Short sleep duration as a risk factor for the development of the metabolic syndrome in adults

By: Jean-Phillipe Chaput, Jessica McNeil, Jean-Pierre Després, Claude Bouchard, and Angelo Tremblay


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Abstract:

Objective: The objective of this study was to investigate the association between self-reported sleep duration and the incidence of features of the metabolic syndrome in adults. Methods: A longitudinal analysis from the Quebec Family Study (Canada) was conducted on 293 participants, aged 18 to 65 years, followed for a mean of 6 years (until 2001). Participants were categorized as short (≤ 6 h), adequate (7–8 h) or long (≥ 9 h) sleepers. The metabolic syndrome was defined according to the American Heart Association/National Heart, Lung, and Blood Institute's criteria. The hypertriglyceridemic waist phenotype was defined as high waist circumference (≥ 90 cm in men and ≥ 85 cm in women) combined with high fasting triglyceride level (≥ 2.0 mmol/L in men and ≥ 1.5 mmol/L in women). Results: The incidence rates of metabolic syndrome and hypertriglyceridemic waist phenotype were 9.9% and 7.5%, respectively. Short sleepers were significantly more at risk of developing the metabolic syndrome (relative risk (RR): 1.74; 95% confidence interval (CI): 1.05–2.72) and the hypertriglyceridemic waist phenotype (RR: 1.82; 95% CI: 1.16–2.79), compared to those sleeping 7 to 8 h per night after adjusting for covariates. However, long sleep duration was not associated with an increased risk of developing the metabolic syndrome or the hypertriglyceridemic waist phenotype (either unadjusted or adjusted models). Conclusion: Short sleep duration is associated with an increased risk of developing features of the metabolic syndrome in adults.

Keywords: Sleep | Metabolic syndrome | Cardiometabolic risk | Longitudinal study | Adults

Article:

Introduction

Chronic sleep deprivation is increasingly common in modern society (Akerstedt and Nilsson, 2003, Matricciani et al., 2012). Insufficient sleep adversely impacts health possibly through deleterious metabolic and endocrine perturbations that affect a variety of systems in the body
Previous epidemiologic studies have shown that both short and long sleep durations are associated with poor health outcomes including obesity (Chaput et al., 2008, Patel and Hu, 2008), type 2 diabetes (Cappuccio et al., 2010a, Chaput et al., 2009a), coronary heart disease (Cappuccio et al., 2011, King et al., 2008), hypertension (Gangwisch et al., 2006, Knutson et al., 2009), and premature death (Cappuccio et al., 2010b, Gallicchio and Kalesan, 2009). Although the association between sleep duration and health indicators has been reported to be U-shaped in many studies, the adverse effects of inadequate sleep appear far more important in today's environment (Knutson and Turek, 2006). In particular, the association between long sleep duration and the metabolic syndrome appears to be explained by residual confounding factors and comorbidity (Ju and Choi, 2013).

The metabolic syndrome represents a cluster of cardiometabolic risk factors and is an important public health concern (Després and Lemieux, 2006). A simple screening tool to help clinicians identify individuals with clustering metabolic abnormalities and those at increased coronary artery disease risk is the hypertriglyceridemic waist phenotype. The combined measurement of waist circumference (≥ 90 cm in men and ≥ 85 cm in women) and fasting triglyceride levels (≥ 2.0 mmol/L in men and ≥ 1.5 mmol/L in women) is an inexpensive and clinically useful test for the early screening of individuals at risk for atherogenic and diabetogenic features of the metabolic syndrome (Blackburn et al., 2009, Blackburn et al., 2012, Lemieux et al., 2000, Lemieux et al., 2007).

Although sleep is important for the normal functioning of metabolic and hormonal processes, only one study to date has examined the association between sleep duration and the incidence of metabolic syndrome. In a Korean cohort of adults aged 40 to 70 years, Choi et al. (2011) showed that short sleep duration was a significant risk factor for the development of metabolic syndrome in women over a follow-up period of 2–4 years. However, they did not control for several important variables that could confound the association between short sleep duration and the development of metabolic syndrome (e.g. energy intake, cardiorespiratory fitness, socioeconomic status) and only relied on Koreans for whom different waist circumference cut-offs are recommended. Therefore, more studies are needed to prospectively examine the association between sleep duration and the metabolic syndrome to better understand the potential contribution of sleep as a predictor of cardiometabolic risk.

