Sleep Science

ORIGINAL ARTICLE

Is there an association between body composition, basal metabolic rate, and sleep in elderly patients with and without obstructive sleep apnea?

Existe associação entre a composição corporal, taxa metabólica basal e sono em pacientes idosos com e sem apneia obstrutiva do sono?

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ABSTRACT

Objectives: Aging is associated with morphological, functional, biochemical, and psychological alterations. The aim of this study was to verify whether basal metabolic rate (BMR) is associated with body composition and sleep in elderly men with or without obstructive sleep apnea (OSA). **Methods:** We evaluated 95 elderly men (69.1 ± 3.4 years old) divided into OSA and non-OSA groups. The protocol included polysomnography, BMR (performed the morning after polysomnography) and body composition evaluations. **Results:** The results showed that body mass index (BMI) and REM sleep percentage were positively correlated with BMR and negatively related to stage 1 sleep percentage. This model can explain 40% of the BMR variation observed. BMR was positively correlated with morphologic variables and sleep efficiency. In addition, we observed negative correlations of BMR with sleep latency and wake after sleep onset. When comparing the groups, the OSA group had higher values for weight, fat mass, arousals, and apnea -hypopnea index. The OSA group had lower percentage of REM sleep and levels of fat-free mass. **Conclusion:** This study identified significant associations among BMR, BMI and some sleep variables (stage 1 and REM). Despite the differences between groups in AHI, arousals some sleep parameters, weight and fat mass, BMR was not different between the OSA and non-OSA group.

Keywords: aging, body composition, obstructive, sleep apnea, sleep disorders.

RESUMO

Objetivos: O envelhecimento está associado a alterações morfológicas, funcionais, bioquímicas e psicológicas. O objetivo deste estudo foi verificar se a taxa metabólica basal (TMB) está associada com a composição corporal e o sono em homens idosos, com ou sem apnéia obstrutiva do sono (AOS). **Métodos:** Foram avaliados 95 homens idosos (69,1 ± 3,4 anos de idade), distribuídos em grupos de AOS e não-AOS. O protocolo incluiu polissonografia, TMB (realizada pela manhã, após a polissonografia) e avaliações de composição corporal. Resultados: Os resultados mostraram que o índice de massa corporal (IMC) e percentual de sono REM foi positivamente correlacionado com a TMB e negativamente relacionado com a porcentagem do estágio 1 do sono. Este modelo pode explicar 40% da variação observada na TMB. A TMB foi positivamente correlacionada com as variáveis morfológicas e eficiência do sono. Além disso, observou-se correlações negativas da TMB com a latência do sono e a vigília após o início do sono. Ao comparar os grupos, o grupo AOS apresentou valores mais elevados de peso, massa gorda, despertares e índice de apnéia e hipopnéia. O grupo OSA apresentou menor porcentagem de sono REM e os níveis de massa livre de gordura. **Conclusão:** Este estudo identificou associações significativas entre a TMB, IMC e algumas variáveis do sono (estágio 1 e REM). Apesar das diferenças entre os grupos em IAH, despertares, alguns parâmetros do sono, peso e massa gorda, a TMB não foi diferente entre o OSA e grupo de não-OSA.

Descritores: apneia do sono tipo obstrutiva, composição corporal, envelhecimento, transtornos do sono.

INTRODUCTION

During the aging process, morphological, functional, biochemical, and psychological alterations generate a progressive decrease in the individual's capacity to adapt to the environment, increasing the vulnerability to and incidence of diseases[1].

Among the morphological alterations, there is a gradual and progressive decrease in fat-free mass (FFM), ranging from 15% to 45% from 30 to 80 years of age[2]. This represents a loss of approximately 6.3% for each decade of life[3]. With respect to the metabolic changes, basal metabolic rate (BMR) decreases approximately 1 to 2% per decade[4,5].

