Changes in objectively measured sleep after a multidisciplinary lifestyle intervention in children with abdominal obesity: A randomized trial

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ARTICLE INFO

ABSTRACT

Background/objective: childhood obesity and sleep disorders have a well-established cross-sectional association, but lifestyle interventions' effects on sleep quality remain under-researched. This study aimed to evaluate the sleep quality of 122 participants (7–16 years) with abdominal obesity after a 2-year necessary lifestyle intervention.
Patients/methods: participants were assigned to either the intervention group (moderate hypocaloric Mediterranean Diet) or the usual care group (standard recommendations on a healthy diet). Sleep was objectively assessed using triaxial accelerometry, and sleep parameters analyzed included latency, efficiency, wake after sleep onset, total time in bed, total sleep time, number of awakenings, and awakening duration.
Results and conclusions: the results showed that the intervention group significantly improved sleep latency at 12 and 24 months and improved sleep efficiency at 2 and 12 months, compared to the usual care group. Wake after sleep onset and the number of awakenings were significantly reduced at 24 months in the intervention group. Wake after sleep onset and leptin levels were positively associated in all participants. Total time in bed was inversely associated with triglycerides and metabolic score, and total sleep time was inversely associated with leptin, triglycerides, and metabolic score after the 2-month intervention. Triglyceride levels were inversely associated with total time in bed and total sleep time at one year, while the metabolic score was directly associated with wake after sleep onset and the number of awakenings and inversely associated with efficiency. In conclusion, the multidisciplinary intervention in children and adolescents with abdominal obesity reduced anthropometric parameters and improved sleep habits.

1. Introduction

Obesity is a chronic disease which prevalence in Spanish children has increased in the last two decades from 7.8% to 10.5% [1]. The main factors that contribute to obesity are genetics, excessive energy intake, decreased physical activity, and sedentarism. Sleep duration, timing, and chronotype have also recently been recognized as possible risk factors for obesity in children [2]. Furthermore, children with obesity have less good sleep habits [3]. Limited longitudinal evidence exists regarding the relationship between sleep disorders and weight gain in children. Two prospective cohort studies observed that chronic short sleep from infancy through school age was associated with increased adiposity indicators and obesity [4,5].

A recent meta-analysis (86 out of 103 studies) reported a significant inverse association between sleep duration and measured weight status [6]. However, few studies examined other dimensions of sleep, such as quality, efficiency, bed/wake times, and their relationship with weight status [6]. Inadequate or disrupted sleep has repeatedly been associated with an elevated likelihood of developing obesity during adolescence [7, 8].

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Furthermore, the role of sleep habits in childhood obesity risk on health status in future age has received considerable scientific attention. Although, most of the analyses included subjective measures of sleep or parent-reported sleep with a high risk of bias. Questionnaires, sleep diaries, or parental reports on their children's sleep may not be accurate, and the precision may be affected by the context of the study [9]. Thus, clinical and research settings should consider objective sleep quality indicators.