The main objective of this observational, longitudinal study was to investigate the relationship between sleep duration and the development of metabolic syndrome in adults from the greater Quebec City area (Canada). We hypothesized that short sleepers (≤ 6 h per night) would be more at risk of developing the metabolic syndrome and the hypertriglyceridemic waist phenotype than those sleeping 7 to 8 h a day, independently of relevant covariates.

Methods

Participants

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 assessed. Recruitment was conducted irrespective of body weight during phase 1, resulting in a cohort with a wide range of body mass index, ranging from 13.8 to 64.9 kg/m². In phases 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 body mass index of 32 kg/m² or higher were added to the cohort. Families were recruited through the media and were all French Canadians from the greater Quebec City area. Details of recruitment procedures have been published elsewhere (Bouchard, 1996). This cohort thus represents a mixture of random sampling and ascertainment through obese individuals. The present analyses are based on participants tested in phases 2 and 3 because some measurements were not available in phase 1. Adults between 18 and 65 years of age were selected for longitudinal analyses (n = 293). A total of 23 participants were excluded because they were outside this age range. The mean duration of follow-up between phases 2 and 3 was 6.0 ± 0.9 years. All subjects provided written informed consent to participate in the study. The project was approved by the Medical Ethics Committee of Laval University.

Sleep duration assessment

The number of hours of sleep was assessed at baseline and year 6 with a question that was inserted into a self-administered questionnaire on physical activity participation. The question was formulated as follows: “On average, how many hours do you sleep a day?” We classified the participants into 3 sleep duration groups: short sleepers (≤ 6 h of sleep), adequate sleepers (7–8 h of sleep) and long sleepers (≥ 9 h of sleep), in agreement with our recent papers (Chaput et al., 2007, Chaput et al., 2009b). We elected to classify the participants into 3 sleep duration groups because of the U-shaped relationship that has been previously noted between sleep duration and several health outcomes. Epidemiological evidence indicates that a daily sleep duration of 7–8 h is optimal, and is associated with an overall good health status in adults (Bixler, 2009).

Metabolic syndrome assessment

Components of the metabolic syndrome were assessed in the morning following a 12-h overnight fast at baseline and year 6. Systolic (SBP) and diastolic (DBP) blood pressure measurements were obtained by trained staff, using a mercury sphygmomanometer and cuff size appropriate to the participant's arm circumference. Blood pressure was calculated as the mean of two consecutive readings obtained on the right arm, in a seated position, and following 10 min of rest. Body weight, height and waist circumference (WC) (midpoint of the lowest rib and iliac crest) were measured according to standardized procedures (The Airlie, VA Consensus Conference, 1988), and body mass index (BMI) was calculated as body weight divided by height squared (kg/m²). Study staff also collected a venous blood sample to measure fasting serum lipids, glucose, and insulin concentrations, as previously described (Chaput et al., 2007). The metabolic syndrome was defined according to the American Heart Association/National Heart, Lung, and Blood Institute's criteria (Grundy et al., 2005) as the presence of three or more of the following risk factors: (i) waist circumference greater than 102 cm in men or greater than 88 cm in women; (ii) fasting serum glucose of 5.6 mmol/L or greater, or the use of oral hypoglycemic medication; (iii) systolic and diastolic blood pressures of at least 130 mm Hg and 85 mm Hg, respectively, or the use of antihypertensive medication; (iv) serum triglyceride levels of 1.7 mmol/L or higher, or the use of medication for hypertriglycerideremia; and (v) high-density
lipoprotein (HDL) cholesterol levels of less than 1.03 mmol/L in men or 1.29 mmol/L in women, or the use of medication for low HDL cholesterol.

For exploratory purposes, we also calculated a continuous metabolic syndrome risk score for each participant by adding up Z scores for each variable as follows:

\[
\text{Continuous metabolic syndrome risk score} = -z_{\text{Insulin}} + z_{\text{Glucose}} + z_{\text{Triglycerides}} + (z_{\text{BMI}} + z_{\text{WC}})/2 + (z_{\text{SBP}} + z_{\text{DBP}})/2.
\]

This clustered cardiometabolic risk score has been used successfully in recent investigations (Carson and Janssen, 2011, Ekelund et al., 2006, Franks et al., 2004) and in contrast to a binary definition for the metabolic syndrome (presence/absence), this approach provides a continuous risk score that increases statistical power. Although the continuous metabolic syndrome risk score is a valid alternative for epidemiological analyses (Wijndaele et al., 2006), the binary definition still remains the method of choice in clinical practice.

