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Exploring the mediating role of energy balance-related behaviours in the association between sleep duration and obesity in European adults. The SPOTLIGHT project

Myrthe Timmermans a, Joreintje D. Mackenbach a,*, Helene Charriere b,c, Helga Bárdos d, Sofie Compernolle e, Ilse De Bourdeaudhuij e, Jean-Michel Oppert b,f, Harry Rutter g, Martin McKee g, Jeroen Lakerveld a

a Department of Epidemiology and Biostatistics, Amsterdam Public Health Research Institute, VU University Medical Center, Amsterdam, The Netherlands
b Equipe de Recherche en Épidémiologie Nutritionnelle (EREN), Centre de Recherche en Épidémiologie et Statistiques, Inserm (U1153), Inra (U1125), ComUE Sorbonne Paris Cité, Université Paris 13, Bobigny, France
c Sorbonne Universités, Université Pierre et Marie Curie, Université Paris 06; Institute of Cardiometabolism and Nutrition, Department of Nutrition, Pitié-Salpêtrière Hospital, Assistance Publique-Hôpitaux de Paris, Paris, France
d Department of Preventive Medicine, Faculty of Public Health, University of Debrecen, Hungary
e Department of Movement and Sport Sciences, Faculty of Medicine and Health Sciences, Ghent University, Ghent, Belgium
f Equipe de Recherche en Epidémiologie Nutritionnelle (EREN), Centre de Recherche en Epidémiologie et Statistiques, Inserm (U1153), Inra (U1125), ComUE Sorbonne Paris Cité, Université Paris 13, Bobigny, France
g European Centre on Health of Societies in Transition, London School of Hygiene and Tropical Medicine, London, UK

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ABSTRACT

Sleep restriction is a risk factor for weight gain and obesity. Few studies have formally investigated the mediating role of energy balance-related behaviours in the sleep–obesity association. The aim of this study was to explore the mediating role of physical activity, sedentary behaviours and dietary habits in the association of sleep duration with obesity in adults in five European urban regions. Data on self-reported sleep duration, energy balance-related behaviours, height and weight and other covariates were collected between February and September 2014 from participants to the SPOTLIGHT survey (N = 5900, mean age 52 years). Participants were recruited from 60 urban neighbourhoods in Belgium, France, Hungary, the Netherlands and the United Kingdom. Multilevel logistic regression analyses were used to assess the associations of sleep duration, energy balance-related behaviours and obesity and mediating effects were calculated using MacKinnon’s product-of-coefficients method. Results indicated that a 1 h increase in sleeping time was associated with a 14% lower likelihood of being obese (OR = 0.86, 95% CI = 0.80; 0.93). Only work-related sedentary behaviour was identified as a statistically significant mediator in the association between sleep duration and obesity for the total sample, and youngest and oldest age group. We did not find evidence for a mediating role of dietary habits and physical activities.

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1. Introduction

The increased prevalence of obesity in recent decades has been accompanied by a decrease in sleep duration (Van et al., 2008; Akerstedt and Nilsson, 2003) with growing evidence linking the two (Gildner et al., 2014; Patel and Hu, 2008; Xiao et al., 2013; Buxton and Marcelli, 2010; Chaput, 2014; Chaput et al., 2007; Kobayashi et al., 2012). A prospective study among adults showed that each hour increase in total sleep reduced the incidence of obesity by 30% (Bo et al., 2011). The underlying mechanisms involved remain unclear. Patel & Hu’s conceptual model (Patel and Hu, 2008) suggests that shorter sleep duration may increase hunger via changes in energy regulatory hormones such as leptin and ghrelin (Magee et al., 2008; Magee and Hale, 2012), longer time awake may offer more time and opportunities to eat (Patel and Hu, 2008; Chaput, 2014; Patterson et al., 2014), a lower core body temperature related to sleep deprivation may lower total energy expenditure and increase food intake (Patel and Hu, 2008; Magee and Hale, 2012; Atkinson and Davenne, 2007), and increased fatigue may decrease energy expenditure (Patel and Hu, 2008; Magee and Hale, 2012). These four pathways may further lead to increased caloric intake and reduced energy expenditure, resulting in obesity.

