International Journal of Obesity (2012) 36, 752–756
 0307-0565/12
www.nature.com/ijo

SHORT COMMUNICATION

Longer sleep duration associates with lower adiposity gain in adult short sleepers

J-P Chaput[1], J-P Després[2], C Bouchard[3] and A Tremblay[4]

[1]*Healthy Active Living and Obesity Research Group, Children's Hospital of Eastern Ontario Research Institute, Ottawa, Ontario, Canada;* [2]*Quebec Heart Institute, Hôpital Laval Research Center, Hôpital Laval, Quebec City, Quebec, Canada;* [3]*Human Genomics Laboratory, Pennington Biomedical Research Center, Baton Rouge, LA, USA and* [4]*Division of Kinesiology, Department of Social and Preventive Medicine, Faculty of Medicine, Laval University, Quebec City, Quebec, Canada*

The objective of this longitudinal, observational study was to verify whether a favorable change in sleep duration over 6 years could impact objective indicators of adiposity in adults aged 18–64 years. Short-duration sleepers ($\leqslant$6 h per day; $n=43$) at baseline were divided into two groups: (i) those who increased their sleep duration to a 'healthy' length of 7–8 h per day at year 6 (mean increase: 1.52 ± 0.66 h per day; $n=23$); and (ii) those who maintained their short sleep duration habits (mean change: -0.11 ± 0.38 h per day; $n=20$). Adult individuals who reported sleeping 7–8 h per day at both baseline and year 6 ($n=173$) were used as a control group. Change in adiposity indicators for each sleep-duration group was compared by analysis of covariance. We observed that the two short-sleep-duration groups had similar baseline characteristics. However, short-duration sleepers who maintained their short sleep duration experienced a greater increase in body mass index (BMI) (difference: 1.1 ± 0.36 kg m^{-2}, $P<0.05$) and fat mass (difference: 2.4 ± 0.64 kg, $P<0.05$) over the 6-year follow-up period than short-duration sleepers who increased their sleep duration, even after adjustment for relevant covariates. We did not observe any significant difference in adiposity changes between the control group and short-duration sleepers who increased their sleep duration. This study suggests for the first time that shifting sleep duration from a short to a healthier length is associated with an attenuation of fat mass gain.
International Journal of Obesity (2012) **36,** 752–756; doi:10.1038/ijo.2011.110; published online 7 June 2011

Keywords: adiposity; body weight; sleep duration; sleep augmentation

Introduction

Evidence that short sleep duration is another determinant of obesity is accumulating. Prospective cohort studies have shown that short sleep duration is associated with weight gain and an increased incidence of obesity in children and adults.[1,2] Intervention studies have begun to provide a mechanistic explanation, that is, that sleep restriction could influence the hormonal regulation of food intake.[3,4] Given that chronic sleep restriction is a common feature of our modern lifestyle, studies aimed at investigating the link between short sleep duration and obesity are relevant from a public health standpoint.

One critical next step in this field of investigation is to conduct randomized controlled trials to determine whether increasing sleep duration in sleep-deprived obese individuals can influence body weight.[5] Although such studies are challenging to implement, they will be instrumental in assessing whether increasing sleep duration is feasible and can significantly impact energy balance. To our knowledge, no observational study to date has attempted to examine the influence of increasing sleep duration to a healthier length (7–8 h per night) on body weight. The objective of this longitudinal, observational study is to verify whether a positive change in sleep duration over 6 years can impact adiposity. We hypothesize that short-duration sleepers ($\leqslant$6 h per day) who increase their sleeping time to a length of 7–8 h per day at year 6 can experience attenuation in fat gain compared with short-duration sleepers who maintain their short sleep duration habits.