*Hypertriglyceridemic waist phenotype assessment*

The hypertriglyceridemic waist phenotype (Lemieux et al., 2000) was also used to identify individuals at increased cardiometabolic risk. For men, the hypertriglyceridemic waist phenotype is defined as a waist circumference of 90 cm or more and a triglyceride level of 2.0 mmol/L or more (Blackburn et al., 2009). For women, the hypertriglyceridemic waist phenotype is defined as a waist circumference of 85 cm or more and a triglyceride level of 1.5 mmol/L or more (Blackburn et al., 2012).

**Covariates**

Numerous variables were measured via self-reported questionnaires at baseline and year 6. These included age, sex, smoking habits (smoker or nonsmoker), total annual family income (Canadian dollars per year), and coffee intake (number of cups per day). Additionally, daily energy intake (kcal/day) and alcohol consumption (g/day) were assessed with a 3-day food record (Tremblay et al., 1983). This method of dietary assessment has been shown to provide a relatively reliable measurement of food intake in this population (Tremblay et al., 1983). Finally, physical work capacity at a heart rate of 150 bpm (PWC$_{150}$), determined by a progressive exercise test on a modified Monark cycle ergometer, was used as an indicator of cardiorespiratory fitness, as previously described (Bouchard et al., 1984). PWC$_{150}$ is expressed in kiloponds/meter (kpm). We originally included moderate-to-vigorous physical activity (MVPA, self-reported from a 3-day physical activity diary) as a covariate in our analyses but decided to use cardiorespiratory fitness instead because (i) MVPA did not fit the definition of a confounding variable (i.e. variable correlated with both the exposure and outcome); (ii) self-reported variables are less accurate than objective measures; and (iii) we were limited by the number of covariates to enter in our models given the small sample size. These 8 covariates have been chosen because they all fit the definition of a confounding variable, i.e. an extraneous variable that correlates with both the independent variable (sleep) and the dependent variable (metabolic syndrome). Given that over-fitting can be a concern with our relatively small sample size, different analyses
were conducted to assess model fit (including testing for multicollinearity) and it resulted that our models had good predictive performance and were not overfit.

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) or chi-squared test (categorical variables). A Tukey post-hoc test was used to contrast mean differences. Multivariate logistic regression analysis was used to evaluate the relative risk for the development of the metabolic syndrome or hypertriglyceridemic waist phenotype in short and long sleepers over the 6-year follow-up period. The 7–8 h sleep duration category was used as the reference group. The model was adjusted for age, sex, smoking habits, total annual family income, alcohol consumption, coffee intake, daily caloric intake, and cardiorespiratory fitness. Relative risks (RR) and 95% confidence intervals (CI) were reported. Because some individuals in this study came from the same nuclear family and are biologically related, we adjusted for clustering in the analyses using the generalized estimating equations statistical method to avoid underestimation of standard deviations. This procedure allowed us to model sleep duration and covariates as repeated measures as two time points (baseline and 6 years later), thus taking into account both measures over time. A 2-tailed P value of less than 0.05 was the threshold to indicate statistical significance. All statistical analyses were performed using JMP version 10 (SAS Institute, Cary, NC).

Results

Baseline characteristics of participants within each sleep duration group are shown in Table 1. Among the 293 Caucasian participants of this study, 10 (3.4%) had diagnosed type 2 diabetes, and 27 (9.2%) had diagnosed hypertension. Over the 6-year follow-up period, 11 new cases of type 2 diabetes and 18 new cases of hypertension were observed. Short sleepers had a significantly higher body mass index, waist circumference, and fasting insulin concentrations compared to those sleeping 7 to 8 h per night. Short sleepers also reported drinking more coffee than long sleepers.

Overall, 64 participants (21.8% of the sample) had the metabolic syndrome at baseline. There were 29 new cases of metabolic syndrome that developed over the 6-year follow-up period (incidence rate of 9.9%). Likewise, 53 participants (18.1% of the sample) had the hypertriglyceridemic waist phenotype at baseline and 22 new cases occurred over this period (incidence rate of 7.5%). As shown in Fig. 1, an inverse J-shaped relationship between sleep duration and the development of the metabolic syndrome or hypertriglyceridemic waist phenotype was observed ($P < 0.05$).