Some authors report during the aging process, BMR decreases as a result of the reduction in lean tissue (about 5% lower in the elderly compared to young adults)[2,6] or the simultaneous increase in fat mass[7]. However, although there is a positive correlation between FFM and BMR, other physiological factors, such as sleep characteristics, may influence body composition and energy

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Received: June 27, 2013; Accepted: January 23, 2014.

consumption. According to Siegel[8], there is an inverse relationship between body weight and energy consumption. Thus, small-sized mammals would have high metabolic rates that could increase sleep quantity. However, verification of these associations in humans requires further evaluation, especially in the elderly population, because these characteristics (reduced muscle mass, decreased BMR, and poor sleep quality) become more common with advancing age.

In addition, there is consensus in the literature that the prevalence of sleep disorders increases with age[9-11], and about 25% of the population over 65 years old have more than five apneic events per hour of sleep[12,13]. However, many sleep disorders, especially obstructive sleep apnea (OSA), appear as a result of comorbidities such as obesity, heart disease, and chronic pain that are very common in the elderly[14].

Thus, when compared to the young population, the elderly show some changes in sleep characteristics. These changes include decreases in slow wave sleep (stages 3 and 4), REM sleep, and total night sleep time and increases in arousals, daytime sleepiness and naps, which affect daily activities[11,15].

It is important to mention that REM sleep, for which the duration progressively decreases with aging, is a cognitively restorative sleep and is related to increased core temperature and increased brain metabolism (about 25% of total energy consumption), and other features of sleep are pronounced in this stage[16].

According to Bernhard[17], REM sleep is involved in the replenishment of depleted ATP reserves in the hippocampus and associated brain structures, so most of the ATP generated during REM sleep is conserved for use during wakefulness. There are probably several other areas of the brain that may also be re-energized during this stage of the sleep cycle. In addition, glucose is transferred from the body to the brain via the bloodstream during phasic REM sleep and during increases in physiological arousal that occur while a person is awake.

Therefore, the objective of this study was to verify whether BMR is associated with body composition and sleep in elderly men with or without OSA.

METHOD

Participant Recruitment and Intervention

Volunteers were recruited by advertisements placed in different types of media (radio, television, and newspaper). The sample consisted of 95 elderly men ages 65 to 80, divided into two groups: those with OSA (apnea-hypopnea index ≥ 15 events/h) and those without OSA (apnea-hypopnea index < 15 events/h), according to the criteria of The International Classification of Sleep Disorders 2[18]. The inclusion criteria were 65 - to 80 - year-old men who were sedentary, nonsmokers, non-users of antidepressants and anti-anxiety drugs, and who were allowed to participate in the study after medical evaluation and approvable after blood and heart tests (thyroid function within normal range).

This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects were approved by the Research Ethics Committee of the *Universidade Federal de São Paulo* (#1107/06). Written informed consent was obtained from all volunteers.

Procedures

The volunteers were subjected to two stages of evaluation. The first stage consisted of evaluation of the inclusion criteria: (a) initial screening (by phone) composed of questions about age, use of medications, and presence of chronic diseases; (b) interview to explain the research protocol and to obtain informed consent; (c) clinical consultation with a geriatrician; (d) resting and stress electrocardiogram; and (e) blood test (total cholesterol, LDL, HDL, VLDL, triglycerides, glucose, T3, T4, free T4, and TSH) to confirm the patient's general health status.

The second stage consisted of the following evaluations: polysomnography, BMR performed the morning after polysomnography, and physical evaluations (body composition and anthropometric measurements).

Polysomnography

Polysomnography consisted of simultaneous evaluation by electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG), electrocardiogram (ECG), nasal airflow with a nasal cannula, transducer and thermistor, respiratory effort (thoracic and abdominal), body movements, and oxygen saturation. The polysomnography recordings were performed and staged according to standardized criteria for sleep studies. Comparative and simultaneous analysis of EEG, EOG, and EMG allowed us to differentiate between different stages of sleep. The equipment used was the EMBLA digital system (EMBLA S7000, Embla Systems Inc., CO, USA)[19].

