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ORIGINAL ARTICLE

Sleep and Metabolic Health

Lifestyle mediators of associations among siestas, obesity, and metabolic health

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Abstract

Objective: The aim of this study was to determine the association between siestas/no siestas and obesity, considering siesta duration (long: >30 minutes, short: ≤30 minutes), and test whether siesta traits and/or lifestyle factors mediate the association of siestas with obesity and metabolic syndrome (MetS).

Methods: This was a cross-sectional study of 3275 adults from a Mediterranean population (the Obesity, Nutrigenetics, TIming, and MEditerranean [ONTIME] study) who had the opportunity of taking siestas because it is culturally embedded.

Results: Thirty-five percent of participants usually took siestas (16% long siestas). Compared with the no-siesta group, long siestas were associated with higher values of BMI, waist circumference, fasting glucose, systolic blood pressure, and diastolic blood pressure, as well as with a higher prevalence of MetS (41%; $p = 0.015$). In contrast, the probability of having elevated SBP was lower in the short-siesta group (21%; $p = 0.044$) than in the no-siesta group. Smoking a higher number of cigarettes

per day mediated the association of long siestas with higher BMI (by 12%, percentage of association mediated by smoking; $p < 0.05$). Similarly, delays in nighttime sleep and eating schedules and higher energy intake at lunch (the meal preceding siestas) mediated the association between higher BMI and long siestas by 8%, 4%, and 5% (all $p < 0.05$). Napping in bed (vs. sofa/armchair) showed a trend to mediate the association between long siestas and higher SBP (by 6%; $p = 0.055$).

Conclusions: Siesta duration is relevant in obesity/MetS. Timing of nighttime sleep and eating, energy intake at lunch, cigarette smoking, and siesta location mediated this association.

INTRODUCTION

Obesity is a risk factor for adverse physical and mental health outcomes and currently affects more than 1 billion people worldwide [1]. Because of the multifactorial nature of obesity [2], its prevention and treatment require different approaches, including healthy sleep patterns [3, 4]. For sleep-deprived individuals and shift workers, daytime naps have been recommended [5] to improve performance, alertness, and health [6], and a midday nap (or siesta) may increase productivity and potentially reduce obesity risk [7]. However, before proposing siestas (i.e., regular naps in the middle of the day) as a solution for obesity prevention in the general population, it is important to know whether they are beneficial. Although the acute benefits of a siesta for increased alertness and cognitive performance [8] during sleep deprivation have been well established [9], the long-term effects of habitual napping on chronic diseases remain controversial. Cross-sectional studies have pointed to napping as both beneficial [10, 11] and detrimental [12–14] for obesity, cardiometabolic risk, and all-cause mortality.

Our large-scale, genome-wide association study in the UK Biobank and 23andMe using Mendelian randomization analysis has indicated that habitual napping can be a causal factor for obesity and other obesity-related traits such as blood pressure [14]. However, in this UK study, information was derived from only one question about the frequency of napping (never, sometimes, or usually), and no information was available on other aspects of napping, such as duration or motives for napping. In addition, the study was conducted only in the UK and the United States, two countries where siestas are not culturally embedded. Midday napping or siesta is a common habit in Mediterranean culture, and its effects on obesity and obesity traits may substantially differ from Anglo-Saxon countries, especially in aged populations in whom daytime napping could be a sign of aging or underlying diseases [15].

Based on recent studies that have shown the relationship between prolonged napping and health risk [15–17], we hypothesize that the association of siestas with obesity depends, in part, on siesta duration (i.e., whether one takes a short or long siesta). Different lifestyle factors previously related to obesity and napping, such as lower levels of physical activity, higher energy intake, delayed meal or sleep behaviors [10], and individual chronotype, as well as other classical obesity risk factors such as smoking, alcohol intake, etc., may mediate the association of siestas with obesity and related metabolic alterations. Other unexplored factors directly related to siestas might also play a role, such as the following: the causes or motives for taking a siesta; how the individual feels after the siesta; whether the individual feels hungry after

Study Importance

What is already known?

- Siesta, or midday napping, is a common practice in numerous countries to recover from the deleterious effects of insufficient sleep. Nevertheless, the relationship between siestas and metabolic health is still not well understood.

What does this study add?

- This study, in a Mediterranean population, demonstrates that long siesta-takers had a higher BMI and were more likely to have metabolic syndrome than those who did not take siestas. In contrast, short siesta-takers were less likely to have elevated systolic blood pressure.
- We identified potential lifestyle mediators in the association between long siestas and metabolic alterations. Those mediating factors are nighttime sleep timing, food timing, energy intake at lunch, cigarette smoking, and place of siesta (bed vs. sofa).

How might these results change the direction of research or the focus of clinical practice?

- Siesta duration may be relevant in clinical practice for the treatment of obesity and metabolic syndrome.
- Results call for studies to investigate whether short siestas are advisable over long siestas, especially in individuals with behaviors that mediate the association between long siestas and obesity, such as delayed meals and sleep schedules, or in those who smoke. In addition, studies are needed to test whether lower caloric intake at lunch decreases the deleterious effects of long napping on obesity and systolic blood pressure.

the siesta; whether the siesta is seasonal (taken only in the summer) or is taken across the whole year; or where the siesta is taken, for example, in bed (lying down) or on a sofa/armchair (sitting with head up), because acute changes in posture of the body with napping were shown to be related to cardiovascular risk [18].

Therefore, our objectives were to investigate the following: 1) the potential association between habitual siestas and obesity, considering the duration of siesta (i.e., long: >30 minutes or short: ≤30 minutes); and 2) whether other siesta characteristics or lifestyle factors mediate the association between siestas and obesity. We used data of a population from a Mediterranean area in Spain, where siestas are culturally embedded, that includes individuals who have the opportunity to nap after lunch.