However, formal evidence for such mediating pathways is lacking. Two observational studies concluded that, among adults, dietary patterns could only partially explain the effect of short sleep duration on the incidence of obesity (Nishiura et al., 2010) and that, among adolescents, the association between short sleep duration and obesity was explained by increased sedentary time and unhealthier dietary habits (Garaulet et al., 2011). This suggests that part of the sleep–obesity association can be explained by energy balance-related behaviours.
However, as the study of Nishiura et al. was limited to men aged 40–59 years and only examined dietary habits, and the study by Garauet et al. was conducted in adolescents, the role of energy balance-related behaviours in the sleep–obesity association in adults is still unknown. Given the considerable costs and efforts associated with conducting experimental studies, cross-sectional analyses of mediating pathways may provide a valuable insight in the sleep obesity association.

Based on the conceptual framework of Patel and Hu, we used a large sample of adults residing in five urban regions across Europe to examine the mediating roles of physical activity, sedentary behaviours and dietary habits in the association of sleep duration with obesity.

2. Methods

This study was part of the SPOTLIGHT project (Lakerveld et al., 2012), conducted in five urban regions across Europe: Ghent and suburbs (Belgium), Paris and inner suburbs (France), Budapest and suburbs (Hungary), the Randstad (a conurbation including the cities Amsterdam, Rotterdam, the Hague and Utrecht in the Netherlands) and Greater London (United Kingdom). Sampling of neighbourhoods and recruitment of participants has been described in detail elsewhere (Lakerveld et al., 2015). Briefly, neighbourhood sampling employed a combination of neighbourhood residential density and socioeconomic status (SES). A random sample of adult inhabitants (age range 18+) from each of the 60 randomly selected neighbourhoods was invited to participate in an online survey. A total of 6037 (10.8%, out of 55,893) individuals participated in the study between February and September 2014.

Participants reported on demographics, neighbourhood perceptions, social environmental factors, health, motivations and barriers for healthy behaviour, energy balance-related behaviours and weight and height. The study was approved by the corresponding local ethics committees of participating countries and all participants provided informed consent.

2.1. Measures

2.1.1. Obesity

Body mass index (BMI) was calculated as self-reported body weight (in kilograms) divided by height (in meters) squared. The variable was dichotomized into obesity (BMI ≥ 30 kg/m²) and no obesity (BMI < 30 kg/m²) consistent with World Health Organization criteria (Hirshkowitz et al., 2015).

2.1.2. Sleep duration

Participants provided information on sleep duration during an average night, with possible responses ranging from 4 to 16 h, in half-hour intervals. Sleep duration was used as a continuous variable. As we wanted to explore whether both short and long sleep duration were associated with higher odds of obesity, we also divided sleep duration into short (<7 h; N = 1686), normal (7 < 9 h; N = 3469) and long sleep duration (≥ 9 h; N = 330) on the basis of common sleep guidelines (Hirshkowitz et al., 2015).

2.1.3. Dietary habits

Dietary habits were assessed by short common food frequency questions that have been linked to BMI in a previous study within the SPOTLIGHT project (Compernolle et al., 2016). Questions were asked about consumption of: fruits, vegetables, fish, sugar sweetened beverages, sweets and fast food. Response options ranged from ‘once a week or less’ to ‘more than twice a day’. Furthermore, participants reported on the number of times per week they ate breakfast and cooked a meal at home using bought ingredients, rather than eating ready or takeaway meals. The food consumption questions used in the SPOTLIGHT survey served as a proxy for healthy and unhealthy dietary habits and all questions asked were used in this study. Variables were not normally distributed and as transformations did not improve the distribution of residuals, variables were dichotomized using the median (fruit consumption ≥ 7 times a week, vegetable consumption ≥ 7, fast food consumption ≥ 2, fish consumption ≥ 2, sweets consumption ≥ 3, sugar sweetened beverage consumption ≥ 2, breakfast consumption ≥ 6, cooking with ingredients ≥ 5). All dietary variables were tested as mediator variables.

2.1.4. Physical activity

Questions about leisure time physical activity and transport-related physical activity were adapted from the validated long version (last seven days) International Physical Activity Questionnaire (IPAQ) (Craig et al., 2003). A previous 12-country study showed good reliability (intra-class correlations range from 0.46 to 0.96) and acceptable criterion validity (median ρ = 0.30). Minutes of leisure time- and transport-related physical activity per day were calculated according to IPAQ guidelines and used as mediator variables. Given non-normal distribution of the variables, they were dichotomized based on the median (≥25 min leisure time- and ≥25 min transport-related physical activity).