Basal Metabolic Rate

We used open-circuit indirect calorimetry while controlling the brightness, noise and temperature (24 to 26 °C) of the polysomnography room. Sampling occurred upon awakening, before the removal of polysomnography electrodes, while the volunteers awake and supine. To perform the tests, volunteers were instructed to fast for 12 hours, not drink caffeinated or alcoholic drinks, and not exercise for 24 hours before evaluation. The O_2 consumption was measured for 15 minutes and was examined the final 10 minutes in intervals of 30 seconds, using a computerized metabolic system (Fitmate® - COSMED - Italy) with a facial mask[20]. BMR was calculated from oxygen consumption with a fixed respiratory quotient of 0.85, using a modified Weir equation[21].

Body Composition and Anthropometric Evaluation

The anthropometric evaluation was performed by measuring height and weight. Body composition was acquired by plethysmography, using the Body Composition System Bod Pod®. This system consists of an electronic scale, a plethysmograph, a calibration cylinder, and computer software. It uses total body density to calculate body measurements[22].

Statistical Analysis

Statistical analysis was performed using the Statistica for Windows software (Statsoft, Inc., version 7.0). Initially, we performed the Kolmogorov-Smirnov test to verify normality before a descriptive

analysis. A Pearson correlation and multivariate linear regression was used to analyze the relationships between main study variables (BMR, sleep architecture, and body composition). The Student's *t*-test for independent samples was used to compare the OSA and non-OSA groups. Data are presented as the mean ± standard deviation, and the significance level was set at 5% ($p \leq 0.05$).

RESULTS

Table 1 shows the descriptive data from the sample and comparison between the study groups. The OSA and non-OSA groups showed significant differences in some morphologic variables. The OSA group had higher weight, BMI, fat mass, body fat and lower body fat-free than the non-OSA group. Regarding sleep variables, REM sleep latency, arousals, and apnea-hypopnea index were higher in the OSA group, while REM sleep percentage was lower in the OSA group when compared to the non-OSA group. In biochemical measurements the OSA group showed significant increased in glucose than the non-OSA group.

Complete Sample

In the linear regression equations with the complete sample (n = 95), independent variables initially included in the model to best describe BMR were weight; fat mass; total sleep time; sleep efficiency; sleep latency; arousals; wake after sleep onset; percentage of stages 1, 2, 3, 4, and REM; apnea-hypopnea index; and periodic leg movements index.

The three variables that significantly influenced BMR were BMI (kg/m^2), stage 1 (%), and REM sleep (%), and the final model was:

BMR = 101.84 + 46.55 BMI - 16.51 stage 1 + 14.95 REM sleep

This causation model estimated that every 1 kg/m^2 increment of BMI increases the BMR by 46 kcal/day at rest. Every 1% decrement of stage 1 sleep increases the BMR by 16 kcal/day at rest, and every increase of 1% of the REM sleep increases the BMR by 15 kcal/day at rest. The model could explain 40% of the variation of BMR ($r^2 = 0.40$). More details are described in Table 2.

Table 1. Comparison between Non-OSA and OSA groups.

