*British Journal of Nutrition* (2022), 128, 646–652 doi:10.1017/S0007114521003615


# Association between sitting/lying down, standing, walking time and number of steps per day with the hormonal profile and resting energy expenditure of women with obesity living in a low-income region

Mateus de Lima Macena[1], André Eduardo da Silva Júnior[2], Dafiny Rodrigues Silva Praxedes[1], Laís Gomes Lessa Vasconcelos[3], Isabele Rejane de Oliveira Maranhão Pureza[2], Telma Maria de Menezes Toledo Florêncio[1] and Nassib Bezerra Bueno[1]*

[1]*Faculdade de Nutrição, Universidade Federal de Alagoas, Maceió, Alagoas, Brasil*
[2]*Escola Paulista de Medicina, Universidade Federal de São Paulo, São Paulo, SP, Brasil*
[3]*Hospital Universitário Professor Alberto Antunes, Maceió, Alagoas, Brasil*

*(Submitted 2 June 2021 – Final revision received 20 August 2021 – Accepted 6 September 2021 – First published online 16 September 2021)*

## Abstract

Reducing sedentary behaviour (SB) and increasing physical activity (PA) by sitting less and standing/walking more is advised to prevent chronic diseases. However, the mechanisms underlying this recommendation are not well established, especially in individuals with obesity living in low-income regions. The present study evaluated whether there are associations between PA indicators (PAI – standing time, walking time and the number of steps/d) and SB indicators (SBI – sitting/lying down time) with the hormonal profile and resting energy expenditure (REE) of adult women living in a low-income region. This is a cross-sectional study. We collected data on hormones (insulin resistance, leptin and thyroid axis), body composition (tetrapolar bioimpedance), REE (indirect calorimetry), and PAI and SBI (triaxial accelerometers, ActivPAL). Multivariable linear models adjusting for age and fat-free mass were performed. Fifty-eight women (mean age of 31 years and BMI of 33 kg/m$^2$) were included. The mean sitting/lying down time and standing time were 16·08 and 5·52 h/d, respectively. Sitting/lying down time showed a direct association with free thyroxine ($FT_4$) ($\beta = 0{\cdot}56$ ng/dl; 95 % CI = −1·10, −0·02). Standing time showed a direct association with $FT_4$ ($\beta = 0{\cdot}75$ ng/dl; 95 % CI = 0·01; 1·48) and inverse association with free triiodothyronine ($\beta = -2{\cdot}83$ pg/ml 95 % CI = −5·56, −0·10). There were no associations between PAI and SBI with the REE, insulin resistance, leptin and thyroid-stimulating hormone. Thus, decreased SB is associated with thyroid hormones levels but not with REE, insulin resistance or leptin in women with obesity living in low-income regions.

**Key words:** **Body composition: Exercise: Hormones: Indirect calorimetry**

Obesity has a multifactorial origin, with increasing prevalence in recent decades worldwide and a high burden on the public health system due to its significant morbidity and mortality[1]. Over the years, increasing physical activity (PA) energy expenditure has become a commonly applied strategy to combat obesity, especially through the practice of physical exercises, a planned structured repetitive PA aimed at improving or maintaining one or more components of physical fitness[2–4].

The practice of physical exercise brings health benefits to individuals[5]. Physical exercise can provide several metabolic changes, including thermogenesis, $\beta$-adrenergic stimulation, hepatic mitochondrial activity, decreased blood pressure, improved insulin resistance and decreased circulating leptin levels[5–10]. Due to its effects on lean mass, physical exercise is purported to increase an individual's resting energy expenditure (REE)[11]. However, there is evidence that differences in REE between individuals are not fully explained by differences in body composition and aerobic fitness alone[12].

On the other hand, it is noteworthy that physical exercise is not the only component of PA that plays a role in human health. PA is defined as any type of movement performed by the skeletal muscles that result in energy expenditure, at various intensities, durations and frequencies of muscle contractions. Low PA, which is related to sedentary behaviour (SB), is gaining attention for its deleterious effects on human health[4]. SB occurs through sitting/lying down or reclining while awake and leads to low energy expenditure (approximately 1·0–1·5 times metabolic equivalent of task (MET)), being associated with an increased

**Abbreviations:** FFM, fat-free mass; FT3, free triiodothyronine; FT4, free thyroxine; HOMA-IR, homoeostatic insulin resistance model; MET, metabolic equivalent of task; PA, physical activity; PAI, PA indicator; REE, resting energy expenditure; SB, sedentary behavior; SBI, SB indicator; TSH, thyroid-stimulating hormone.

