short sleep duration predicted higher weight gain independent of baseline weight.¹⁵ However, sleep duration was assessed at only 1 time point, body weight was assessed from a questionnaire, and the study was limited to middle-aged women in the nursing profession.

The present study examines longitudinal associations between sleep duration and adiposity indices in the Quebec Family Study. We hypothesize that short sleep duration predisposes to increased body weight and fat gain due to a combination of increased caloric intake and reduced physical activity.

METHODS

Subjects

The Quebec Family Study was initiated at Laval University in 1978. The primary objective of this study was to investigate the role of genetics in the etiology of obesity and related cardiovascular risk factors. In phase 1 of the study (1978 to 1981), a total of 1650 individuals from 375 families were recruited and measured. Recruitment was conducted irrespective of body weight during Phase 1, resulting in a cohort with a wide range of body mass index (BMI), ranging from 13.8 to 64.9 kg/m². In Phase 2 (1989-1994) and 3 (1995-2001), 100 families from Phase 1 were retested, and an additional 123 families with at least 1 parent and 1 offspring with a BMI of 32 or higher were added to the cohort. Families were recruited through the media and were all French Canadians from the greater Quebec City area. From the sample of 223 Caucasian nuclear families (totaling 951 subjects involved in Phases 1, 2, and 3), 163 men and 199 women were potentially eligible for longitudinal analyses between Phase 2 and 3. Longitudinal analyses were not possible with Phase 1 because the assessments were different at this moment. Additional details about the Quebec Family Study have been previously published.¹⁶ In the current study, baseline corresponded to Phase 2, and the mean duration of follow-up between Phase 2 and 3 was 6.0 (SD 0.9) years. The following exclusion criteria were applied: (1) aged less than 21 years or greater than 64 years (27 men and 26 women excluded); (2) diabetic, defined as use of insulin or a hypoglycemic agent, a fasting plasma glucose level of 126 mg/dL or more (≥ 7.0 mmol/L), or a 2-hour postload plasma glucose level of 200 mg/dL or more (≥ 11.1 mmol/L) (8 men and 3 women excluded); and (3) body-weight change greater than 2 kg during the 6 months prior to baseline testing (5 men and 7 women excluded). In addition, subjects with missing data on 1 or more of the variables investigated in 1 of the 2 testing sessions (baseline and 6 years later) were excluded (6 men and 4 women). The final longitudinal sample with full data included 276 individuals (117 men and 159 women). All subjects provided written informed consent to participate in the study. The project was approved by the Medical Ethics Committee of Laval University and is in accordance with the Helsinki II Declaration.

Sleep Duration Assessment

The number of hours of sleep was assessed at baseline and year 6 through a question inserted in a self-administered questionnaire on physical activity participation. The question formulation was: "On average, how many hours do you sleep a day?" Because of the fact that only 2 subjects (1 man and 1 woman) reported sleeping 4 hours a day and 5 subjects (2 men and 3 women) reported sleeping 11 or 12 hours a day and that the majority of subjects reported sleeping 7 or 8 hours a day, we decided to classify the subjects into 3 sleep-duration groups in order to have sufficient sample sizes in each category, in agreement with our recent paper.⁹ These groups are short-duration sleepers (5-6 hours of sleep; 21 men and 22 women), average-duration sleepers (7-8 hours of sleep; 85 men and 112 women), and long-duration sleepers (9-10 hours of sleep; 11 men and 25 women). Since there was no sex interaction with the other factors, data for both sexes were combined.

Anthropometric and Body Composition Measurements

Height was measured to the nearest 0.1 cm using a standard stadiometer, and body weight was measured to the nearest 0.1 kg using a digital panel indicator scale (Beckman Industrial Ltd, Model 610/612, Scotland, UK). BMI was calculated as body weight divided by height squared (kg/m²). Waist circumference was measured at the line between the lower border of the last rib and the upper border of the iliac crest. These anthropometric measurements were performed according to standardized procedures recommended at The Airlie Conference.¹⁷ Furthermore, body density was obtained from the mean of 6 valid measurements derived from underwater weighing.¹⁸ Before immersion in the hydrostatic tank, the helium dilution method of Meneely and Kaltreider¹⁹ was used to determine the pulmonary residual volume. The percentage of total body fat was determined from body density with the equation of Siri.²⁰ Body fat mass was estimated from body weight and the percentage of body fat. These measurements were performed in the same way at both baseline and after 6 years.

