ORIGINAL ARTICLE

SHORT SLEEP DURATION IN RELATION TO BODY WEIGHT AND BLOOD PRESSURE AMONG CZECH ADULTS

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Abstract

Study objectives: The aim of this study was to investigate possible associations between obesity related anthropometric determinants and blood pressure measurements according to the self-recorded number of daily hours of sleep within the adult Czech population.

Methods: A total of 478 women and men aged 18–65 years were included between the years 2006–2010. The research methodology consisted of anthropometric measurements and bioelectrical impedance for the classification of body parameters, and the measurement of blood pressure (BP). Data on sleep duration were obtained using 24-hour time diaries in which the total hours of sleep per day for seven consecutive days were recorded. Logistic regression and multivariable logistic regression analysis were applied to calculate crude odds ratios (ORs), adjusted ORs after controlling for potential confounders, and 95% confidence intervals (CIs) of obesity, high normal BP and hypertension for short sleep defined as <6.9 hours per day.

Results: No associations were found within the total cohort. Only participants aged 18–39 years, sleeping <6.9 hours per day had a significantly increased risk of abdominal obesity (OR = 2.67; 95% CI: 1.09–6.52 based on waist circumference and OR = 5.92; 95% CI: 2.46–14.24 based on waist-hip ratio) and hypertension (OR = 3.10; 95% CI: 1.33–7.22 for diastolic BP ≥90 mmHg) after adjustments.

Conclusions: Our findings suggest that short sleep duration appears independently associated with central fat distribution and high blood pressure particularly in younger age groups.

Key words: *sleep; obesity; body mass index; waist circumference; waist-hip ratio; adiposity; blood pressure; hypertension*

Abbreviations:
BMI, body mass index
BF, body fat
BP, blood pressure
dBP, diastolic blood pressure
sBP, systolic blood pressure
CI, confidence interval
HT, hypertension
OR, odd ratio
WC, waist circumference
WHR, waist-hip ratio

INTRODUCTION

Sleep is a complex and essential biological process that is important not only for maintaining mood, memory and cognitive performance, but also for the normal functioning of the endocrine, immune and metabolic systems. Given that one third of our life we spend sleeping, the quality of sleep directly impacts our overall health and quality of life. Sleep is an integral

part of one's lifestyle. Both have changed rapidly over the last five decades. During this period there has been an observeble reduction in both sleep duration and quality, while the prevalence of obesity and associated outcomes has increased. Despite the current depth knowledge of etiological factors corresponding to obesity, the number of obese individuals still has not decreased. Thus, it was hypothesized that the increasing prevalence of obesity and inadequate sleep might be related.

This hypothesis has been supported by laboratory-based evidence showing that a short amount of sleep is associated with multiple neuro-hormonal changes, including increased cortisol and ghrelin levels and decreased leptin levels, impaired glucose metabolism and activation of inflammatory pathways (Aldabal and Bahammam 2011, Nedeltcheva and Scheer 2014). Furthermore, a body of epidemiological studies in past decade have revealed a growing link between sleep duration and a variety of serious health problems such as obesity, diabetes, dyslipidemia, metabolic syndrome, hypertension, cardiovascular diseases, depression and increased morbidity and mortality (Aldabal and Bahammam 2011, Nedeltcheva and Scheer 2014). A recent meta-analysis of cross-sectional and prospective studies confirmed a strong significant association between insufficient sleep and higher risk of obesity, which is in relation to sleep one of the most frequently studied issue in last years (Cappuccio et al. 2008, Wu et al. 2014, Sperry et al. 2015). Based on these findings, sleep deprivation has been recognised as another behavioral risk factor contributing to the above mentioned, non-communicable disease.

Little information is available on the relationship between sleep duration and obesity or blood pressure in Czechs. The aim of the present study was to investigate possible associations between obesity related anthropometric determinants and blood pressure measurements in according to total hours of sleep per day in a sample of Czech adult population.

