Body composition impact on sleep in young adults: The mediating role of sedentariness, physical activity, and diet

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Abstract

Background

Obesity and short sleep duration have both been related to endocrine and metabolic alterations, type II diabetes mellitus, life-threatening cardiovascular diseases, and impaired daytime functioning and mood. However, the bidirectional relationship between these conditions and underlying mechanisms still remain unclear, especially in young adults.

Objective

This cross-sectional study therefore was aimed at elucidating the potential association of anthropometric and body composition parameters with objective and subjective sleep duration and quality in young sedentary adults, considering the potential mediating role of objectively-measured sedentariness, physical activity, and diet.

Methods

A total of 187 adults aged 18-25 (35.29% men) were included in the study. Body mass index (BMI) and waist-hip ratio were calculated through weight, height, waist and hip circumferences measures. Dual-energy X-ray absorptiometry scanner was used to assess body composition parameters such as lean mass index, fat mass index and visceral adipose tissue mass. Sedentary time, physical activity, and sleep duration and quality were objectively measured using accelerometry, sleep quality also being subjectively measured with the Pittsburgh Sleep Quality Index. Dietary intake was assessed by means of 24h recall questionnaires.

Results

BMI, waist-hip ratio and lean mass index were inversely associated to objectively-measured total sleep time and sleep efficiency (p < 0.05). Sedentary time moderated by sex explained the effects of BMI on total sleep time such that a high BMI was related to higher sedentariness in men which, in turn, was significantly associated with shorter sleep duration.

Discussion

Sedentary time is a link-risk factor mediating the adverse consequences of high BMI on short sleep duration in healthy young men. However, not until the complex association between body
composition and sleep in young population is properly understood will it be possible to establish appropriate therapeutic goals addressing the early morbidity and mortality that obesity and short sleep duration certainly determine.

1. Introduction

Obesity epidemic has globally been recognised as a substantial health concern in modern society, becoming an important risk factor for increased morbidity and mortality with vast clinical and economic adverse consequences on the health system [1]. According to the Global Burden of Diseases, Injuries and Risk Factors Study 2017 [2]—which includes exposure, attributable deaths and disability-adjusted life-years estimations of 84 risk factors for 195 countries—the global exposure risk to a high body-mass index (BMI) has ominously increased 70.39% since 1990 in the overall population. In 2017, 4.72 million of deaths (36.3% higher since 2007) and 148 million disability-adjusted life-years were attributable to a BMI higher or equal to 25 kg/m². Therefore, obesity has become the fourth leading risk factor worldwide for mortality predominantly due to the well-established association between high BMI and cardiovascular diseases [2].

Similarly, sleep duration and quality disturbances such as voluntary sleep curtailment or sleep restriction are being considered emerging public health issues due to their adverse health-related consequences, increasing rate of prevalence and, thus, their significant major economic cost [3, 4]. Short sleep duration (commonly < 7 h per night) [5]—included in the most recent International Classification of Sleep Disorders (ICSD-3) as Insufficient Sleep Syndrome [6]—is also significantly related to a greater risk of mortality from cardiovascular diseases and all causes [7]. Specifically, epidemiologic evidence suggest that short sleepers have up to 12% greater risk of all-cause mortality [8] potentially due to the vast number of severe comorbidities triggered by metabolic and endocrine alterations caused by insufficient sleep, such as obesity and, in turn, other non-communicable diseases [9, 10].

A bidirectional relationship between sleep disorders and obesity therefore has been widely accepted throughout the body of literature in this field. However, although the effects of short sleep on obesity have been shown in clinical and epidemiological research [9, 10], the underlying mechanisms
explaining the effects of obesity on sleep, and thus the potential bidirectional association, still remaining unclear [11, 12]. Potential mediating factors may be sedentariness and physical activity, which have been closely related to both obesity and sleep duration and quality [13-15]. Although the relationship between BMI and short sleep duration may be explained by hormonal and neuroendocrine alterations related to obesity [11], those who have a high BMI are usually less active and display greater sitting time and screen viewing, which is inversely associated to sleep quality and duration [15, 16] as well as to sleep time needed to recover [13]. Regarding physical activity, specifically moderate-vigorous physical activity, although it has been related to better BMI and sleep duration and quality [17, 18], some studies did not find this association in younger ages [19]. These behavioural factors (i.e. sedentariness and physical activity) therefore may be significant key-link elements explaining the closed association between BMI and sleep, although there is a need for further research, especially in young adults.