Measurement of Energy Intake

Diet was evaluated with a 3-day record, including 2 weekdays and 1 weekend day, at baseline and year 6. Participants were shown how to complete this record by a dietician who provided instruction about measuring the quantities of ingested foods. This method of dietary assessment has been shown to provide a reliable measure of diet in this population.²¹ Mean daily energy intake was estimated by a dietician using a computerized version of the Canadian Nutrient File.²²

Measurement of Energy Expenditure

Resting metabolic rate was measured by indirect calorimetry in the morning, after a 12-hour overnight fast, with a ventilated hood as previously described.²³ The Weir formula was used to calculate energy expenditure, and values were expressed in kcal/24 hours.²⁴ On the other hand, physical activity level was estimated using a physical activity record.²⁵ Subjects had

Table 1—Baseline Characteristics of Subjects According to Sleep-Duration Group^a

Variable	5-6 hours (n = 43)	7-8 hours (n = 197)	9-10 hours (n = 36)
Age, y	39.9 ± 14.9	38.9 ± 14.8	37.3 ± 18.2
Sex			
Male	22 (51)	94 (48)	15 (47)
Female	21 (49)	103 (52)	21 (53)
Body weight, kg	76.9 ± 14.7	69.3 ± 14.9 ^b	72.4 ± 23.3
BMI, kg/m ²	27.8 ± 5.5	25.0 ± 4.7 ^b	26.1 ± 6.4
Body fat mass, kg	22.6 ± 10.8	17.8 ± 9.3 ^b	19.6 ± 11.1
Body fat, %	28.5 ± 9.1	25.2 ± 9.3 ^b	26.6 ± 9.3
Waist circumference, cm	90.1 ± 15.0	82.5 ± 14.0 ^b	83.8 ± 17.0
RMR, kcal/24-h	1531 ± 295	1474 ± 253	1470 ± 311
Smoking habits			
Nonsmoker or exsmoker	36 (83)	172 (87)	32 (89)
Light smoker ^c	2 (5)	12 (6)	3 (8)
Heavy smoker ^d	5 (12)	13 (7)	1 (3)
Employment status			
Student	7 (16)	43 (22)	7 (19)
Paid employment	25 (58)	116 (59)	18 (50)
Looking for work	2 (5)	5 (2)	2 (6)
Home duties	4 (9)	21 (11)	4 (11)
Retired	3 (7)	10 (5)	4 (11)
Disabled	2 (5)	2 (1)	1 (3)
Highest educational level			
High school	21 (49)	83 (42)	18 (50)
College ^e	14 (32)	72 (37)	12 (33)
University	8 (19)	42 (21)	6 (17)
Total annual family income ^f			
70,000 or more	14 (32)	76 (38)	13 (36)
50,000-69,000	13 (30)	55 (28)	9 (25)
30,000-49,000	12 (28)	60 (30)	12 (33)
10,000-29,000	2 (5)	2 (1)	1 (3)
< 10,000	2 (5)	4 (2)	1 (3)
Menopausal status			
In menopause	6 (14)	22 (11)	4 (11)
Not in menopause	37 (86)	175 (89)	32 (89)
Alcohol intake, g/d	7.58 ± 15.2	6.97 ± 13.3	7.55 ± 17.8
Coffee intake, cups/d	3.00 ± 2.35	2.61 ± 2.11	2.51 ± 2.53
Energy intake, kcal/d	2505 ± 676	2314 ± 713	2253 ± 570
Vigorous PA, min/d ^g	14.7 ± 28.9	6.4 ± 9.8 ^b	7.9 ± 11.3

BMI refers to body mass index; RMR, resting metabolic rate.

^aUnless otherwise stated, values represent number, with percentage in parentheses.