MATERIAL AND METHODS

The research design corresponds to a cross-sectional type of survey. Volunteers for the survey were recruited between 2006–2010 through a mass media campaign advertising research project focusing primarily on the genetic background of obesity. Details regarding respondent recruitment, inclusion and exclusion criteria and other characteristics for this study were reported elsewhere (Bienertova-Vasku et al. 2009). Because it is proved that the effect of sleep on obesity diminishes with age (Najafian et al. 2010), participants older than 65 years were not included (n = 23), as well participants with incomplete data regarding their sleep (n = 24), with prescription of hypnotics in anamnesis (n = 14) and with extreme body mass index (≤17 and ≥60 kg/m^2) representing outliners (n = 3). From the initial cohort of 542 subjects, the compiled final sample of 478 respondents (26% male, 74% female) aged 18–65 years was used.

For the purpose of the present study, data on sleep were collected. Information about sleep duration was obtained from the 24-hour record of daily activities (time diaries) of seven consecutive days in which sleep was defined as the total hours of sleep per day. The average sleep duration from the whole week was calculated for each individual. Because the mean sleep duration (±s.d.) was 7.45 (±0.9) hours/day, the subjects sleeping ≥7.0 hours per day were used as the reference. There was no difference in sleep duration between men and women.

Anthropometric characteristics included weight, height, body mass index, total body fat, waist and hip circumferences and waist-hip ratio. Body parameters were measured by Seca 764 digital weighing and measuring scale (Seca gmbh&co.kg, Hamburg, Germany) and circumference measuring tape Seca 201. Body composition was assessed by bioelectrical impedance analysis, using the BodyStat®1500 (Bodystat Ltd., Douglas, Isle of Man, UK). Four types of obesity were defined: (1) body mass index (BMI) ≥30 kg/m^2, (2) waist circumference (WC) ≥88 cm in women and ≥102 cm in men, (3) waist-hip ratio (WHR) ≥0.85 in women and ≥1.0 in men and (4) body fat (BF) ≥35% in women and ≥30% in men.

Blood pressure (BP) was measured three times using a digital tonometer Omron M7 (Omron Healthcare Co. Ltd., Kyoto, Japan). The average value was calculated from the last two measurements. As defined by

the European society of hypertension and European society of cardiology (Mancia et al. 2007) high normal blood pressure was classified by values 130–139 mmHg for systolic BP or 85–89 mmHg for diastolic BP. Hypertension (HT) was defined as systolic or diastolic blood pressure ≥140 or ≥90 mmHg, respectively, or the use of anti-hypertensive medications.

There was additional information collected in the initial project for each subject, e.g. the number of comorbidities, smoking status, energy and nutrient intake, working hours and number of steps and aerobic steps per day measured by a pedometer. These were included in statistical models as confounding factors, as well as the age and sex, due to their close relationship to the primary outcome variables. Shift work and occupation status were not monitored in our investigation (only the number of hours spent at work per week).

Four types of obesity (BMI, WC, WHR, BF), high normal BP and hypertension were defined as binary indicators and were compared among the two categories of sleep: <6.9 h vs ≥7.0 h using χ^2-test. A logistic regression analysis was used to calculate the crude odds ratios (ORs) and 95% confidence intervals (CIs). Multivariable logistic analysis was carried out to calculate the adjusted ORs after controlling for the above mentioned potential confounders. Stratified analysis was realized to compare crude ORs and adjusted ORs between three age subgroups: 18–39, 40–49 and 50–65 years. All the analyses were performed using Epi Info™ program (version 3.4.3; CDC, Atlanta, GA, USA) at the significance level defined as $p<0.05\%$ and $p<0.01\%$.

The study was approved by the Committee for Ethics of Medical Experiments on Human Subjects, Faculty of Medicine, Masaryk University, Brno and was performed in adherence to the Declaration of Helsinki Guidelines. Participants gave their written informed consent before they entered the study and this consent was archived.

RESULTS

The basic data for the individuals included in the study are summarized in Table 1. The participants of our study had a mean age of 44.50 ±12.59 years, a mean BMI was 32.36 ± 7.64 kg/m² (included 40% non-obese and 60% obese) and a mean sleep duration was 7.45 ± 0.91 hours [included 26% of short sleepers (<6.9 h, $n = 123$) and 74% of normal and long sleepers (≥7.0 h, $n = 355$)]. There were 71% obese classified by the WC, 62% obese based on % of BF and 52% obese based on WHR. The highest prevalence of short sleepers was found in a group aged between 40–49 years (42%). Conversely, the lowest prevalence was in group between the age of 18–29 years (11%). There was a strong positive correlation of age with BMI ($r = 0.39$, $p = 0.000$) and negative correlation of age with sleep duration ($r = -0.14$, $p = 0.003$).