Accelerometers are one of the most precise tools to assess sleep in children's daily living conditions over multiple days. In a cross-sectional study of children using actigraphy, later bedtime, greater variability in bedtime, and wake time were associated with greater adiposity and an increased risk of obesity [10,11]. Implementing lifestyle interventions that include advice regarding sleep improvement or evaluation of sleep quality alongside promoting other healthy lifestyle behaviors are necessary to tackle childhood obesity. In this line, the IDEFICS
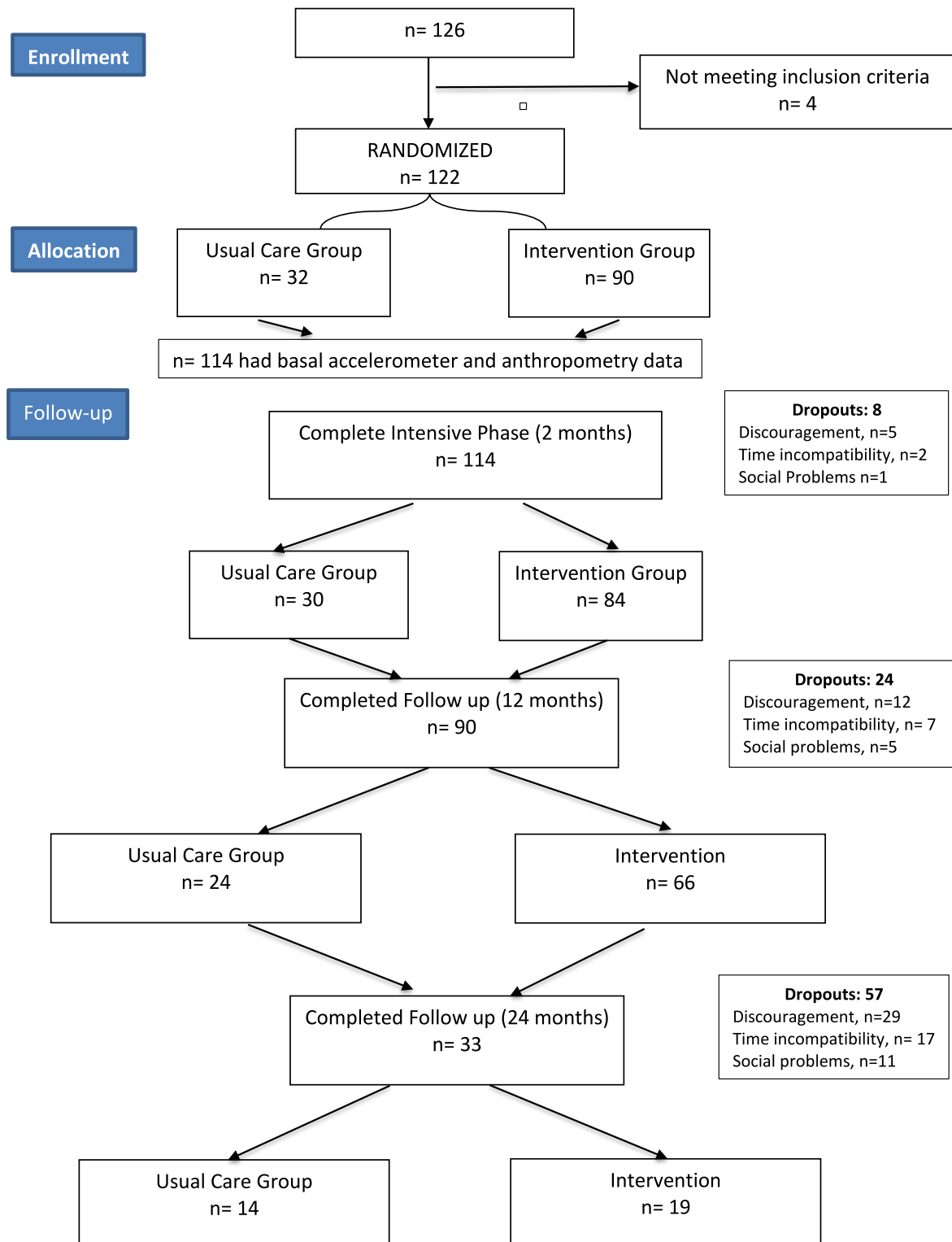

Fig. 1. Flow chart of participants of IGENOI study.

(Identification and Prevention of Dietary-induced and lifestyle-induced health Effects in Children and infantS') implemented a multilevel intervention that included sleep duration as a critical behavioral target in children from 2 to 9.9 years of age. They observed a small intervention effect on weeknight sleep duration (measured by a parental 24-h recall) over two years [12].

Based on this, objective measures are necessary to evaluate sleep quantity and quality. To our knowledge, no studies have objectively assessed changes in sleep parameters in pediatric subjects with obesity after a lifestyle intervention. For this reason, our study aimed to assess the effectiveness of an intervention (usual care vs. intervention group) on sleep quality and its relationship with changes in biochemical and Metabolic Syndrome related anthropometric parameters.

2. Material and methods

2.1. Participants

The IGENOI *(Intervention Grupo Estudio Navarro de Obesidad Infantil)* study is a randomized controlled trial (NCT031472) conducted in Pamplona, Navarra (Spain). It is a two-year family-based lifestyle intervention program involving children and adolescents with abdominal obesity. Participants were recruited at the Pediatric Endocrinology Units of *Clínica Universidad de Navarra,* Hospital Universitario de *Navarra,* and Primary Care Health Centers in Pamplona. According to national data, the study population comprised children aged seven to sixteen with a waist circumference above the 90th percentile [13]. The exclusion criteria included prevalent prediabetes or any other endocrine disorders, food intolerance, eating disorders or psychiatric disease, pharmacological treatment, regular alcohol consumption, or special diet treatment. The study followed the ethical standards recognized in the 2013 Declaration of Helsinki (Fortaleza, Brasil, October 2013) and was approved and supervised by the Human Research Ethics Committee of the University of Navarra (Reference number 044/2014). The parents or legal guardians and children involved in the trial received detailed explanations about the aim of the study. Informed assent or consent was obtained from every participant, and all the parents or legal guardians signed an informed consent according to the Declaration of Helsinki.