2.1.5. Sedentary behaviours

Sedentary behaviours were measured using questions from the Marshall questionnaire (Marshall et al., 2010). Domain-specific sedentary time was estimated by asking the average time spent sedentary while travelling, working, watching television, using a computer or tablet and during other leisure time activities on both weekdays and weekend days during the last seven days. Due to non-normal distribution, the variables were dichotomized based on the median (work related ≥ 275 min of sitting per day, transport related ≥ 60, watching television ≥ 138, using a computer ≥ 78 and leisure related ≥ 65) and used as mediator variables.

2.1.6. Socio-demographic and health information

Participants further reported age, gender, level of education (higher (either a college or university degree) and lower (all lower educational levels)), self-rated health (using a Visual Analogue Scale ranging from 0 (very unhealthy) to 100 (very healthy)), work type (sitting or standing occupation, manual work, heavy manual work), alcohol consumption (the number of glasses per week a respondent drank an alcoholic beverage), smoking (yes, no, but I have previously been a regular smoker and no, I have never been a regular smoker), longstanding illness, disability or infirmity (yes or no), screen time (amount of hours watching television and using a computer at home in week days and weekends) and work hours (0–10, 11–20, 21–30, 31–40, 41–50 and more than 50).

2.2. Statistical analyses

We used Kruskal-Wallis ANOVAs and Chi-Squared tests to examine differences between short (<7 h/night), normal (7 < 9 h/night) and long sleepers (≥9 h/night), and report p-values in Table 1.

Multilevel logistic regression was used to test associations between sleep duration, dietary habits, physical activity, sedentary behaviours and obesity with individuals as the first level, and neighbourhoods as the second level. Age, gender and education were a priori added to the model as potential confounding factors. Work type, alcohol consumption, smoking, self-rated health, longstanding illness, disability or infirmity, screen time and work hours were tested as potential additional confounders. Only self-rated health was found to be a confounder on the basis of a ≥ 10% change in the coefficient of sleep after adding self-rated health to the model. Age, gender, education and country were tested as effect modifiers in the main analyses (c-path). Age, divided into 18–40 years, 41–65 years and >65 years old, significantly interacted with sleep duration, so analyses stratified by age bands are also presented in the tables.

Mediating roles of dietary habits, physical activity and sedentary behaviours were examined using Mackinnon’s product-of-coefficients
method (MacKinnon et al., 2007). We first assessed the association between the independent variable sleep duration and the dependent variable obesity (c-path). Second, we assessed the association between the independent variable sleep duration and the potential mediators ‘dietary habits’ (a1-path, consisting of 8 different variables), ‘physical activity’ (a2-path, consisting of 2 different variables) and ‘sedentary behaviours’ (a3-path). Then, we assessed associations between potential mediators (dietary activity, physical activity and sedentary behaviours) and the dependent variable obesity, adjusted for sleep duration (b-path). Lastly, we assessed the association between the independent variable sleep duration and the dependent variable obesity, adjusted for each mediator (c’-path). Mediators were added separately, resulting in eleven different single mediation models. An overview of the analyses is in Fig. 1.

MacKinnon’s product-of-coefficients (ab) was calculated, representing single mediation effects (MacKinnon et al., 2007). Given the possibility of multiple opposing mechanisms whose influences are of opposite direction and cancel out the main effect (Cerin and MacKinnon, 2009), a statistically significant main effect is no requirement for this method. We therefore continued mediation analyses even when no association between the independent and outcome variable was found. Odds ratios were standardized and the Sobel test was used to test significance of the mediated effect (p < 0.05) by dividing ab (multiplied standardized odds ratios of the a- and b-paths) by its standard error $SE_{ab} = \sqrt{(a^2 \times SE_{b2}) + (b^2 \times SE_{a2})}$ (MacKinnon et al., 2007). We used MacKinnon’s method to calculate the proportion mediated (ab/(ab + c′)) (MacKinnon et al., 2007).

A total of 137 individuals had missing data on the neighbourhood identifier and were thus excluded for further analysis (final N = 5900). Assuming that data were missing at random, missing values for all individual variables used (ranging from <1% to 27%) were imputed using Predictive Mean Matching in SPSS version 22.0. All variables described in the methods section were used in the model with 20 imputed datasets, and country- and neighbourhood identifiers were used as auxiliary variables.