^bSignificantly different from the 5-6-hours sleeping group, $P < 0.05$.

^c≤10 cigarettes per day.

^d>10 cigarettes per day.

^eIn Quebec, it is a level of education generally of 2 or 3 years between high school and university termed *CEGEP* (*Collège d'Enseignement Général et Professionnel*), an acronym that does not have any translation in English.

^fCanadian dollars (CAD).

^gMean time spent in vigorous physical activity (PA) participation estimated as the number of periods graded 8 and 9 over the 3 days.

to complete a physical activity diary for 3 days, including 2 weekdays and 1 weekend day. Each day was divided in 96 periods of 15 minutes each. For each 15-minute period, subjects had to code the main activity performed on a scale from 1 to 9. Participation in vigorous physical activity was estimated as the number of periods graded 8 and 9 over the 3 days. The reliability and the validity of the record have been previously reported.²⁵ These measurements were performed both at baseline and after 6 years.

Measurement of Covariates

Numerous covariates were measured via self-reported questionnaires at baseline and year 6. These include age, sex, smoking habits (nonsmoker or exsmoker, light smoker [≤ 10 cigarettes per day], heavy smoker [> 10 cigarettes per day]), employment status (student, paid employment, looking for work, home duties, retired, disabled), highest educational level (high school, college [CEGEP for Quebec], university), total annual family income (categorized into 5 groups ranging from

Table 2—Mean Weight Gain (kg) Above Baseline Weight for Short- and Long-Duration Sleepers Relative to the Gain in those Sleeping 7 to 8 Hours

	Sleep-duration group				
	5-6 hours		7-8 hours	9-10 hours	
	Mean	95% CI	Mean	Mean	95% CI
Age- and sex-adjusted model	1.86	1.13-2.62	0.00	1.51	0.93-2.44
Age, sex and BMI-adjusted model	1.98	1.16-2.82	0.00	1.58	1.02-2.56
Fully adjusted model ^a	1.84	1.08-2.61	0.00	1.49	0.92-2.48

CI refers to confidence interval.

^aFull model adjusted for age, sex, baseline body mass index (BMI), study phase, length of follow-up, resting metabolic rate, smoking habits, employment status, education level, total annual family income, menopausal status, shift-working history, alcohol intake, coffee intake, total caloric intake, and participation in vigorous physical activity.

< \$10,000 to \$70,000 or more), menopausal status, shift-working history (none, < 5 years, ≥ 5 years), alcohol intake (g/day), and coffee intake (number of cups per day).

Statistical Analysis

Differences in baseline characteristics by sleep-duration categories were compared by use of the analysis of variance (continuous variables) and χ^2 test for comparison of frequencies (categorical variables). Multiple regression models were used to determine which of these baseline characteristics are significantly and independently associated with sleep duration at baseline. For each sleep-duration group, weight gain, change in waist circumference, and change in percentage of body fat 6 years later were computed. An analysis of variance was performed on the means of these variables, followed by a Tukey HSD posthoc test aiming to contrast mean differences among the sleep-duration groups. In addition, multivariate linear regression modeling was used to estimate the mean weight gain above baseline weight for short- and long-duration sleepers relative to the gain in those sleeping 7 to 8 hours per night. The models were adjusted for age, sex, baseline BMI, study phase, length of follow-up, resting metabolic rate, smoking habits, employment status, educational level, total annual family income, menopausal status, shift-working history, alcohol intake, coffee intake, total caloric intake, and participation in vigorous physical activity. Because some individuals in this family study are biologically related, we adjusted for clustering in the analyses to avoid underestimation of standard deviations using the generalized estimating equations statistical method.²⁶ This procedure allowed us to model sleep duration and covariates as repeated measures at 2 time points (baseline and 6 years later). Data are given as mean and SD unless otherwise noted. Statistical significance was set at a P value < 0.05. All statistical analyses were performed using the JMP version 3.2.2 program (SAS Institute, Cary, NC).

