Observational Study

Medicine®

OPEN

Assessment of sleep and obesity in adults and children

Observational study

Lilla Bonanno, MSc, PhD[a,*], Daniela Metro, MD[b], Mattia Papa, MD[c], Giuseppa Finzi, Psy[d], Antonia Maviglia, MD[b], Fabrizio Sottile, MD, PhD[a], Francesco Corallo, Psy[a], Luigi Manasseri, MD[b]

Abstract

The sleep allows many psychological processes, such as immune system activity, body metabolism and hormonal balance, emotional and mental health, learning, mnemonic processes. The lack of sleep could undermine mental and physical purposes, causing an alteration in cognitive functions or metabolic disorders. In our study, we have examined the irregular sleep effects with the overweight and obesity risk in children and adults.

The sample was composed of 199 subjects, of which 71 adults, (29 males and 42 females), and 128 children (73 males and 55 females). We have measured the weight and height with standard techniques; we also have measured the body mass index dividing the weight in kg with the height square expressed in meters (kg/m^2). Subjects were divided into underweight, normal weight, overweight, and obese. Were administered some questionnaires to measure the quantity and quality of sleep, and eating habits and individual consumption of food.

Analysis of demographic variables not showed significant differences between male and female groups but highlighted a significant trend differences in normal-weight score. The clinical condition has a substantial impact on body mass index score and sleep hours were significant predictor on this.

Quantity and quality sleep can also represent a risk factor of overweight and obesity, so sufficient sleep is a factor that influence a normal weight. Adults and children that sleep less, have an increase in obesity and overweight risk with dysfunctional eating behaviors, decreased physical activity, and metabolic changes.

Abbreviation: BMI = body mass index.

Keywords: adult, children, obesity, sleep

1. Introduction

Sleep is essential to support the functions and health of the entire body. Indeed, it guarantees the optimal functioning of a multiple physiological processes, such as immune system activity, body metabolism and hormonal balance, emotional and mental health, various forms of learning, the memorization processes. Numerous studies have highlighted negative effects of lack of sleep on mental and physical balance, indeed they could change cognitive functions, such as memory and emotions control, and hunger regulation. It has been shown that upset or insufficient sleep weakens immune system and favors metabolic disorders. Several studies have underlined that decreased duration and/or quality of sleep can affect glucose metabolism and interfere with the secretion of anabolic hormones like growth hormone (GH),[1] prolactin,[2] and testosterone[3]; also it can alter amount and timing of catabolic hormones like glucocorticoids,[4] catecholamines[5]; lastly, can changes in dietary regulation.[6,7]

Many epidemiological studies have shown that decreased sleep duration and quality (6–7 hours per night) is associated with increased obesity incidence,[8–10] diabetes,[11,12] cardiovascular diseases,[13,14] and mortality.[15,16]

In our study, we investigated the association between sleep duration and quality, and overweight risk and obesity in children and adults. The examined subjects diet and eating habits were also evaluated.

2. Method

The study was conducted on secondary school children, and on practice dental clinic patients. Were involved 199 subjects, of which 71 adults (29 males and 42 females) with age between 29 and 65 years, and 128 children (73 males and 55 females) with age between 10 and 13 years. Every subject was instructed about the goals and the procedures and signed an informed consent form. The study was approved by the local ethics committee. The data were picked in a period between October 2016 and November 2017. Were evaluated weight, height, and body mass

Editor: Marcello Iriti.

The authors have no conflicts of interest to disclose.

[a] IRCCS Centro Neurolesi Bonino-Pulejo, [b] Department of Biomedical and Dental Sciences and Morphofunctional Imaging University of Messina, [c] Food and Nutrition Hygiene Service (SIAN) ASP 5 – Provincial Health Authority 5, [d] AO Papardo, Neurology Unit, Messina, Italy.

[*] Correspondence: Lilla Bonanno, IRCCS Centro Neurolesi "Bonino-Pulejo," S.S. 113, Via Palermo, Ctr. Casazza, 98124 Messina, Italy (e-mail: lillabonanno@gmail.com).