Variables	Total Group (n = 95)	Non-OSA group (n = 41)	OSA group (n = 54)	*p*
Age (years)	69.10 ± 3.41	68.78 ± 3.22	69.35 ± 3.56	0.421
Morphological Variables				
Weight (kg)	73.70 ± 11.63	70.62 ± 9.21	76.04 ± 12.77*	0.024
Height (m)	1.69 ± 0.07	1.70 ± 0.06	1.69 ± 0.08	0.208
BMI (kg/m2)	25.54 ± 4.33	24.50 ± 2.72	26.90 ± 3.63*	< 0.001
Body Fat (%)	26.93 ± 8.53	24.64 ± 7.31	27.93 ± 7.06*	0.029
Body Fat-free (%)	73.18 ± 8.54	75.36 ± 7.31	72.25 ± 7.11*	0.040
Fat Mass (kg)	20.19 ± 8.15	17.72 ± 6.56	21.56 ± 7.96*	0.014
Fat-free Mass (kg)	53.49 ± 7.94	52.84 ± 6.21	54.49 ± 7.90	0.275
Metabolic Variable				
Basal Metabolic Rate (kcal/day)	1483.82 ± 340.94	1418.20 ± 322.00	1533.64 ± 357.78	0.102
Sleep Variables				
Total Sleep Time (min)	319.04 ± 56+14	315.85 ± 55.27	321.46 ± 57.18	0.633
Sleep Efficiency (%)	75.35 ± 11.71	75.36 ± 12.62	75.34 ± 11.08	0.994
Sleep Latency (min)	15.59 ± 16.58	13.92 ± 11.21	16.86 ± 19.72	0.395
REM Sleep Latency (min)	96.93 ± 65.62	77.68 ± 47.65	111.53 ± 73.61*	0.012
Arousals (events/h)	18.84 ± 10.54	13.75 ± 6.84	22.70 ±11.25*	< 0.001
WASO (min)	88.83 ± 44.06	89.80 ± 49.08	88.10 ± 40.29	0.854
Stage 1 (%)	6.84 ± 5.28	5.75 ± 3.42	7.67 ± 6.24	0.080
Stage 2 (%)	58.34 ± 9.01	56.76 ± 8.73	59.54 ± 9.12	0.137
Slow Wave Sleep (%)	15.35 ± 7.24	15.68 ± 6.77	15.09 ± 7.62	0.697
REM Sleep (%)	19.47 ± 7.09	21.81 ± 7.13	17.69 ± 6.58*	0.005
AHI (events/h)	20.52 ± 16.88	7.17 ± 4.32	30.65 ± 15.77*	< 0.001
PLM (events/h)	11.84 ± 21.69	12.03 ± 21.25	11.70 ± 22.23	0.941
Epworth (value)	7.34 ± 4.10	7.34 ± 3.58	7.33 ± 4.48	0.992
Biochemical Measurements				
Glucose (mmol/L)	105.41 ± 17.21	100.22 ± 13.79	109.14 ± 18.55*	0.017
total cholesterol	195.70 ± 32.21	194.00 ± 27.54	196.92 ± 35.42	0.680
HDL	49.98 ± 11.58	50.47 ± 13.17	49.62 ± 10.41	0.738
LDL	118.06 ± 30.28	118.22 ± 21.87	117.94 ± 35.34	0.966

Continued Table 1.

VLDL	26.85 ± 10.97	26.14 ± 12.41	27.36 ± 9.90	0.613
Triacilglycerols	134.40 ± 54.81	131.00 ± 61.85	136.84 ± 49.65	0.629
T3	129.54 ± 18.88	128.76 ± 17.22	130.05 ± 20.20	0.834
T4	7.87 ± 1.34	7.65 ± 1.39	8.00 ±1.32	0.416
TSH	2.21 ± 1.61	2.39 ± 1.95	2.10 ±1.38	0.589
Free T4	1.11 ± 0.17	1.06 ± 0.17	1.14 ± 0.17	0.160

Kg: Kilogram; m: Meter; BMI: Body mass index; kcal: Kilocalories; WASO: Wake after sleep onset (min); REM: Rapid eye movement; AHI: Apnea-hypopnea Index (events/h); OSA: Obstructive sleep apnea; PLM: Periodic leg movements index (events/h); HDL: High-density lipoprotein; LDL: Low-density lipoprotein; VLDL: Very-low-densitylipoprotein; T3: Triiodothyronine; T4: Thyroxine; TSH: Hyroid-stimulating hormone; Free T4: Free thyroxine. Data presented as the mean ± standard deviation. *t*-Test for independent samples, * significant results, $p \leq 0.05$.

Table 2. Multiple linear regression model of all subjects (OSA and non-OSA groups) with the basal metabolic rate as dependent variable.