RESULTS

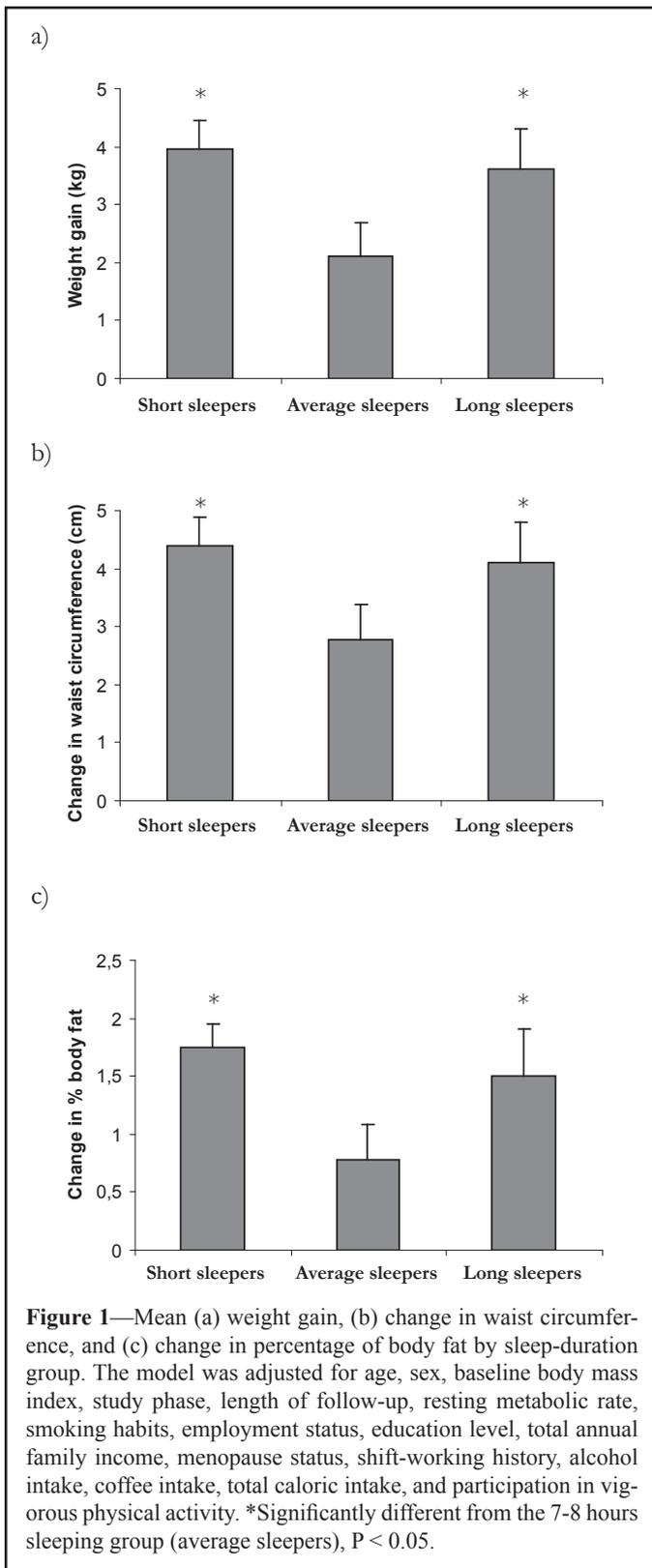
Table 1 presents baseline characteristics of subjects by sleep-duration category. Short-duration sleepers presented higher body weight, BMI, percentage of body fat, and abdominal circumference values at baseline, as compared with average-duration sleepers. However, we were not able to detect any significant difference between the 3 sleeper groups for

demographic characteristics, resting metabolic rate, and total caloric intake. Short-duration sleepers reported more vigorous physical activity participation than did average-duration sleepers. In addition, multiple regression models were used to determine which baseline characteristics (except those related to body composition) were significantly and independently associated with sleep duration at baseline. Among these variables, shift-working history, participation in vigorous physical activity, employment status, smoking habits, and coffee intake were the main independent predictors of sleep duration. This model accounted for 14.8% of the variance in sleep duration (P < 0.01).