How to cite this article: Bonanno L, Metro D, Papa M, Finzi G, Maviglia A, Sottile F, Corallo F, Manasseri L. Assessment of sleep and obesity in adults and children. Medicine 2019;98:46(e17642).

Received: 9 July 2019 / Received in final form: 23 September 2019 / Accepted: 25 September 2019

http://dx.doi.org/10.1097/MD.0000000000017642

index (BMI) data. The weight and height were measured with standard techniques, the BMI was assessed dividing the weight in kg with the height square expressed in meters (kg/m^2). The hormones involved in the hunger and satiety mechanisms can be measured during sleep, thereby, we not analyzed. Subjects were divided into underweight, normal weight, overweight, and obese. Were administrated several questionnaires to evaluate the quantity and quality of sleep, with which were highlighted hours of sleep overall, the mode of falling asleep, and the quality of sleep. The survey of eating habits and individual consumption of food was carried out through the administration of questionnaires and the use of a reasoned atlas of nutrition.[17]

2.1. Statistical analysis

We considered 2 groups: adults and children. The Shapiro normality test was carried out to analyze the distribution of the variables. Continuous variables were expressed as mean ± standard deviation, whereas as median, and first-third quartile in no normal distribution. The Student unpaired *t* test and the Mann–Whitney *U* test were used to compare 2 group (males and females groups), when appropriate. For intragroup analysis, correlations between variables were computed by Pearson correlation or Spearman coefficient. We performed a multiple regression analysis on the BMI score (dependent variables). At first, we focused on the influence of demographic and clinical variables, by using patient's age and sleep hours scores as predictors. We applied a backward elimination stepwise procedure for the choice of the best predictive variables according to the Akaike information criterion. Analyses were performed using an open-source R3.0 software package. A 95% of confidence level was set with a 5% alpha error. Statistical significance was set at $P < .05$.

3. Results

3.1. Adult group

Analysis of demographic variables not showed significant differences between male and female groups but highlighted a significant trend differences in normal-weight score ($P = .07$) (Table 1). In male group found a significative positive correlation between age and BMI score ($r = 0.48$; $P = .009$) and a significative negative correlation between sleep hours and BMI score ($r = -0.64$; $P < 0.001$) (Fig. 1A and B). In female group, we found a significative negative correlation between sleep hours and BMI score ($r = -0.78$; $P < .001$) (Fig. 1C). Results in Table 2 showed that the clinical condition of the patients has a significant impact on BMI score. Sleep hours was significant predictor on BMI score for male and female group.

Table 1

Socio-demographic and clinical characteristics of adults group.

	Sample	Male	Female	*P*-value
N. Subjects	71	29	42	
Age (mean ± SD)	43.65 ± 11.39	42.21 ± 11.55	44.64 ± 11.31	.38
BMI (mean ± SD)	25.25 ± 3.87	25.97 ± 3.41	24.76 ± 4.12	.2
Sleep hours (mean ± SD)	7.5 ± 0.91	7.42 ± 0.88	7.56 ± 0.93	.52
BMI				
Underweight (median (I–III))	17 (17.0–17.0)	–	17 (17.0–17.0)	–
Normal-weight (median (I–III))	23 (21.0–24.0)	23 (22.0–24.0)	22 (20.0–23.0)	.07
Overweight (median (I–III))	27 (26.0–28.0)	27 (27.0–28.0)	27 (26.0–27.75)	.20
Obese (median (I–III))	32 (31.0–33.0)	32 (31.0–34.0)	32 (31.25–32.75)	.90
Sleep hours				
Underweight (median (I–III))	8 (8.0–8.0)	–	8 (8.0–8.0)	
Normal-weight (median (I–III))	8.3 (7.6–8.5)	8 (7.5–8.5)	8.3 (8.0–8.5)	.29
Overweight (median (I–III))	7 (6.5–7.3)	7 (6.5–7.3)	7.05 (6.62–7.3)	.98
Obese (median (I–III))	6.5 (6.0–7.0)	6.5 (6.25–6.75)	6.4 (5.7–6.87)	.70

I = first quartile, III = third quartile, BMI = body mass index, SD = standard deviation.