Correspondingly, scientific literature in this field has also indicated that dietary patterns may similarly show a relationship with sleep duration and quality [20, 21]. According to the evidence, high-carbohydrate intake is related to longer sleep duration and better sleep architecture due to shorter sleep-onset latency and increased REM sleep, whereas high-fat intake may produce lower sleep efficiency, shorter periods of REM sleep and increased arousal index [20]. Low protein intake (< 16% of total energy intake) has been associated to longer sleep latency and poor sleep quality, while high intake of protein (≥ 19% of total energy intake) has been linked to increased sleep fragmentation [22]. As short sleep duration, in turn, may yield to higher energy intake and weight gain mainly via dysregulation of ghrelin and leptin hormones —increasing appetite and decreasing satiety [9, 10]— diet quality may also be a mediating factor between body composition and total sleep time.

Apart from BMI, cross-sectional studies have also exposed that specific body composition parameters can be closely related to sleep duration and quality. Concretely, lean mass has been positively related to sleep duration and quality, whereas a high fat mass seems to be linked to short and poorer sleep [23–25]. Indeed, a combination of both low skeletal muscle mass and increased fat mass, i.e. sarcopenic obesity [26], has been strongly associated with general sleep disorders and, in particular,
with short sleep duration [27]. Similar to sleep impairments, epidemiological studies have emphasised that the imbalance between high BMI and muscle impairment is also independently linked to accelerated functional decline and high risk of chronic conditions and mortality [26, 28]. Thus, sarcopenic obesity may not only be a potential risk factor of sleep disorders but also a syndrome that exacerbates the adverse consequences of these conditions [29].

Understanding the relationship of body weight and composition with sleep duration and quality therefore is of clinical interest in order to design appropriate strategies of health promotion in young populations and, in turn, to prevent early morbidity and mortality. Although as aforementioned there are studies showing independent relationships of markers of obesity with sleep duration and quality, and physiologic and behavioural factors [13–16], at least one of these outcomes were solely assessed by subjective measures such as questionnaires or simple questions in these studies. Moreover, to the best of our knowledge, there are no studies clarifying the potential mechanisms which may enclose all these key elements in an individual model. Therefore, our study was mainly aimed at elucidating the association of anthropometry and body composition—including BMI, waist-hip ratio (WHiR), fat mass index, visceral adipose tissue, and lean mass index parameters—with objective and subjective sleep duration and quality in young sedentary adults. Additionally, we also pursued to investigate the potential mediating role of objectively-measured sedentariness, physical activity, and diet in the specific relationship between BMI and sleep duration and quality. Particularly, we hypothesised that there would be a significant relationship of anthropometric and body composition parameters with sleep duration and quality and that, in the specific association of BMI and sleep, this relationship would be mediated by sedentariness, physical activity and/or diet.

2. Methods
2.1. Study protocol and participants
A total of 187 healthy young adults (n = 121 women), aged 18–25 years and with a BMI ranging from 18 to 35 kg/m², were selected from the Activating Brown Adipose Tissue Through Exercise (ACTIBATE) study (Table 1), an exercise-based randomized controlled trial (ClinicalTrials.gov identifier NCT02365129). A comprehensive explanation of the study design and methodology can be found
The inclusion/exclusion criteria included (i) to be non-smokers, (ii) not to be enrolled in a weight loss program or to be engaged in regular physical activity > 20 min on > 3 days/week during the prior 12 weeks, (iii) to have a stable body weight (body weight changes < 3 kg) over the previous 3 months, (iv) not to be physically active (< 20 minutes on < 3 days/week), (v) not to take any medication or drugs, and (vi) not to suffer from any acute or chronic illness. The study was conducted in the South of Spain between October 2015 and November 2016 at the Sport and Health University Research Institute (iMUDS). The study protocol and written informed consent procedures were accordingly performed with the last revised Declaration of Helsinki (2013), and approved by the Human Research Ethics Committee of the University of Granada (i.e. 924) and Junta de Andalucía.