METHODS

Research participants

The population used for the current study was part of the Obesity, Nutrigenetics, TIming, and Mediterranean (ONTIME) study (ClinicalTrials.gov: NCT02829619; University Ethical Committee, ID-632/2017; University of Murcia, Spain), which consists of healthy individuals with no diagnosed illnesses except for obesity. All participants were voluntarily attending one of five weight loss clinics in Spain for dietetic and behavioral treatment based on the principles of a Mediterranean diet. All participants came from the Spanish region of Murcia, located on the southeast coast of the Mediterranean Sea. Individuals receiving treatment with thermogenic or lipogenic drugs, those diagnosed with bulimia, and those who underwent treatment with anxiolytic or antidepressants were excluded. Because this study was aimed at investigating the timing of behaviors, and because the number of shift workers attending these clinics was very low (<0.5%), in the macro study of ONTIME, shift workers were initially excluded.

Participants included in this study were adult volunteers (aged 18–65 years) who had completed the "Siesta characteristics questionnaire" (Supporting Information Table S1). A total of 31.2% of the participants were recruited in winter, 24% in spring, 22% in summer, and 22.9% in autumn. A total of 70% of the population had a university education (i.e., the highest education level), 24.2% had only a secondary school education, and 5.8% had only a primary school education.

General characteristics and metabolic status

Height, weight, waist circumference, hip circumference, waist-hip ratio, and body composition were determined as previously described [19]. Fasting glucose concentration was determined in serum by the glucose oxidase method [20]. Plasma concentrations of triglycerides, total cholesterol, and high-density lipoprotein (HDL) cholesterol were determined using commercially available kits (Roche), whereas low-density lipoprotein (LDL) cholesterol was calculated according to the Friedewald equation [21]. Fasting insulin concentration was determined using a solid-phase, two-site chemiluminescent immunometric assay (IMMULITE 2000 Insulin). As an estimate of insulin resistance, the homeostatic model assessment of insulin resistance was calculated using the following standard formula: fasting glucose (millimoles per liter) × fasting insulin (milli-international units per liter)/22.5. As a measure of insulin sensitivity, the quantitative insulin-sensitivity check index (QUICKI) was calculated with the following formula: 1/(log[fasting insulin in microunits per milliliter] + log[fasting glucose in milligrams per deciliter]) [22]. Arterial systolic and diastolic blood pressure (SBP, DBP) was measured with a mercury sphygmomanometer according to international guidelines [23].

Body mass index (BMI) was categorized as follows: normal weight (<25 kg/m^2); overweight (≥25 to <30 kg/m^2); and obesity (≥30 kg/m^2). For cardiometabolic risk (non-healthy values), the Adult Treatment Panel (ATP)-III criteria [24] were as follows: SBP and DBP: ≥130 and ≥85 mm Hg, respectively; fasting glucose ≥ 110 mg/dL; HDL cholesterol < 40 and <50 mg/dL in men and women, respectively; triglycerides ≥ 150 mg/dL; and abdominal obesity > 102 cm and >88 cm in men and women, respectively [24]. We also assessed the components of metabolic syndrome (MetS) and calculated the MetS score (number of altered components). If the score was ≥3, then the participants were classified as presenting with MetS (MetS score from 0–6 points). We also included a cutoff for elevated SBP (≥120 mm Hg) [25].

Siesta characteristics

Habitual siestas in study participants were quantified using the "Siesta characteristics questionnaire" (Supporting Information Table S1). A positive answer to the question "Do you usually take siesta during the week?" was used to determine habitual nappers.

Among habitual nappers (i.e., on average, those who take a siesta at least once per week during weekdays), the question "What is the duration in minutes?" allowed us to quantify habitual siestas and to categorize as follows: 1 = short siesta; and 2 = long siesta, ≤30 and >30 minutes, respectively. A short siesta was defined as ≤30 minutes based on previous studies [16, 17, 26].

The questionnaire evaluated other variables related to siesta behavior that were answered not only by those classified as habitual nappers but also by those who took siestas occasionally (for example, once every 2 weeks, once a month, or only during weekends). The variables were as follows: causes/motives for napping; feelings related to not being able to take a siesta; how individuals feel after a short or long siesta; whether they are hungry after waking from a siesta; where they usually take the siesta (bed vs. sofa/armchair); and whether their siestas follow a seasonal pattern (yes/no seasonal siesta, summer).

We estimated the timing of night sleep onset and offset, as well as the nighttime sleep duration, using the following questions: "On weekdays (and weekends) (1) at what time do you usually go to bed?" and "(2) at what time do you usually get up in the morning?" Nighttime sleep duration was determined as the difference between nighttime sleep onset and offset. Because no participants were shift workers, weighted weekly sleep duration (SL) was calculated as follows: ([weekday SL × 5] + [weekend SL × 2])/7 [14].

Lifestyle factors

Classical lifestyle factors involved in obesity

To assess daily energy intake, all volunteers completed a single 24-hour dietary recall (type, amount, and preparation of each recorded eating episode). Data on energy intake per day and per meal were obtained using the software program "Grunumur 2.0" (Murcia, Spain) based on Spanish food composition tables [27]. Alcohol consumption (in grams) was also determined. Physical activity level was determined with the International Physical Activity Questionnaire (IPAQ), which assesses physical activity in the last 7 days [28]. Smoking status was assessed by the number of cigarettes per day as "≥1 cigarette as smoker" or "=0 cigarettes as nonsmoker" [29].

Timing of meals and sleep

Participants were asked, "On weekdays (and weekends), at what time do you usually eat breakfast (lunch and dinner)?" Responses were in 30-minute increments. A weighted weekly average of mealtimes was calculated (5/7 weight for weekdays and 2/7 weight for weekends). We determined the midpoint of meal intake by calculating the midpoint between the weekly averages for breakfast and dinner times (hours) and adding this value to the average breakfast time [30]. To calculate the dinner timing relative to sleep onset, participants recorded sleep onset timing on the 5 days of the week and on the 2 days of the weekend, and the weighted mean was calculated. To categorize individual chronotype, we used the validated 19-item scale Morningness-Eveningness Questionnaire (MEQ) scale [31].

Statistical analyses

We performed statistical analyses with siesta classification in three groups (coded as "no-siesta," including non-habitual nappers [0]; and "short siesta" [1] or "long siesta" [2], including habitual nappers). Quantitative variables are reported as means and standard deviations (SD) and qualitative variables as numbers of participants and percentages. *P* values were derived from ANOVA for quantitative variables among the three groups (no-siesta, long siesta, and short siesta) and from χ^2 test for the qualitative variables. When significant, we explored differences among groups using Bonferroni correction.

