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Short sleep duration is associated with high energy and total lipid intake in obese women: a pilot study

A curta duração do sono está associada ao elevado consumo energético e de lipídios totais em mulheres com obesidade: um estudo piloto

Erika Duarte Grangeiro[1], Mariana Silva Trigueiro[1], Leysimar de Oliveira Siais[1], Hilana Moreira Paiva[1], Eliane Lopes Rosado[1]

[1] Universidade Federal do Rio de Janeiro, Instituto de Nutrição Josué de Castro, Departamento de Nutrição e Dietética. Rio de Janeiro, RJ, Brasil. Correspondence to: ED GRANGEIRO. E-mail: <erikawduartegs@gmail.com>.

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ABSTRACT

Objective
To evaluate the influence of self-reported sleep duration on ghrelin secretion and nutritional indicators in obese women.

Methods
This is an observational study, including 36 adult women with obesity. Sleep duration was reported while completing the general questionnaire. Dietary, laboratory, anthropometric, and body composition indicators, and resting metabolic rate, were evaluated. For statistical analysis, sleep duration data were grouped into tertiles: less than six (first tertile); equal to or above six; and less than eight (second tertile); equal to or greater than eight hours of sleep per day (third tertile). The indicators were compared for the different ranges of the sleep duration.

Results
There was no significant difference when comparing anthropometric, laboratory, and energy expenditure indicators between sleep tertiles. However, women with shorter sleep duration (less than 6 hours per day) had a higher mean caloric intake, compared with the tertile of eight hours or more of sleep per day. For total lipid intake, the mean consumption was higher in the first tertile (up to six hours a day).

Conclusion
Sleeping less than six hours a day led to an increase in energy and lipid intake in obese women. However, it did not change the plasma ghrelin concentration.

Keywords: Eating. Ghrelin. Obesity. Resting metabolic rate. Sleep.

RESUMO

Objetivo

Avaliar a influência da duração de sono autorrelatada na secreção de grelina e indicadores nutricionais na obesidade.

Métodos

Trata-se de um estudo observacional, incluindo 36 mulheres adultas com obesidade. A duração do sono foi relatada durante o preenchimento do questionário de dados gerais. Foram avaliados indicadores dietéticos, laboratoriais, antropométricos e de composição corporal, além da taxa metabólica de repouso. Para análise estatística, os dados de duração de sono foram agrupados em tercis, sendo menor do que seis (primeiro tercil), igual ou acima seis e menor do que oito (segundo tercil), igual ou maior do que oito horas de sono por dia (terceiro tercil). Os indicadores supracitados foram comparados entre as diferentes faixas dos tercis de duração de sono.

Resultados

Não houve diferença significativa ao comparar os indicadores antropométricos, laboratoriais e do gasto de energia, entre os tercis de sono. Porém, mulheres com menor tempo de duração do sono (menos de 6 horas por dia) apresentaram maior média da ingestão calórica, comparado com o tercil de oito horas ou mais de sono por dia. Para a ingestão de lipídios totais, a média de consumo foi maior no primeiro tercil (até seis horas por dia).

Conclusão

Dormir menos do que seis horas por dia levou ao aumento na ingestão energética e de lipídios em mulheres com obesidade, porém, não alterou a concentração de grelina plasmática.

Palavras-chave: *Ingestão de alimentos. Grelina. Obesidade. Taxa metabólica de repouso. Sono.*

INTRODUCTION

Obesity is a chronic disease with rising prevalence in Brazil and the world [1]. In 2016, more than 1.9 billion adults around the globe presented elevated body mass. Among those, more than 650 million were diagnosed with obesity [2]. In Brazil, the number of individuals with high body weight grew 30.8% in the last 12 years, corresponding to 55.7% of the total population, more than half of it. For obesity, the percentage rose 8% in 12 years, especially for women (20.7%) compared with men (18.7%) [3].

A complex illness unleashed by multiple factors and frequently resulting from a positive energy balance, obesity is related to the imbalance between the ingestion and expenditure of body energy [4,5]. Other etiological agents, such as genetic modifications, environmental factors, and hormonal deregulations, are relevant [6-8].

Moreover, evidence shows that alterations in sleep duration disrupt the circadian rhythm, possibly favoring body weight gain and obesity [9]. Transversal studies suggest a relationship between shorter sleep durations (less than six hours a night) and elevated body weight. In contrast, prospective studies show inconclusive results, leaving this association unclear [10-13].

Possible relations between reduced sleep duration and excessive weight are reported in connection to hormonal deregulation. They might lead to alterations in the secretion of regulatory appetite hormones, such as increasing ghrelin levels (orexigenic hormone) and energy expenditure, thus modifying food consumption [14]. Analyses are heterogenous in the connection between declared reduced sleep duration and ghrelin secretion in individuals with excessive weight and suggest the need for more studies on the theme [15,16].