First using a χ^2-test, we found a statistically significant higher prevalence of obesity based on % of BF and WHR only within participants sleeping less than 6.9 hours/day (29% and 32% obese vs 20% and 19% non-obese respectively, $p = 0.03$ and $p = 0.003$). After inclusion of age as a confounding factor in multivariable logistic regression, no association between obesity nor elevated BP and short sleep was observed in the entire population. This is presented in Table 2.

Age had been found to be a powerful confounding factor which biased the examined associations within the entire sample due to a higher age involving both obese/hypertensive individuals and those suffering from little sleep. For this reason, an age stratified analysis was applied. This step revealed statistically significant associations in the youngest age group (18–39 years). Sleeping less than 6.9 hours/day had been associated with increased risk of central obesity based on WC (OR = 2.86; 95% CI: 1.23–6.67, $p<0.05$) and WHR (OR = 5.56; 95% CI: 2.42–12.7, $p<0.01$) and with increased dBP (OR = 2.50; 95% CI: 1.05–5.98, $p<0.05$ for dBP ≥80 mmHg and OR = 3.10; 95% CI: 1.33–7.22, $p<0.01$ for dBP ≥90 mmHg) as well with a higher prevalence involving a history of hypertension (OR = 4.67; 95% CI: 1.58–13.77, $p<0.01$); results (unadjusted ORs by age groups) are shown in Table 2.

Since the primary interest of the original study was the investigation of obesity, we further focused on the association of sleep and obesity. From the data presented in Fig. 1, it is evident that the adjustment for other potential confounding factors, in multivariable logistic

Table 1. Basic characteristic of the analysed individuals

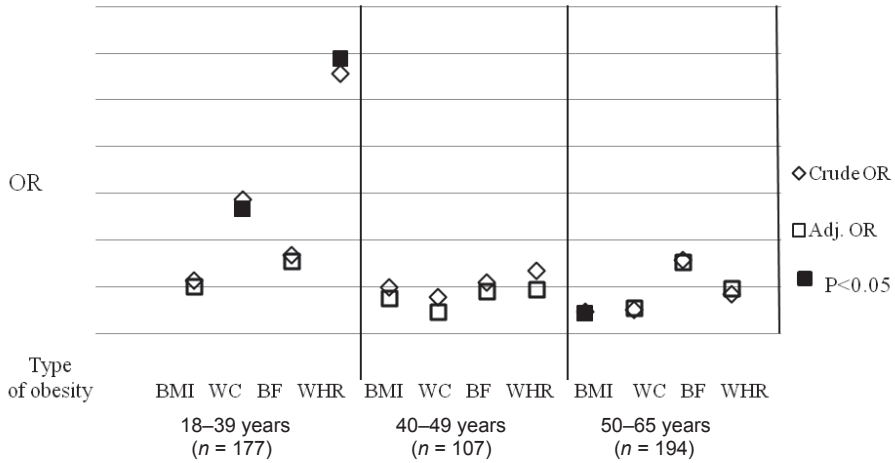
Sex / Characteristics	Women *n* = 355 (74%)	Men *n* = 123 (26%)	Total *n* = 478 (100%)
Age (years)			
Average (± SD)	44.73 (± 12.57)	43.84 (± 12.67)	44.50 (± 12.59)
Median (min, max)	45.90 (18.0; 65.7)	43.60 (18.0; 64.7)	45.25 (18.0; 65.7)
BMI (kg/m²)			
Average (± SD)	32.23 (± 7.93)	32.76 (± 6,74)	32.36 (± 7.64)
Median (min, max)	31.60 (17.1; 59.1)	32.10 (21.3; 53.3)	31.70 (17.1; 59.1)
Waist circumference (cm)			
Average (± SD)	97.73 (± 17.79)	110.67 (± 18.44)	101.06 (± 18.83)
Median (min, max)	97.00 (58.0; 147.0)	110.00 (72.0; 162.0)	100.00 (58.0; 162.0)
Waist-hip ratio			
Average (± SD)	0.85 (± 0.08)	0.98 (± 0.09)	0.88 (± 0.1)
Median (min, max)	0.85 (0.7; 1.1)	1.01 (0.8; 1.2)	0.88 (0.7; 1.2)
Body fat (%)			
Average (± SD)	39.82 (± 9.76)	28.19 (± 8.56)	36.84 (± 10.74)
Median (min, max)	40.80 (15.0; 63.8)	27.85 (9.4; 47.3)	37.70 (9.4; 63.8)
Systolic BP (mmHg)			
Average (± SD)	129.97 (± 20.04)	137.13 (± 18.39)	131.82 (± 19.86)
Median (min, max)	128.00 (87.0; 203.0)	133.00 (102.0; 203.0)	130.00 (87.0; 203.0)
Diastolic BP (mmHg)			
Average (± SD)	86.63 (± 12.35)	88.58 (± 13.54)	87.13 (± 12.68)
Median (min, max)	86.00 (54.0; 134.0)	86.00 (62.0; 131.0)	86.00 (54.0; 134.0)
Sleep (hours/day)			
Average (± SD)	7.45 (± 0.93)	7.43 (± 0.87)	7.45 (± 0.91)
Median (min, max)	7.50 (3.4; 10.8)	7.45 (4.8; 9.4)	7.50 (3.4; 10.9)