2.2. Outcome measurements

2.2.1. Sleep duration and quality

Objective sleep outcomes were assessed using a wrist-worn accelerometer (ActiGraph GT3X+, Pensacola, FL, US) continuously 24 hours a day for during 7 consecutive days [23, 30, 31]. Subjects were instructed to wear the accelerometers on the non-dominant wrist as much as possible, removing them only when swimming or bathing. A 7-day sleep diary was provided to the participants in order to register their bedtime, wake up time, and the time they removed the device every day. The accelerometer was programmed to store raw accelerations at 100 Hz of sampling frequency [32] and its derived raw data were stored and downloaded using the ActiLife software (version 6.13.3, ActiGraph, Pensacola, FL, US). A conversion of the resulted GT3X + files was performed to get 1” epoch csv files containing x, y and z vectors to improve raw data processing. Subsequently, these files were processed in R (version 3.1.2, https://www.cran.r-project.org/) using the GGIR package (version 1.5-12, https://cran.r-project.org/web/packages/GGIR/) which included (i) a signal auto-calibration using local gravity as a reference [33], (ii) an evaluation of sustained abnormally high values, (iii) a detection of non-wear time, (iv) a calculation of the Euclidean Norm minus 1 g (ENMO), and (iv) an assessment of waking and sleeping time using an automatized algorithm [34].

Actigraphy recordings were used to determine: (i) total sleep time (defined as the total amount of time spent in bed excluding sleep latency), (ii) sleep efficiency (defined as the percentage of sleep
time over the bedtime) and (iii) wake after sleep onset (WASO; defined as the sum of time awaken from sleep onset to the final awakening)[35]. Those participants registering less than 16 hours/day of wear time for less than 4 days and/or not having data from at least 1 weekend day were excluded from the final analysis.

The Spanish validated version of the Pittsburgh sleep quality index (PSQI) scale was used to determine participant’s subjective sleep quality [36, 37]. This scale includes a total of 19 self-rated items combined to form seven component scores (i.e. subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction), each of which has a range from 0 to 3 points. The sum of these seven PSQI component scores are considered to obtain a global PSQI score (ranging from 0 to 21), higher scores indicating poorer sleep quality.

2.2.2. Anthropometry and body composition

Body weight and height were determined with participants wearing light clothing and without shoes using a SECA scale and stadiometer (model 799; Electronic Column Scale, Hamburg, Germany).

Subsequently, the BMI was calculated as weight (kilograms) divided by height squared (meters$^2$). Waist circumference (centimetres) was assessed midway between the lowest rib and the top of the iliac crest after exhalation, whereas hip circumference (centimetres) was determined over the great trochanters. Each measurement was performed twice and averaged. WHiR was subsequently calculated as waist divided by hip circumferences.

Body composition was measured by dual-energy x-ray absorptiometry (Discovery Wi; Hologic, Inc., Marlborough, Massachusetts) strictly following the manufacturer instructions and obtaining lean mass, fat mass and visceral adipose tissue mass [38]. Lean mass index and fat mass index were calculated as lean mass (kilograms) / fat mass (kilograms), respectively, divided by height squared (meters$^2$).

2.2.3. Sedentary time and physical activity intensity levels

Sedentariness and physical activity levels were determined by accelerometry (see specific details about the procedure above) [39, 40]. The GGIR package (v.1.5–12, https://cran.r-project.org/web/packages/GGIR/) in R (v.3.1.2, https://www.cran.r-project.org/) was used to discern
among (i) sedentary time, (ii) light physical activity (LPA), (iii) moderate physical activity (MPA), (iv) vigorous physical activity (VPA), and (v) moderate-vigorous physical activity (MVPA), using age-specific thresholds for ENMO [41, 42].

2.2.4. Dietary intake
An average of three 24-h recalls performed on non-consecutive days (including one weekend day) were used to register dietary intake. This technique is considered a valid method to assess energy intake with an 8 to 10% margin of error. The 24-h recalls were conducted by experienced dietitians-nutritionists who obtained a detailed description of the food consumed by the participants through a personal interview. Coloured photographs containing different foods and portion sizes were used in order to assist in estimating the quantity of food consumed. Energy intake and macronutrient content (i.e. fat, protein and carbohydrate intake) were obtained using the EvalFINUT ® software.

2.3. Statistical analysis
Shapiro-Wilk test, Q-Q plots, and visual checking of histograms were used to confirm the normal distribution of all variables. Descriptive characteristics of participants are presented as mean ± standard deviation. Sex differences were determined by unpaired Student t- tests. Given that non-significant sex interaction (p < 0.05) was observed in regression analyses, data from men and women were simultaneously included in all the analyses performed.