Thus, investigating the association between reported sleep duration and obesity is greatly important, including the analysis of the hormones connected with hunger, food consumption, biochemical and anthropometric parameters, and Energy Expenditure (EE). This is the present study's

objective, which contains a review of the updated and pertinent literature adequate to presenting the problem and highlighting its relevance.

METHODS

The study was approved by the Research Ethics Committee of the Hospital Clementino Fraga Filho (HUCFF) under protocol CAAE 97290918.2.0000.5257, opinion 3.015.648, and published in the Brazilian Record of Clinical Trials under number RBR-7k6v3v. The present study is observational and analytic, and it was carried out in a sample of a larger study. It is a randomized and parallel clinical trial.

We evaluated adult women from 20 to 45 years old, with degrees I and II of obesity (BMI between 30 and 39.9 kg/m^2) [17], of any race or color, before menopause, without recent alterations in body weight (± three kilos in the last three months), and that have not gone into diets for losing weight in the last three months or undergone bariatric surgery. Athletes, users of medication or supplementation for weight control, smokers, drinkers, women with cardiac or respiratory complications, diagnosed with diabetes *Mellitus* types 1 and 2, uncontrolled hypothyroidism (laboratory markers out of normality), pregnant women, lactating women, illiterate, or those with cancer, and severe hepatopathy and nephropathy were considered ineligible. Women who reported severe binge eating, as assessed by the Binge Eating Scale (BES), were also not selected.

Participants who did not comply with the entire protocol proposed during the intervention or with intercurrences that interfered with their participation in the larger study (clinical trial) were excluded. The research was published on the webpage of the *Instituto de Nutrição Josué de Castro* (Institute of Nutrition Josué de Castro), where the recruitment took place.

Data collection happened in Institute of Nutrition Josué de Castro's *Laboratório de Avaliação Nutricional* (Laboratory for Nutritional Assessment). The first meeting aimed to check eligibility criteria, apply BES, check body weight and Body Mass Index (BMI). At the occasion, participants received general orientation on the research, filling in the three-day dietary registers, preparation for blood collection, EE, and body composition evaluation. The researchers also collected information like sleep duration with questionnaires on general, nutritional, and clinical data. Forms for registering their diets for three days were handed in to the participants, who were expected to return them on the first day of the intervention.

In the second meeting, the women came to the *Laboratório de Análises Clínicas - Faculdade de Farmácia* (Laboratory of Clinical Analyses - Faculty of Pharmacy) for drawing blood after fasting for 12 hours. They were subsequently conducted to Laboratory for Nutritional Assessment for anthropometric, body composition, and EE evaluations.

Sleep duration was investigated in the interviews (participant and responsible researcher), as the general questionnaire was filled up. The duration of habitual sleep at night was questioned.

Participants filled in forms of dietary records for three non-consecutive days – two typical and one atypical day – for the assessment of eating habits. The nutritionists revised the records to check for the possible omission of information (number of meals, added sugar, size of spoon, consumed portions, and type of preparation) and notes that could generate posterior doubt (few registered items, unknown food items, mistakes in informing home measures, and absence of any meal) [18].

The assessments of energy, protein, carbohydrates, lipids, Monounsaturated (MUFA), Polyunsaturated (PUFA), and Saturated (SFA) fatty acids, cholesterol, and fibers were carried out in the software for diet analyses DietProClínico® (version 6.1). The chemical composition of food

items, obtained from the dietary registers, was compared to the Recommended Daily Intake (RDI). The total consumption of fiber and fatty acids was contrasted with the recommendation, and the values considered adequate were as follows: 25 g/day of dietary fiber; SFA below 10% of the Total Energy Intake (TEI); MUFA from 15 to 20% of TEI; and PUFA from 6 to 11% of TEI [19,20].

The BES was applied, translated, adapted, and validated to observe the magnitude of eating behaviors in each individual at the beginning of the research, given that 47% of the obese population may present a pattern of binge eating that compromises adequate eating habits [21,22].

The body weight, height, Waist Circumference (WC), and body composition were measured by trained professionals in the morning after fasting for 12 hours. The body weight was evaluated with light clothes and without shoes, using the electronic platform scale Filizola® model Personal Line 200, with a 200 kg capacity and 50-gram precision. The height was determined with a portable vertical anthropometer Alturexata® with a one-millimeter precision. To verify the stature, the participant stood up without shoes, joined feet, relaxed arms, and head in the Frankfort plane [23].

The WC was analyzed using a flat steel anthropometric tape with a total extension of two meters and one-millimeter precision, brand Sanny®, model TR4011, and verified at the medium point between the last rib and iliac crest [17]. The BMI was classified according to the cutting points suggested by the World Health Organization [24,25].

The evaluation of body composition was performed with a multi-frequency BIA (Biodynamics® model 450) [26]. For estimating Fat-Free Mass (FFM), a predictive equation validated for obese women was employed: FFM (kg) = 0.0015 (height [cm] 2) – 0.344 (resistance) + 0.140 (weight) – 0.158 (age) + 20.387 [27,28]. After the calculation, The Body Fat Mass (BFM) was determined by the difference of the total body mass, considering the model of two body compartments (fatty and fat-free).