Table 2. Relative risk (OR) of certain types of disease (obesity, high normal BP and hypertension) for short sleepers (<6.9 hours/d) vs normal and long sleepers (≥7.0 hours per day) in the total cohort (after adjusting for age) and in the subsets according to age of participants

Disease		Age (years)		
	All subjects (*n* = 478)	18–39 (*n* = 177)	40–49 (*n* = 107)	50–65 (*n* = 194)
	Adj. OR (95% CI)	*Odds ratio (95% confidential interval)*		
BMI ≥30 kg/m²	0.71 (0.45–1.12)	1.14 (0.52–2.53)	0.98 (0.44–2.16)	**0.46 (0.22–0.96)***
WC ≥94 cm M, ≥80 cm F	1.04 (0.61–1.77)	**2.86 (1.23–6.67)***	0.79 (0.33–1.86)	0.47 (0.17–1.32)
BF ≥30% M, ≥35% F	1.30 (0.79–2.15)	1.68 (0.75–3.74)	1.10 (0.48–2.48)	1.58 (0.56–5.68)
WHR ≥1.0 M, ≥0.85 F	1.58 (0.99–2.51)	**5.56 (2.42–12.76)****	1.34 (0.61–2.94)	0.84 (0.41–1.70)
sBP ≥130 mmHg	0.92 (0.59–1.43)	1.21 (0.52–2.80)	0.91 (0.42–1.98)	0.76 (0.38–1.54)
sBP ≥140 mmHg	1.08 (0.68–1.71)	2.06 (0.68–6.30)	1.16 (0.51–2.36)	0.85 (0.45–1.62)
dBP ≥85 mmHg	1.32 (0.82–2.11)	**2.50 (1.05–5.98)***	0.77 (0.10–1.91)	0.66 (0.29–1.48)
dBP ≥90 mmHg	1.42 (0.93–2.15)	**3.10 (1.33–7.22)****	1.10 (0.50–2.38)	0.75 (0.39–1.42)
HT in anamnesis	1.29 (0.84–1.99)	**4.67 (1.58–13.77)****	0.77 (0.31–1.94)	0.52 (0.27–1.01)

BMI – body mass index, WC – waist circumference, BF – body fat, WHR – waist-hip ratio, sBP – systolic blood pressure, dBP – diastolic blood pressure, HT – hypertension; M – male, F – female; * $p<0.05$, ** $p<0.01$

regression in each age stratum, did not significantly affect the association between short sleep and risk of obesity showing a decreasing tendency with an increasing age. The youngest generation up to 39 years with short sleep was in significantly higher risk of abdominal obesity independently of many factors of lifestyle (e.g. sex, smoking, number of comorbidities, fat intake, working hours or physical activity expressed as number of steps) as compared with the same old individuals sleeping at least 7 hours per day. Adjusting OR for WC obesity slightly decreased to 2.67 (95% CI: 1.09–6.52, p = 0.031) whilst for WHR obesity increased to 5.92 (95% CI: 2.46–14.24, p = 0.0001). On the other hand, among older respondents, short sleep had paradoxically rather protective effect against BMI ≥30 kg/m² (adj. OR = 0.44; 95% CI: 0.19–0.96, p = 0.039).