As a sensitivity analysis, complete case analysis was conducted on the original dataset; results are shown in supplementary tables S1a, S1b1, S1b2 and S1c. Additionally, we conducted a sensitivity analysis with sleep categorized into short, normal and long sleep duration (Hirshkowitz et al., 2015). Results of the a- and c-paths are shown in supplementary tables S2a1, S2a2 and S2b. All multilevel logistic regression analyses were conducted using STATA version 12.1. We also performed linear regression analyses using a continuous BMI variable as dependent variable; this resulted in similar results as the main analyses (results not shown).

### 3. Results

Characteristics of the study participants according to sleep categories are shown in Table 1. A total of 9000 individuals with a mean age of 52 years (44% male) were included in the analyses.

Table 2 shows the results of the association between sleep duration and obesity (c-path). One hour increase in total sleeping time was associated with a 14% decreased odds of obesity (OR = 0.86, 95%CI = 0.80; 0.93). In analyses stratified by age, sleep duration was negatively associated with obesity in the youngest and middle age groups (OR = 0.70, 95%CI = 0.85; 0.86 and OR = 0.88, 95%CI = 0.80; 0.98, respectively). No significant associations were found in the oldest group (OR = 0.93, 95%CI = 0.82; 1.05). (See Tables 3a1 and 3a2)
The a-path of the total sample (Tables 3a1 and 3a2) indicated that sleeping 1 h longer was associated with a 17% increase in breakfast consumption (OR = 1.17, 95% CI = 1.10; 1.25). Sleeping longer was also associated with lower ORs for transport- and work related sedentary behaviour (OR = 0.93, 95% CI = 0.87; 0.98, OR = 0.93, 95% CI = 0.92; 0.95 respectively). For the youngest age group, sleeping longer was associated with a 18% higher breakfast consumption (OR = 1.18, 95% CI = 1.05; 1.33) and a lower consumption of sweetened beverages (OR = 0.89, 95% CI = 0.80; 0.99). In this age group we also found a negative association between sleep duration and work related sedentary behaviour (OR = 0.97, 95% CI = 0.94; 0.99) For the middle age group we found a significant positive association between sleep duration and breakfast consumption (OR = 1.20, 95% CI = 1.09; 1.33) and a significant negative association between sleep duration and transport- and work related sedentary behaviour (OR = 0.91, 95% CI = 0.84; 0.99, OR = 0.92, 95% CI = 0.90; 0.94, respectively). For the oldest age group we found that longer sleep duration was associated with lower fruit consumption (OR = 0.89, 95% CI = 0.79; 0.99) and lower work-related sedentary behaviour (OR = 0.50, 95% CI = 0.39; 0.64).

Table 3b shows the association between physical activity, sedentary behaviours and dietary habits with obesity. In the total sample there was a negative association between consumption of fish and sweets with obesity (OR = 0.64, 95% CI = 0.47; 0.89 and OR = 0.80, 95% CI = 0.67; 0.96 respectively). Also leisure time physical activity and transport related physical activity (OR = 0.79, 95% CI = 0.66; 0.93 and OR = 0.73, 95% CI = 0.62; 0.87) was negatively associated, and work-, TV-watching and computer related sedentary behaviours (OR = 1.10, 95% CI = 1.04; 1.16, OR = 1.75, 95% CI = 1.43; 2.13, OR = 1.26, 95% CI = 1.04; 1.54, respectively) were positively associated with obesity. Work-related sedentary behaviour was positively associated with obesity in the youngest age group (OR = 1.46, 95% CI = 1.31; 1.62). For the middle age group, leisure time physical activity was negatively associated with obesity (OR = 0.77, 95% CI = 0.61; 0.97) and TV-related sedentary behaviour was positively associated with obesity (OR = 1.85, 95% CI = 1.42; 2.42). Fish consumption and transport-related physical activity were both negatively associated with obesity in the oldest age group (OR = 0.44, 95% CI = 0.24; 0.80 and OR = 0.62, 95% CI = 0.44; 0.86 respectively). TV-watching and computer/tablet-related sedentary behaviour were positively associated with obesity (OR = 1.56, 95% CI = 1.05; 2.31, OR = 1.51, 95% CI = 1.02; 2.24, respectively) and work-related sedentary behaviour was negatively associated with obesity (OR = 0.20, 95% CI = 0.11; 0.36).