We analyzed linear regression models (adjusted for sex, age, clinical center, and year of recruitment) to examine the associations of short and long siestas with body composition, cardiometabolic profiles, and MetS score, with the no-siesta group as the reference group. These statistical analyses were performed using Stata (StataCorp LLC).

Lifestyle factors associated with both obesity and siesta duration were tested as mediators: bed versus sofa/armchair as place for siesta (percent); well-being after siesta (percent); midpoint meal (hours); physical activity (hours per week); energy of lunch (kilocalories per day); time of breakfast (hours); chronotype score (MEQ); time between dinner and sleep (hours); time of nighttime sleep onset (hours); duration of nighttime sleep (hours); number of cigarettes per day; and alcohol consumption (yes, percent).

We used the R package "mediation" for "mediation analyses." We calculated estimates for the total effect, average direct effect, and average causal mediation effect using the quasi-Bayesian Monte Carlo method based on a normal approximation with 5000 simulations. When significant, a mediated fraction expresses the percentage of the possible effect mediated by the hypothesized mediator. We performed further multicollinearity tests among significant mediators using Pearson correlation coefficients and stepwise regression models. We found no interaction with sex or age in the association between long siestas and BMI; therefore, men and women were pooled, and sex and age were included as covariates in addition to center (which refers to the nutritional clinic) and the year of recruitment. We performed further sensitivity analyses with the level of education as a socioeconomic indicator. A two-sided $p < 0.05$ was considered statistically significant.

RESULTS

General characteristics of the population and metabolic status

The study population included 3275 adults aged 18 to 65 years with an average BMI of 31.1 kg/m^2 and of whom 78% were women. Even though 87.4% of the population had overweight/obesity, metabolic parameters related to obesity, such as fasting glycemia, homeostatic model assessment of insulin resistance, total cholesterol, and triglycerides, were below the cutoff points of MetS risk [24]. Descriptive data (anthropometric, metabolic status, siesta-related behaviors, and other lifestyle factors) are shown in Table 1.

Siesta characteristics

Thirty-five percent of participants usually had siestas (habitual nappers), with an average frequency of four times per week and a similar average duration on weekdays and weekends (~43 minutes; Table 1). Of the studied population, only 16% usually took long siestas, whereas 20% usually took short siestas.

In general, the main cause or motive for napping was relaxing (49%), followed by tiredness (36%); 19% felt bad (drowsy and/or moody) after long siestas compared with only 8% who felt bad after short siestas. Eleven percent had siestas only in the summer, with no differences between long and short nappers ($p > 0.05$). After napping, 42% expressed being hungry, and 63% of them felt like eating something sweet (vs. 7.5% who felt like eating something salty). Additionally, whereas no significant differences were found in hunger between short and long nappers, there was a trend toward lower appetite for salty foods among short nappers ($p = 0.06$). Most participants (including habitual and occasional nappers) napped on the

TABLE 1 Main characteristics of study participants and their siesta behaviors and characteristics (in the total sample)

	n	Mean[a]	SD
General characteristics			
Age (y)	3275	41	12
Women (%)[a]	2568	78	
BMI (kg/m^2)	3265	31	6
BMI classification			
No obesity (<30 kg/m^2, %)[a]	1601	49	
Obesity (≥30 kg/m^2, %)[a]	1669	51	
Body fat (%)	3154	37	7
Waist circumference (cm)	3198	101	15
Waist-hip ratio	3197	0.90	0.09
MetS score	2135	2.04	1.20
MetS (%)[a]	651	30.5	
Metabolic status			
Glucose (mg/dL)	2451	87.50	15.24
Insulin (mIU/L)	2375	7.68	6.33
HOMA-IR	2338	1.72	1.71
QUICKI	2338	0.37	0.04
Total cholesterol (mg/dL)	2482	194	37
LDL cholesterol (mg/dL)	2466	116	33
VLDL cholesterol (mg/dL)	2398	20	11
HDL cholesterol (mg/dL)	2433	58	16
Triglycerides (mg/dL)	2481	102	53
Uric acid (mg/dL)	2394	4.8	1.5
SBP (mm Hg)	2361	117	15
DBP (mm Hg)	2361	73	10
Siesta characteristics			
Do you usually take siesta during the week? (yes, %)[a]	1161	35	
Siesta duration (total week, min)	1161	43	30
Siesta duration (total weekend, min)	656	45	33
Siesta frequency (times per week)	1145	4.5	1.9
Short siesta frequency (times per week)	630	4.6	1.9
Long siesta frequency (times per week)	515	4.3	1.9
Short siesta (% of population)[a]	641	20	
Long siesta (% of population)[a]	520	16	
Causes or motives of siesta			
Tiredness (%)[a]	495	36	
Relax (%)[a]	685	49	
Disconnect from work (%)[a]	85	6	
Need (%)[a]	127	9	
If you could choose between taking siesta or not? (yes, %)[a]	1100	34	
If you could not take siesta, how would you feel?			
Irritated (%)[a]	150	5	
Fatigue (%)[a]	700	21	
No effect (%)[a]	545	17	

(Continues)

TABLE 1 (Continued)

	n	Mean[a]	SD
Associated feelings with siesta			
How do you feel after a siesta if it is short (≤30 min; bad, %)[a]	247	8	
How do you feel after a siesta if it is long (>30 min; bad, %)[a]	634	19	
When you wake up from a siesta, are you hungry? (yes, %)[a]	570	42	
What do you feel like eating after a siesta?			
Sweet (%)[a]	334	63	
Salty (%)[a]	40	7	
Indifferent (%)[a]	159	30	
Where do you have siesta?			
Bed (as place for siesta, %)[a]	214	22	
Sofa (as place for siesta, %)[a]	767	78	
When do you have siesta?			
Seasonal (summer) siesta (%)[a]	365	11	
Classical lifestyle factors involved in obesity			
Energy intake (kcal/d)	2625	2016	819
Energy of breakfast (kcal/d)	1296	314	241
% Energy of breakfast (from total energy)	1297	17	11
Energy of lunch (kcal/d)	1485	769	422
% Energy of lunch (from total energy)	1484	39	14
Energy of dinner (kcal/d)	1455	603	431
% Energy of dinner (from total energy)	1454	30	13
Physical activity (h/wk)	1173	4.40	5.94
Physical activity (Met-min/wk)	2036	3916	6475
Tobacco smoking (yes, %)[a]	560	18	
Number of cigarettes per day	557	11	8
Alcohol drinking (yes, %)[a]	1293	67	
Timing of meals and sleep			
Chronotype score (MEQ)	2177	53	10
Midpoint of meals (h)	2930	14.91	0.66
Timing of breakfast	3066	8.49	2.00
Timing of lunch	3189	14.60	0.59
Timing of dinner	3166	21.33	0.62
Time difference between dinner and sleep onset (h)	2854	2.57	0.91
Midpoint of sleep (h)	2952	3.75	0.73
Sleep onset	2963	23.9	0.91
Sleep offset	2955	7.53	0.99
Nighttime sleep duration (h)	2952	7.60	1.05