Variables	Beta (β)	T	*p*
BMI (kg/m^2)	46.55	5.76	< 0.001*
Stage 1 (%)	-16.51	-2.88	0.005*
REM Sleep (%)	14.95	3.47	0.001*

Multiple Linear Regression; * Significant results; OSA: Obstructive sleep apnea; BMI: Body mass index; REM: Rapid eye movement; $r^2 = 0.40$.

In the analysis of associations based on all subjects, BMR was positively correlated with the following variables: weight ($r = 0.38$, $p < 0.001$), BMI ($r = 0.42$, $p < 0.001$), FFM ($r = 0.29$, $p = 0.004$), fat mass ($r = 0.29$; $p = 0.004$) and sleep efficiency ($r = 0.30$, $p = 0.003$). In addition, BMR was negatively correlated with sleep latency ($r = -0.20$, $p = 0.004$) and with wake after sleep onset ($r = -0.29$; $p = 0.004$). The significant positive correlation between FFM and BMR is shown in Figure 1.

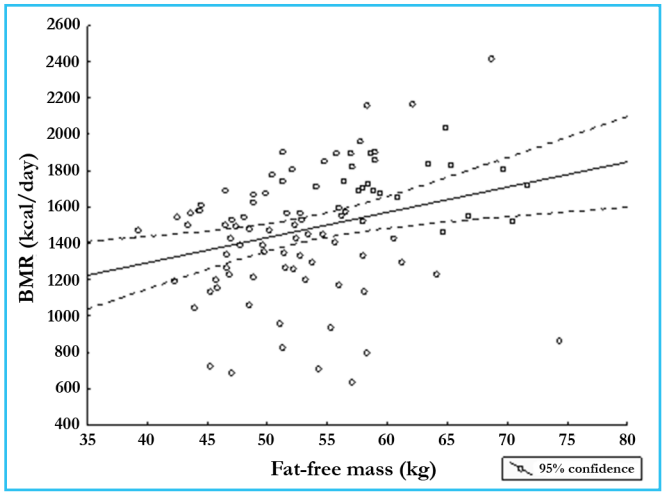

Figure 1. Association between BMR (kcal/day) and fat-free mass (kg) in the complete sample ($r = 0.29$, $p = 0.004$).

DISCUSSION

Aging is associated with several factors that influence energy expenditure and lead to decreases in BMR, including physiological and hormonal changes, alterations in body composition, and decreased peripheral action, food intake and physical activity level[5].

In the present study, the significant results were the positive associations between BMR and weight, BMI, FFM (kg), and fat mass (kg). Some of these associations have also been observed in previous studies. In particular, the decrease in FFM and concomitant increase in body fat have been associated with BMR[7,23,24].

However, it is not clear whether the decline in BMR is due entirely to a decrease in FFM or whether it also reflects a reduction in the metabolic activity of lean tissue. Thus, FFM and fat mass cannot fully account for the lower BMR in the elderly, suggesting that aging per se is associated with an alteration in tissue energy metabolism[7].

Furthermore, many factors confound BMR measurements, such as food, temperature, alcohol, and caffeinated beverages[21], but these factors were controlled in this study.

The results in both groups confirmed previous findings of BMR values between 1350 and 1550 kcal/day among the healthy elderly[25-28]. However other studies have observed higher values, between 1550 and 1700 kcal/day, in elderly men with similar characteristics to the ones in this study[7,29,30].

Other significant associations were observed between BMR and some stages of sleep. BMR was positively correlated with percentage of REM sleep and negatively correlated with percentage of stage 1 sleep in our entire elderly sample. These results highlight the importance of good sleep quality, especially for older men who show more sleep derangements, including increased respiratory disorders and arousals[31,32].

REM sleep was significantly reduced in the OSA group, possibly because of the higher frequency of apneic events in this group. There is no evidence in the literature implicating decreased REM sleep as a predisposing factor or causal contributor to BMR decreases in the elderly, as observed in this study. Nevertheless, the sleep deprivation due to OSA could be influencing the reduction of BMR.