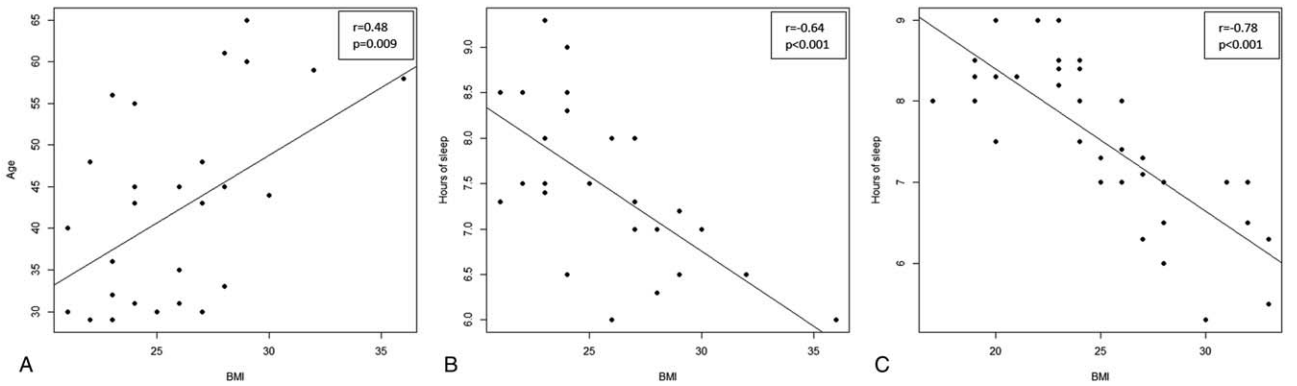

Figure 1. Correlation between BMI score and demographic and clinical variables. (A) Scatter plot of BMI score and age in male adult group. (B) Scatter plot of BMI score and sleep hours in male adult group. (C) Scatter plot of BMI score and sleep hours in female adult group. BMI = body mass index.

Table 2

Backward linear regression: predictors on BMI score in adult group.

Dependent variables	Predictors	β	Std β	*P*-value	Adjusted R^2
BMI male group	Age	0.08	0.04	.09	0.43
	Sleep hours	−2.07	0.6	.002	
BMI female group	Age	0.04	0.04	.28	0.59
	Sleep hours	−3.49	0.44	<.001	

β = regression coefficient, BMI = body mass index, Std β = standardized regression coefficient.

3.2. Children group

Analysis of demographic variables not showed significant differences between male and female groups but highlighted a significant differences in hours of sleep in underweight group ($P = .03$) (Table 3). A significative negative correlation between sleep hours and BMI score ($r = -0.6$; $P < .001$) and ($r = -0.56$; $P < .001$) in male and female group respectively were found (Fig. 2A and B). Results in Table 4 showed that the clinical condition of the patients has a significant impact on BMI score. Sleep hours were significant predictor of BMI score for male and female group.

Table 3

Socio-demographic and clinical characteristics of children group.

	Sample	Male	Female	*P*-value
N. Subjects	128	73	55	
Age (mean ± SD)	11.32 ± 1.14	11.33 ± 1.16	11.31 ± 1.14	.92
BMI (mean ± SD)	19.49 ± 3.12	19.63 ± 3.28	19.30 ± 2.92	.56
Sleep hours (mean ± SD)	9.03 ± 0.91	9.07 ± 0.92	8.98 ± 0.89	.55
BMI				
Underweight (median (I–III))	16.2 (16.0–16.6)	16.3 (16.0–16.6)	16.1 (16.0–16.6)	.68
Normal-weight (median (I–III))	18 (17.7–18.6)	28.15 (17.8–18.7)	18 (17.7–18.4)	.45
Overweight (median (I–III))	21.4 (20.6–22.0)	21.2 (20.2–21.8)	21.5 (21.0–22.0)	.47
Obese (median (I–III))	26.5 (25.2–27.0)	26.5 (25.6–27.0)	26.5 (25.2–27.0)	.91
Sleep hours				
Underweight (median (I–III))	9.5 (9.0–10.1)	10 (9.4–10.5)	9.3 (9.0–9.6)	.03*
Normal-weight (median (I–III))	9.3 (9.0–10.0)	9.3 (9.0–10.0)	9.5 (9.3–10.0)	.15
Overweight (median (I–III))	8.1 (8.0–8.3)	8 (8.0–8.3)	8.2 (8.0–8.3)	.92
Obese (median (I–III))	8.3 (8.0–8.6)	8.3 (8.0–8.7)	8.3 (8.0–8.5)	.82