* **Corresponding author:** Nassib Bezerra Bueno, email nassib.bueno@fanut.ufal.br

risk of overweight/obesity, arterial hypertension and type 2 diabetes[13–15]. A decrease in SB is associated with decreased postprandial levels of glucose and insulin[16]. In addition, sitting/lying down time alone has also been linked to diseases and conditions associated with obesity, such as greater risks of morbidity and mortality from type 2 diabetes, cancer and CVD and is sometimes mentioned as being as harmful as or even worse than smoking[17].

Thus, decreasing SB is an essential goal for improving the health of populations, and the practice of physical exercises is the most usual measure adopted[18]. However, several governmental organisations, such as the National Health Services of the UK[19], and expert panels, such as the Mayo Clinic[20] advises that to decrease SB, individuals could implement simple tasks throughout their days, such as stand while in public transport and while talking on the phone, work in a standing desk, etc. Hence, it is important to investigate the associations between these SB indicators (SBI) and PA indicators (PAI), such as standing time, walking time, the number of steps/d and not only physical exercise , on the human health. Considering the recommendations to decrease SB and increase PAI to prevent chronic diseases[19,20], it is interesting to investigate the relationship between these indicators with REE, insulin resistance, leptin and thyroid hormones, especially in individuals with obesity.

The study of these associations gains further importance in the context of low-income regions. Individuals of lower socioeconomic status are the ones with the lowest probability of following fitness advice, given their difficulties in having access to adequate facilities to practice physical exercises or their inability to go for a walk in a poor and violent neighbourhood, even in developed countries[21]. In Brazil, adult women living in low-income regions have the highest obesity prevalence and are the group with the lowest frequency of physical exercise in their free time[22]. Furthermore, taking objective measurements of PAI and SBI in these populations may be difficult given the high costs associated with the use of properly developed devices such as triaxial accelerometers. Thus, the objective of this study was to evaluate whether there is an association between PAI (standing, walking time and the number of steps/d) and SBI (sitting/lying down time) with the hormonal profile and REE of adult women with obesity living in a low-income region.

## Materials and methods

### Ethical aspects

The study was approved by the Research Ethics Committee of the Universidade Federal de Alagoas (no. 2535·99). All participants were informed about the procedures and signed an informed consent form.

### Study type

This was a cross-sectional study.

### Study population

The sampling was non-probabilistic, and recruitment was performed through advertisements in the community and direct invitations to women who had some ties with the Center for Recovery and Nutritional Education (CREN) located in the 7th administrative region of Maceió-Alagoas, Brazil, which has the lowest human development index in the municipality. The CREN serves about 100 children with chronic malnutrition in 24 slums in the city and provides health care to their mothers and other guardians.

Adult women (19–44 years old) with obesity and stable weight for at least 1 month were included. Obesity was defined as the presence of two of the three following criteria: I. BMI $\geq 30$ kg/m$^2$ but $< 45$ kg/m$^2$; II. waist circumference $\geq 88$ cm and III. percentage of fat defined by a bioimpedance of $\geq 35$ %. The exclusion criteria were use of chronic medications (antidiabetic, antihypertensive, antiretroviral, immunosuppressive and antidepressants); currently postmenopausal, pregnant or breastfeeding, a history of having undergone surgical intervention for weight loss, or currently performing a physical exercise regimen.

### Anthropometric evaluation and body composition

Body weight data were collected using a digital scale (Filizola, São Paulo, Brazil), while height was determined using a portable stadiometer. BMI was calculated as recommended by the WHO (1995)[23]. Body composition was assessed using tetrapolar bioimpedance, mono-frequency (50 kHz) Sanny BI 1010 (Sanny). The participants were instructed to fast for 10 h and not perform any type of physical exercise or drink alcohol in the preceding 24 h. For the examination, four electrodes were attached to the patient's right hemibody while in a supine position, wearing light clothing, barefoot and not wearing metallic ornaments. The resistance and reactance data obtained in the evaluation were inserted in the Bio Tectronic Sanny software version 1.2.2 for the determination of the percentage and quantity (in kilograms) of fat-free mass (FFM) and body fat.

### Hormonal analysis

Through blood sample collection by peripheral venepuncture, the hormones insulin, leptin, thyroid-stimulating hormone (TSH), free thyroxine ($FT_4$) and free triiodothyronine ($FT_3$) were measured. The chemiluminescence technique (Unissel DxI 800, Beckman Coulter) was used to determine the insulin and thyroid hormone (TSH, $FT_4$ and $FT_3$) levels. The enzyme immunoassay method (Linco Research) was used to determine leptin levels. The homoeostatic insulin resistance model (HOMA-IR) was calculated from plasma levels of insulin and fasting glucose using the equation developed by Matthews *et al.* (1985) to assess insulin sensitivity[24].