The results of the multivariate logistic regression analysis assessing the relationship between sleep duration and the development of metabolic syndrome or hypertriglyceridemic waist phenotype are summarized in Table 2, Table 3. Short sleepers were significantly more at risk of developing the metabolic syndrome or the hypertriglyceridemic waist phenotype compared to those sleeping 7 to 8 h per night, after adjusting for covariates. However, long sleep duration was
not associated with an increased risk of developing the metabolic syndrome or the hypertriglyceridemic waist phenotype (either unadjusted or adjusted models). Likewise, the continuous metabolic syndrome risk score increased significantly more in short sleepers (but not long sleepers) compared to adequate sleepers over the 6-year follow-up period after adjustment for covariates (data not shown). Of note, the use of another definition for the metabolic syndrome (e.g. International Diabetes Federation) did not change the associations. Among the individual components of the metabolic syndrome, abdominal adiposity (followed by HDL cholesterol) was the component that changed to the greatest extent in short-duration sleepers over the 6-year follow-up period (data not shown).

### Table 1. Baseline characteristics of participants according to sleep duration group.

<table>
<thead>
<tr>
<th></th>
<th>≤ 6 h per night (n = 38)</th>
<th>7–8 h per night (n = 215)</th>
<th>≥ 9 h per night (n = 40)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>41.0 ± 13.6</td>
<td>39.2 ± 14.3</td>
<td>39.4 ± 14.8</td>
<td>0.78</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men (%)</td>
<td>55</td>
<td>46</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>Women (%)</td>
<td>45</td>
<td>54</td>
<td>65</td>
<td>0.20</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>28.4 ± 5.1 a</td>
<td>25.6 ± 5.7</td>
<td>26.8 ± 6.7</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>91.6 ± 14.4 a</td>
<td>83.8 ± 15.6</td>
<td>84.9 ± 17.5</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Fasting glucose (mmol/L)</td>
<td>5.44 ± 1.15</td>
<td>5.21 ± 1.21</td>
<td>4.93 ± 0.49</td>
<td>0.18</td>
</tr>
<tr>
<td>Fasting insulin (pmol/L)</td>
<td>86.7 ± 68.1 a</td>
<td>60.8 ± 48.8</td>
<td>81.3 ± 68.9</td>
<td>0.01</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>115 ± 12</td>
<td>117 ± 15</td>
<td>121 ± 19</td>
<td>0.20</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>74 ± 11</td>
<td>72 ± 10</td>
<td>74 ± 11</td>
<td>0.45</td>
</tr>
<tr>
<td>Fasting triglycerides (mmol/L)</td>
<td>1.63 ± 1.18</td>
<td>1.58 ± 2.11</td>
<td>1.48 ± 0.76</td>
<td>0.85</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.21 ± 0.35</td>
<td>1.25 ± 0.34</td>
<td>1.26 ± 0.36</td>
<td>0.72</td>
</tr>
<tr>
<td>Smoking habits</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonsmoker (%)</td>
<td>74</td>
<td>86</td>
<td>83</td>
<td></td>
</tr>
<tr>
<td>Smoker (%)</td>
<td>26</td>
<td>14</td>
<td>17</td>
<td>0.14</td>
</tr>
<tr>
<td>Total annual family income ($C)</td>
<td>61,389 ± 29,510</td>
<td>58,578 ± 24,351</td>
<td>56,750 ± 26,446</td>
<td>0.72</td>
</tr>
<tr>
<td>Alcohol consumption (g/day)</td>
<td>9.3 ± 15.7</td>
<td>6.8 ± 12.9</td>
<td>9.4 ± 19.7</td>
<td>0.53</td>
</tr>
<tr>
<td>Coffee intake (cups/day)</td>
<td>3.03 ± 2.31 b</td>
<td>2.61 ± 2.11</td>
<td>2.19 ± 1.81</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Energy intake (kcal/day)</td>
<td>2498 ± 682</td>
<td>2313 ± 760</td>
<td>2218 ± 587</td>
<td>0.22</td>
</tr>
<tr>
<td>PWC150 (kpm)</td>
<td>753 ± 349</td>
<td>625 ± 268</td>
<td>588 ± 241</td>
<td>0.07</td>
</tr>
</tbody>
</table>