Note: Values are means and SD for quantitative variables and percentages for qualitative variables for each characteristic in the total sample.
Abbreviations: DBP, diastolic blood pressure; HDL, high-density lipoprotein; HOMA-IR, homeostatic model assessment of insulin resistance; LDL, low-density lipoprotein; MEQ, Morningness-Eveningness Questionnaire (scores of 41 and below indicate evening type; scores of 59 and above indicate morning type; scores between 42 and 58 indicate intermediate [neither type]); Met, metabolic equivalents; MetS, metabolic syndrome; QUICKI, quantitative insulin-sensitivity check index, (1/log[fasting insulin in microunits per milliliter] + log[fasting glucose in milligrams per deciliter]); SBP, systolic blood pressure; VLDL, very low-density lipoprotein.
[a]Percentage for a qualitative variable. Hours (h) are represented as decimal clock hours.

TABLE 2 Differences among the three classification groups of nappers in obesity-related traits (1): siesta characteristics (2) and obesity-related lifestyle factors further tested as potential mediators between siestas and obesity (3 and 4)

	No siesta		Short siesta (≤30 min)		Long siesta (>30 min)		
	Mean	SD	Mean	SD	Mean	SD	*p* value
General characteristics (1)							
n	2114		641		520		
Age (y)	40	11[a]	44	11[b]	40	12[a]	<0.00005
Women (%)	80[a]		76 [a]		76[a]		0.042
BMI (kg/m^2)	31	5.75[a]	31	5[a]	32	6[b]	0.009
No obesity (BMI < 30 kg/m^2, %)	50		49		44		*0.052*
Obesity (≥30 kg/m^2, %)	50		51		56		
MetS score	2.0	1.2[a]	2.1	1.2[a,b]	2.2	1.2[b]	0.037
MetS (%)	28[a]		33[a,b]		36[b]		0.010
Siesta characteristics (2)							
Bed as place for siesta (%)	22[a]		14[b]		32[c]		<0.0005
Classical lifestyle factors involved in obesity (3)							
Tobacco smoking (yes, %)	17[a]		17[a]		23[b]		0.003
Number of cigarettes per day	10	8[a]	108	8[a]	13	8[b]	0.009
Alcohol drinking (yes, %)	65[a]		72[b]		67[a,b]		0.041
Timing of meals and sleep (4)							
Midpoint of meals (h)	14.91	0.62[a]	14.84	0.64[a]	14.97	0.78[b]	0.018
Physical activity (h/wk)	4.15	3.19[a]	4.33	2.58[a,b]	5.51	13.53[b]	0.027
Energy of lunch (kcal/d)	746	395[a]	790	441[a,b]	840	496[b]	0.031
Chronotype score (MEQ)	52	10 [a]	54	9[a]	51	10[b]	0.001
Timing of dinner with respect to sleep (h)	2.54	0.87[a]	2.56	0.87[a,b]	2.70	1.08[b]	0.009
Sleep onset (h)	23.86	0.89[a]	23.88	0.85[a]	24.07	1.05[b]	0.0002

Note: Values are means and SD for quantitative variables and percentages for qualitative variables. *P* values were derived from one-way ANOVA for quantitative variables among the three groups (no siesta, short siesta, and long siesta) and from χ^2 test for qualitative variables. When significant, differences among groups were explored using Bonferroni correction. Different superscript letters indicate significant differences among groups with post hoc Bonferroni correction.

Abbreviations: MetS, metabolic syndrome; MEQ, Morningness-Eveningness Questionnaire (scores of 41 and below indicate evening type; scores of 59 and above indicate morning types; scores between 42 and 58 indicate intermediate [neither type]). Hours (h) are represented as decimal clock hours.

sofa/armchair (78%), and only 22% napped in bed. Habitual short nappers napped on the sofa 2.5 times more than long nappers (odds ratio [OR]: 2.50, 95% confidence interval [CI]: 1.7–3.8; $p < 0.001$).

Relationship of siesta duration with obesity and MetS traits

As a first approach, we tested the potential association of average siesta duration with BMI. That relationship was found to be positive and significant ($\beta = 0.022$; $p < 0.0001$), which suggests that longer average siesta duration is associated with higher BMI. However, the siesta frequency (times per week) was not significantly associated with BMI ($\beta = -0.009$; $p = 0.915$). Further classification of the population in the no-siesta, short-siesta, and long-siesta groups showed significant differences in BMI and MetS score between groups, i.e., toward higher values in the long-siesta group (Table 2). Along the same lines, regression models showed that long siestas were associated with more obesity- and MetS-related traits compared with no siesta (Table 3). These included BMI, body fat percentage, waist circumference, body weight, fasting blood glucose, SBP, and DBP, as well as a trend toward lower HDL cholesterol (Table 3). In general, individuals who habitually took long siestas had a higher BMI than those who did not take siestas (i.e., a 0.648-kg/m^2 higher BMI; $p = 0.015$), equivalent to an increase of 2.1%. Furthermore, those who habitually took long siestas had a higher MetS score than those who did not (i.e., 0.157-point higher MetS score; $p = 0.014$), equivalent to an increase of 8.1%. In contrast, taking short siestas was nominally associated with lower SBP (a trend; $p = 0.08$) compared with no siesta (Table 3).