A total of 126 children were initially recruited. One hundred twenty-two met the inclusion criteria, and 114 had basal accelerometer and anthropometric data available (see Fig. 1). The study population includes 122 children with obesity (BMI-SDS: 2.91 $\pm$ 1.08) (mean age 11.25 $\pm$ 2.45 years, 37.7% boys) (76 girls, 46 boys), and waist circumference over the 90th percentile. Children were predominantly white/Caucasian. During the follow-up, accelerometer and anthropometric data were available as follows: one hundred and fourteen at two months, 90 at 12 months, and 33 at 24 months (dropout rate: 6,6%, 26,3%, and 88,5% respectively), and the main reasons were discouragement, social problems, inability to comply the visits (school exam periods, and intercurrent infections) as described in other pediatric trials [14].

2.2. Experimental design and lifestyle intervention

The multidisciplinary intervention consisted of a two-year program that comprised a 2-month intensive phase with individual and group sessions and a follow-up period at 12 and 24 months. A multidisciplinary team, including registered dieticians, pediatricians, physical activity experts, and nurses, conducted the intervention in a clinical setting. Parents or legal guardians accompanied them to the visits.

Subjects were randomly assigned to the usual care (UCG) or intervention group (IG) at a ratio of 1:3. The randomization was performed using computer-generated randomization.

UCG received one 30-min individual session with the dietitian and standard pediatric recommendations for a healthy diet (SENPE 2016) (Aranceta Bartrina et al., 2016) and ten monitoring visits to assess anthropometric measurements during the first year of intervention. During the two months, usual care subjects and their parents received a 30 min individual session with the dietician and five monitoring visits to assess anthropometric parameters.

IG was advised to follow a fully-day meal plan during the intensive phase, consisting of a moderately hypocaloric Mediterranean diet [15]. The dietary pattern was based on high consumption of fruits (3 portions per day) and vegetables (2 portions per day), legumes, whole grains, and olive oil; moderate consumption of dairy products, poultry, and fish; and the reduction of processed and red meats, limiting them to 1 portion per week. The energy expenditure was calculated using the Schofield equation (adapted to age and sex) [16]. The calorie restriction applied varied from 10 to 40% depending on the standard deviation score of Body Mass Index (SDS-BMI) and trying not to interfere with the participant's body growth [17,18]. Caloric diets below 1300 Kcal and above 2200 Kcal were not prescribed. Energy intake (percentage) was distributed into five meals according to the Spanish Society of Community Nutrition [19]: breakfast 20%, morning snack 5–10%, lunch 30–35%, afternoon snack 10–15% and dinner 20–25% of total energy. The distribution of main macronutrients was as follows: carbohydrates 55%, lipids 30%, and proteins 15% of total energy intake. During the 2-month intensive phase, patients were prescribed to follow up on a diet, and they received six 30-min sessions every two weeks, conducted by dieticians, to evaluate the accomplishment of diet and anthropometric measurements. In addition, they received one parallel group session where children and adolescents learned about healthy lifestyles, including eating behavior (portion control) and the importance of being physically active (sedentary activities and physical activity) [15]. The parents or legal guardians were taught about their role in the study and obesity-related problems. During the follow-up period, intervention participants had monitoring visits at 3, 4, 5, 6, 9, and 12 months, where nutrition educational topics about healthy breakfast and food choices were given at months 3 and 5 from baseline and at month 4 a group session about groups of foods and frequency of food consumption was taught to children and parents or legal tutors.

No advice was given to participants regarding to the timing of meals that could potentially affect the timing of sleep.

Regarding physical activity, both groups were advised to accumulate 200 min of physical activity per week at 60–75% of their maximum heart rate as recommended by The American College of Sports Medicine [20]. The subjects were advised to enroll in biweekly physical activities organized by their schools or public or private sports centers. No advice was given to participants regarding sleep habits.

2.3. Anthropometric, clinical, and biochemical measurements

Trained personnel conducted all anthropometric measurements (body weight, height, waist, and hip circumferences) using calibrated equipment at the beginning, 2, 12, and 24 months. Participants were barefoot and wearing a gown.