### Resting energy expenditure

REE was measured by indirect calorimetry using a gas analyser (Quark, Cosmed). The equipment was calibrated before each test session according to the manufacturer's specifications, with gases at a concentration of 20·9 % $O_2$ and 5 % $CO_2$ and a 3 l syringe with a secondary manometer adjustable to 40–60 psi.

The participants were transported by car to the Laboratory of Sciences Applied to Sports at the Universidade Federal de

British Journal of Nutrition

Alagoas. Measurements were made between 07.00 and 09.00 in a room with lighting and temperature adjusted to ensure their comfort. The participants followed the same preparation used to perform the electrical bioimpedance test, since the measurements took place at the same time. Vital signs of blood pressure (HEM-4030, Omron) and axillary temperature (Techline, São Paulo) were measured to identify fluctuations in hyperthermia (> 37·5°C) and tachycardia (> 100 bpm). The volumes of inspired oxygen ($VO_2$) and the volume of expired carbon dioxide ($VCO_2$) were captured for 15 min using a silicone mask. The data from the first 5 min were discarded to avoid discrepancies due to unfamiliarity with the location or equipment[25]. After $VO_2$ and $VCO_2$ were measured, the REE was calculated using the equation proposed by Weir (1949)[26].

### Physical activity

PA was estimated using triaxial accelerometer motion sensors (ActivPAL, Glasgow, UK) that measured the acceleration in three body axes: anteroposterior, lateral and vertical. These accelerometers were placed on the participants in the CREN facilities and fixed at the midpoint between the inguinal line and the upper edge of the patella exactly on the anterior muscles of the right thigh. The region was previously sanitised using cotton and 70 % hydrated ethyl alcohol, and then the accelerometers were fixed to the skin with the aid of two transparent film dressings (VitaMedical®) that were both hypoallergenic and impermeable to water. The participants used the accelerometers for three consecutive days in the same week in which they underwent the electrical bioimpedance and indirect calorimetry tests. The participants were advised to continue their routine activities as normal and remove the equipment only before engaging in any water-based activities. The data were exported to ActivPAL3™ software version 7.2.32. The system calculates the periods spent sitting/lying down (awake or sleeping), standing and walking every 10 s for the entire time the device was used based on the acceleration of the three-body axes previously mentioned. It also provides an estimated MET value for the entire period in which individuals used the device by multiplying the MET value for each activity by its duration based on the standard values: sitting/lying down (1·25 MET), standing (1·40 MET) and walking at 120 steps per minute (4 MET). For cadences that differed from 120 steps per minute, the following equation was used to calculate the MET estimate:

$$MET.h = (1.4 \times d) + (4 - 1.4) \times \left(\frac{c}{120}\right) \times d$$

whose value of $c$ corresponds to the cadence (steps per minute) and $d$ is the duration of the activity (in hours)[27]. The MET value expressed by the accelerometer analysis is closely related to the concept of the PA ratio (total energy expenditure/REE)[28]. In this study, the total MET value obtained for the 3 d was divided by the total number of hours that individuals used the accelerometer (72 h) to reach an estimated daily MET/h. Such a method for estimating the daily MET/h has already been applied to a sample similar to the one used in the present study and showed good agreement with the total energy expenditure measured by doubly labelled water[29].

### Statistical analysis

All categorical variables are presented as frequencies and continuous variables as means and standard deviations. We performed multivariable linear models to assess the association of SBI (sitting/lying down) and PAI (standing, walking time, number of steps and total MET) with the hormonal profile and REE with the adjustment for age and FFM as a proxy for body composition. A value of $\alpha = 5\,\%$ was adopted. All analyses were performed using the software R v 3·6·1, with the package 'Rcmdr'.

The sample size was determined based on the feasibility of recruitment. Hence, considering our sample size ($n$ 58), an α value of 5 % and a statistical power of 80 %, we would be able to detect an effect size ($f^2$) of 0·14. In a multiple linear regression model, including age, FFM and sitting/lying down time as predictors and REE as outcome, this effect size would yield a total $R^2$ of 30 % and a partial $R^2$ of the main variable (sitting/lying down time) of 10 %. Calculations were conducted using Gpower v. 3.1.9.2 (University of Dusseldorf, Dusseldorf, Germany).

## Results

Fifty-eight women with a mean age of 31 ± 7 years were included. The participants had an average BMI of 33·34 ± 4·11 kg/m$^2$ and an average sitting/lying down time of 16·08 ± 1·44 h/d. The other descriptive characteristics of the sample are presented in Table 1.