Similar results are presented in Table 4 after categorizing individuals into high or low risk based on various cutoff values for obesity and MetS characteristics (see *Methods*). The data show that, among those who habitually took long siestas (compared with no siesta), MetS was more frequent (by 41% more; $p = 0.015$), especially for the blood pressure categories ($p < 0.05$; Table 4). A similar trend was found with obesity, which was higher by 23% (a trend; $p = 0.051$). In contrast,

TABLE 3 Regression coefficients among anthropometric, biochemical, and clinical variables in short nappers and long nappers related to individuals who do not take siesta

		No siesta		Short siesta (≤30 min)			Long siesta (>30 min)		
	Total	*n*	β (95% CI)	*n*	β (95% CI)	*p* value	*n*	β (95% CI)	*p* value
BMI (kg/m^2)	3265	2106	ref.	641	−0.321 (−0.807 to 0.164)	0.194	518	**0.648 (0.124 to 1.172)**	**0.015**
Body fat (%)	3154	2034	ref.	614	−0.066 (−0.6311 to 0.499)	0.820	506	**0.749 (0.144 to 1.353)**	**0.015**
Waist circumference (cm)	3198	2055	ref.	629	−0.973 (−2.136 to 0.189)	0.101	514	**1.402 (0.156 to 2.647)**	**0.027**
WHR	3197	2055	ref.	629	−0.003 (−0.009 to 0.003)	0.364	513	0.006 (−0.001 to 0.013)	*0.085*
WHR[a]	3192	2052	ref.	629	−0.002 (−0.008 to 0.004)	0.582	511	0.004 (−0.003 to 0.104)	0.312
Body weight (kg)	3265	2106	ref.	641	−0.325 (−1.707 to 1.056)	0.645	518	**2.244 (0.754 to 3.733)**	**0.003**
MetS score	2135	1346	ref.	430	−0.008 (−0.126 to 0.109)	0.891	359	**0.157 (0.032 to 0.281)**	**0.014**
Glucose (mg/dL)	2451	1553	ref.	506	−0.496 (−1.951 to 0.959)	0.504	392	**2.388 (0.791 to 3.986)**	**0.003**
Insulin (mg/dL)	2375	1503	ref.	491	−0.394 (−1.027 to 0.239)	0.223	381	0.087 (−0.606 to 0.781)	0.805
HOMA-IR	2338	1476	ref.	487	−0.097 (−0.269 to 0.075)	0.269	375	0.077 (−0.112 to 0.266)	0.426
QUICKI	2338	1476	ref.	487	0.003 (−0.001 to 0.07)	0.104	375	−0.001 (−0.006 to 0.003)	0.534
Cholesterol (mg/dL)	2482	1575	ref.	509	2.020 (−1.620 to 5.662)	0.277	398	−2.336 (−6.316 to 1.643)	0.250
LDL cholesterol (mg/dL)	2466	1566	ref.	506	1.271 (−1.930 to 4.473)	0.436	394	−1.209 (−4.714 to 2.295)	0.499
VLDL cholesterol (mg/dL)	2398	1527	ref.	490	0.062 (−0.971 to 1.096)	0.906	381	0.706 (−0.426 to 1.838)	0.222
HDL cholesterol (mg/dL)	2433	1545	ref.	500	0.519 (−0.942 to 1.980)	0.486	388	−1.527 (−3.129 to 0.074)	*0.062*
Triglycerides (mg/dL)	2481	1576	ref.	510	0.481 (−4.590 to 5.552)	0.853	395	3.275 (−2.291 to 8.841)	0.249
Uric acid (mg/dL)	2394	1519	ref.	491	0.004 (−0.121 to 0.129)	0.947	384	−0.016 (−0.152 to 0.121)	0.827
SBP (mm Hg)	2361	1520	ref.	472	−0.123 (−0.261 to 0.014)	*0.080*	369	**0.232 (0.082 to 0.382)**	**0.002**
DBP (mm Hg)	2361	1521	ref.	471	0.023 (−0.073 to 0.119)	0.640	369	**0.183 (0.078 to 0.287)**	**0.001**

Note: Values are regression coefficients (95% CI) derived from linear regression models adjusted for sex, age, center, and year of recruitment. Bold represents significant values (statistical differences with $p < 0.05$); italics represent nonsignificance but a trend ($p < 0.1$).
Abbreviations: DBP, diastolic blood pressure; HDL, high-density lipoprotein; HOMA-IR, homeostatic model assessment of insulin resistance; LDL, low-density lipoprotein; MetS, metabolic syndrome; QUICKI, quantitative insulin-sensitivity check index, (1/log[fasting insulin in microunits per milliliter] + log[fasting glucose in milligrams per deciliter]); SBP, systolic blood pressure; VLDL, very low-density lipoprotein; WHR, waist-hip ratio.
[a]Further adjusted for BMI.

TABLE 4 Associations between metabolic risk categories and short- or long-siesta groups related to the no-siesta group

		No siesta		*Short siesta (≤30 min)*			*Long siesta (>30 min)*		
	N/cases	*n*	OR (95% CI)	*n*	OR (95% CI)	*p* value	*n*	OR (95% CI)	*p* value
Obesity (BMI ≥ 30 kg/m^2)	3265/1667	2106	1.00 (ref.)	641	0.90 (0.74–1.09)	0.273	518	1.23 (0.99–1.51)	*0.051*
Abdominal obesity (≥88 cm)	3200/2563	2056	1.00 (ref.)	630	0.88 (0.69–1.12)	0.306	514	1.01 (0.78–1.31)	0.939
High triglycerides (≥150 mg/dL)	2481/332	1576	1.00 (ref.)	510	0.93 (0.68–1.27)	0.671	395	1.20 (0.87–1.65)	0.262
Low HDL cholesterol (Spain guidelines)	2433/168	1545	1.00 (ref.)	500	1.20 (0.80–1.81)	0.374	388	1.34 (0.88–2.03)	0.173
Low HDL cholesterol (US guidelines)	2433/605	1545	1.00 (ref.)	500	0.98 (0.77–1.25)	0.887	388	1.11 (0.86–1.43)	0.435
Elevated SBP (≥130 mm Hg)	2361/502	1520	1.00 (ref.)	472	0.79 (0.60–1.05)	0.102	369	**1.35 (1.01–1.80)**	**0.043**
Elevated SBP (≥120 mm Hg)	2361/1310	1520	1.00 (ref.)	472	**0.79 (0.63–0.99)**	**0.044**	369	**1.36 (1.05–1.75)**	**0.020**
Elevated DBP (≥80 mm Hg)	2361/979	1521	1.00 (ref.)	471	0.99 (0.78–1.24)	0.909	369	**1.54 (1.20–1.97)**	**0.001**
Elevated glycemia (≥110 mg/dL)	2451/115	1553	1.00 (ref.)	506	0.86 (0.52–1.42)	0.56	392	1.47 (0.90–2.41)	0.125
Elevated glycemia (≥120 mg/dL)	2451/66	1553	1.00 (ref.)	506	0.81 (0.42–1.58)	0.546	392	1.59 (0.86–2.96)	0.141
MetS (≥ 3 components)	2135/651	1346	1.00 (ref.)	430	0.98 (0.76–1.27)	0.886	359	**1.41 (1.07–1.86)**	**0.015**