Height was measured using a Harpenden's stadiometer of 1 mm precision (Seca 220, Vogel & Halke, Hamburg, Germany). Body weight and fat mass were measured to the nearest 0.1 kg/% using a digital scale (BC-418 Segmental Body Composition Analyzer, TANITA, Tokyo, Japan). Body Mass Index (BMI) was calculated as weight divided by height squared (Kg/m^2) and converted into standard deviation scores (SDS) for sex and age derived from Spanish reference data according to specific cut-off points for BMI [21]. Waist (W) and hip (H) circumferences (C) were assessed with a non-elastic measuring tape (Type Seca 200) following standard procedures (placed horizontally halfway between the last floating rib and the top of the iliac crest, and the measurement was taken at the end of a normal breath). Finally, pubertal development was evaluated according to Tanner stages [22]. The pubertal stage and the presence of acanthosis nigricans were examined by pediatric endocrinologists of the team [23].

Venous blood samples were obtained after an overnight fast.

Standard autoanalyzer techniques determined glucose, insulin, and lipid profile. Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated from fasting glucose and insulin values. Leptin serum levels were measured by ELISA (R&D Systems, Minneapolis, MN). All the measurements were taken at baseline and after 2, 12 and 24 months.

Blood pressure was measured using an electronic sphygmomanometer (OMRON M6, Hoofddrop, The Netherlands) on the right arm after the children rested quietly for 15 min, according to the American Heart Association guidelines [24].

A metabolic syndrome score was calculated using the following formula [25]:

(2xwaist)/height + (glucose/5.6) +triglycerides/1.7 + (systolic bp/130)-HDL/1.02.

(Glucose, triglycerides, and HDL in mmol/L).

2.4. Objective sleep assessment

Sleep was objectively assessed using triaxial accelerometry (Actigraph wGT3XBT, Actigraph LLC, Pensacola, FL, USA) at baseline, 2, 12, and 24 months. Participants wore the accelerometer for five days, including at least two weekdays. Participants and parents were instructed on wearing the device on an elastic belt positioned over the non-dominant waist at the right mid-axillary line, above the iliac crest in line with current recommendation [26], including sleep time, and removing it just for water-related activities (bathing, showering, and swimming). The monitors were initialized using 60-s epochs, as described elsewhere [27].

Accelerometry data were analyzed using Actilfe 6.0 software (Actigraph wGT3X-BT, Actigraph LLC, Pensacola, FL, USA). Continuous 24-h accelerometer data was recorded from weekdays and weekends and analyzed separately.

Sleep data were analyzed using the Sadeh algorithm derived from fundamental research performed by Avi Sadeh et al. [28]. This algorithm was commonly used in younger adolescents (10–11 years old) [29]. The following sleep variables were calculated: latency (time since lying in bed to start sleep), sleep onset (time to go to sleep), sleep offset (time awake in the morning), wakefulness after sleep onset (WASO: total number of minutes that a person is awake after having initially fallen asleep), number of awakenings and awakenings duration (total minutes awake), total sleep time (also referred as true sleep time, minutes between sleep onset and offset, excluding wakes), total time in bed and sleep efficiency (percentage of time asleep between sleep onset and offset expressed as a percentage). Sleep variables were calculated for five days and during weekdays and weekends separately. Weighing data for all the sleep variables was also done by weighing five times on weekdays plus two times on weekend days and dividing this result by seven, as previously reported [12]. Once sleep was accounted for, non-wear time in different activity intensities was only calculated from the awake data. A day was scored as valid if at least eight valid awake hours existed. Non-wear time was calculated as at least 20 min of consecutive zeros.

2.5. Dietary intake assessment

Trained dieticians collected dietary intake data at baseline, after two months and the two points of follow-up (at 1-year and 2-year) using a baseline semi-quantitative 136-item Food-Frequency Questionnaire (FFQ) [18], which was previously validated in Spain and latterly re-evaluated [30,31].

2.6. Statistical analysis

The primary endpoint of the IGENOI study was to assess the effectiveness of lifestyle interventions based on BMI-SDS values. Changes in BMI-SDS are the primary outcome since successful interventions should decrease BMI-SDS near 0.5 units to decrease cardiometabolic risk [32].