In univariable analyses, PAI and SBI were not associated with REE, HOMA-IR, leptin and TSH. Sitting/lying down time was only associated with $FT_4$ ($\beta = -0{\cdot}56$ ng/dl; 95 % CI = −1·08, −0·03; $P = 0{\cdot}03$), while standing time was only associated with $FT_3$ ($\beta = -2{\cdot}85$ ng/dl; 95 % CI = −5·48, −0·22; $P = 0{\cdot}03$).

Regarding multivariable analyses adjusted for age and FFM, the PAI and SBI still did not show association with REE, HOMA-IR, leptin and TSH. In contrast, sitting/lying down time showed a statistically significant association with $FT_4$ ($\beta = -0{\cdot}56$ ng/dl; 95 % CI = −1·10, −0·02, $P = 0{\cdot}04$) as well as and standing time ($\beta = 0{\cdot}75$ ng/dl; 95 % CI = 0·01, 1·48; $P = 0{\cdot}04$). Standing time was also associated with $FT_3$ ($\beta = -2{\cdot}83$ pg/ml; 95 % CI = −5·56, −0·10; $P = 0{\cdot}04$) (Table 2).

About statistical power, the model including sitting/lying down time, age and FFM as predictors, and REE as outcome showed a total $R^2$ of 10 % and the sitting/lying down time showed a partial $R^2$ of 0·3 %.

## Discussion

The present study showed that the sitting/lying down time showed an inverse association with the thyroid hormone $FT_4$, while standing time was directly associated with $FT_4$ and inversely associated with $FT_3$ after adjusting for age and FFM in women with obesity living in low-income regions. PAI and SBI were not associated with REE, HOMA-IR, leptin and TSH levels.

Studies of the relationship between PAI and SBI with thyroid hormones are scarce. Commonly, studies have evaluated the effects of physical exercise on these hormones; however, findings are still controversial[30]. The main associations between thyroid hormone levels and PA could be related to the role of

British Journal of Nutrition

**Table 1.** Descriptive characteristics (Mean values and standard deviations, *n* 58)

| Variables | Mean | Standard deviation |
|---|---|---|
| Age (years) | 31·44 | 7·00 |
| Body weight (kg) | 80·79 | 11·69 |
| Height (cm) | 155·60 | 6·02 |
| BMI (kg/m$^2$) | 33·34 | 4·11 |
| WC (cm) | 100·82 | 10·31 |
| Body fat (%) | 43·81 | 5·09 |
| Body fat (kg) | 35·74 | 8·57 |
| Fat-free mass (kg) | 45·04 | 4·81 |
| REE (kcal/d) | 1538·53 | 290·47 |
| Temperature (ºC) | 35·96 | 0·46 |
| MET/h | 1·45 | 0·06 |
| Sitting/lying time (h/d) | 16·08 | 1·44 |
| Standing time (h/d) | 5·52 | 1·20 |
| Walking time (h/d) | 1·92 | 0·72 |
| Number of steps (thousand/d) | 9·83 | 3·98 |
| Blood glucose (mg/dl) | 79·10 | 9·49 |
| Insulin (μUI/ml) | 14·59 | 7·52 |
| HOMA-IR | 2·89 | 1·63 |
| Leptin (ng/ml) | 45·24 | 25·44 |
| TSH (μUI/ml) | 1·95 | 1·21 |
| $FT_4$ (ng/dl) | 0·92 | 0·14 |
| $FT_3$ (pg/ml) | 2·82 | 0·51 |

WC, waist circumference; REE, resting energy expenditure; MET, metabolic equivalent of task; HOMA-IR, homoeostatic insulin resistance model; TSH, thyroid stimulating hormone; $FT_3$, free triiodothyronine; $FT_4$, free thyroxine.

$FT_3$ in the skeletal muscle, mainly by activating the expression of uncoupling protein-3 genes, which could lead to an increase in REE[31,32]. In addition, $FT_3$ mediates the expression of important genes related to the metabolic activity of muscles, such as the genes of the *α*-myosin heavy chain and sarcoplasmic reticulum Ca ATPase, which are positively regulated, and the genes of the β-myosin heavy chain and phospholamban, negatively regulated, which could increase the efficiency of muscle contraction[33,34].

It is important to note that in our analyses, the SBI 'sitting/lying down time' encompasses sleeping time, which can further confound our findings. However, it is difficult to isolate the possible effects of sleep on the thyroid axis hormonal profile of individuals with obesity. Most studies that correlate these variables assess this relationship in individuals with sleep deprivation or thyroid disorders. Some studies have already shown that sleep deprivation, which was not the case of our sample, could reduce circulating levels of leptin and be associated with increased TSH, $FT_3$ and $FT_4$ levels[35–37].