On the other hand, stage 1 of NREM sleep exerts a negative influence on BMR, such that the less time an elderly subject remains in stage 1, the higher the basal metabolism the next day. This result, which is extremely interesting, still has no scientific explanation. However, one may speculate that stage 1 does not contribute directly to a good restoration of sleep, being only a route of passage from wakefulness to sleep. Therefore, it is possible to hypothesize that an increase in this stage would cause a decrease in basal metabolism due to possible alterations in the two stages of consciousness (sleep and wakefulness).

The OSA group was characterized as having more negative modifications in the polysomnography than the non-OSA

group, despite not showing any changes in BMR. Ucok et al.[33] demonstrated the increased Resting Metabolic Rate (RMR) in patients with OSAS than Simple Snorer, however the population evaluated wasn't elderly patients. Regarding morphological variables, the OSA group showed higher weight, BMI, fat mass, and body fat-free (%). In terms of the sleep architecture, the number of arousals and REM sleep latency increased in the OSA group, reducing the percentage of REM sleep when compared to the non-OSA group[14,34].

OSA appears to be an influential factor because although the altered sleep pattern, BMI and weight of the elderly in the OSA group, is also accompanied by large amounts of fat mass (kg) and FFM (kg). Furthermore, changes in sleep did not influence BMR when comparing both groups in this study. This lack of effect on BMR may have been due to sleep parameters exerting less influence on basal metabolism than morphological variables. For instance, the abundant lean tissue (kg) in the OSA group means that the main component of FFM is very metabolically active, as observed in this and other studies[5,7,23].

Despite differences between groups, the three variables that influenced BMR in both groups were BMI, stage 1 and REM sleep. For this reason, the regression model that best represents the whole group included these variables.

When the regression model was performed separately there was no statistically significant values possibly due to apnea not be the variable that most directly influences BMR. Thus, there is evidence that REM sleep similarly affected both groups in this study independently of sleep disturbances, suggesting that age is more important in metabolic changes. In this sense, new studies are needed to clarify these findings.

Another association investigated in several studies was the relationship between sleep pattern and obesity[35,36]. Hasler et al.[37] argued that there is a longitudinal association between short sleep duration and future weight gain, which occurred in young patients with an average age of 35, though this rate was decreased in the elderly population.

According to Gangwisch et al.[35] and Kohatsu et al.[38], possible explanations for the differences in the relationship between sleep patterns and body composition in the middle-aged and elderly might be due to some characteristic factors of aging, such as increased mortality associated with obesity, longitudinal sleep changes, or demographic and lifestyle differences between these populations. These distinctions between age groups could partially explain why this study did not find more alterations, especially BMR alterations in the elderly, even when these individuals had no chronic diseases.

In conclusion, this study identified significant associations between BMR and BMI and between BMR and some sleep variables (stage 1 and REM sleep). The OSA group showed lower quality sleep patterns and higher body weight with more fat mass, without a difference in BMR when compared to the non-OSA group.

This study was the first to present a model for the relationships among variables (metabolic, morphologic, and sleep variables) for a specific population, i.e., the elderly. This demonstrates there is a possible influence of REM sleep on energy metabolism, which had not been previously demonstrated. Therefore, the prospect of similar relationships in other age groups warrants investigation. In addition, effects of non-pharmacological treatments of OSA, such as physical exercise[39] and use of CPAP[40], should be evaluated with respect to these parameters.

Acknowledgements

We would like to acknowledge all the volunteers for participating in this study; geriatrician Carlos André, M.D. for evaluating the volunteers' health status; and Professor of Statistics Angela Paes for her assistance with the statistical analysis. This study was supported by Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP #06/05210-0; CEPID #98/14303-3), Associação de Fundo e Incentivo à Pesquisa (AFIP), Centro de Estudos em Psicobiologia e Exercício (CEPE), Centro de Estudo Multidisciplinar em Sonolência e Acidentes (CEMSA) and Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq). MTM and ST are recipients of the CNPq fellowship.

The authors declare that they have no conflict of interest.

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