I = first quartile, III = third quartile, BMI = body mass index, SD = standard deviation.
* $P < .05$.

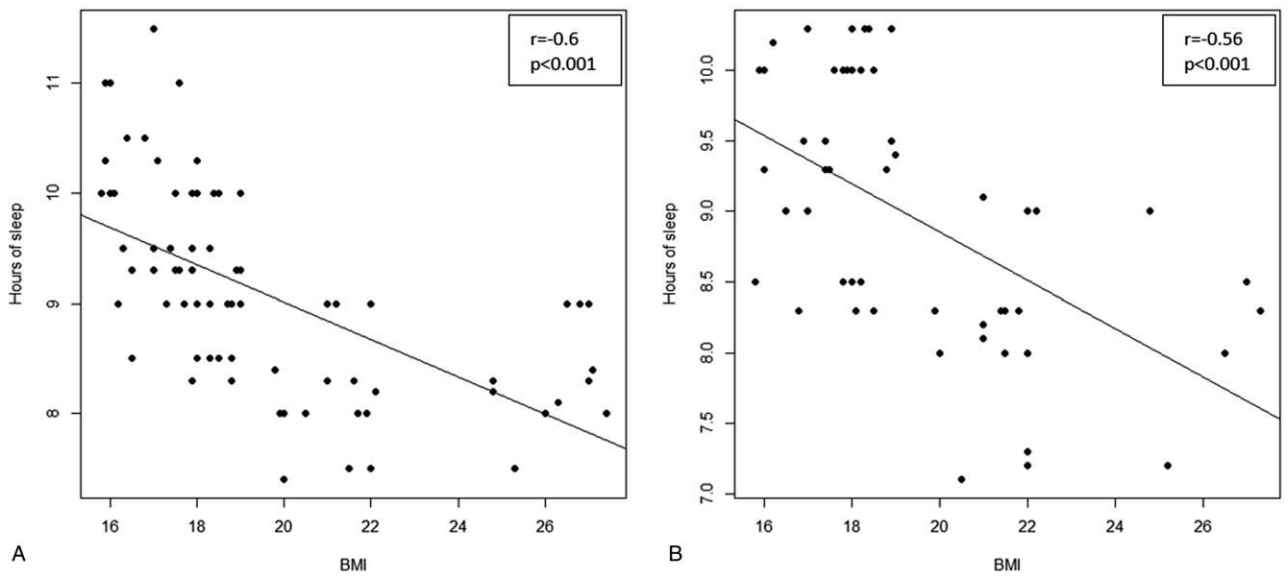

Figure 2. Correlation between BMI score and demographic and clinical variables. (A) Scatter plot of BMI score and sleep hours in male children group. (B) Scatter plot of BMI score and sleep hours in female children group. BMI = body mass index.

Table 4

Backward linear regression: predictors on BMI score in children group.

Dependent variables	Predictors	β	Std β	*P*-value	Adjusted R^2
BMI male group	Age	−0.2	0.27	.45	0.35
	Sleep hours	−2.12	−0.34	<.001	
BMI female group	Age	−0.17	0.3	.57	0.29
	Sleep hours	−1.81	0.38	<.001	

β = regression coefficient, BMI = body mass index, Std β = standardized regression coefficient.