We hypothesise that the finding that standing time is directly associated with $FT_4$ levels and inversely associated with $FT_3$ levels is due to a regulatory action of these hormones on the REE to keep it within a normal range. $FT_4$ is considered a pro-hormone that depends on the deiodination process to become $FT_3$, the main hormone acting on this axis[38]. One of the main functions of $FT_3$ is to increase REE in several ways, highlighting the decoupling of cellular metabolism from the synthesis of ATP[39]. As standing time could reflect a greater energy need than in sitting/lying down activities, the body may decrease the deiodination process to avoid an increase in energy expenditure, keeping it low instead. This may also explain why the REE did not seem to have been affected by PAI or SBI in the present study.

**Table 2.** Effects of physical activity and sedentary behaviour indicators on the hormonal profile and resting energy expenditure (Coefficient values and 95 % confidence intervals, *n* 58)*

| | REE (kcal/d) | | | HOMA-IR | | | Leptin (ng/ml) | | | TSH (μUI/ml) | | | $FT_4$ (ng/dl) | | | $FT_3$ (pg/ml) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Indicators | *β* | 95 % CI | *P*-value | *β* | 95 % CI | *P*-value | *β* | 95 % CI | *P*-value | *β* | 95 % CI | *P*-value | *β* | 95 % CI | *P*-value | *β* | 95 % CI | *P*-value |
| Sitting/lying down time (h/d) | −250·5 | −1356·9, 855·8 | 0·65 | −2·7 | −9·0, 3·5 | 0·38 | 37·7 | −67·7, 137·2 | 0·45 | −0·8 | −5·7, 4·0 | 0·72 | −0·5 | −1·1, −0·1 | 0·04 | 1·5 | −0·4, 3·6 | 0·13 |
| Standing time (h/d) | −106·9 | −1612·7, 1398·7 | 0·88 | 5·3 | −3·1, 13·8 | 0·21 | −28·4 | −164·1, 107·1 | 0·67 | 1·0 | −5·6, 7·6 | 0·76 | 0·7 | 0·1, 1·4 | 0·04 | −2·8 | −5·5, −0·1 | 0·04 |
| Walking time (h/d) | 451·5 | −2031·0, 2934·0 | 0·71 | 2·0 | −12·1, 16·2 | 0·77 | −7·8 | −232·0, 216·3 | 0·94 | −0·7 | −11·7, 10·2 | 0·89 | 0·4 | −0·8, 1·7 | 0·47 | 0·1 | −4·5, 4·8 | 0·95 |
| Number of steps (thousand/d) | 0·57 | −18·49, 19·63 | 0·95 | 0·02 | −0·08, 0·13 | 0·66 | 0·29 | −1·42, 2·00 | 0·34 | 0·01 | −0·07, 0·09 | 0·73 | 0·01 | −0·01, 0·01 | 0·79 | −0·01 | −0·04, 0·03 | 0·78 |
| MET total | −1·76 | −7·05, 3·52 | 0·50 | −0·01 | −0·03, 0·02 | 0·92 | −0·01 | −0·48, 0·47 | 0·98 | 0·01 | −0·01, 0·03 | 0·55 | −0·01 | −0·01, 0·01 | 0·62 | −0·01 | −0·01, 0·01 | 0·34 |

REE, resting energy expenditure; HOMA-IR, homoeostatic insulin resistance model; TSH, thyroid stimulating hormone; $FT_4$, free thyroxine; $FT_3$, free triiodothyronine; MET, metabolic equivalent of task.
* Multivariable analyses adjusting for age and fat-free mass.

British Journal of Nutrition

Furthermore, we are unaware of any study to date that has evaluated the effects of PAI and SBI on REE. Given that decreasing SBI and increasing PAI is advised to prevent obesity, it would be expected that they could be associated with REE, but the limited existing evidence fail to show this association. Johannsen *et al.*[40] evaluated twenty community-dwelling women, ten with and ten without obesity, and observed that the group with obesity had a longer sedentary time (+2·7 h/d) on weekdays. However, no intergroup differences in REE were found after the adjustment for FFM, indicating that differences in body composition alone could explain the differences in REE. Nevertheless, the authors recognise the lack of statistical power to detect significant differences despite the high effect sizes found. In another investigation, evaluating REE and PA levels in routine contexts without interventions and in individuals of both sexes with mixed body weight classifications and occupations, there seems to be no association between PA measured as counts per minute and REE[41].

Individuals with obesity have disorders in HOMA-IR and leptin levels, and the practice of physical exercise associated with the loss of body fat helps with hormonal control[8,42,43]. Nevertheless, our study, which did not evaluate physical exercise , did not find significant associations between these hormones and the PAI and SBI. Once again, as decreasing SBI and increasing PAI is advised as a way to prevent type 2 diabetes mellitus, it would be expected an association with HOMA-IR. We highlight the lack of studies that have assessed the impact of PAI and SBI time rather than physical exercise on these outcomes.