Table 4 shows the results for the formal test of the mediating effects of dietary habits, sedentary behaviours and physical activity in the association between sleep duration and obesity. The results from the Sobel test did not reveal a statistically significant mediating effect of dietary habits (Sobel Z-scores ranging from −0.94 to 1.05) or of physical activity (Sobel Z-scores ranging from −1.39 to 1.01) in either the total sample or any of the age groups. In analyses conducted in employed participants only, we did find evidence for a statistically significant
mediating effect of work-related sedentary behaviours in the association between sleep and obesity for the total, youngest and oldest age group (Z-score = −3.05, −2.75, 3.88, respectively).

Sensitivity analyses with sleep divided in short, normal and long sleep duration are presented in Tables S2a and S2b. We found that short sleepers had higher odds of being obese than normal sleepers (OR = 1.47, 95% CI = 1.23; 1.75). However, after the categorization of sleep duration into three groups, no significant mediation for work-related sedentary behaviour was found.

### 4. Discussion

In the present study, we used a cross-sectional design to explore the mediating role energy balance-related behaviours in the association between sleep duration and obesity in adults. We found that sleep duration was associated with obesity, with 1 hour increase in total sleeping time associated with a 14% lower odds of being obese. Stratification by age group showed that this association was strongest in younger and middle aged adults. These findings are consistent with previous observational and experimental studies showing a link between sleep duration and obesity (Gildner et al., 2014; Patel and Hu, 2008; Buxton and Marcelli, 2010; Nishiura et al., 2010; Anic et al., 2010; Spaeth et al., 2013; Capers et al., 2015; Shechter et al., 2012). We found work-related sedentary behaviours to be a significant mediator in the association between sleep and obesity for the total, youngest and oldest age group.

For leisure time- and transport-related physical activity we found significant negative associations between physical activity and obesity in the total sample, middle and oldest age group, as found in other studies (Stamatakis et al., 2009; Pate et al., 2015). We found no association between sleep duration and physical activity in any of the age groups, corresponding to the findings of a systematic review (Shlisky et al., 2012). It was found that the relation between sleep duration and physical activity varies between individuals (Klingenberg et al., 2012), that is, some people show lower levels of physical activity after sleep restriction, some higher levels and some did not change at all (Klingenberg et al., 2012). As such, physical activity may not be the main underlying mechanism in the sleep-obesity association.

It may be that energy intake, rather than energy expenditure, is the key mechanism underlying the sleep obesity association (Shlisky et al., 2012; Bel et al., 2013; Chapman et al., 2012). However, in our study we did not find evidence to support this hypothesis. A possible explanation could be the use of relatively simple questions with regard to dietary habits, which do not provide information on caloric intake. Also, timing and quantity of food intake may play a substantial role in obesity. Irregular food intake and larger portion size can disrupt the circadian system, which may lead to weight gain (Garaulet et al., 2013; Berg and Forslund, 2015). We found unexpected results for the relation between sweets consumption and obesity, such that an increased frequency of sweets consumption was associated with decreased odds of obesity. Interestingly, this in line with the study by Just & Wansink (Just and Wansink, 2015). They advise that frequency of intake of unhealthy food products may not be a strong indicator of healthy weight.

In the analyses with work-related sedentary behaviour as mediator – performed in a subsample of employed participants only – we found shorter sleep duration to be associated with increased work-related sedentary behaviour in the total sample and oldest age group. Moreover, increased work-related sedentary behaviour was associated with higher odds of obesity in the total sample and youngest employed participants, whereas in the oldest employed group increased work-related sedentary behaviour was associated with lower odds of obesity. As the 70 participants aged 65+ and still employed were more active and experienced less illness (data not shown), it may be that increased work-related sedentary behaviour actually represents a healthy state instead of a health-risk in this oldest group. The mediation effect of the oldest employed group was a suppressor, which means that the magnitude of the total effect increases when the mediator is included.

| Table 3a1 Associations between the independent variable sleep duration and each potential mediator of dietary habits and physical activity (a-path) N = 5900. |
| Table 3a1 Associations between the independent variable sleep duration and each potential mediator of dietary habits and physical activity (a-path) N = 5900. |