4. Discussion

We have highlighted, in previous surveys on a Sicilian student population with overweight/obese subjects compared to normal-weight subjects, a lower adherence to the Mediterranean diet. Observational studies and clinical experiences have shown that adherence to the Mediterranean diet is inversely associated with obesity. Furthermore, we have found a lower consumption of breakfast and a higher consumption of snacks and out-course meals.[17,18]

Our study has highlighted that the duration and quality of sleep can also represent a risk factor of overweight and obesity in examined subjects. Therefore, sufficient sleep is required to maintain a normal weight.

One of the first indications of the link between reduced sleep duration and increased body weight came from the observation that the increase in the number of overweight or obese subjects, in industrial societies, was accompanied by an equal decrease in the average sleep duration of about 1 or 2 hours. Several studies[4,19–25] have shown that children aged 5 to 9 years who sleep less than 10 hours per night run a 1.5 to 2-fold risk of becoming obese compared to those who sleep properly; while adults that sleep less than 6 hours per night run a 50% risk of becoming obese. The association between decreased duration and quality of sleep with increased incidences of obesity and type II diabetes has been highlighted by epidemiological and experimental studies.[4,19–25] In addition, people who sleep less than 8 hours have an increase in BMI proportionate to the decrease in sleep.[26] The association between sleep duration and weight gain in women is supported by 16 longitudinal studies.[27] Moreover, studies in school children and adolescents (8–17 years) have shown that going to bed late was associated with adiposity, regardless of the sleep length.[28,29] Biological and behavioral mechanisms have been proposed to explain the association between obesity risk and sleep deprivation. The relationship between decreased sleep hours with increased body weight and risk of type II diabetes mellitus may include increased food intake, decreased physical activity and energy expenditure, and changes in glucose metabolism.[30,31]

Several studies have argued that excessive introduction of food, which leads to an increased risk of obesity in people with short-term sleep, is associated with changes in hormones responsible for hunger and appetite control, especially leptin and ghrelin.[32]

Grelin is the hormone produced by the stomach during fasting and sends signals to the hypothalamus stimulating the centers of hunger, while leptin, produced by the adipocytes, sends signals of satiety.

An adult study showed that the reduction of sleep for 2 nights caused a reduction of 18% in leptin, a hormone with an anorectic effect, an increase of 28% in ghrelin, an obesifying factor, and an increase of about 24% in hunger, with a preference for the intake of foods containing carbohydrates with high caloric density over controls.[6]

In addition, numerous epidemiological studies have shown that short chronic duration of sleep, even in school children, was associated with low levels of leptin.[33]

Observational studies by Peter Jones and his team, at the University of Manitoba, also have shown that during the morning breakfast, subjects undergoing 4 hours of sleep compared to a control group with a rest of 9 hours, took an average of 300 Kcalories and about 30% more saturated fat.

Moreover to eating habits, physical activity also affects body weight. Sleep deprivation contributes to weight gain by reducing energy expenditure and physical activity.[30,34] Indeed, people who sleep less are less active.

The lateral hypothalamus plays an important role in many motivated behaviors, in sleep/wake cycle, in food introduction, in energy balance. It is also home-based to a heterogeneous population of neurons that expresses and co-expresses many neuropeptides, including hypocretin.[35,36]

Hypocretin 1 (Orexin A) is a key signal in the regulation of the wake/sleep cycle, regulating the introduction of food by stimulating appetite and energy metabolism.[37,38] Neurons that produce hypocretin are sensitive to glucose and leptin concentrations; indeed, when glucose concentration decreases, hypocretin neurons are activated, which induce an increase in food intake. On the contrary, when leptin levels increase, hypocretin neurons are inhibited and the amount of food ingested decreases.

In case of sleep deprivation, decrease of leptin may contribute to hypocretine increase which prolongs wakefulness, stimulates appetite and allows the search for food.