The EE was evaluated with a calorimeter with a pulmonary function analyzer (Vmax 29®, ViasysHealthcare, EUA) [29]. The orientations were for starting preparation three days before the evaluation, including not drinking alcohol, maintaining daily activities, avoiding intense physical activity and exercise in general, and following specific nutritional guidelines which stressed not consuming food with excessive fat, protein, and the main sources of caffeine, as well as fasting for 12 hours at night [30].

The measures were taken in the morning, in a room with ambient temperature maintained at the thermoneutral zone, controlled humidity, soft lighting, lack of noise, and the participant in dorsal decubitus or a slightly elevated posture, according to the need [31,32]. Before the analyses began, the participant rested for 20 minutes, and the mask (canopy) connected to the calorimeter was positioned. The Volumes of Carbon Dioxide (VCO_2) and Oxygen (VO_2) were measured for 30 minutes with the participant laying down without moving and not considering the first five minutes. An equation described by Weir (1949) [33] was also employed to determine the body's Resting Metabolic Rate (RMR): ((3.9 x VO_2 L/minute) + (1.1 x VCO_2 L/minute)) x 1440.

Blood samples were collected in the morning after a minimum fasting period of 12 hours and a maximum of 14 hours. Other factors evaluated were glucose (Glucose PAP Liquiform, Labtest Diagnóstica®), insulin (Beckham®), total cholesterol (TC) (Cholesterol Liquiform, Labtest Diagnóstica®), triglycerides (TG) (Triglycerides Liquiform, Labtest Diagnóstica®), and High-Density Lipoproteins (HDL-c) (HDL LE, Labtest Diagnóstica®). Active ghrelin was analyzed with Elisa, using the commercial kit E-EL-H2002®.

The considered concentrations of Low-Density Lipoprotein (LDL-c) were calculated by Laboratory of Clinical Analyses-Faculty of Pharmacy with the equation proposed by Friedwald et

al. [34]. Insulin Resistance (IR) was estimated with the HOMA-IR method = fasting insulin serum concentration (μU/mL) x fasting glycemia (mmol/L) / 22.5, and analyzed according to Stern et al. (2005) and Matthews et al. (1985) [35,36]. Glycemia results were analyzed according to the *Sociedade Brasileira de Diabetes* (Brazilian Society of Diabetes) guidelines. Lipidemia was evaluated according to the Update for Brazilian Guidelines for Dislipidemia and Preventing Atherosclerosis [37,38]. Insulin Sensitivity (IS) was estimated using the Quantitative Insulin Sensitivity Check Index (QUICKI) method, QUICKI = 1/(LOG (fasting insulin serum concentration) + LOG (fasting glycemia) [39].

The results were expressed as mean and standard deviation. Normality was assessed by comparing the mean values with the Kolmogorov-Smirnorv test. The test for outliers in active ghrelin was carried out with GraphPad Prism 8.3. Afterward, the results for the variable sleep duration were grouped in tertiles between percentile 33 (P33) and percentile 66 (P66). The distribution of tertiles was as follows: the first included women with less than six hours of sleep a night; the second comprised those who slept six to eight hours a night; and the third included eight or more hours a night.

After evidencing the gaussian distribution (normal), the homogeneity of variables with Levene's test, and the division of tertiles, the variables were compared among the different tertiles of sleep duration with variance analysis (ANOVA one way), followed by Tukey's post-hoc tests. The program of statistical analysis was SPSS (Statistical Package for Social Science, IBM Corporation, NY) version 22.0, considering p-value <0.05.

RESULTS

Among the 276 women who were interested in participating in the study, 156 met the eligibility criteria, but 120 gave up or presented intercurrences and were excluded from the study. The main reasons for non-inclusion were: BMI of less than 30 kg/m^2 or more than 40 kg/m^2, associated chronic diseases, menopause, smoking, lactating, alcoholism, bariatric surgery post-operatory procedures, weight loss of more than three kilos in the last three months, continuous use of medication for controlling anxiety and appetite, and corticotherapy.

Among the eligible participants, 58 were included and 40 women concluded the study. After the ghrelin analysis, outlier values were excluded, and 36 participants finalized the study (Figure 1).

The tertile groups presented similarities in age and anthropometric indicators, in body weight (p=0.452) and WC (0.607). The participants presented diagnoses of obesity degrees I and II (BMI between 30.00 and 39.99 kg/m^2), as well as an increased risk for metabolic complications associated with the accumulation of fat in the abdominal region (WC of more than 88 cm) [40].

No difference was observed when comparing the anthropometric indicators of GE among the sleep tertiles. Biochemical results show that different sleep strands did not lead to significant alterations in these indicators. The comparisons between concentrations of fasting active ghrelin in the tertiles also did not show significant statistical differences. The values of glucose were normal. However, the HOMA-IR results presented IR. The plasmatic lipidic profile was according to the recommendations for all tertiles [38,41] (Table 1).