Regarding the sample size of our study, we should note that our recruitment used a criterion based on feasibility. We chose as primary analysis the relationship between sitting/lying down time and REE with adjustments for age and FFM, which was the model that we used to estimate our statistical power, expecting a partial $R^2$ of 10 % for the sitting/lying down time. However, the effect of this relationship was much smaller than we expected, with a Partial $R^2$ of only 0·3 %. Thus, for our primary variable, we had no statistical power to find significance. It is noteworthy that for this effect size to be statistically significant, 2611 individuals would be needed, which would be impractical. We also emphasise that we were able to find significant associations with secondary variables in our study, indicating that for greater effect sizes, our sample was adequately powered.

The limitations of the present study include the inaccuracy of the triaxial accelerometers for detecting upper-body movements. However, the accelerometers used here are already widely validated in adults and produce reliable estimates for light physical activities, such as walking, sitting and getting up, which was the focus in our current sample[44–47]. Another limitation is that the women spent 72 straight hours with the accelerometers, which raises some concerns. First, we were unable to distinguish the exact sleeping time throughout the period in which the women used the accelerometer, which may prevent us from following the exact definition of SB. Second, 3 d of accelerometer use may be insufficient to accurately represent the PA pattern of each individual, as some studies suggested 5-d use for older adults[48]. A Brazilian study using a wrist-worn accelerometer suggested that, for younger adults, 3 d would sufficiently show light PA patterns[49]. A third limitation is that individuals did not wear the accelerometer on weekend days when they usually show lower PA levels. Ricardo *et al.*[49] also indicated that, for younger adults, the 'week *v* weekend' difference pattern is not observed; rather, it is more common in adults older than 30 years of age. Finally, we emphasize that because our study is cross-sectional, we cannot make inferences of causality between the analysed variables nor we can disregard reverse causation bias.

Despite the limitations of our study, some strengths may be highlighted, such as the fact that our sample was a group of interest, women with obesity of lower economic classes in a developing country, the group with the highest prevalence of obesity worldwide. In addition, we used a reliable objective method to assess five PAI and one SBI in this population living in low-income regions, which in conjunction with REE and the hormonal profile of these women increased our ability to document this debated relationship.

In conclusion, sitting/lying down time was inversely associated with the $FT_4$ levels while standing time was directly associated with $FT_4$ and inversely associated with $FT_3$ levels after the adjustment for age and FFM, in women with obesity living in low-income regions. There were no associations between the other indicators of SB and PA evaluated with REE, leptin and insulin resistance. We highlight the need for more studies that explore the effect of these PAI and SBI on energy expenditure and hormonal profile of individuals in order to elucidate the possible mechanisms that justify the beneficial effects of decreasing SB and increasing PA on the human health.

## Acknowledgements

The authors are grateful to Professors Gustavo Gomes Araújo and João Araújo de Barros Neto for making access to the Laboratory of Applied Science to Sport available for analysis of resting energy expenditure and the use of electrical bioimpedance to estimate body composition, respectively.

This study was partially funded by Fundação de Amparo à Pesquisa do Estado de Alagoas (no. 14/2016) and by the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior with a research grant for MLM and IRP (under the numbers of process 23065.005919/2021-75 and 1793050).

M. L. M. wrote the original draft preparation and was responsible for the reviewing and editing of the manuscript and the formal analysis. A. E. S. J., D. R. S. P., I. R. O. M. P., L. G. L. V. and M. L. M. were responsible for the investigation, methodology and data curation as well as the writing, reviewing and editing of the manuscript. T. M. M. T. F. was responsible for the resources and the writing, and reviewing of the manuscript. N. B. B. provided resources, supervision, conceptualisation, methodology, and the writing, reviewing, and editing of the manuscript. All the authors read and approved the final version of the manuscript.

There are no conflicts of interest.