BMI obesity – body mass index ≥30 kg/m², WC obesity – waist circumference ≥94 cm in male and ≥80 cm in female, BFobesity – % of body fat ≥30 % in male and ≥35 % in female, WHR obesity – waist-to-hip ratio ≥1.0 in male and ≥0.85 in female. Adjusted OR – relative risk of certain types of obesity in individuals sleeping <6.9 hours compared to those sleeping ≥7.0 hours controlling for sex, number of comorbidities, smoking status, percent of energy intake in fat, working hours and number of total and aerobic steps.

Fig. 1. Relative risk of certain type of obesity expressed by crude and adjusted odds ratios (OR) for individuals sleeping <6.9 hours per day by age groups

DISCUSSION

There are not many studies in the Czech Republic focusing on sleep duration in the general population in relationship to health outcomes such as obesity or hypertension with which we would be able to compare our results. First Adámková et al. (2009) in a group of 3,970 probands aged 18–65 years detected a significant negative correlation between the duration of sleep and body weight and BMI only with optimal values of the BMI when sleep lasted at least seven hours per night; circumference of waist and hip, WHR, systolic and diastolic BP, heart rate and biochemical parameters from blood samples were not significantly associated with sleep duration. Second Vosátková et al. (2012) in group of 805 participants aged 18–65 years investigated the relationship between length of sleep and metabolic syndrome, which is characterized among other criteria by the presence of central obesity. This study did not confirm a relationship between sleep duration and metabolic syndrome.

However, comparability of these results is limited due to different methodological approaches and also primarily due to the discrepancies in age distribution of the cohorts, when taking into account that both characteristics (sleep and anthropometric measurements) are strongly dependent on

age. Adámková et al. (2009) mentioned rather young individuals compiling the sample, while our sample included mainly participants of middle age, among whom, as a whole, we did not detect any associations.

The variety of methods used for analyzing obesity may also be problematic. The majority of epidemiological cross-sectional surveys assessed the obesity using a BMI. BMI and body fat percentage are very useful and practical information for classifying obesity, but only WC or WHR can tell more about fat distribution. Central adiposity is well known to be associated with metabolic and cardiovascular diseases. There is a study from Theorell-Haglöw et al. (2012) dealing with WC in women, in which an independent association between short sleep (<5.0 h) and central obesity was found and was strongest in younger women (<50 years). As well Najafian et al. (2010) reported, that short sleep (<5.0 h) increased the odds ratio for abdominal obesity in both men and women, but only individuals under the age of 60 years, independently of its relation to BMI. Our results are similar, as we ascertained that a short amount of sleep (<6.9 h) being linked with central obesity (WC and WHR) after adjustments in participants younger than 39 years. A significant negative relationship between sleep duration and WC, indicating short sleep duration inversely correlates with central adiposity, has been documented in very recent meta-analysis of cross-sectional studies from Sperry et al. (2015).

A stronger association of sleep with obesity in young adults is in line with the literature, where systematic reviews and meta-analysis of studies from around the world show a consistent increased risk of obesity among short sleepers in children and younger age groups (Gangwisch et al. 2005, Cappuccio et al. 2008, Patel and Hu 2008, Magee and Hale 2012, Fatima et al. 2015). Age-related changes in sleep architecture, body composition and hormonal changes in the elderly, may explain why sleep is not as strongly associated with obesity in the elderly, as in the young. Sleep deprivation is hypothesized to be a powerful stressor particularly for sensitive young organism. In young individuals, it may contribute to the development of central obesity through affecting carbohydrate metabolism and metabolic functions, impaired glucose tolerance, elevated cortisol concentrations or increased sympathetic activity. In addition, metabolic hormonal changes such as decreased leptin and elevated ghrelin levels may stimulate appetite, hyperphagia and cause weight gain (Aldabal and Bahammam 2011, Nedeltcheva and Scheer 2014).