Abbreviations: HDL, high-density lipoprotein; PWC150, physical work capacity at a heart rate of 150 bpm. Values are mean ± SD or n (%). Statistical significance was assessed by analysis of variance with continuous variables and by a chi-squared test with categorical variables. A Tukey post-hoc test was used to contrast mean differences.

a Significantly different from the 7–8 h sleeping group.
b Significantly different from the ≥ 9 h sleeping group.
Fig. 1. Development of the metabolic syndrome (A) and the hypertriglyceridemic waist phenotype (B) between sleep duration groups over the 6-year follow-up period (until 2001) in adults from the greater Quebec City area (Canada). Data are presented as percentage. The metabolic syndrome was defined according to the American Heart Association/National Heart, Lung, and Blood Institute's criteria. Statistical significance was assessed by a chi-squared test ($P < 0.05$ for both analyses). Short sleepers ($\leq 6$ h of sleep per night; $n = 38$), adequate sleepers (7–8 h of sleep per night; $n = 215$), and long sleepers ($\geq 9$ h of sleep per night; $n = 40$).

Table 2. Relative risk for the incidence of metabolic syndrome by sleep duration group over the 6-year follow-up period (until 2001) in adults from the greater Quebec City area (Canada).

<table>
<thead>
<tr>
<th>Sleep duration group</th>
<th>$\leq 6$ h per night ($n = 38$)</th>
<th>7–8 h per night ($n = 215$)</th>
<th>$\geq 9$ h per night ($n = 40$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RR</td>
<td>2.01</td>
<td>1.31</td>
<td>0.80–2.12</td>
</tr>
<tr>
<td>95% CI</td>
<td>1.30–3.05</td>
<td>1.00</td>
<td>0.51–1.98</td>
</tr>
<tr>
<td>RR</td>
<td>1.74</td>
<td>1.10</td>
<td></td>
</tr>
<tr>
<td>95% CI</td>
<td>1.05–2.72</td>
<td>0.51–1.98</td>
<td></td>
</tr>
</tbody>
</table>

The metabolic syndrome was defined according to the American Heart Association/National Heart, Lung, and Blood Institute's criteria.

Model 1: adjusted for age and sex.

Model 2: adjusted for age, sex, smoking habits, total annual family income, alcohol consumption, coffee intake, daily caloric intake, and cardiorespiratory fitness.

Abbreviations: RR, relative risk; CI, confidence interval.
Table 3. Relative risk for the incidence of the hypertriglyceridemic waist phenotype by sleep duration group over the 6-year follow-up period (until 2001) in adults from the greater Quebec City area (Canada).

<table>
<thead>
<tr>
<th>Sleep duration group</th>
<th>7–8 h per night (n = 215)</th>
<th>≥9 h per night (n = 40)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td>RR</td>
<td>95% CI</td>
</tr>
<tr>
<td>≤6 h per night (n = 38)</td>
<td>2.32</td>
<td>1.41–3.51</td>
</tr>
<tr>
<td>Model 2</td>
<td>1.82</td>
<td>1.16–2.79</td>
</tr>
</tbody>
</table>

The hypertriglyceridemic waist phenotype was defined as having both a high waist circumference (≥90 cm in men and ≥85 cm in women) and increased fasting triglyceride levels (≥2.0 mmol/L in men and ≥1.5 mmol/L in women).

Model 1: adjusted for age and sex.
Model 2: adjusted for age, sex, smoking habits, total annual family income, alcohol consumption, coffee intake, daily caloric intake, and cardiorespiratory fitness.
Abbreviations: RR, relative risk; CI, confidence interval.

Discussion

Overall, we observed that sleeping less than 6 h per night was associated with an increased risk of developing features of the metabolic syndrome in this sample of adults from the Quebec Family Study. In contrast, long sleep duration was not associated with a higher incidence of metabolic syndrome or hypertriglyceridemic waist phenotype compared to sleeping between 7 and 8 h a day. Our results strongly suggest that short sleeping habits should be further investigated as a lifestyle factor that may predispose individuals to cardiometabolic disease risk.