<table>
<thead>
<tr>
<th>Dietary habits</th>
<th>Physical activity</th>
<th>Total</th>
<th>Youngest age</th>
<th>Middle age group</th>
<th>Oldest age group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fish consumption (≥ 7 times a week)</td>
<td>1.02 (0.89;1.17)</td>
<td>0.93 (0.79;1.10)</td>
<td>0.98 (0.81;1.18)</td>
<td>1.14 (0.94;1.39)</td>
<td></td>
</tr>
<tr>
<td>Fruit consumption (≥ 7 times a week)</td>
<td>0.89 (0.79;0.99)</td>
<td>0.95 (0.78;1.14)</td>
<td>0.92 (0.80;1.05)</td>
<td>1.05 (0.88;1.26)</td>
<td></td>
</tr>
<tr>
<td>Vegetable consumption (≥ 7 times a week)</td>
<td>0.89 (0.79;0.99)</td>
<td>0.95 (0.78;1.14)</td>
<td>0.92 (0.80;1.05)</td>
<td>1.05 (0.88;1.26)</td>
<td></td>
</tr>
<tr>
<td>Home cooked meals (≥ 5 times a week)</td>
<td>1.09 (1.05;1.33)</td>
<td>1.09 (1.05;1.33)</td>
<td>1.09 (1.05;1.33)</td>
<td>1.09 (1.05;1.33)</td>
<td></td>
</tr>
<tr>
<td>Sweetened beverages consumption (≥ 2 times a week)</td>
<td>1.09 (0.98;1.20)</td>
<td>1.05 (0.92;1.19)</td>
<td>1.02 (0.90;1.16)</td>
<td>1.06 (0.92;1.23)</td>
<td></td>
</tr>
<tr>
<td>Breakfast consumption (6 times a week)</td>
<td>1.09 (0.98;1.20)</td>
<td>1.05 (0.92;1.19)</td>
<td>1.02 (0.90;1.16)</td>
<td>1.06 (0.92;1.23)</td>
<td></td>
</tr>
<tr>
<td>Fast food consumption (≥ 2 times a week)</td>
<td>1.09 (0.98;1.20)</td>
<td>1.05 (0.92;1.19)</td>
<td>1.02 (0.90;1.16)</td>
<td>1.06 (0.92;1.23)</td>
<td></td>
</tr>
<tr>
<td>Transport-related physical activity (≥ 25 min/day)</td>
<td>1.09 (0.98;1.20)</td>
<td>1.05 (0.92;1.19)</td>
<td>1.02 (0.90;1.16)</td>
<td>1.06 (0.92;1.23)</td>
<td></td>
</tr>
<tr>
<td>Leisure time physical activity (≥ 25 min/day)</td>
<td>1.09 (0.98;1.20)</td>
<td>1.05 (0.92;1.19)</td>
<td>1.02 (0.90;1.16)</td>
<td>1.06 (0.92;1.23)</td>
<td></td>
</tr>
</tbody>
</table>

Data collected between February and September 2014. Bold values represent significant (p < 0.05) associations. OR = Odds ratio CI = Confidence interval. Models are adjusted for age, education, gender and self-rated health.


Marcelli, 2010; Nishiura et al., 2010; Anic et al., 2010; Spaeth et al., 2013; Gildner et al., 2014; Patel and Hu, 2008; Buxton and Marcelli, 2010; Nishiura et al., 2010; Anic et al., 2010; Spaeth et al., 2013; Capers et al., 2015; Shechter et al., 2012. Further studies are needed to investigate the underlying mechanisms of the association between sleep and obesity.
Data collected between February and September 2014. Bold values represent significant (p < 0.05) associations. OR = Odds ratio, CI = Confidence interval. Models are adjusted for age, education, gender and self-rated health.

(Mackinnon et al., 2007). This result may be explained by the negative association between work-related sedentary behaviour and obesity (b-path). Due to the small group size we were unable to calculate the proportion of the mediated effect (Mackinnon et al., 2007).

We also found a mediating effect of work-related sedentary behaviour in the total and youngest employed group, with small proportions of the association mediated (0.004% and 3.1%, respectively). This indicates that the association between sleep duration and obesity could be partly explained by work-related sedentary behaviour, in correspondence with the model described by Patel and Hu, in which less sleep causes more fatigue, resulting in more sedentary behaviour. That we only found mediation effects for work-related sedentary behaviour may be because this sedentary domain is the biggest contributor of total sedentary behaviour, making it easier to detect effects.