Table 2 allows the comparison of the habitual dietetic ingestion between different sleep tertiles. Analyzing the total energy consumption, a statistical difference between tertiles was found, with the first tertile being different from the third (p=0.021), but not from the second (p=0.176). This shows that the average ingestion of energy is smaller when the sleep duration is reduced (less than six hours a day).

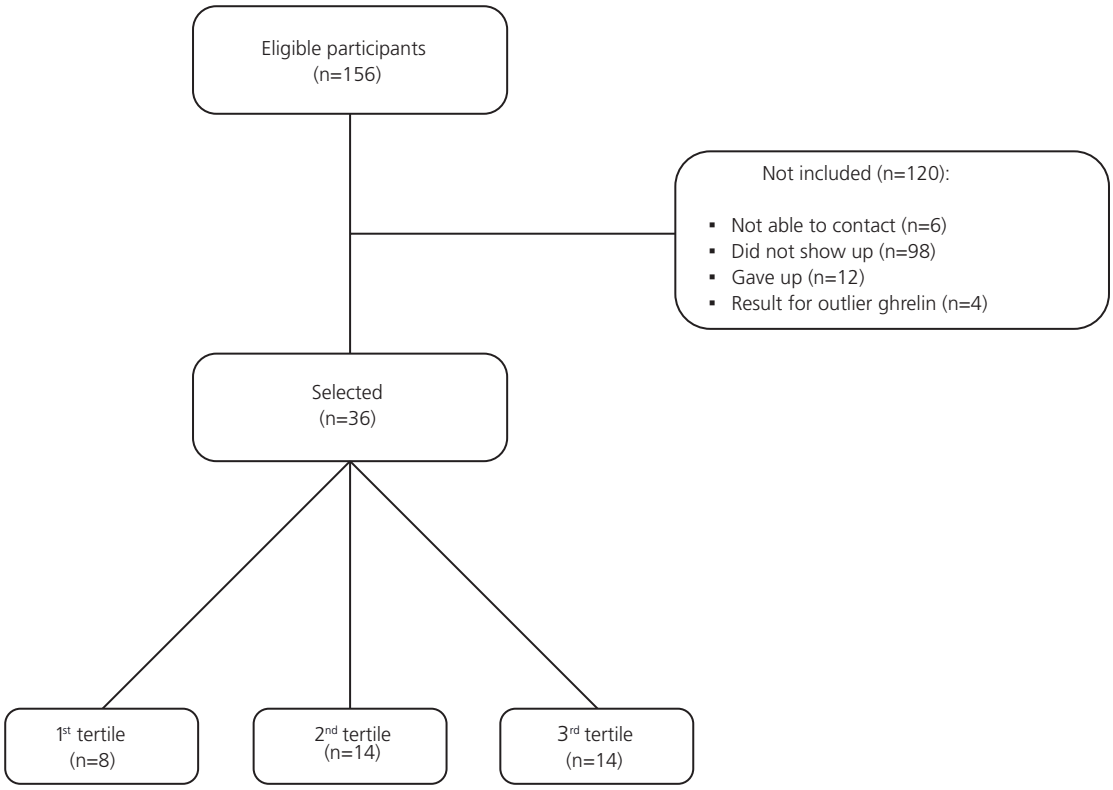

Figure 1 – Recruitment and selection flowchart.

Table 1 – Comparison of anthropometric, energetic expenditure, and laboratory indicators among sleep duration tertiles.

Indicators	First tertile (n=8)	Second tertile (n=14)	Third tertile (n=14)	*p*-value
Age (years)	33.37 (±6.58)	29.64 (±8.88)	29.14 (±7.85)	0.468
Anthropometric and energy expenditure				
Weight (Kg)	92.75 (±7.62)	93.85 (±10.48)	89.34 (±9.70)	0.452
BMI (Kg/m²)	35.24 (±2.81)	35.07 (±4.07)	34.99 (±4.30)	0.990
WC (cm)	97.05 (±10.85)	99.63 (±9.20)	100.97 (±6.93)	0.607
RMR (kcal)	1491.58 (±116.58)	1515.28 (±182.80)	1437.63 (±137.26)	0.405
Laboratory				
Ghrelin (pg/mL)	98.13 (±103.62)	113.45 (±97.24)	139 (±100.52)	0.614
TC (mg/dL)	188.87 (±38.03)	164.14 (±23.94)	174.57 (±32.43)	0.219
LDL (mg/dL)	114.87 (±10.85)	95.71 (±17.74)	106.14 (±26.03)	0.258
HDL (mg/dL)	52.00 (±12.46)	48.28 (±11.89)	47.35 (±9.00)	0.626
TG (mg/dL)	110.50 (±33.83)	100.71 (±22.53)	105.42 (±32.03)	0.747
Glucose (mg/dL)	87.75 (±11.29)	87.35 (±7.88)	91.28 (±22.10)	0.778
Insulin (μU/mL)	19.81 (±8.05)	24.41 (±14.35)	19.15 (±8.97)	0.432
HOMA IR	4.30 (±1.83)	5.32 (±3.16)	4.30 (±2.12)	0.508