## References

1. Apovian CM (2016) Obesity: definition, comorbidities, causes, and burden. *Am J Manag Care* **22**, s176–s185.

2. Spiegelman BM & Flier JS (2001) Obesity and the regulation of energy balance. *Cell* **104**, 531–543.
3. Archer E, Lavie CJ & Hill JO (2018) The contributions of 'Diet', 'Genes', and physical activity to the etiology of obesity: contrary evidence and consilience. *Progr Cardiovasc Dis* **61**, 89–102.
4. Caspersen CJ, Powell KE & Christenson GM (1985) Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. *Public Health Rep* **100**, 126–131.
5. Ruegsegger GN & Booth FW (2018) Health benefits of exercise. *Cold Spring Harb Perspect Med* **8**, a029694.
6. Carpio-Rivera E, Moncada-Jiménez J, Salazar-Rojas W, *et al.* (2016) Acute effects of exercise on blood pressure: a meta-analytic investigation. *Arq Bras Cardiol* **106**, 422–433.
7. Chung N, Park M-Y, Kim J, *et al.* (2018) Non-exercise activity thermogenesis (NEAT): a component of total daily energy expenditure. *J Exerc Nutr Biochem* **22**, 23–30.
8. Fedewa MV, Hathaway ED, Ward-Ritacco CL, *et al.* (2018) The effect of chronic exercise training on leptin: a systematic review and meta-analysis of randomized controlled trials. *Sports Med* **48**, 1437–1450.
9. Stevanović J, Beleza J, Coxito P, *et al.* (2020) Physical exercise and liver 'fitness': role of mitochondrial function and epigenetics related mechanisms in non-alcoholic fatty liver disease. *Mol Metab* **32**, 1–14.
10. Zouhal H, Jacob C, Delamarche P, *et al.* (2008) Catecholamines and the effects of exercise, training and gender. *Sports Med* **38**, 401–423.
11. Speakman JR & Selman C (2003) Physical activity and resting metabolic rate. *Proc Nutr Soc* **62**, 621–634.
12. Arciero PJ, Goran MI & Poehlman ET (1985) Resting metabolic rate is lower in women than in men. *J Appl Physiol* **75**, 2514–2520.
13. Tremblay MS, Aubert S, Barnes JD, *et al.* (2017) Sedentary behavior research network (SBRN) – terminology consensus project process and outcome. *Int J Behav Nutr Phys Act* **14**, 75.
14. Ekelund U, Steene-Johannessen J, Brown W, *et al.* (2016) Does physical activity attenuate, or even eliminate, the detrimental association of sitting time with mortality? A harmonised meta-analysis of data from more than 1 million men and women. *Lancet* **388**, 1302–1310.
15. Guo C, Zhou Q, Zhang D, *et al.* (2020) Association of total sedentary behaviour and television viewing with risk of overweight/obesity, type 2 diabetes and hypertension: a dose-response meta-analysis. *Diabetes Obes Metab* **22**, 79–90.
16. Dempsey PC, Larsen RN, Sethi P, *et al.* (2016) Benefits for type 2 diabetes of interrupting prolonged sitting with brief bouts of light walking or simple resistance activities. *Diabetes Care* **39**, 964–972.
17. Vallance JK, Gardiner PA, Lynch BM, *et al.* (2018) Evaluating the evidence on sitting, smoking, and health: is sitting really the new smoking? *Am J Public Health* **108**, 1478–1482.
18. World Health Organization (2010) *Global Recommendations on Physical Activity for Health*. Geneva: World Health Organization.
19. National Health Services of the United Kingdom (2019) Why We Should Sit Less. https://www.nhs.uk/live-well/exercise/why-sitting-too-much-is-bad-for-us/ (accessed August 2021).
20. Mayo Clinic (2020) What are the Risks of Sitting Too Much? https://www.mayoclinic.org/healthy-lifestyle/adult-health/expert-answers/sitting/faq-20058005 (accessed August 2021).
21. Booth GL, Creatore MI, Moineddin R, *et al.* (2013) Unwalkable neighborhoods, poverty, and the risk of diabetes among recent immigrants to Canada compared with long-term residents. *Diabetes Care* **36**, 302–308.
22. Ministério da Saúde (2020) Vigitel Brazil 2019: surveillance of risk and protective factors for chronic diseases by telephone survey: estimates of frequency and sociodemographic distribution of risk and protective factors for chronic diseases in the capitals of the 26 Brazilian states and the Federal District in 2019. Brasília. http://bvsms.saude.gov.br/bvs/publicacoes/vigitel_brasil_2019_vigilancia_fatores_risco.pdf (accessed August 2021). ISBN 978-85-334-2765-5.
23. World Health Organization (1995) *Physical Status: the Use and Interpretation of Anthropometry*. Geneva: World Health Organization.
24. Matthews DR, Hosker JP, Rudenski AS, *et al.* (1985) Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* **28**, 412–419.
25. Borges JH, Langewanmattsr RD, Cirolini VX, *et al.* (2016) Minimum time to achieve the steady state and optimum abbreviated period to estimate the resting energy expenditure by indirect calorimetry in healthy young adults. *Nutr Clin Pract* **31**, 349–354.
26. Weir JB (1949) New methods for calculating metabolic rate with special reference to protein metabolism. *J Physiol* **109**, 1–9.
27. Ainsworth BE, Haskell WL, Whitt MC, *et al.* (2000) Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc* **32**, S498–S504.
28. Food and Agriculture Organization (2001) *Human Energy Requirements. Report of a Joint FAO/WHO/UNU Expert Consultation, Rome. Food and Nutrition Technical Report Series*. Rome: FAO.
29. Macena ML, Pureza IM, Melo IV, *et al.* (2019) Agreement between the total energy expenditure calculated with accelerometry data and the BMR yielded by predictive equations *v.* the total energy expenditure obtained with doubly labelled water in low-income women with excess weight. *Br J Nutr* **122**, 1398–1408.
30. Hackney AC, Davis HC & Lane AR (2016) Growth hormone-insulin-like growth factor axis, thyroid axis, prolactin, and exercise. *Front Horm Res* **47**, 1–11.
31. Cunningham O, McElligott AM, Carroll AM, *et al.* (2003) Selective detection of UCP 3 expression in skeletal muscle: effect of thyroid status and temperature acclimation. *Biochim Biophys Acta* **1604**, 170–179.
32. Schrauwen P & Hesselink M (2003) Uncoupling protein 3 and physical activity:the role of uncoupling protein 3 in energy metabolism revisited. *Proc Nutr Soc* **62**, 635–643.
33. Clement K, Viguerie N, Diehn M, *et al.* (2002) *In vivo* regulation of human skeletal muscle gene expression by thyroid hormone. *Genome Res* **12**, 281–291.
34. Arruda AP, da Silva WS, Carvalho DP, *et al.* (2003) Hyperthyroidism increases the uncoupled ATPase activity and heat production by the sarcoplasmic reticulum Ca2+ −ATPase. *Biochem J* **375**, 753–760.
35. Gary KA, Winokur A, Douglas SD, *et al.* (1996) Total sleep deprivation and the thyroid axis: effects of sleep and waking activity. *Aviat Space Environ Med* **67**, 513–519.
36. Koehler C, Ginzkey C, Kleinsasser NH, *et al.* (2013) Short-term severe thyroid hormone deficiency does not influence sleep parameters. *Sleep Breath* **17**, 253–258.
37. Taheri S, Lin L, Austin D, *et al.* (2004) Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Med* **1**, e62.
38. Kohrle J (2018) Thyroid hormones and derivates: endogenous thyroid hormones and their targets. *Methods Mol Biol* **1801**, 85–104.
39. Kim B (2008) Thyroid hormone as a determinant of energy expenditure and the basal metabolic rate. *Thyroid* **18**, 141–144.