Subjects and methods

Subjects

Subjects were participants in the Quebec Family Study. Details of recruitment procedures have been published elsewhere.[6] This cohort represents a mixture of random

Correspondence: Dr J-P Chaput, Healthy Active Living and Obesity Research Group, Children's Hospital of Eastern Ontario Research Institute, 401 Smyth Road, Ottawa, Ontario, Canada K1H 8L1.
E-mail: jpchaput@cheo.on.ca
Received 7 January 2011; revised 2 March 2011; accepted 3 May 2011; published online 7 June 2011

sampling and ascertainment through obese (body mass index (BMI) $\geqslant 32\,kg\,m^{-2}$) probands. The present analyses are based on participants in phases 2 (1989–1994) and 3 (1995–2001). Adult individuals who were between 18 and 64 years of age and who had short sleep duration (that is, $\leqslant 6$ h per day) at baseline (22 men and 21 women) were selected for prospective analyses (mean duration of follow-up: 6.0 ± 0.9 years). Adult individuals who reported sleeping 7–8 h per day at both baseline and year 6 ($n = 173$) were included as a control group. Additional inclusion criteria were (i) not pregnant; (ii) stable body weight (± 2 kg) over the 6 months preceding testing; and (iii) no metabolic disease (for example, diabetes, hypertension) or no medication that could interfere with the outcome variables. All subjects provided written informed consent to participate in the study. The project was approved by the Medical Ethics Committee of Laval University.

Anthropometric and body composition data

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, Scotland, UK). BMI was calculated as body weight divided by height squared ($kg\,m^{-2}$). 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.[7] Furthermore, body density was obtained from the mean of six valid measurements derived from underwater weighing.[8] Before immersion in the hydrostatic tank, the helium dilution method of Meneely and Kaltreider[9] was used to determine the pulmonary residual volume. The percentage of total body fat was determined from body density using the equation of Siri.[10] 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.

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?' We classified individuals sleeping $\leqslant 6$ h per day as 'short-duration sleepers', in agreement with the available literature.[11,12] We divided the short-duration sleepers into two groups: (i) short sleepers who increased their sleep duration to a healthy length of 7–8 h per day at year 6 ($n = 23$); and (ii) short sleepers who maintained their short sleep duration habits ($n = 20$). As a sleep duration of 7–8 h per day has been reported to be associated with the prevention of common diseases and premature death,[13,14] a control group was also added to facilitate comparisons. As mentioned above, the control group included individuals who reported sleeping 7–8 h per day at both baseline and year 6.

Covariates

Numerous covariates were measured via self-reported questionnaires at baseline and year 6. These include age, sex, smoking habits, employment status, highest educational level, total annual family income and menopausal status. Additionally, the diet of participants was evaluated with a 3-day food record, including 2 week days and 1 weekend day. Participants were shown how to complete the record by a dietician who provided instructions 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.[15] Mean daily energy intake was estimated by a dietician using a computerized version of the Canadian Nutrient File.[16] Finally, daily physical activity level and pattern were evaluated with a 3-day physical activity diary, as previously described.[17] Moderate-to-vigorous physical activity participation over the 3 days was used for statistical analyses. The validity and reliability of the physical activity record have been previously reported.[17]

Statistical analysis

Since there was no statistically significant gender interaction between sleep duration and the outcome variables, data for both sexes were combined to improve clarity and maximize power. Baseline characteristics of participants by sleep duration group were compared by analysis of variance (continuous variables) and χ^2 test (categorical variables). A Tukey HSD *post-hoc* test was conducted to investigate which of the different sleep duration groups differed from the others. Furthermore, change in adiposity indicators over 6 years was computed for each sleep-duration group. An analysis of covariance was performed on the means of these variables, followed by a Tukey HSD *post-hoc* test. The model was adjusted for age, sex, baseline BMI, smoking habits, employment status, highest educational level, total annual family income, menopausal status, energy intake and moderate-to-vigorous physical activity as covariates. A two-tailed *P*-value of less than 0.05 was considered to indicate statistical significance. All statistical analyses were performed using the JMP version 8 program (SAS Institute, Cary, NC, USA).