Hypocretin-producing neurons project into the ventrolateral preoptic area and induce the transition from sleep to wakefulness, significantly increase awakening, physical activity and energy expenditure, and reduce sleep. In addition, partial sleep deprivation reduces the effectiveness of hypocretine in stimulating physical activity and energy expenditure.[39]

Finally, observational and experimental studies have shown an association between decreased duration and quality of sleep with reduced insulin sensitivity, insulin resistance, and decreased glucose tolerance,[40,41] with an increased risk of type II diabetes. The risk of falling ill with type II diabetes increases by 50% in subjects with less than 5 hours of sleep, compared to those who sleep 8 hours. Restricting sleep by 4 hours for 6 nights causes a decrease in glucose tolerance as measured by the glucose tolerance test (19). A long period of sleep restriction (<6.5 hours per night) can lead to a 40% decrease in glucose tolerance. Studies conducted by Bergman and Yaggi have shown a significant reduction in insulin sensitivity during night-time sleep[42,43] and the insulin sensitivity index in subjects with short sleep duration is lower than in subjects with normal sleep duration.[42]

Sleep deprivation causes an increase in cortisol (4) and GH (1), hormones that contribute to determining resistance to the action of insulin. High cortisol secretion and a shift in the circadian rhythm of cortisol; therefore, contribute to significant alterations in glucose tolerance, insulin-sensitivity, and insulin secretion.[44] Adipocytes, through the production of hormones called adipokines or adipocytokines, can also directly modulate insulin sensitivity. Circulating adipocytokine patterns are profoundly altered in obese patients. An increase in fat mass is associated with a general increase in adipocytokines that can reduce insulin sensitivity and have a direct effect on the genesis of insulin resistance (eg, tumor necrosis factor-alpha, interleukin 6, resistins[45–48]).

On the other hand, adinopectin levels are inversely related to obesity and insulin resistance.[49]

Indeed, adiponectin increases tissue insulin sensitivity and circulating levels of adiponectin are commonly decreased in obese subjects and are associated with insulin resistance, inadequate insulin response, and type II diabetes.[50,51]

Levels of Visfatin, an Adipoquine with insulin-like action, are also considered a link between obesity and insulin resistance.[52] In fact, sleep deficiency is associated with high BMI, high Visfatin levels and high insulin resistance.[53]

Author contributions

Conceptualization: Daniela Metro, Luigi Manasseri.
Data curation: Lilla Bonanno, Giuseppa Finzi.
Formal analysis: Lilla Bonanno.
Funding acquisition: Daniela Metro, Mattia Papa, Giuseppa Finzi, Antonia Maviglia, Fabrizio Sottile, Francesco Corallo.
Investigation: Daniela Metro, Giuseppa Finzi.
Methodology: Daniela Metro, Francesco Corallo.
Supervision: Daniela Metro, Luigi Manasseri.
Validation: Luigi Manasseri.
Visualization: Mattia Papa, Giuseppa Finzi, Fabrizio Sottile, Luigi Manasseri.
Writing – original draft: Lilla Bonanno, Daniela Metro.
Writing – review & editing: Giuseppa Finzi, Antonia Maviglia, Fabrizio Sottile, Francesco Corallo.