Short sleep duration has been on the basis of convincing evidence proved as significant risk factor for obesity. Some of the studies also show long sleep duration associated with higher BMI (Buxton and Marcelli 2010), but meta-analysis of prospective studies did not confirm this (Wu et al. 2014). In our study, this was only the case of the oldest age group (50–65 years), where individuals sleeping less than 6.9 hours per day, were at lower risk of being obese (based on BMI) than individuals sleeping longer. This interesting finding can be explained by the above mentioned age-dependent sleep and body changes and also by the fact, that our shortly sleeping young generation showed the highest number of working hours per week, but a lower number of daily steps compared with the same older, yet longer sleeping individuals. While the elderly short sleepers showed a significantly higher number of daily steps than their same old and longer sleeping contemporaries. That could mean, that the younger generation probably spend their prolonged alertness more with passive activities (transport, PC, TV, sitting), in contrary to the older generation spending their prolonged alertness time more active. But this hypothesis is denied by the multivariable logistic analysis controlled also for the number of working hours and the number of steps.

There is also a theory proclaiming that the older and younger cohorts lived through distinct historical time periods with different stressors. A dramatic increase in the prevalence of chronic partial sleep deprivation in the past half century, in parallel with the growing epidemic of obesity, occurred due to societal and technological changes, including increases in shift work, 24-hour stores, cable television and use of the Internet. It is, therefore, possible that an even stronger association currently exists between short sleep duration and obesity (Gangwisch et al. 2005).

Not sleep as continuous parameter, but also its certain parts might have a key role in pathological processes. Loss of SWS ("slow wave sleep") sleep is discussed as an important contributor of hypertension particularly in younger population, which have normally proportionally more SWS sleep than older ones (Palagini et al. 2013). A systematic review on the relationship of sleep and BP showed that experimental sleep deprivation as well short sleep duration and persistent insomnia are associated with increased BP and an increased risk of hypertension, even after controlling for other risk factors (Palagini et al. 2013). Gangwisch (2014) adds that the association between short sleep duration with higher BP and HT appears stronger in middle-aged adults and in women. This is also confirmed with our results. The possible mechanism may consist of activated sympathetic nervous system activity and elevated cortisol levels as a result of a prolonged exposure to physical and psychosocial stressors after a short night of sleep.

Our findings are subject to several limitations. The first limitation is unrepresentative population sample which does not enable us any generalization for our conclusion. Nevertheles, it was a fairly extensive set of men and women involving a wide range of ages without serious diseases (e.g. oncological or mental) compiled on precise inclusion criteria from the entire country. A skewed age distribution of the cohort was corrected using stratified analysis and statistical tests were further controlled for numerous potential confounding factors (e.g. sex, number of comorbidities, smoking status, percent of energy intake in fat, work load and number of total and aerobic steps).

Another limitation of the study was using sleep duration as the sole measurement for sleep because such a measurement does not address the quality of sleep. Even people who sleep for a relatively long period of time may not get an adequate quality of sleep because their sleep is disrupted by sleep apnea, sleep disorders such as insomnia, the side-effects of various medications, the presents of chronic diseases, or other unknown causes.

Finally, the cross-sectional design of the survey prevented us from attempting to determine the causal relationship between obesity and hypertension and sleep. However, the most likely scenario is that excess weight and insufficient or poor quality of sleep, may have a reciprocal causal relationship.

CONCLUSION

In conclusion, our results suggest that sleep less than 6.9 hours per day is significantly associated with an increased risk of abdominal obesity and high blood pressure, especially in young Czech adults. These findings support the theory of the interaction between sleep and obesity and high BP in younger ages. Given the severity of the disease at such a young age (<39 years), adequate sleep duration might be another of the potential modifiable factors, in addition to a balanced diet and physical activity, in prevention of obesity and hypertension.

CONFLICT OF INTEREST

The authors have no conflict of interest to disclose.

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