Pearson correlations were preliminarily conducted to examine the relationship among objective and subjective sleep parameters, anthropometric and body composition outcomes, sedentary time, physical activity levels and dietary intake variables. The associations of anthropometric (i.e. WHiR and BMI) and body composition parameters (i.e. lean mass index, fat mass index and visceral adipose tissue) with sleep parameters were studied by simple lineal regression (Model 0) and multiple lineal regression models adjusted by sex (Model 1).

In order to analyse the potential mediating role of sedentary time, physical activity levels and dietary intake outcomes in the relationship between BMI and sleep parameters, mediation analyses were performed following the steps for establishing mediation proposed by Baron and Kenny [43], using corresponding models in the most recent PROCESS macro version 3.4 developed by Andrew F. Hayes
According to Baron and Kenny, the independent variable X must firstly be associated to the dependent variable Y (total effect; path c), and secondly with the mediator variable M (path a); thirdly, the mediator variable M must also be significantly correlated to the dependent variable Y even after adjusting by the causal variable X (path b); and, fourthly, the effects of X on Y should drop in strength and/or significance after controlling by M (direct effect path c’) for the mediation to be established. Preacher and Hayes’s SPSS macro incorporates the stepwise procedure described by Baron and Kenny and includes the estimation of the indirect effect ab (i.e. ab = c – c’; the amount of mediation or reduction of the effects of X on Y), which is the recommended way of robustly measuring the mediation [44-46]. This latter estimation is based on the bootstrapping method, a non-parametric resampling method which estimates the indirect effect through 5,000 bias-corrected bootstrap samples and 95% confidence intervals. If these confidence intervals do not include zero, the indirect effect ab can be considered as different from this value and therefore the mediation is assumed.

Calculations were performed using the Statistical Package for the Social Sciences v.22.0, (IBM SPSS Statistics, IBM Corporation). GraphPad Prism 5 software (GraphPad Software, San Diego, CA, USA) was used to draw plots. Significance was set at p < 0.05.

3. Results

Descriptive characteristics of the study participants are shown in Table 1. Overall, men presented statistically significant higher values of WASO, BMI, WHiR, lean mass, lean mass index, visceral adipose tissue mass, energy intake, fat intake, protein intake and carbohydrate intake compared with those observed in women (all p < 0.05). In contrast, women showed greater levels of sleep efficiency and fat mass percentage than men.

| Table 1. Descriptive characteristic of participants. |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Age (years)     | Sleep quality   | Sleep quantity  | Total sleep time |                 |                 |                 |
| N               | All             | Men             | N               | Men             | N               | Women           |
| Age (years)     | 187             | 22.0 7 ± 2.1 9  | 66              | 22.25 ± 2.24    | 121             | 21.97 ± 2.17    |
| Sleep quality   | 187             | 385.63 ± 44.02  | 66              | 378.38 ± 44.70  | 121             | 389.58 ± 43.32  |
| Parameter                              | Description                      | Mean | SD  | Mean | SD  | Mean | SD  |
|----------------------------------------|-----------------------------------|------|-----|------|-----|------|-----|
| Sleep efficiency (%)                  | Minute                            | 86.99| ±   | 5.00 | ±   | 85.86| ±   |
| Wake after sleep onset (min)           |                                  | 59.68| ±   | 26.29| ±   | 65.53| ±   |
| PSQI total score                      |                                  | 5.63 | ±   | 2.60 | ±   | 6.02 | ±   |
| Anthropometry and body composition    | Body mass index (kg/m²)           | 24.97| ±   | 4.80 | ±   | 26.84| ±   |
|                                       | Waist-Hip ratio                   | 0.80 | ±   | 0.10 | ±   | 0.87 | ±   |
|                                       | Lean mass (kg)                    | 41.93| ±   | 9.81 | ±   | 53.11| ±   |
|                                       | Lean mass index (kg/m²)           | 14.74| ±   | 2.44 | ±   | 17.14| ±   |
|                                       | Fat mass (kg)                     | 24.83| ±   | 8.89 | ±   | 25.37| ±   |
|                                       | Fat mass (%)                      | 35.50| ±   | 7.37 | ±   | 30.13| ±   |
|                                       | Fat mass (%)                      | 35.50| ±   | 7.37 | ±   | 30.13| ±   |