The results of the present study agree with those recently published by Choi et al. (2011) showing that only short sleep duration (less than 6 h per day) was a significant risk factor for the development of the metabolic syndrome in midlife women. However, the present study had a longer follow-up period (6 years vs. 2–4 years), included important covariates that were not assessed in the Choi et al. study (e.g. daily caloric intake, cardiorespiratory fitness, socioeconomic status), and we added the concept of hypertriglyceridemic waist phenotype. Furthermore, Choi et al. (2011) relied on a different ethnic group (Korean cohort) in which different waist circumference cut-offs are recommended.

Our findings are also consistent with the few cross-sectional studies published on the association between sleep duration and the metabolic syndrome. While short sleep duration has been shown to be associated with the presence of metabolic syndrome in some studies (Kobayashi et al., 2011, Wu et al., 2012), both short and long sleep durations were related to an increased risk of the metabolic syndrome and its components in other studies (Choi et al., 2008, Hall et al., 2008). Conversely, Arora et al. (2011) observed that long sleep duration was associated with a greater risk of metabolic syndrome in older Chinese. Although the latter study is not in line with the evidence published to date on this topic, the authors of the study speculated that the difference could perhaps be explained by an attenuation of the sleep–metabolic syndrome relationship with age or possibly the fact that long sleep may be a consequence of ill health.

There are a number of biological mechanisms through which reduced sleep durations may lead to the metabolic syndrome. Experimental sleep restriction has been shown to decrease glucose
tolerance and increase insulin resistance, sympathetic nervous system activity, blood pressure and blood cortisol concentrations (Cappuccio et al., 2011, Knutson, 2010, Leproult and Van Cauter, 2010, Spiegel et al., 2009). Low-grade inflammation has also been shown to increase with short sleep and could possibly impact cardiometabolic risk (Miller and Cappuccio, 2007). Moreover, lack of sleep is generally associated with an increase in food intake and can contribute to weight gain and obesity (Chaput and Tremblay, 2012). However, future studies will be needed to elucidate the pathways between insufficient sleep and cardiometabolic risk, and to rule out reverse causation.

Accumulating evidence suggests that a sleep duration of 7–8 h per night in adults is associated with the maintenance of good health (National Sleep Foundation, 2002). Both short sleep duration and poor sleep quality are pervasive in modern societies and many individuals still believe that sleep is a waste of time (Matricciani et al., 2012, National Sleep Foundation, 2002). Insufficient sleep adversely affects physiological and psychological health and impacts certain behaviors of individuals (e.g. eating habits, physical activity participation, alcohol consumption) so that the maintenance of overall health is potentially compromised by insufficient sleep (Chaput and Tremblay, 2012, McNeil et al., 2013). The fact that short sleep duration was still associated with the development of the metabolic syndrome or the hypertriglyceridemic waist after adjusting for many possible confounding factors suggests that the relationship was independent of these covariates.

Strengths and limitations of the study

Strengths of this study include its longitudinal design and the adjustment for many important variables that could confound the relationship between short sleep duration and the incidence of metabolic syndrome (e.g. caloric intake, cardiorespiratory fitness and socioeconomic status). Furthermore, data were obtained from both men and women and we used an approach that should minimize confounding with repeated measures of sleep duration and covariates. We also added the hypertriglyceridemic waist phenotype to provide another simple screening tool of clustering metabolic abnormalities. However, our results need to be interpreted in light of the following limitations. First, the relatively small sample size limits statistical power and more high quality and well powered studies are needed to better appreciate the influence of sleeping habits on features of the metabolic syndrome. Second, although good agreement has been found in previous studies between self-reported sleep durations and those obtained through actigraphic monitoring (Hauri and Wisbey, 1992, Lockley et al., 1999), the single question approach does not provide information on sleep quality. Third, the incidence rate of metabolic syndrome or hypertriglyceridemic waist phenotype was low over the 6-year follow-up period and could impact the results reported in the present study. Fourth, the external generalizability of our findings may be restricted to adults of Western European descent. Finally, the possibility of residual confounding by unmeasured variables is always a possibility in observational studies (e.g. depression, obstructive sleep apnea, insomnia, medications).

Conclusion

In summary, the present study provides evidence that short sleep duration is associated with an increased risk of developing features of the metabolic syndrome in adults. Replication studies are
warranted and future studies should examine pathways by which reduced sleep duration affects the development of metabolic syndrome.

Conflict of interest statement

The authors declare that there are no conflicts of interest.

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

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