Results

Baseline characteristics of participants according to sleep-duration group are shown in Table 1. We observed that the two short-sleep-duration groups were similar at baseline and did not significantly differ for any of the variables. In contrast, individuals in the control group had significantly higher sleep duration and were leaner than short-duration sleepers, as expected from previous observations.

Table 1 Baseline characteristics of participants according to sleep-duration group

		Short-duration sleepers	
	Control group (n = *173*)	*Increased sleep* (n = *23*)	*Maintained sleep* (n = *20*)
Age (years)	39.0 ± 14.5	40.3 ± 12.5	40.5 ± 12.6
Sex			
Men	85 (49)	12 (52)	10 (50)
Women	88 (51)	11 (48)	10 (50)
Body weight (kg)	70.9 ± 16.3	77.1 ± 12.9*	75.4 ± 17.6
BMI (kg m^{-2})	25.5 ± 5.8	27.7 ± 4.8*	27.7 ± 6.2*
Fat mass (kg)	18.6 ± 9.9	22.8 ± 10.5*	22.8 ± 11.7*
Waist circumference (cm)	83.9 ± 15.8	90.1 ± 13.8	87.9 ± 16.7
Smoking habits			
Nonsmoker or ex-smoker	149 (86)	20 (87)	16 (80)
Light smoker[a]	9 (5)	1 (4)	1 (5)
Heavy smoker[b]	15 (9)	2 (9)	3 (15)
Employment status			
Student	34 (19)	4 (17)	3 (15)
Paid employment	109 (63)	13 (56)	12 (60)
Looking for work	4 (2)	1 (4)	1 (5)
Home duties	18 (10)	2 (9)	2 (10)
Retired	6 (3)	2 (9)	1 (5)
Disabled	2 (1)	1 (4)	1 (5)
Highest educational level			
High school	63 (36)	10 (43)	11 (55)
Pre-university level	67 (39)	8 (35)	6 (30)
University	43 (25)	5 (22)	3 (15)
Total annual family income ($C)	57 811 ± 23 903	56 315 ± 31 878	64 545 ± 28 058
Menopausal status			
In menopause	15 (9)	3 (13)	3 (15)
Not in menopause	158 (91)	20 (87)	17 (85)
Sleep duration (h)	7.7 ± 0.4	5.7 ± 0.2*	5.6 ± 0.4*
Energy intake (kcal per day)	2358 ± 791	2546 ± 782	2466 ± 533
MVPA (min per day)	19.4 ± 28.5	25.2 ± 41.2	24.8 ± 54.3