Note: Caption: First tertile: <6 hours of sleep/night; Second tertile: ≥6<8 hours of sleep/night; Third tertile: ≥8 of sleep/night. Results expressed as means and standard deviation. The comparison between groups was carried out with the ANOVA one way test, considering $p<0.05$ as statistically significant. BMI: Body Mass Index; HDL: High-Density Lipoprotein; HOMA IR: Homeostasis Model Assessment Index; LDL: Low-Density Lipoprotein; RMR: Resting Metabolic Rate; TC: Total Cholesterol; TG: Triglycerides; WC: Waist Circumference.

Table 2 – Comparison of feeding consumption indicators among sleep tertiles.

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Indicators	First tertile (n=8)	Second tertile (n=14)	Third tertile (n=14)	*p*-value
Energy (kcal)	2310.54 (±544.63)	1934.14 (±503.98)	1729.81 (±366.20)	**0.028**
Carbohydrates (% TEI)	47.45 (±8.20)	52.34 (±5.79)	50.87 (±5.07)	0.212
Protein (% TEI)	16.48 (±4.51)	16.71 (±4.55)	17.61 (±3.01)	0.769

Table 2 – Comparison of feeding consumption indicators among sleep tertiles.

Indicators	First tertile (n=8)	Second tertile (n=14)	Third tertile (n=14)	*p*-value
Lipids (% TEI)	37.02 (±5.02)	31.28 (±5.39)	33.00 (±2.77)	**0.023**
Cholesterol (mg)	420.07 (±171.22)	301.52 (±142.12)	304.40 (±113.17)	0.124
SFA (% TEI)	11.75 (±2.21)	9.92 (±2.08)	10.53 (±1.96)	0.152
MUFA (% TEI)	10.22 (±2.27)	9.02 (±2.47)	9.62 (±1.81)	0.461
PUFA (% TEI)	5.98 (±2.88)	6.79 (±2.16)	7.33 (±2.12)	0.433
Fibers (g)	18.09 (±7.74)	20.27(±5.47)	18.19 (±8.07)	0.685
Meals a day	4.37 (±0.91)	4.57 (±0.93)	3.85 (±0.86)	0.118

Note: First tertile: <6 hours of sleep/night; Second tertile: ≥6<8 hours of sleep/night; Third tertile: ≥8 of sleep/night. Results expressed as means and standard deviation. The comparison between groups was carried out with the ANOVA one way test, considering $p<0.05$ as statistically significant. MUFA: Monounsaturated Fatty Acids; PUFA: Polyunsaturated Fatty Acids; SFA: Saturated Fatty Acids; TEI: Total Energy Intake.

Regarding the consumption of carbohydrates, total fibers, and proteins, as well as the number of meals a day, the results were similar among tertiles. In general, carbohydrate and protein ingestion was within the recommended levels, but fiber consumption did not reach 25 grams a day.

A significant difference was observed among the tertiles of total lipid ingestion, and the first tertile was different from the second ($p=0.017$), but not from the third ($p=0.119$). The average consumption of lipids was larger when sleep was in the first tertile (up to six hours a day) when compared to the second tertile (six to eight hours a day). No difference was found for the ingestion according to the sleep tertile for lipidic fractions. Cholesterol, SFA, and MUFA ingestion were inadequate in every tertile, with no difference among them [37,38].

DISCUSSION

The literature is inconsistent regarding the results of short sleep duration's influence on ghrelin secretion in obese individuals [15,42]. Ghrelin is an orexigenic peripherical hormone that activates receptors mostly found in the center of appetite (hypothalamus and pituitary gland), and acts in energetic homeostasis, possibly influencing body weight [43,44]. Thus, our data may contribute to research and intervention measures for the growing problem in the world that is overweight, which elevates the risk of metabolic complications [1].

Some studies show that short sleep duration did not change the concentration of ghrelin in obesity [15,45]. The present work also did not associate sleep with growing plasmatic ghrelin in obese women.

A systematic review with meta-analysis including 2,250 individuals showed that obese people with short sleep durations did not show differences in ghrelin values. A limitation, however, is the small number of studies including only obese people [42]. The review affirms the need for more studies confirming these findings. Although the feeding state is the most relevant factor for ghrelin emission, other factors, not sleep, may be related to this result. Thus, caution is recommended for analyzing the issue.

In the literature, another potential mechanism is that short sleep duration may reduce energy expenditure and influence body weight [46]. We did not observe alterations in the TMR, verified by indirect calorimetry, for different sleep durations in obese women. Thus, we also highlight the need for more studies explaining the possible mechanisms involved in this association for adults [47].