British Journal of Nutrition

40. Johannsen DL, Welk GJ, Sharp RL, *et al.* (2008) Differences in daily energy expenditure in lean and obese women: the role of posture allocation. *Obesity* **16**, 34–39.
41. Pontzer H, Durazo-Arvizu R, Dugas LR, *et al.* (2016) Constrained total energy expenditure and metabolic adaptation to physical activity in adult humans. *Curr Biol* **26**, 410–417.
42. Boyer WR, Johnson TM, Fitzhugh EC, *et al.* (2016) The associations between increasing degrees of HOMA-IR and measurements of adiposity among euglycemic U.S. adults. *Metab Syndr Relat Disord* **14**, 108–113.
43. Farr M, Gavrieli A & Mantzoros C (2015) Leptin applications in 2015: what have we learned about leptin and obesity? *Curr Opin Endocrinol Diabetes Obes* **22**, 353–359.
44. Florez-Pregonero A, Meckes N, Buman M, *et al.* (2017) Wearable monitors criterion validity for energy expenditure in sedentary and light activities. *J Sport Health Sci* **6**, 103–110.
45. Kozey-Keadle S, Libertine A, Lyden K, *et al.* (2011) Validation of wearable monitors for assessing sedentary behavior. *Med Sci Sports Exerc* **43**, 1561–1567.
46. Ryan CG, Grant PM, Tigbe WW, *et al.* (2006) The validity and reliability of a novel activity monitor as a measure of walking. *Br J Sports Med* **40**, 779–784.
47. van Nassau F, Chau JY, Lakerveld J, *et al.* (2015) Validity and responsiveness of four measures of occupational sitting and standing. *Int J Behav Nutr Phys Act* **12**, 144.
48. Aguilar-Farias N, Martino-Fuentealba P, Salom-Diaz N, *et al.* (2019) How many days are enough for measuring weekly activity behaviours with the ActivPAL in adults? *J Sci Med Sport* **22**, 684–688.
49. Ricardo LI, Wendt A, Galliano LM, *et al.* (2020) Number of days required to estimate physical activity constructs objectively measured in different age groups: findings from three Brazilian (Pelotas) population-based birth cohorts. *PLOS ONE* **15**, e0216017.

British Journal of Nutrition