* indicates statistical significance.
| Index (kg/m²) | Visceral adipose tissue mass (g) | Sedentariness and physical activity levels |
|--------------|----------------------------------|---------------------------------------------|
| 150          | 340 ± 179.95 49 427 ± 179.18 101 299.15 ± 164.80* |
| Sedentary time (min) | 926 ± 59.9 55.1 66 935 ± 59.1 121 922.4 ± 51.9 |
| Light physical activity (min) | 24.5 ± 12.7 66 24.7 ± 12.6 121 24.4 ± 12.7 |
| Moderate physical activity (min) | 59.8 ± 24.7 66 56.5 ± 23.2 121 61.6 ± 25.4 |
| Vigorous physical activity (min) | 1.8 ± 3.3 66 1.6 ± 2.1 121 1.9 ± 3.8 |
| Moderate-vigorous physical activity (min) | 61.7 ± 26.5 66 58.2 ± 23.8 121 63.6 ± 27.8 |
| Dietary intake | Energy intake (kcal) | Fat |
| 170          | 186 ± 532 59 210 ± 519 111 1741.0 ± 497.7* |
| 170          | 83. ± 29. 59 94. ± 29. 111 77.7 ± 27.7* |
The associations of anthropometric and body composition outcomes with sleep parameters are shown in Fig. 1, Fig. 2, and Additional file 1. There was a significant negative association between BMI and total sleep time ($\beta = -0.165; R^2 = 0.027; p = 0.029$; Fig. 1A), which was attenuated after controlling by sex ($p = 0.092$). WHiR was also negatively associated with total sleep time ($\beta = -0.222; R^2 = 0.049; p = 0.007$; Fig. 1E) and sleep efficiency ($\beta = -0.174; R^2 = 0.030; p = 0.037$; Fig. 1F), and positively related to WASO ($\beta = 0.169; R^2 = 0.029; p = 0.042$; Fig. 1G); although these associations disappeared after adjusting by sex in the case of sleep efficiency ($p = 0.409$) and WASO ($p = 0.481$). Regarding lean mass index, a significant negative association was observed between this outcome and total sleep time ($\beta = -0.219; R^2 = 0.048; p = 0.004$; Fig. 2A) which persisted after controlling by sex ($p = 0.039$). Similarly, a significant negative association was noted between lean mass index and sleep efficiency ($\beta = -0.188; R^2 = 0.035; p = 0.013$; Fig. 2B), which disappeared after adjusting by sex ($p = 0.255$). No further associations were found between the rest of anthropometric and body composition parameters with sleep-related outcomes (Figs. 1 and 2).

Correlation analyses for anthropometric and body composition outcomes, sleep parameters, sedentary time, physical activity levels and dietary intake can be found in Additional file 2, 3 and 4. Considering required correlations in order to proceed with the mediational analyses, only sedentary time was found to be related to both BMI and total sleep time. Thus, while a significant positive relationship of BMI and sedentary time was observed only in men ($p < 0.05$; Additional file 3), a negative correlation was found between sedentary time and total sleep time in both men and women ($p < 0.01$; Additional file 2). Other relevant correlations found among sleep parameters and dietary...
and physical activity outcomes were a statistically significant positive relationship between MVPA and sleep efficiency ($p < 0.05$), and an inverse correlation between MVPA and WASO ($p < 0.05$; Additional File 2).

Regarding the mediational analyses, Fig. 3 and Table 2 show the moderated mediating effect of sedentary time in the relationship between BMI and total sleep time. As BMI was only significantly and positively related to sedentary time in men, we proceeded to perform a moderated mediation analysis (model 7), including sex as a moderator variable of the mediational effect of sedentary time.

Considering this model, BMI was negatively associated with total sleep time (total effect $c = -13.667$; $p < 0.05$), and positively related to sedentary time only in men (effect $a = 37.866$; $p < 0.01$).

Sedentary time, in turn, was also negatively associated to total sleep time (effect $b = -0.551$; $p < 0.01$). After including the moderator and mediator variable (i.e. sex and sedentary time, respectively) in the model, the association between BMI and total sleep time, although remaining significant, was significantly reduced (direct effect $c' = -8.894$; $p < 0.05$), suggesting therefore that a partial moderated mediation was reached. The confidence interval of the conditional indirect effect $ab$ (amount of mediation) for men, as well as the final moderated mediation index (the quantification of the association between the indirect effect and the moderator), did not include the value zero, so the mediating role of sedentary time moderated by sex in the significant relationship between BMI and total sleep time was further confirmed.