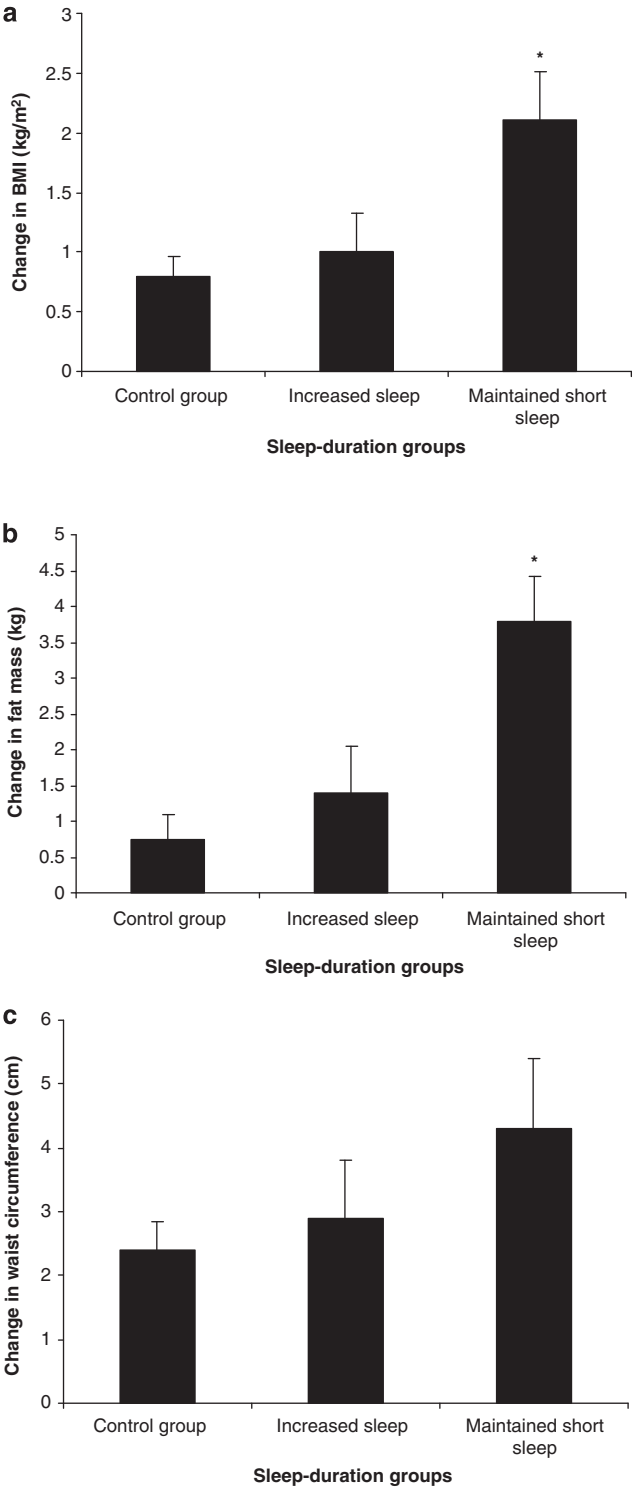
Abbreviations: BMI, body mass index; MVPA, moderate-to-vigorous physical activity. Control group: individuals who reported sleeping 7–8 h per day at both baseline and year 6. Short-duration sleepers who increased their sleep duration: from short sleep duration (⩽6 h per day) at baseline to a healthier length of 7–8 h per day at year 6. Short-duration sleepers who maintained their short sleep duration: ⩽6 h per day at both baseline and year 6. Values are mean ± s.d. or *n* (%). *$P<0.05$ versus control group. Statistical significance was assessed by analysis of variance with continuous variables and by a χ^2 test with categorical variables, followed by a Tukey HSD *post-hoc* test. [a]⩽10 cigarettes per day. [b]>10 cigarettes per day.

We observed that short-duration sleepers who maintained their short sleep duration habits experienced a greater increase in BMI and fat mass over the 6-year follow-up period compared with short-duration sleepers who increased their sleep duration, even after adjustment for covariates ($P<0.05$; Figure 1). A similar pattern was observed for weight gain; however, change in waist circumference did not reach statistical significance ($P=0.09$; Figure 1). We did not observe any significant difference in adiposity indicators between the control group and short-duration sleepers who increased their sleep duration. Variations in energy intake and moderate-to-vigorous physical activity over the follow-up period did not significantly differ between the two short-sleep-duration groups. Changes in sleep duration were −0.11 ± 0.38 and 1.52 ± 0.66 h per day for short-duration sleepers who maintained and increased their sleep duration, respectively.

Discussion

This study is the first to test whether an increase in sleep duration (from ⩽6 h per day to 7–8 h per day) could impact body weight and adiposity. We observed that short-duration sleepers who reported sleeping 7–8 h per day 6 years later were less likely to gain weight compared with those who maintained their habitual short-sleep-duration habit. This observation is of clinical relevance and justifies the inclusion of sleep duration in the list of environmental factors that contribute to weight gain.

Furthermore, this study suggests that shifting sleep duration from a short length to a healthier length is associated with lower adiposity gain, independently of a number of covariates.