After adjustment for covariates, the average weight gain of short-duration sleepers was 88% more than the weight gain of average-duration sleepers (see Figure 1). Similarly, short-duration sleepers experienced a 58% and 124% higher increase in waist circumference and percentage of body fat, as compared with average-duration sleepers, respectively. On the other hand, long-duration sleepers also exhibited a significantly higher increase in body weight (71%), waist circumference (47%), and percentage of body fat (94%), as compared with the increase observed in average-duration sleepers.

Table 2 shows the mean weight gain above baseline weight for short- and long-duration sleepers relative to the gain in those sleeping 7 to 8 hours. After adjustment for age, sex, and baseline BMI, short-duration sleepers gained 1.98 kg (95% confidence interval [CI]: 1.16-2.82) more and long-duration sleepers gained 1.58 kg (95% CI: 1.02-2.56) more than did average-duration sleepers over 6 years. Adjustment for potential covariates and further inclusion of energy intake and physical activity participation had no material effect on the relationship between sleep duration and weight gain.

Over the 6-year follow-up period, 86 of the 276 subjects (31%) experienced a weight gain of 5 kg or more. Short- and long-duration sleepers were 35% and 25% more likely to have a 5-kg weight gain, respectively, as compared with normal-duration sleepers, P < 0.05 (data not shown). In addition, a total of 52 subjects were obese at baseline, defined as a BMI of greater than 30 kg/m². Among the 224 subjects who were not obese at baseline, 32 new cases of obesity (14.3%) were identified over the 6-year follow-up period. The risk of developing obesity was elevated for short- and long-duration sleepers, as compared with average-duration sleepers, with a 27% and 21% increase in risk, respectively, P < 0.05 (data not shown).



DISCUSSION

The primary result of this study is that both short and long sleep times predict higher body weight and fat gain, independent of baseline weight or other covariates. This finding is partially in line with the results of 3 other longitudinal studies cited above.^{8,14,15} However, the latter studies found that only reduced sleep duration was associated with an increased risk for future

obesity. Our results nevertheless concur with a growing body of epidemiologic evidence showing a U-shaped relationship between sleep duration and BMI,⁷ type 2 diabetes,²⁷ coronary heart disease,²⁸ and all-cause mortality.²⁹ Thus, a body of data suggests that there may be an “optimal sleeping time” for the prevention of common diseases and premature death.

The observation that long sleep time was associated with greater increases in adiposity is in line with some epidemiologic data.^{7,9} In this regard, one plausible explanation is that long-duration sleepers are characterized by reduced energy expenditure due to increased time in bed. However, the present study could not detect such an effect. In addition, it should be noted that long-duration sleepers had higher BMIs at baseline, making it plausible that they would be more likely to gain weight over the follow-up period. Another possibility pertains to the fact that obesity is associated with increased proinflammatory cytokines, which promote sleep.³⁰ Finally, we need to consider the possibility that self-reported long-duration sleepers are spending a lot of time in bed but not getting a lot of sleep, i.e., they might have poor sleep quality possibly due to sleep disorders or other health issues. On the other hand, our study showed that short sleep duration influenced body weight, as has been previously observed in several studies. Ultimately, in order to cause weight gain, short sleeping hours must favor a positive energy balance. In this respect, one potential explanation may be that when we sleep less, we simply have more time and/or more opportunities to eat. In addition, restricted sleep may lead to daytime fatigue and perhaps reduced physical activity. However, no significant difference was observed between the 3 sleeper groups for energy intake and resting metabolic rate, whereas short-duration sleepers reported higher vigorous physical activity participation. Similarly, Patel et al¹⁵ were not able to find a relationship between short sleep duration and increased caloric intake. They also found, in accordance with the study of Hasler et al,¹⁴ that changes in voluntary physical activity could not explain the sleep–body weight association. However, we have to keep in mind the limitations of questionnaire-based measurements and the fact that minor daily energy surplus not detected by our questionnaire could result in weight gain over time. Furthermore, we need to realize that short sleep duration does not seem to have a direct measurable impact on energy intake or expenditure but, rather, may influence one or several regulatory steps, with the consequence of a mismatch between energy input and output.