Table 2

| Sedentariness (M) | Total Sleep Time (Y) |
|-------------------|----------------------|
| **Body mass index (X)**<br> $a_1 \rightarrow$ | **Coefficient (SE)**<br>83.8837**<br>(28.1842) | **95% CI**<br>28.2498, 139.5175 | **Coefficient (SE)**<br>$c' \rightarrow$ | **-8.8941***<br>(4.3049) | **95% CI**<br>-17.3914, -0.3969 |
| **Sex (V)**<br> $a_2 \rightarrow$ | **Coefficient (SE)**<br>51.4133*<br>(24.9181) | **95% CI**<br>2.2266, 100.6000 | **Coefficient (SE)**<br>$b \rightarrow$ | **-0.5511***<br>(0.0430) | **95% CI**<br>-0.6360, -0.4663 |
| **$X^*W$**<br> $a_3 \rightarrow$ | **Coefficient (SE)**<br>-46.0177**<br>(16.1402) | **95% CI**<br>-77.8774, -14.1581 | **Moderated Mediation Index** | **25.3618**<br>(0.0013) | **7.8916, 43.8275** |

*p < 0.05, **p < 0.01, ***p < 0.001

The mediating role of physical activity levels and dietary intake in the relationship between BMI and...
total sleep time were not statistically significant in any case (see Additional file 5).

4. Discussion
Our study sought to elucidate the potential association of anthropometry and body composition parameters with sleep duration and quality and, in turn, whether behavioural factors such as sedentariness, physical activity and diet were potential mediating mechanisms explaining the closed association between BMI and sleep in young adults. Out of all included body composition and sleep parameters, only BMI, WHiR, and LMI were associated with objective sleep parameters, these associations being attenuated after adjusting by sex. As we expected, although physical activity and diet did not mediate the relationship between BMI and sleep duration, sedentariness significantly explained the association between these two variables. According to our results, sedentary time moderated by sex partially explained the effects of BMI on total sleep time such that a higher BMI was related to higher sedentariness in men which, in turn, was significantly associated with shorter sleep duration.

High BMI and short sleep duration caused by sleep disorders are both public health concerns due to their high and increasing prevalence in the overall population [1–4] and their broad-ranging adverse health-related consequences such as endocrine and metabolic alterations, life-threatening cardiovascular diseases, and impaired daytime functioning and mood [47, 48]. Although the bidirectional relationship between obesity and sleep disturbances is well-accepted [49], and the effects of short sleep on BMI have been widely shown and explained [10, 50], the underlying mechanism explaining the effects of high BMI on sleep still remains uncertain [11, 12]. According to our results, sedentary time may be one of the mediating factors explaining the negative impact of high BMI on sleep duration in young men, which is in accordance with previous studies where sedentariness was independently and closely associated with higher BMI and shorter sleep duration [13–16]. Regarding sex differences, previous epidemiological/experimental studies also showed that the beneficial effects of acute or regular exercise on sleep were significantly higher in men than women [17, 51] and, in general, the association between BMI and sleep was lower in women possibly due to other cofounders such as psychological stress rather than exercise/sedentary time [52, 53].
Concerning moderate-vigorous physical activity, we did not find a significant association between this variable and total sleep time, which is in accordance with results from a recently published cross-sectional study where an increased moderate-vigorous physical activity, although associated with improved subjective sleep quality, was not related to better or longer objective sleep duration [54]. Thus, differing from sedentary time, physical activity did not explain the association between BMI and objective or subjective sleep, which is consistent with the overwhelming and major negative impact of sedentary behaviour in sleep and overall metabolic risk independent to physical activity [15].

Nevertheless, we found that increased moderate-vigorous physical exercise was significantly associated to higher sleep efficiency and lower wake after sleep onset. Therefore, while the practice of physical activity at this intensity may not be related to longer sleep duration, it is related to a reduced number of awakenings or time spent awake after sleep onset which, in turn, is reflected in higher sleep efficiency. These results are also supported by previous empirical evidence which emphasized that, although total sleep time may not be modified by an increased moderate to vigorous physical activity, a reduction on this intensity seems to be linked to reduced slow-wave sleep and, in turn, increased rapid eye movement (REM) sleep [55].