Some studies point to gender and age as factors interfering with the association between sleep, hormone secretion, food ingestion, and excessive body fat [48,49]. Thus, one of the present

study's positive points is having analyzed only adult women with obesity. A study by association demonstrated that sleep duration was related to weight gain among women, for which short sleep duration appears to be a risk factor [50]. The present work did not show significant differences between sleep hours and weight, WC, and body composition. Ning et al. [47] showed that among 21,958 adult Chinese individuals, those who slept six or less hours had greater chances of gaining weight and having central adiposity. A limitation of that study is the reduced number of women among those who participated.

As for laboratory markers of glycemia, studies suggest insufficient sleep duration may lead to glycemic deregulation, with blood insulin and glucose rising [51-53]. St-Onge et al. [54], however, show that short sleep duration did not affect glycemia or insulin levels for both women and men. These results are similar to those in our study.

Concerning indicators related to energy consumption, a study by Hart et al. [45] showed that overweight or obese women with short sleep durations had increased protein ingestions and no significant alteration in total energy consumption. Grandner et al. [55] also show a complex relationship between feeding, sleep, and nutritional state, with individuals who sleep five to six hours a night tending to consume more proteins, lipids, carbohydrates, and simple sugar, as well as a smaller quantity of fibers, compared to those who sleep six to eight hours a day.

Studies assessing post-menopausal women showed a negative correlation between sleep duration and total lipid, MUFA, PUFA, SFA, trans fat, and cholesterol consumption. On the other hand, no association between (self-reported) sleep and dietary consumption was observed [56]. Although inconsistent results still exist, the evidence points to an increased tendency of elevated ingestion of total lipids in short sleep duration [14]. Accordingly, the present study observed larger energy and total lipid ingestion in short sleep duration.

CONCLUSION

The study suggests that different sleep durations a day did not influence ghrelin secretion, body weight, BMI, glycidic and lipid profile, carbohydrate, protein, fiber, and lipidic fraction consumption, as well the number of meals a day. However, sleeping less than six hours a day increased the caloric and total lipidic profile in obese women, which may hinder the reduction and maintenance of body weight in the mid and long term. We highlight that, despite the limitation of the sample size in the study, the results of the association demonstrate it is a positive pilot, and more studies are needed on this theme.

REFERENCES

1. Afshin A, Forouzanfar MH, Reitsma MB, Sur P, Estep K, Lee A, et al. Health Effects of Overweight and Obesity in 195 Countries over 25 Years. N Engl J Med. 2017;377(1):13-27.
2. World Health Organization. Obesity and overweight [Internet]. Genève: Organization; 2016 [cited 2018 Aug. 25]. Available from: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight
3. Ministério da Saúde (Brasil). VIGITEL Brasil 2019: vigilância de fatores de risco e proteção para doenças crônicas por inquérito telefônico [Internet]. Brasília: Ministério; 2020 [cited 2021 Apr 19]. Available from: http://bvsms.saude.gov.br/bvs/publicacoes/vigitel_brasil_2019_vigilancia_fatores_risco.pdf
4. Associação Brasileira para Estudo da Obesidade e da Síndrome Metabólica. Diretrizes brasileiras de obesidade 2016 / ABESO. [Internet]. São Paulo: Associação; 2016 [cited 2019 Oct 19]. Available from: http://www.abeso.org.br/diretrizes
5. Hall KD, Guo J. Obesity Energetics: Body Weight Regulation and the Effects of Diet Composition. Gastroenterology. 2017;152(7):1718-27.