4.1. Strengths & limitations

This study has several limitations that should be addressed. First, self-reported sleep duration, measured with one item, does not provide information about sleep quality and often overestimates true sleep duration (Silva et al., 2007). Second, much more detailed data may be necessary to provide information on total caloric intake, or total energy expenditure. We used self-reported questionnaires, which are known to imperfectly capture both exposure and outcome measures (Coughlin, 1990). In combination with the dichotomization of the mediators, our variables may have been somewhat crude. To reduce the participant burden of completing an already lengthy questionnaire, we chose to use relatively simple questions with regard to dietary habits. However, these questions have not been validated. In addition, our shortened version of the internationally validated IPAQ (Craig et al., 2003), only provided information on two domains of physical activity. Third, additional factors potentially confounding the sleep - obesity association not measured in the present study are the presence of sleep disorders, depression, diseases like hypertension and diabetes mellitus and the use of medication. Lastly, the cross sectional design precludes causal inference. In addition, a cross-sectional study design may not be the most optimal design for the identification of mediating effects. Longitudinal analyses would allow for the identification of temporal effects and thus changes within as well as between individuals over time. However, as sleep duration and energy balance-related behaviours may change repeatedly over time, this may obscure predictive and mediation effects in longitudinal data. Disentangling the mediating effects of energy

### Table 3a2

<table>
<thead>
<tr>
<th>Sedentary behaviours</th>
<th>Total sample OR (95% CI)</th>
<th>Age group 18–40 OR (95% CI)</th>
<th>Age group 41–65 OR (95% CI)</th>
<th>Age group 66–109 OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transport-related sedentary behaviours (≥60 min/day)</td>
<td>0.93 (0.87; 0.98)</td>
<td>0.91 (0.92; 0.95)</td>
<td>1.01 (0.96; 1.07)</td>
<td>1.02 (0.96; 1.08)</td>
</tr>
<tr>
<td>Work-related sedentary behaviours (≥279 min/day)</td>
<td>0.91 (0.89; 0.95)</td>
<td>0.97 (0.94; 0.99)</td>
<td>1.00 (0.90; 1.12)</td>
<td>1.00 (0.98; 1.11)</td>
</tr>
<tr>
<td>Leisure-related sedentary behaviours (≥65 min/day)</td>
<td>0.93 (0.89; 0.97)</td>
<td>0.99 (0.92; 1.07)</td>
<td>1.00 (0.90; 1.12)</td>
<td>0.97 (0.87; 1.09)</td>
</tr>
<tr>
<td>TV-related sedentary behaviours (≥138 min/day)</td>
<td>0.95 (0.89; 1.00)</td>
<td>0.95 (0.89; 1.00)</td>
<td>1.03 (0.96; 1.11)</td>
<td>1.01 (0.94; 1.08)</td>
</tr>
<tr>
<td>Computer/tablet-related sedentary behaviours (≥78 min/day)</td>
<td>0.95 (0.89; 1.00)</td>
<td>0.95 (0.89; 1.00)</td>
<td>1.03 (0.96; 1.11)</td>
<td>1.01 (0.94; 1.08)</td>
</tr>
</tbody>
</table>

Data collected between February and September 2014. Bold values represent significant (p < 0.05) associations. OR = odds ratio, CI = Confidence interval. Models are adjusted for age, education, gender, self-rated health.

* Analyses are done with only those who indicated to be employed. Total: N = 3240, youngest age group: N = 1252, middle age group: N = 1918, oldest age group: N = 70.
Table 4