Similarly, dietary intake has previously been found to be linked to sleep architecture and quality [20–22]. Whereas high-fat intake and low-protein intake seem to produce reductions in REM sleep, increased arousal index and, thus, poorer sleep quality [21, 22], high-carbohydrate intake has been related to increased REM sleep, and improved sleep architecture and quality [20]. However, we did not find any significant relationship between dietary intake — including energy, fat, protein and carbohydrates intake — and objective or subjective sleep duration and quality in our sample. These results may be explained by the use of accelerometry as a sleep measurement which, although providing reliable measures of objective sleep, does not allow to discern more potential specific changes in sleep architecture rather than duration. The use of dietary recalls for the energy intake estimations may also potentially suffer from significant bias [56, 57], which could similarly explain the lack of association between these dietary outcomes and sleep.

Apart from BMI, only WHiR and LMI were related to objectively measured sleep parameters. According
to our results, higher values of WHiR were associated with shorter sleep duration, lower sleep efficiency and increased time awake after sleep onset. These results are in accordance with previous research emphasizing that, besides being major risk factors for cardiometabolic alterations, cancer and all-cause mortality [58, 59], WHiR and abdominal obesity are significantly related to severe sleep disturbances such as short sleep duration and obstructive sleep apnoea [23, 60], especially in younger adults [58]. Regarding LMI, we found that those participants with higher LMI exhibited shorter sleep and lower sleep efficiency. Although controversial to previous studies where LMI was positively associated with sleep quality [23], these results may be explained by evidence exposing that lower LMI was related to longer or excessive sleep duration, potentially due to increased levels of cortisol triggered by sleep disturbances which, in turn, has been related to muscle degradation [61]. An inverted u-shaped relationship between LMI and sleep duration therefore may occur, although the testing of this association in our sample was not feasible due to the sole inclusion of healthy young adults, i.e. non-inclusion of extreme LMI values.

Interestingly, no body composition parameters were related to subjective sleep quality. Although these results may seem controversial with those regarding BMI and objective sleep duration, they emphasise the well-established discrepancy between objective and subjective sleep [62]. Unlike objective sleep duration, which is the actual time the individual is asleep, subjective sleep quality refers to the self-evaluation or one’s perception of sleep duration and satisfaction, independently to the more objective aspect of sleep. According to the evidence, subjective sleep quality is highly related to the individual’s emotional status and cognitive function [63–65], which could explain the non-significant associations found between body composition parameters and subjective sleep, potentially due to underlying uncontrolled psychological cofounders.

However, our results should be cautiously interpreted as these are restricted to the sample included, methodology followed, and study design. Although the analysis of mediator variables enables approach to causal inferences [43–46], our main limitation was the cross-sectional study design which does not allow to completely determine cause-effect relationships. The use of accelerometry/actigraphy for the objective measurement of sleep, although widely used and validated
[34-35], may have underestimate/overestimate sleep outcomes such as sleep duration, sleep efficiency, sleep onset latency and wake after sleep onset [32]. Finally, our sample only included healthy young adults so, although there was a lack of studies including this population in this field of research, the generalisation of our findings may be limited to this population. Future well-designed longitudinal studies should be performed in order to robustly establish causal relationships between body composition parameters and sleep. Furthermore, studies in this field of research should include polysomnography, which is the gold-standard method to appropriately assess not only sleep duration and efficiency but also potential associations between behavioural factors and polysomnographic sleep outcomes such as sleep architecture. Finally, as psychological conditions such as mood and anxiety disturbances have been related to both obesity and sleep disorders [66, 67], upcoming studies should also include measurements of these psychological variables and thus control their potential mediating/mediating role.