Note: Values are OR and 95% CI, for the development of MetS criteria according to siesta categories. All models adjusted for sex, age, center, and year of recruitment. Bold represents significant values (statistical differences with $p < 0.05$); italics represent nonsignificant data but those that showed a trend ($p < 0.1$). Spain guidelines for HDL cholesterol: <40 and <50 mg/dL in men and women, respectively. US guidelines for HDL cholesterol: <40 mg/dL.
Abbreviations: DBP, diastolic blood pressure; HDL, high-density lipoprotein; MetS, metabolic syndrome; OR, odds ratio; SBP, systolic blood pressure.

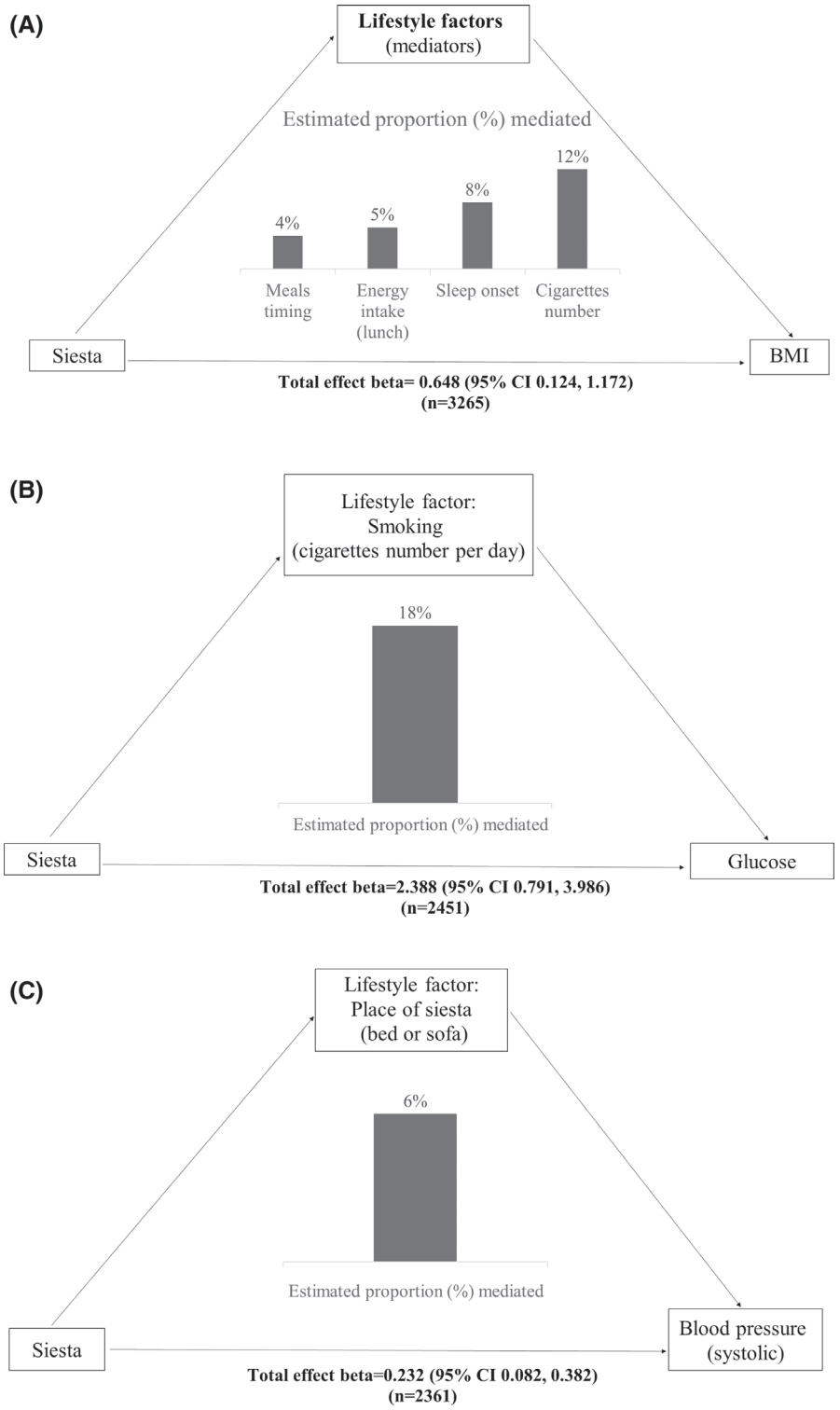

FIGURE 1 (A) Mediated total effect of long siestas vs. no siesta on BMI. (B) Mediated total effect of long siestas vs. no siesta on glucose. (C) Mediated total effect of long siestas vs. no siesta on systolic blood pressure. All models were adjusted for sex, age, center, and year of recruitment

elevated SBP (≥120 mm Hg) was significantly less frequent (by 21%) with short siestas compared with those who did not take siestas ($p = 0.044$). Further sensitivity analyses, including the level of education as a covariate, showed no changes in significance (data not shown).