References

[1] Spiegel K, Leproult R, Colecchia EF, et al. Adaptation of the 24-h growth hormone profile to a state of sleepdebt. Am J Phisiol Regul Integr Comp Physiol 2000;279:874–83.
[2] Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on physiological rhythms. Rev Neurol (Paris) 2003;159(11 Suppl):S11–20.
[3] Penev P, Spiegel K, L'Hermite-Balériaux M, et al. Relationship between REM sleep and testosterone secretion in older men. Ann Endocrinol (Paris) 2003;64:157.
[4] Leproult R, Copinschi G, Buxton O, et al. Sleep loss results in an elevation of cortisol levels the next evening. Sleep 1997;20:865–70.
[5] Penev P, Spiegel K, Marcinkowski T, et al. Impact of carbohydrate-rich meals on plasma epinephrine levels: dysregulation with aging. J Clin Endocrinol Metab 2005;90:6198–206.
[6] Spiegel K, Tasali E, Penev P, et al. Brief communication: sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. Ann Intern Med 2004;141:846–50.
[7] Nedeltcheva AV, Kilkus JM, Imperial J, et al. Sleep curtailment is accompanied by increased intake of calories from snacks. Am J Clin Nutr 2009;89:126–33.
[8] Taheri S. The link between short sleep duration and obesity: we should recommend more sleep to prevent obesity. Arch Dis Child 2006;91:881–4.
[9] Watanabe M, Kikuchi H, Tanaka K, et al. Association of short sleep duration with weight gain and obesity at 1-year follow-up: a large-scale prospective study. Sleep 2010;33:161–7.
[10] Xiao Q, Arem H, Moore SC, et al. A large prospective investigation of sleep duration, weight change, and obesity in the NIH-AARP Diet and Health Study cohort. Am J Epidemiol 2013;178:1600–10.
[11] Holliday EG, Magee CA, Kritharides L, et al. Short sleep duration is associated with risk of future diabetes but not cardiovascular disease: a prospective study and meta-analysis. PLoS One 2013;8:e82305.
[12] McNeil J, Doucet E, Chaput JP. Inadequate sleep as a contributor to obesity and type 2 diabetes. Can J Diabetes 2013;37:103–8.
[13] Sabanayagam C, Shankar A. Sleep duration and cardiovascular disease: results from the National Health Interview Survey. Sleep 2010;33: 1037–42.
[14] Grandner MA, Chakravorty S, Perlis ML, et al. Habitual sleep duration associated with self-reported and objectively determined cardiometabolic risk factors. Sleep Med 2014;15:42–50.
[15] Grandner MA, Hale L, Moore M, et al. Mortality associated with short sleep duration: the evidence, the possible mechanisms, and the future. Sleep Med Rev 2010;14:191–203.
[16] Grandner MA, Patel NP. From sleep duration to mortality: implications of meta-analysis and future directions. J Sleep Res 2009; 18:145–7.
[17] Metro D, Tardugno R, Papa M, et al. Adherence to the Mediterranean diet in a Sicilian student population. Nat Prod Res 2017;32:1775–81.
[18] Metro D, Papa M, Manasseri L, et al. Mediterranean diet in a Sicilian student population. Second part: breakfast and its nutritional profile. Nat Prod Res 2018;19:1–7.
[19] Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. Lancet 1999;354:1435–9.
[20] Van Cauter E, Holmback U, Knutson K, et al. Impact of sleep and sleep loss on neuroendocrine and metabolic function. Horm Res 2007;67 (Suppl 1):2–9.
[21] Tasali E, Leproult R, Ehrmann DA, et al. Slow-wave sleep and the risk of type 2 diabetes in humans. Proc Natl Acad Sci U S A 2008;105:1044–9.
[22] Jiang F, Zhu S, Yan C, et al. Sleep and obesity in preschool children. J Pediatr 2009;154:814–8.
[23] Ho TF. Cardiovascular risks associated with obesity in children and adolescents. Ann Acad Med Singapore 2009;38:48–9.
[24] Padez C, Mourao I, Moreira P, et al. Long sleep duration and childhood overweight/obesity and body fat. Am J Hum Biol 2009;21:371–6.
[25] Durán Agüero S, Haro Rivera P. Association between the amount of sleep and obesity in Chilean school children. Arch Argent Pediatr 2016;114:114–9.
[26] Taheri S, Lin L, Austin D, et al. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med 2004;1:e62.
[27] Patel SR, Malhotra A, White DP, et al. Association between reduced sleep and weight gain in women. Am J Epidemiol 2006;164:947–54.
[28] Golley R, Maher C, Matricciani L, et al. Sleep duration or bedtime? Exploring the association bedtime sleep timing behaviour, diet and BMI in children and adolescents. Int J Obes 2013;37:546–51.
[29] Jarrin D, McGrath J, Drake C. Beyond sleep duration: distinct sleep dimensions are associated with obesity in children and adolescents. Int J Obes 2013;37:552–8.
[30] Anton-Paduraru DT, Teslariu O, Mocanu V. Influence of sleep on obesity in children. Rev Med Chir Soc Med Nat Iasi 2016; 120:239–43.
[31] Ren H, Zhou Z, Liu WK, et al. Excessive homework, inadequate sleep, physical inactivity and screen viewing time are major contributors to high paediatric obesity. Acta Paediatr 2017;106:120–7.
[32] Taheri S, Lin L, Austin D, et al. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med 2004;1:210–7.
[33] Boeke CE, Storfer-Isser A, Redline S, et al. Childhood sleep duration and quality in relation to leptin concentration in two cohort studies. Sleep 2013;37:613–20.
[34] Parrish JB, Teske JA. Acute partial sleep deprivation due to environmental noise increases weight gain by reducing energy expenditure in rodents. Obesity (Silver Spring) 2017;25:141–6.
[35] Tyree SM, de Lecea L. Lateral hypothalamic control of the ventral tegmental area: reward evaluation and the driving of motivated behavior. Front Syst Neurosci 2017;11:50.
[36] Chieffi S, Carotenuto M, Monda V, et al. Orexin system: the key for a healthy life. Front Physiol 2017;31:8–357.