To the best of our knowledge, this is the first study acutely analysing modifiable behavioural risk factors as potential underlying mechanisms through which BMI and sleep may be related, including not only reliable objective measures of body composition and sleep but also the gold-standard test of physical activity, i.e. accelerometry. Apart from independent associations of body composition parameters and physical activity with sleep, we essentially found that sedentary behaviour seems to significantly explain part of the adverse impact of high BMI on sleep duration in young men. Our study therefore has significant contributions to the clinical and research practice in this field, further clarifying the recognised bidirectional relationship between obesity and sleep disorders [9] and, thus, supporting the need of designing appropriate strategies of health promotion through physical activity and/or sedentary behaviour avoidance in young populations. In this regard, the replacement of sedentary time with physical activity may be the most effective strategy for general sleep and cardio-metabolic benefits [68, 69]. As sedentariness has also been independently linked to cardiovascular disease, cancer incidence and mortality, and all-cause mortality in adults [70], modifying this behavioural risk factor could not only improve the current obesity and sleep disorder epidemics in young adults but also prevent this population from suffering chronic health conditions and early
5. Conclusions
Our study further highlights the potential underlying mechanisms through which body composition, especially BMI, has a significant impact on objective and subjective sleep. According to our findings, sedentary behaviour is the link-risk factor mediating the adverse consequences of high BMI on short sleep duration in healthy young men. Other potential modifiable risk factors such as physical activity and diet, although not mediating the impact of BMI on sleep, have also been independently related to both these current public health concerns, i.e. obesity and sleep curtailment. Due to the vast and adverse health-related consequences of these two conditions and thus the resulting resource utilization and/or health care costs, the identification of these potential modifiable risk factors and affected young patients should become an essential goal for researchers and clinicians. To this end, researchers and health care professionals from different fields should collaborate in multidisciplinary research and interventions. Not until the complex association between body composition and sleep in young population is properly understood will it be possible to establish appropriate therapeutic goals addressing and limiting the early morbidity and mortality that the interaction of health conditions such as obesity and short sleep duration certainly determine.

Declarations

Ethics approval and consent to participate: The study protocol and written informed consent procedures were accordingly performed with the last revised Declaration of Helsinki (2013), and approved by the Human Research Ethics Committee of the University of Granada (i.e. 924) and Junta de Andalucía.

Consent for publication: Not applicable.
Availability of data and materials: The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no competing interests.

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**Additional Files**
The following additional files are available online, Additional file 1: Association of body mass index, waist-hip ratio, lean mass index, fat mass index, and visceral adipose tissue mass with total sleep time, sleep efficiency, wake after sleep onset, and Pittsburgh total score before and after adjusting for sex (Model 0 and Model 1, respectively); Additional file 2: Correlations of sleep parameters with sedentary time, physical activity levels and dietary intake; Additional file 3: Correlations of body composition with sedentary time, physical activity variables, and dietary intake; Additional file 4: Correlations for sedentary time, physical activity and dietary intake; Additional file 5: Mediation model of the relationship between BMI and Total Sleep Time (min) with energy intake (A), fat intake (B), protein intake (C), carbohydrates intake (D), sedentary time (E), and moderate-vigorous physical activity (F) as mediator variables.

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Figures

Figure 1

Linear regression graphs of BMI and WHiR with total sleep time (Panels A and E), sleep efficiency (Panels B and F), wake after sleep onset (Panels C and G) and PSQI total score (Panels D and H), before and after adjusting by sex (model 0 and model 1, respectively). β, standardised linear regression coefficient; R², coefficient of determination; p value. Closed and open circles represent men and women, respectively. Abbreviations: BMI, body mass index; WHiR, waist-hip ratio; PSQI, Pittsburgh sleep quality index.
Figure 2

Linear regression graphs of LMI, FMI, and VAT with total sleep time (Panels A, E, and I), sleep efficiency (Panels B, F, and J), wake after sleep onset (Panels C, G and K) and PSQI total score (Panels D, H, and L), before and after adjusting by sex (model 0 and model 1, respectively). β, standardised linear regression coefficient; R², coefficient of determination; p value. Closed and open circles represent men and women, respectively. Abbreviations: LMI, lean mass index; FMI, fat mass index; VAT, visceral adipose tissue mass; PSQI, Pittsburgh sleep quality index.
Figure 3

Moderated mediation model of the relationship between Body Mass Index and Total Sleep Time (min) with sedentary time (min) and sex as mediator and moderator variables, respectively. Paths a, b, c, and c’ are presented as unstandardized coefficients (standard error). [Lower-limit CI; upper-limit CI], lower and upper levels for 95% bias-corrected CIs of the indirect effects based on 5,000 bootstraps. BMI, body mass index; CI, confidence interval; \( \beta(X) \), unstandardized coefficient of the association of BMI with sedentariness; \( \beta(W) \), unstandardized coefficient of the association of sex with sedentariness; \( \beta(X*W) \), unstandardized coefficient of the association between BMI and sedentariness moderated by sex.

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