The mechanisms by which short sleep duration influences body weight are under scrutiny and might involve both sides of the energy balance equation. Lack of sleep has been reported to decrease plasma leptin levels, increase plasma ghrelin and cortisol levels, alter glucose homeostasis and activate the orexin system, all of which impact the regulation of food intake.[18] Lack of sleep could also lead to weight gain and obesity by increasing the time available for eating and by making the maintenance of a healthy physically active lifestyle more difficult. In an environment in which food is highly palatable and readily available, caloric intake may be directly proportional to time spent awake, especially if a large proportion of the waking hours is spent in sedentary activities, such as watching television, when snacking is common.[19] Furthermore, the increased fatigue and tiredness associated with not having enough sleep may impact the overall physical activity participation. Interestingly, recent results have shown that sleep might be an important factor in successful weight loss, suggesting that a narrow focus on diet and exercise might not be sufficient.[20]

It is important to keep in mind that the association between short sleep duration and obesity does not in any way diminish the importance of diet and physical activity in promoting healthy body weight. Many fail to realize the complex etiology of obesity. Based on decades of research, the effect of any single factor on body weight should be relatively small. If confirmed by other studies, an attenuation in fat mass gain associated with a positive change in sleep duration could have important clinical and translational implications. However, more research is needed to better understand the effects of adequate sleep on food intake, appetite control, metabolic rates and physical activity participation before advocating sleep habit modification as a means of weight control.

The strengths of this study include its longitudinal design and the use of objective measures of adiposity on both men and women. However, although observational cohort studies are well suited for the identification of associations, they cannot establish causality. Additionally, sleep duration was only assessed at two time points. It cannot be excluded that sleep duration could have varied over the 6-year follow-up period. Furthermore, it would have been interesting to know

Figure 1 Mean (**a**) change in body mass index, (**b**) change in fat mass and (**c**) change in waist circumference by sleep-duration group. Control group: individuals who reported sleeping 7–8 h per day at both baseline and year 6. Short-duration sleepers who increased their sleep duration: from short sleep duration ($\leqslant$6 h per day) at baseline to a healthier length of 7–8 h per day at year 6. Short-duration sleepers who maintained their short sleep duration: $\leqslant$6 h per day at both baseline and year 6. Values are mean ± s.e.m. Statistical significance was assessed by analysis of covariance, followed by a Tukey HSD *post-hoc* test. The model was adjusted for age, sex, baseline BMI, smoking habits, employment status, highest educational level, total annual family income, menopausal status, energy intake and moderate-to-vigorous physical activity as covariates. *$P<0.05$ versus two other groups.

how and why some short-duration sleepers increased their sleep duration. The small sample size and preliminary nature of this investigation preclude any definitive conclusion regarding the influence of a change in sleep duration on adiposity. However, we hope that it will generate larger and well-designed studies to address this issue in a timely fashion. Finally, we also have to keep in mind the limitations of questionnaire-based measurements that are inherent in epidemiological studies, as well as the possibility of residual confounding.

In summary, the present study provides evidence that a positive change in sleep duration (from $\leqslant$6 h per day to 7–8 h per day) is associated with lower fat mass gain over time. These results are novel and emphasize the need to conduct randomized controlled trials aimed at assessing the effectiveness of increasing sleep duration on energy balance, appetite control and metabolic profile.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgements

We express our gratitude to the subjects for their participation in the Quebec Family Study and the staff of the Physical Activity Sciences Laboratory at Laval University for their contribution to this study. We especially thank Dr Germain Thériault, Guy Fournier, Monique Chagnon, Lucie Allard and Claude Leblanc for their help in the collection and analysis of the data. J-PC designed the study, conducted the analyses and wrote the manuscript. CB and AT designed and created the Quebec Family Study and CB, AT and J-PD helped revise the manuscript. We acknowledge the financial support over 20 years of the Medical Research Council of Canada (now the Canadian Institutes of Health Research) through several grants for the Quebec Family Study, as well as other agencies from the governments of Quebec and Canada. J-PC holds a Junior Research Chair in Healthy Active Living and Obesity Research. J-PD is scientific director of the International Chair on Cardiometabolic Risk held at Université Laval. CB is partially supported by the John W Barton Sr Chair in Genetics and Nutrition. AT is partly funded by the Canada Research Chair in Environment and Energy Balance.