6. Arroyo-Johnson C, Mincey KD. Obesity epidemiology trends by race/ethnicity, gender, and education: National Health Interview Survey, 1997-2012. Gastroenterol Clin North Am. 2016;45(4):571-9.
7. Garaulet M, Ordovás JM, Madrid JA. The chronobiology, etiology and pathophysiology of obesity. Int J Obes. 2010;34(12):1667-83.
8. Speakman JR. Functional analysis of seven genes linked to body mass index and adiposity by genome-wide association studies: a review. Hum Hered. 2013;75(2):57-79.
9. McHill AW, Wright KP. Role of sleep and circadian disruption on energy expenditure and in metabolic predisposition to human obesity and metabolic disease. Obes Rev Off J Int Assoc Study Obes. 2017;18:15-24.
10. Nishiura C, Hashimoto H. A 4-Year Study of the Association between Short Sleep Duration and Change in Body Mass Index in Japanese Male Workers. J Epidemiol. 2010;20(5):385-90.
11. Sperry SD, Scully ID, Gramzow RH, Jorgensen RS. Sleep duration and waist circumference in adults: a meta-analysis. Sleep. 2015;38(8):1269–76.
12. Vgontzas AN, Fernandez-Mendoza J, Miksiewicz T, Kritikou I, Shaffer ML, Liao D, et al. Unveiling the longitudinal association between short sleep duration and the incidence of obesity: the penn state cohort. Int J Obes. 2014;38(6):825-32.
13. Xiao Q, Arem H, Moore SC, Hollenbeck AR, Matthews CE. 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(11):1600-10.
14. Dashti HS, Scheer FA, Jacques PF, Lamon-Fava S, Ordovás JM. Short sleep duration and dietary intake: epidemiologic evidence, mechanisms, and health implications. Adv Nutr Bethesda Md. 2015;6(6):648-59.
15. Littman AJ, Vitiello MV, Foster-Schubert K, Ulrich CM, Tworoger SS, Potter JD, et al. Sleep, ghrelin, leptin and changes in body weight during a 1-year moderate-intensity physical activity intervention. Int J Obes. 2007;31(3):466-75.
16. St-Onge M-P, Pizinger T, Kovtun K, RoyChoudhury A. Sleep and meal timing influence food intake and its hormonal regulation in healthy adults with overweight/obesity. Eur J Clin Nutr. 2019;72(Suppl 1):76-82.
17. World Health Organization. Obesity: preventing and managing the global epidemic [Internet]. Genève: Organization; 2000 [cited 2019 Oct 19]. Available from: https://apps.who.int/iris/handle/10665/42330
18. Yokoo EM, Pereira RA, Veiga GV, Nascimento S, Costa RS, Marins VMR, et al. Proposta metodológica para o módulo de consumo alimentar pessoal na pesquisa brasileira de orçamentos familiares. Rev Nutr. 2008;21(6):767-76.
19. Fats and fatty acids in human nutrition. Report of an expert consultation. FAO Food Nutr Pap. 2010 [cited 2019 Nov 19];91:1-166. Available from: https://www.ncbi.nlm.nih.gov/pubmed/21812367
20. Trumbo P, Schlicker S, Yates AA, Poos M, Food and Nutrition Board of the Institute of Medicine, The National Academies. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. J Am Diet Assoc. 2002;102(11):1621-30. https://doi.org/10.1016/s0002-8223(02)90346-9
21. Blundell JE, Gillett A. Control of food intake in the obese. Obes Res. 2001;(9 Suppl 4):263S-270S.
22. Freitas S, Lopes CS, Coutinho W, Appolinario JC. Tradução e adaptação para o português da Escala de Compulsão Alimentar Periódica. Braz J Psychiatry. 2001;23(4):215-20.
23. Lohman T, Roche A, Martorell R, editors. Anthropometric standardization reference manual. Champaign: Human Kinetics Books; 1991. p. 90.
24. World Health Organization. Physical status: the use and interpretation of anthropometry [Internet]. Genève: Organization; 1995 [cited 2019 Nov 19]. Available from: https://apps.who.int/iris/handle/10665/37003
25. Centers for Disease Control and Prevention. Defining adult overweight & obesity [Internet]. Atlanta: CDC. [Last reviewed 2022 June 3; cited 2019 Oct 21]. Available from: https://www.cdc.gov/obesity/basics/adult-defining.html#print
26. Lukaski HC, Johnson PE, Bolonchuk WW, Lykken GI. Assessment of fat-free mass using bioelectrical impedance measurements of the human body. Am J Clin Nutr. 1985;41(4):810-7.
27. Segal KR, Van Loan M, Fitzgerald PI, Hodgdon JA, Van Itallie TB. Lean body mass estimation by bioelectrical impedance analysis: a four-site cross-validation study. Am J Clin Nutr. 1988;47(1):7-14.
28. Gray NT, Bartlett JM, Kolasa KM, Marcuard SP, Holbrook CT, Horner RD. Nutritional status and dietary intake of children with sickle cell anemia. Am J Pediatr Hematol Oncol. 1992;14(1):57-61.

29. Ferrannini E. The theoretical bases of indirect calorimetry: a review. Metabolism. 1988;37(3):287-301.

30. Casas-Agustench P, López-Uriarte P, Bulló M, Ros E, Gómez-Flores A, Salas-Salvadó J. Acute effects of three high-fat meals with different fat saturations on energy expenditure, substrate oxidation and satiety. Clin Nutr Edinb Scotl. 2009;28(1):39-45.

31. Frankenfield D, Roth-Yousey L, Compher C. Comparison of predictive equations for resting metabolic rate in healthy nonobese and obese adults: a systematic review. J Am Diet Assoc. 2005;105(5):775-89.

32. Wahrlich V, Anjos LA. Aspectos históricos e metodológicos da medição e estimativa da taxa metabólica basal: uma revisão da literatura. Cad Saude Publica. 2001;17(4):801-17.

33. Weir JBDB. New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol. 1949;109(1-2):1-9.

34. Friedwald W, Levy R, Fredrickson D. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18:499-502.

35. Stern SE, Williams K, Ferrannini E, De Fronz RA, Bogardus C, Stern MP. Identification of individuals with insulin resistance using routine clinical measurements. Diabetes. 2005;54(2):333-9.

36. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28(7):412-9.

37. Faludi AA, Izar MCO, Saraiva JFK, Chacra APM, Bianco HT, Afiune A, et al. Atualização da Diretriz Brasileira de Dislipidemias e Prevenção da Aterosclerose – 2017. Arq Bras Cardiol. 2017;109:1-76.