Lifestyle factors mediating the association of long siestas with BMI and MetS traits

When analyzing potential differences between the three siesta categories and obesity-related lifestyle factors, data showed that those

who habitually took long siestas (compared with no siesta; Table 2) smoked more frequently and delayed behaviors such as meals, physical activity, and sleep. In general, long-siesta nappers were more evening types than the short nappers or non-nappers. Long nappers chose the bed (vs. sofa or armchair) as the place of napping more frequently (Table 2). Interestingly, nighttime sleep duration was not significantly different between the three siesta categories (mean [SD], no-siesta = 7.69 [1.02] hours; short siesta = 7.76 [0.85] hours; and long siesta = 7.68 [1.01] hours; $p = 0.480$) or between individuals with different degrees of obesity (normal weight = 7.64 [0.96] hours; overweight = 7.65 [0.96] hours; and obesity = 7.57 [1.13] hours; $p = 0.819$) in the ANOVA test. Similarly, the level of education was not significantly different between the three siesta categories ($p = 0.324$). Nevertheless, significant differences were found in the level of education between individuals with different degrees of obesity (i.e., university education percent: normal weight = 50%; overweight = 42%; and obesity = 35%; $p < 0.00001$) in the χ^2 test.

Those lifestyle factors that were significantly different between the three siesta categories were further tested as potential mediators in the association between long siestas (vs. no siesta) and obesity. The results of the mediation analyses are shown in Supporting Information Tables S2–S7 and in Figure 1. Smoking (higher number of cigarettes per day), timing of meals and sleep (i.e., delayed midpoint of meals and delayed onset of sleep), and distribution of energy intake throughout the day (higher energy intake at lunch, the meal preceding siestas) mediated the association between long siestas and higher BMI, as shown by the significant average causal mediated effect (Figure 1A; Supporting Information Table S2).

The same trend was found for the delayed midpoint of meals on the association of long siestas with abdominal adiposity (greater waist circumference; $p = 0.08$; Supporting Information Table S4). Dinner timing relative to (nighttime) sleep onset was also a significant mediator of the association of long siestas with BMI. However, multicollinearity tests among significant mediators using Pearson correlation coefficients showed a strong correlation between the variables dinner timing relative to sleep onset and sleep onset ($r = 0.75$; $p < 0.001$), and stepwise regression models removed this variable.

The number of cigarettes mediated the effect of long siestas on blood glucose ($p = 0.044$; Supporting Information Table S3). We found the same trend ($p = 0.055$) for the place where siestas were held, i.e., sofa/armchair or bed, toward higher values in SBP among those who took long siestas in bed (Figure 1B,C; Supporting Information Table S6).

Physical activity level, breakfast timing, sleep offset, post-siesta feelings, and alcohol consumption were not significant mediators of the association of long siestas with BMI (Supporting Information Table S2). None of the variables tested mediated the association between long siestas and MetS (Supporting Information Table S5). Furthermore, no significant mediators were identified in the beneficial association of short siestas with the reduction of elevated SBP (≥120 mm Hg; data not shown).

DISCUSSION

In the present study, long siestas (>30 minutes) were associated with higher BMI and increased odds for having MetS (41% higher), in particular, in waist circumference, fasting blood glucose, and blood pressure (both SBP and DBP), compared with no siesta. In contrast, short siestas were associated with a lower frequency (21% lower) of elevated blood pressure, i.e., SBP ≥ 120 mm Hg, compared with no siesta. In this Mediterranean population, we identified several lifestyle factors as potential mediators of the association between long siestas and BMI, including a higher number of cigarettes smoked per day, later timing of behaviors (later meals and later sleep), and higher energy intake at lunch. Interestingly, the usual napping place (in bed or on the sofa/armchair) showed a trend for mediating the association between long siestas and SBP.

This study provides comprehensive information on napping in volunteers who had no history of pathology and who were not taking medication. The percentage of nappers in this Mediterranean region of Spain was 35%, and the average number of naps taken was four naps per week; these numbers were similar to other countries in Europe, America, or Asia [15]. The main reason for napping in the current cohort was looking to relax (in 49% of nappers), followed by tiredness (36%). Although napping is traditionally thought to be a good habit and beneficial to health, this notion has become controversial because of studies that have shown that daytime napping is associated with a greater prevalence of obesity and other metabolic alterations [13]. Here, we hypothesize that siesta duration and different lifestyle factors classically related to obesity may help explain this controversy.

In agreement with previous studies [13, 16, 17], our data showed that long siestas were associated with higher values of obesity- and MetS-related traits, particularly BMI, waist circumference, glucose, and blood pressure (SBP and DBP), as compared with no siesta, whereas short siestas were nominally associated with lower values of SBP and with a significantly lower prevalence of elevated blood pressure, i.e., SBP ≥ 120 mm Hg (cutoff point for "elevated" blood pressure) [25]. Of relevance, the changes detected were primarily in the long nappers versus non-nappers and the short nappers versus non-nappers, not necessarily in the long nappers versus short nappers.

To our knowledge, this is the first study to explore whether different lifestyle factors or siesta traits mediate the association between long siestas and obesity. Our data suggested that a higher number of cigarettes smoked per day, later timing of behaviors (later meals and later sleep), and higher energy intake at lunch (the meal preceding siestas), mediate this association.

Alterations in the circadian system, e.g., through changes in cortisol daily rhythms, may be involved in the connections between long siestas and obesity [32]. The cortisol response upon awakening (CAR) has been shown to increase after longer naps, producing elevated evening cortisol [33]. The daily cortisol rhythm is strongly influenced by the circadian system and is involved as a zeitgeber for peripheral tissues [32], and alterations in cortisol rhythmicity have been shown to induce circadian disruption leading to insulin resistance, central obesity, and MetS [34]. In addition, the increase in CAR after napping [33] may help explain why 42% of the current population were hungry when awakening from a siesta, given the role of cortisol in motivating food intake [35]. Along these lines, the higher number of cigarettes smoked per day, which mediated, by 12%, the association between

long siestas and higher BMI, has been reported to associate with an increase in CAR [36]. In addition, the high energy intake at lunch, which mediated the positive association between BMI and long siestas by 5%, might affect the lunch-induced cortisol response and also modify CAR after napping. Additionally, the distension of the gut that follows a copious meal may increase postprandial sleepiness that depends on the amount of food consumed [37] and, therefore, might increase siesta duration.