Much attention has recently been focused on the responses of ghrelin, leptin, and orexin to sleep restriction.^{7,13,31} Alterations in these hormone levels or patterns of secretion may affect hunger and appetite, increasing the risk of overeating and consequently weight gain. They may also affect thermogenesis from activities other than exercise.³² Non-exercise activity thermogenesis (NEAT) (such as is seen in fidgeting and posture changes) is a variable component of energy expenditure and has been reported to account for differential weight gain in rats.³³ In addition, sleep loss results in changes in levels of several other hormones such as cortisol and growth hormone.³⁴ However, the precise mechanisms by which the brain modulates hormone release with sleep loss is unknown, but one possibility is increased sympathetic nervous system activity.^{35,36}

Abnormal sleep-wake patterns likely alter intracellular circadian clocks, which are molecular mechanisms enabling the cell,

tissue, or organism to anticipate diurnal variations in its environment.³⁷ The environment may include circulating levels of nutrients (e.g., glucose, fatty acids, and triglycerides) and various hormones (e.g., insulin, glucocorticoids). Alterations in these molecular mechanisms, in particular within the adipocyte, likely induce metabolic changes that may potentiate disrupted metabolism and weight gain.³⁷ Interestingly, in a study of subjects working alternate day and night shifts, levels of antioxidants were significantly lower following the night shift, compared with the day shift, suggesting that oxidative stress may be more significant following a night shift.³⁸ Whether it has anything to do with positive energy balance and weight gain remains unknown. The role of the peripheral circadian clock mechanism within the adipocyte represents an exciting new field of study in pursuit of the causes of increasing obesity prevalence.

The prevalence of short sleep duration has increased substantially over the same period in which we have observed the increase in the prevalence of obesity. Short sleep duration is apparently explained by factors such as longer work days and longer commuting time, an increase in evening and night work, and the advent of television, the personal computer, and the internet.³⁹ Hence, modernity, and its “24-hour-a-day” turbulent lifestyle, has favored the dominance of factors favoring positive energy balance in which short sleep duration is potentially associated with alterations in the hormonal profile that impair the regulation of energy balance. Furthermore, short sleeping hours in children have been shown to predict overweight or obesity to a greater extent than does low physical activity participation and long television viewing¹⁰ and to preferentially favor abdominal adiposity.⁴⁰ Thus, sleep time must now be considered as a new and potentially important determinant of obesity in the current way of living.

Strengths of this study include its longitudinal design and use of objective measures of adiposity. Furthermore, data were obtained from both men and women, and we used an approach that should serve to minimize confounding with repeated measures of sleep duration. However, the small sample size limits statistical power and generalizability of these results. In addition, caution must be exercised with regard to the heterogeneity in age, since age might act as an effect modifier in the relationship between sleep duration and obesity.¹⁴ Furthermore, we have to keep in mind that it was not a random sample and that the Quebec Family Study was originally conducted to explore the role of genetics in the etiology of obesity, suggesting that the current sample could be predisposed to obesity and weight gain due to genetic influences and through modeling of parental behavior. Another limitation of the study was the use of self-reported sleep durations, as opposed to measured sleep durations. Although good agreement has been found in previous studies between self-reported sleep durations and those obtained through actigraphic monitoring,^{41,42} the validity of a single question is still to be determined. Finally, the possibility of a confounding effect in the relationship between sleep time and adiposity, by unmeasured variables such as sleep-disordered breathing, insomnia, or depression, cannot be excluded. On the other hand, it seems that much of the reduction in sleep time reflects voluntary sleep restriction, with 43% of adults reporting that they often stay up later than they should watching television or using the internet and 45% reporting that they sleep less to get more work done.¹⁻⁴

In summary, the present study provides evidence to the effect that both short and long sleep times predict higher body weight and fat gain in adults, even after adjustment for potential covariates. These results emphasize the need to add sleep duration to the list of environmental factors that are prevalent in our society and that may contribute to the obesity epidemic.

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JPC designed the study, conducted the analyses, and wrote the manuscript. CB, JPD, and AT designed and created the Quebec Family Study and helped revise the manuscript.

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