References

1 Nielsen LS, Danielsen KV, Sørensen TI. Short sleep duration as a possible cause of obesity: critical analysis of the epidemiological evidence. *Obes Rev* 2011; **12**: 78–92.
2 Chaput JP, Klingenberg L, Sjödin AM. Do all sedentary activities lead to weight gain: sleep does not. *Curr Opin Clin Nutr Metab Care* 2010; **13**: 601–607.
3 Brondel L, Romer MA, Nougues PM, Touyarou P, Davenne D. Acute partial sleep deprivation increases food intake in healthy men. *Am J Clin Nutr* 2010; **91**: 1550–1559.
4 Spiegel K, Tasali E, Leproult R, Van Cauter E. Effects of poor and short sleep on glucose metabolism and obesity risk. *Nat Rev Endocrinol* 2009; **5**: 253–261.
5 Cizza G, Marincola P, Mattingly M, Williams L, Mitler M, Skarulis M *et al*. Treatment of obesity with extension of sleep duration: a randomized, prospective, controlled trial. *Clin Trials* 2010; **7**: 274–285.
6 Bouchard C. Genetic epidemiology, association and sib-pair linkage: results from the Quebec Family Study. In: Bray G, Ryan D (eds). *Molecular and Genetic Aspects of Obesity*. Louisiana State University Press: Baton Rouge, LA, USA, 1996, pp 470–481.
7 The Airlie (VA) Consensus Conference. In: Lohman T, Roche A, Martorel R (eds). *Standardization of Anthropometric Measurements*. Human Kinetics: Champaign, IL, USA, 1988, pp 39–80.
8 Behnke AR, Wilmore JH. Evaluation and regulation of body build and composition. In: Behnke AR (ed.). *Evaluation and Regulation of Body Build and Composition*. Prentice-Hall: Englewood Cliffs, NJ, USA, 1974, pp 20–37.
9 Meneely GR, Kaltreider NL. Volume of the lung determined by helium dilution. *J Clin Invest* 1949; **28**: 129–139.
10 Siri WE. The gross composition of the body. *Adv Biol Med Phys* 1956; **4**: 239–280.
11 Patel SR. Reduced sleep as an obesity risk factor. *Obes Rev* 2009; **10** (Suppl 2): 61–68.
12 Chaput JP, Leblanc C, Pérusse L, Després JP, Bouchard C, Tremblay A. Risk factors for adult overweight and obesity in the Quebec Family Study: have we been barking up the wrong tree? *Obesity (Silver Spring)* 2009; **17**: 1964–1970.
13 Gangwisch JE. Epidemiological evidence for the links between sleep, circadian rhythms and metabolism. *Obes Rev* 2009; **10** (Suppl 2): 37–45.
14 Gallicchio L, Kalesan B. Sleep duration and mortality: a systematic review and meta-analysis. *J Sleep Res* 2009; **18**: 148–158.
15 Tremblay A, Sévigny J, Leblanc C, Bouchard C. The reproducibility of a three-day dietary record. *Nutr Res* 1983; **3**: 819–830.
16 Government of Canada. *The Canadian Nutrient File*. Health and Welfare Canada: Ottawa, Canada, 1990.
17 Bouchard C, Tremblay A, Leblanc C, Lortie G, Savard R, Thériault G. A method to assess energy expenditure in children and adults. *Am J Clin Nutr* 1983; **37**: 461–467.
18 Leproult R, Van Cauter E. Role of sleep and sleep loss in hormonal release and metabolism. *Endocr Dev* 2010; **17**: 11–21.
19 Chaput JP, Klingenberg L, Astrup A, Sjödin AM. Modern sedentary activities promote overconsumption of food in our current obesogenic environment. *Obes Rev* 2011; **12**: e12–e20.
20 Nedeltcheva AV, Kilkus JM, Imperial J, Schoeller DA, Penev PD. Insufficient sleep undermines dietary efforts to reduce adiposity. *Ann Intern Med* 2010; **153**: 435–441.