38. Sociedade Brasileira de Diabetes. Diretrizes da Sociedade Brasileira de Diabetes [Internet]. São Paulo: SDB; 2022 [cited 2022 Feb 1]. Available from: https://diretriz.diabetes.org.br/

39. Katz A, Nambi SS, Mather K, Baron AD, Follmann DA, Sullivan G, et al. Quantitative insulin sensitivity check index: a simple, accurate method for assessing insulin sensitivity in humans. J Clin Endocrinol Metab. 2000;85(7):2402-10.

40. World Health Organization. Obesity: preventing and managing the global epidemic [Internet]. Geneve: Organization; 1998 [cited 2018 Aug 25]. Available from: https://apps.who.int/iris/handle/10665/63854

41. Stern RC, Eisenberg JD, Wagener JS, Ahrens R, Rock M, Pico G, et al. A comparison of the efficacy and tolerance of pancrelipase and placebo in the treatment of steatorrhea in cystic fibrosis patients with clinical exocrine pancreatic insufficiency. Am J Gastroenterol. 2000;95(8):1932-8.

42. Lin J, Jiang Y, Wang G, Meng M, Zhu Q, Mei H, et al. Associations of short sleep duration with appetite-regulating hormones and adipokines: a systematic review and meta-analysis. Obes Rev. 2020;21.

43. Mihalache L, Gherasim A, Niță O, Ungureanu MC, Pădureanu SS, Gavril RS, et al. Effects of ghrelin in energy balance and body weight homeostasis. Hormones (Athens). 2016;15(2):186-96.

44. Khatib MN, Gaidhane S, Gaidhane AM, Simkhada P, Zahiruddin QS. Ghrelin O Acyl Transferase (GOAT) as a Novel Metabolic Regulatory Enzyme. J Clin Diagn Res JCDR. 2015;9(2):LE01-5.

45. Hart CN, Carskadon MA, Demos KE, Van Reen E, Sharkey KM, Raynor HA, et al. Acute Changes in Sleep Duration on Eating Behaviors and Appetite-Regulating Hormones in Overweight/Obese Adults. Behav Sleep Med. 2015;13(5):424-36.

46. Hursel R, Rutters F, Gonnissen HKJ, Martens EAP, Westerterp-Plantenga MS. Effects of sleep fragmentation in healthy men on energy expenditure, substrate oxidation, physical activity, and exhaustion measured over 48 h in a respiratory chamber. Am J Clin Nutr. 2011;94(3):804-8.

47. Ning X, Lv J, Guo Y, Bian Z, Tan Y, Pei P, et al. Association of sleep duration with weight gain and General and Central Obesity Risk in Chinese adults: a prospective study. Obes Silver Spring Md. 2020;28(2):468-74.

48. Gallegos JV, Boege HL, Zuraikat FM, St-Onge M-P. Does sex influence the effects of experimental sleep curtailment and circadian misalignment on regulation of appetite? Curr Opin Endocr Metab Res. 2021;17:20-5.

49. Watanabe M, Kikuchi H, Tanaka K, Takahashi M. Association of short sleep duration with weight gain and obesity at 1-year follow-up: a large-scale prospective study. Sleep. 2010;33(2):161-7.

50. Lyytikäinen P, Rahkonen O, Lahelma E, Lallukka T. Association of sleep duration with weight and weight gain: a prospective follow-up study. J Sleep Res. 2011;20(2):298-302.

51. Spiegel K, Leproult R, L'hermite-Balériaux M, Copinschi G, Penev PD, Van Cauter E. Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin. J Clin Endocrinol Metab. 2004;89(11):5762-71.

52. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. Lancet. 1999;354(9188):1435-9.

53. Tsereteli N, Vallat R, Fernandez-Tajes J, Delahanty LM, Ordovas JM, Drew DA, et al. Impact of insufficient sleep on dysregulated blood glucose control under standardised meal conditions. Diabetologia. 2022;65(2):356-65.

54. St-Onge M-P, O'Keeffe M, Roberts AL, RoyChoudhury A, Laferrère B. Short sleep duration, glucose dysregulation and hormonal regulation of appetite in men and women. Sleep. 2012;35(11):1503-10.

55. Grandner MA, Jackson N, Gerstner JR, Knutson KL. Dietary nutrients associated with short and long sleep duration. Data from a nationally representative sample. Appetite. 2013;64:71-80.

56. Grandner MA, Kripke DF, Naidoo N, Langer RD. Relationships among dietary nutrients and subjective sleep, objective sleep, and napping in women. Sleep Med. 2010;11(2):180.

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CONTRIBUTORS

ED GRANGEIRO and EL ROSADO worked in the study's conception and design, analysis and interpretation of data, revision and approval of the manuscript's final version. LO SIAIS, MS TRIGUEIRO and HM PAIVA worked in the analysis and interpretation of data and in the approval of the manuscript's final version.