<table>
<thead>
<tr>
<th>Dietary habits</th>
<th>Total</th>
<th>Age group 18–40</th>
<th>Age group 41–65</th>
<th>Age group 66–109</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fruit consumption</td>
<td>0.86</td>
<td>−0.001</td>
<td>0.21 (0.83)</td>
<td>0.70 &lt; 0.001</td>
</tr>
<tr>
<td>Vegetable consumption</td>
<td>0.86</td>
<td>−0.001</td>
<td>−0.13 (0.90)</td>
<td>0.70 &lt; 0.001</td>
</tr>
<tr>
<td>Fish consumption</td>
<td>0.86</td>
<td>0.002</td>
<td>−0.11 (0.91)</td>
<td>0.70 &lt; 0.001</td>
</tr>
<tr>
<td>Home-cooked meals</td>
<td>0.86</td>
<td>−0.001</td>
<td>−0.47 (0.46)</td>
<td>0.70 &lt; 0.001</td>
</tr>
<tr>
<td>Breakfast consumption</td>
<td>0.86</td>
<td>−0.001</td>
<td>0.30 (0.76)</td>
<td>0.70 &lt; 0.001</td>
</tr>
<tr>
<td>Sweetened beverages</td>
<td>0.86</td>
<td>−0.001</td>
<td>−0.74 (0.46)</td>
<td>0.71 &lt; 0.005</td>
</tr>
<tr>
<td>Sweets consumption</td>
<td>0.86</td>
<td>−0.001</td>
<td>0.94 (0.35)</td>
<td>0.70 &lt; 0.001</td>
</tr>
<tr>
<td>Fast food consumption</td>
<td>0.86</td>
<td>−0.001</td>
<td>−0.21 (0.83)</td>
<td>0.70 &lt; 0.001</td>
</tr>
<tr>
<td>Leisure time physical activity</td>
<td>0.86</td>
<td>−0.001</td>
<td>−0.02 (0.99)</td>
<td>0.70 &lt; 0.001</td>
</tr>
<tr>
<td>Transport-related physical activity</td>
<td>0.87</td>
<td>−0.002</td>
<td>−0.12 (0.90)</td>
<td>0.70 &lt; 0.001</td>
</tr>
<tr>
<td>Sedentary behaviours</td>
<td>0.86</td>
<td>0.001</td>
<td>−1.28 (0.20)</td>
<td>0.70 &lt; 0.004</td>
</tr>
<tr>
<td>Work-related sedentary behaviour</td>
<td>0.76</td>
<td>−0.001</td>
<td>−3.05 (&lt; 0.01)</td>
<td>0.63 0.02</td>
</tr>
<tr>
<td>Leisure-related sedentary behaviour</td>
<td>0.86</td>
<td>0.003</td>
<td>−0.40 (0.69)</td>
<td>0.70 &lt; 0.004</td>
</tr>
<tr>
<td>TV-related sedentary behaviours</td>
<td>0.86</td>
<td>0.006</td>
<td>0.58 (0.56)</td>
<td>0.70 0.004</td>
</tr>
<tr>
<td>Computer/tablet-related sedentary behaviours</td>
<td>0.86</td>
<td>0.004</td>
<td>−1.44 (0.15)</td>
<td>0.70 0.004</td>
</tr>
</tbody>
</table>

Data collected between February and September 2014. Bold values indicate significant (p < 0.05) associations. Models are adjusted for education, gender and self-rated health. c’ = Odds Ratio of the c’ path (association between the predictor and the outcome, adjusted for the mediator).

a Analyses are done with only those who indicated to be employed. Total: N = 3240, youngest age group: N = 1252, middle age group: N = 1918, oldest age group: N = 70.

Table 4 Mediating effect of dietary habits, sedentary behaviours and physical activity in the association between sleep duration (hours/night) and obesity (N = 5900).

balance-related behaviours in the sleep-obesity association may thus require intervention studies that induce sleep changes in adults.

Our study also has a number of strengths. We conducted an extensive survey that allowed the (albeit crude) measurement of various energy balance-related behaviours. The fact that our study was conducted across five different European countries, and the fact that we did not find moderation by country, strengthens the existing evidence for the sleep - obesity association in adults. We used a relatively large sample different European countries, and the fact that we did not find moderation by country, strengthens the existing evidence for the sleep - obesity association in adults. We used a relatively large sample

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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.ypmed.2017.03.021.

References


Competing interests

The authors declare that they have no competing interest. Funders had no role in the study design; collection, analysis and interpretation of data; the writing of the manuscript; or the decision to submit the manuscript for publication.

Author contributions

MT, JDM and JL were involved in the conception of the study, the analysis and data interpretation. MT drafted the first version of the manuscript. JDM, HC, HB, SC, IDB, JMO HR, MM and JL were involved in the data acquisition, data interpretation and critically revising the manuscript, together with MT. All authors read and approved the final manuscript. The data manager of the SPOTLIGHT project (JL) can be contacted for access to the dataset underlying the current analysis.

Transparency Document

The Transparency document associated with this article can be found, in online version.

5. Conclusion

In this study we explored the mediating role of energy balance-related behaviours in the association between sleep duration and obesity in European adults. The data confirm a negative association between sleep duration and the odds of obesity and work-related sedentary behaviours explained a small proportion of this association.

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.ypmed.2017.03.021.

References