[37] Rani M, Kumar R, Krishan P. Role of orexins in the central and peripheral regulation of glucose homeostasis: evidences & mechanisms. Neuropeptides 2018;68:1–6.
[38] Santiago JCP, Otto M, Kern W, et al. Relationship between cerebrospinal fluid concentrations of orexin A/hypocretin-1 and body composition in humans. Peptides 2018;102:26–30.
[39] DePorter DP, Coborn JE, Teske JA. Partial sleep deprivation reduces the efficacy of orexin-A to stimulate physical activity and energy expenditure. Obesity (Silver Spring) 2017;25:1716–22.
[40] Punjabi NM. Do sleep disorders and associated treatments impact glucose metabolism? Drugs 2009;69:13–27.
[41] Spiegel K, Knutson K, Leproult R, et al. Sleep loss: a novel risk factor for insulin resistance and Type 2 diabetes. J Appl Physiol 2005;99:2008–19.
[42] Lihn AS, Pedersen SB, Richelsen B, et al. Adiponectin action, regulation and association to insulin sensivity. Obes Rev 2005;6:13–21.
[43] Yaggi H, Araujo AB, McKinlay JB, et al. Sleep duration as a risk factor for the development of type 2 diabetes. Diabetes Care 2006;29:657–61.
[44] Bergman RN. Toward physiologicalunderstanding of glucose tolerance-minimal-model approach. Diabetes 1989;38:1512–27.
[45] Lyon CJ, Law RE, Hsueh WA. Minireview: adiposity, inflammation, and atherogenesis. Endocrinology 2003;144:2195–200.
[46] Rajala MW, Scherer PE. Minireview: the adipocyte at the crossroads of energy homeostasis, inflammation, and atherosclerosis. Endocrinology 2003;144:3765–73.
[47] Fain JN, Bahouth SW, Madan AK. TNFalpha release by the nonfat cells of human adipose tissue. Int J Obes Relat Metab Disord 2004;28: 616–22.
[48] Lazar MA. How obesity causes diabetes; not a tall tale. Science 2005;307:373–5.
[49] Buijs RM, Escobar C. Corticosterone and activity: the long arms of the clock talk back. Endocrinology 2007;148:5162–4.
[50] Berg AH, Combs TP, Du X, et al. The adipocyte-secreted protein Acrp30 enhances hepatic insulin action. Nat Med 2001;7:947–53.
[51] Weyer C, Funahashi T, Tanaka S, et al. Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and hyperinsulinemia. J Clin Endocrinol Metab 2001;86:1930–5.
[52] Trakada G, Steiropoulos P, Nena E, et al. Plasma visfatin levels in severe obstructive sleep apnea-hypopnea syndrome. Sleep Breath 2009;13: 349–55.
[53] Kazem YM, Shebini SM, Moaty MI, et al. Sleep deficiency is a modifiable risk factor for obesity and cognitive impairment and associated with elevated visfatin. J Med Sci 2015;3:315–21.