The timing of lunch also mediated the association between long siestas and obesity (BMI) by 4%. Eating late can simultaneously increase hunger, decrease energy expenditure, and modify lipid metabolic pathways toward decreased lipolysis/increased adipogenesis [38]. We also identified delayed nighttime sleep onset as a significant mediator between long siestas and obesity. Longer siestas may delay bed timing, which has been associated with general obesity [39]. Interestingly, the place of napping, i.e., sofa/armchair or bed, tended to mediate the effect of long siestas on SBP. As previously introduced, acute changes in posture of the body with napping, as happens when a siesta is taken in bed (but less on the sofa or armchair, because the individual is usually sitting), have been related to cardiovascular risk [18].

Results are in line with previous studies that have shown that short naps were not associated with cardiovascular disease, whereas longer naps (>1 hour) were associated with higher cardiovascular risk, and a significant J-curve dose-response relationship among the length of the nap and cardiovascular diseases has been observed, with the relative risk ratio decreasing from 0- to 30-minute nap duration, indicating a protective effect against cardiovascular risk of short naps of 30 minutes or less [15]. Although we were not able to identify significant lifestyle mediators in the beneficial association of short siestas with lower frequency of elevated SBP, previous studies have suggested that the decreased release of sympathetic system mediators such as catecholamines with short naps may be involved in the beneficial effects on SBP [40]. Furthermore, short siestas are mainly composed of stages 1 and 2 of sleep. The transition to stage 1 sleep has been reported to lower blood pressure acutely [41], and stage 2 sleep (a minimum of 3-minute stage) has been shown to play an important role in the restorative function of a nap [42]. Short siestas of less than 30 minutes, also called power naps, end before deep slow-wave sleep onset and they have been shown to limit sleep inertia [43], increase reaction times, and improve memory performance [15, 44, 45]. This may be especially beneficial for the Spanish population because of the frequent long-working-hours schedules (usually starting at 8:00 a.m. and ending at 8:00 p.m. in this Mediterranean area). In addition, short siestas may be advisable in this Mediterranean population because current data showed that more people felt well after taking a short siesta (than after a long siesta). In contrast to short naps, during a long nap, the individual may have entered a deeper stage of sleep, and forced awakenings could lead to feeling unwell upon awakening and, at least transiently, even sleepier (i.e., groggy) than when not napping [46].O

Limitations

Our results are based on a cross-sectional design. Although our previous study, using Mendelian randomization, showed that daytime napping is causally linked to obesity [14], some components of napping may be a consequence of obesity. We will not be able to conclude causality or directionality from our results. Future longitudinal cohort studies or experimental studies are needed to confirm the relationship between nap duration and the development of obesity and MetS and to test the mechanisms involved. Also, self-reported daytime napping data might result in recall bias; we collected information on siesta duration based on self-reports and not objective assessment (such as actigraphy). However, a previous study showed relatively good agreement between objective and subjective measures of sleep-related parameters [47]. In the current study, none of the volunteers included was diagnosed with obstructive sleep apnea. However, we cannot discard the possibility that participants could have had undiagnosed sleep disorders or subclinical alterations.

Strengths

This study provides novel information on the mediating effect of some lifestyle factors among long siestas, obesity, and MetS. We used data from a population coming from a Mediterranean area in Spain, where siestas are relatively accessible because it is part of the culture. Ours is a large, healthy, and relatively young population. Therefore, we expect that the results obtained are related to siestas and not to aging or comorbidities. Most studies on napping are conducted in older individuals and show that an increased duration of napping is associated with reduced nighttime sleep duration [17]. However, as previously reported in younger and healthy populations where napping does not influence nocturnal sleep [48], we did not find significant differences in nighttime sleep duration among individuals who usually do not take siestas or those who take short or long siestas. We also have a precise phenotype of siesta characteristics and motives for napping. Finally, we have a detailed phenotype of lifestyle factors mediating the effect of long siestas on obesity and MetS traits.

In conclusion, siesta duration is a relevant aspect to consider in obesity traits and MetS. In the current Mediterranean population, long siestas were associated with higher values of obesity traits and of MetS than no siesta, whereas a shorter duration of siesta was associated with a lower prevalence of having elevated SBP values. We identified several potential lifestyle mediators in the association between long siestas and obesity. These results call for studies to investigate whether short siestas are advisable over long siestas, especially in those individuals who usually have delays in eating and sleeping schedules or in those who smoke. Furthermore, studies are needed to test whether a lower caloric intake at lunch may decrease any harmful effects of long siestas on obesity and SBP.O

AUTHOR CONTRIBUTIONS

All authors listed fully meet the criteria for authorship. Conceptualization: Marta Garaulet and Barbara Vizmanos; methodology: Marta Garaulet and Barbara Vizmanos; formal analysis: Marta Garaulet, Barbara Vizmanos, Ana Isabel Cascales, Diego Salmerón, and Eva Morales; resources: Marta Garaulet; data curation: Marta Garaulet, Barbara Vizmanos, Ana Isabel Cascales, Diego Salmerón, and Eva Morales; writing, original draft

preparation: Barbara Vizmanos, Ana Isabel Cascales, María Rodríguez-Martín, Aurora Aragón-Alonso, Frank A. J. L. Scheer, and Marta Garaulet; writing, review and editing: Barbara Vizmanos, Ana Isabel Cascales, María Rodríguez-Martín, Aurora Aragón-Alonso, Marta Garaulet, and Frank A. J. L. Scheer; visualization: Marta Garaulet, Barbara Vizmanos, and María Rodríguez-Martín; supervision: Marta Garaulet and Frank A. J. L. Scheer. All authors have read and agreed to the final version of the manuscript.

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CONFLICT OF INTEREST STATEMENT

Frank A. J. L. Scheer served on the Board of Directors for the Sleep Research Society and has received consulting fees from the University of Alabama at Birmingham. Frank A. J. L. Scheer's interests were reviewed and managed by Brigham and Women's Hospital and Partners HealthCare in accordance with their conflict of interest policies. Frank A. J. L. Scheer's consultancies are not related to the current work. The other authors declared no conflict of interest.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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