Considering this information, we based the sample size on the following assumptions: an alpha error of 5%, a power of 90%, a 1:3 ratio, and a mean difference of 0.50 (SD 0.47) units in BMI-SDS after the lifestyle intervention. The sample size calculation indicated that 15 and 45 subjects were needed for UCG and IG respectively. The rationale for the difference in the size of both groups relies on the fact that a high number of subjects could benefit from intensive intervention as indicated in other programs for obese children [33,34]. The randomization of the patient groups (UCG or IG) was performed by computer-generated randomization using standardized protocols. This study was analyzed under the principle of "intention-to-treat".

The means for the measurements of interest obtained from the multiple day's accelerometer data were calculated for each participant. The means for weekday and weekend days were used to calculate a weighted average for each variable. Total sleep variables were obtained by weighing five times on weekdays plus two times on weekend days and dividing the result by seven, as previously reported for objectively measured physical activity [12].

Student's unpaired and paired t-tests were used to compare changes between or within groups. The change in each variable was calculated as the difference between post and pre-intervention values for each subject. The Bonferroni method was used to perform pairwise comparison following a significant overall test [35]. To maintain the $\alpha = .05$ significance level for the table comparisons using a Bonferroni adjustment, a p-value must be less than $p = 0.01667$. McNemar was test used to analyze paired nominal data. Pearson or Spearman correlations were performed to study the association between sleep variables and anthropometrical or biochemical outcomes. Furthermore, we fitted a multivariable-adjusted linear regression model to examine whether baseline sleep data or changes could predict changes in anthropometry and biochemical outcomes at 2, 12, and 24 months after lifestyle intervention. The potential confounders were sex, age, and Tanner stage.

All statistical analyses were two-tailed, and a p-value <0.05 was considered statistically significant. Clinical and biochemical data were described using mean ± SD. Given their basal sleep values, no participants were identified as an extreme outlier.

STATA 12.0 for Windows (version 12.0, College Station, TX: StataCorp LP, USA) was used for statistical analyses.

3. Results

3.1. Effects of lifestyle intervention on anthropometric and biochemical parameters

Anthropometric and biochemical parameters in each timepoint are presented in Table 1. There were no significant differences between groups for age, sex, and Tanner stage. Most anthropometric and biochemical parameters were similar at baseline except for glucose levels. The UCG group (n = 32) had significantly (p < 0.001) higher mean glucose levels compared with the IG (n = 90). The decrease in BMI-SDS was statistically significant at 2 months, and 12 months of follow-up in both groups and at 24 months in the IG. Fat mass (%) significantly decreased (p < 0.001) at 2, 12 and 24 months in the IG and at 2 (p < 0.001) and 12 months (p < 0.05) in the UCG, finding statistically significant differences in fat mass changes (%) between groups at 2 months, with a greater fat mass reduction in the IG (p < 0.05). By contrast, lean mass (Kg) significantly increased (p < 0.001) in both groups at 12 and 24 months. Waist circumference was reduced by 4 cm in both groups at 2 months. At 12-month only in the IG remained the significant reduction. Finally, hip circumference was reduced at 2 months compared to baseline in both groups. However, UCG increased its values over the follow-up, and the IG maintained a similar value to the baseline.

Regarding biochemical parameters (Table 1), glucose levels significantly decreased after 2 months in both groups, with a greater significant change (p < 0.001) in the UCG (p < 0.05), probably due to the

Table 1
Changes in anthropometric and biochemical parameters after lifestyle intervention.

	Usual Care Group (n = 32)				Intervention Group (n = 90)			
Age (years)	10.7 ± 2.3				11.5 ± 2.5			
Sex (n, M/F)	20/12				56/34			
Tanner Stage (%)	I 40.6/II 9.4/III 28.1/IV 3.1/V 18.8				I 32.2/II 22.2/III 13.3/IV 7.8/V 24.4			
Time Point	*0 (n = 32)*	*1 (n = 30)*	*2 (n = 24)*	*3 (n = 14)*	*0 (n = 90)*	*1 (n = 84)*	*2 (n = 66)*	*3 (n = 19)*
Anthropometric								
BMI-SDS	2.6 ± 0.9	**2.1 ± 0.9**	**1.9 ± 1.3**	1.5 ± 1.5	2.3 ± 1.1	**2.5 ± 1.2**	**2.5 ± 1.3**	**2.0 ± 0.7**
Fat mass (%)	37.7 ± 8.5	**36.2 ± 8.7**	**35.5 ± 10.0**	34.8 ± 6.7	36.9 ± 5.4	**34.0 ± 6.0**	**33.1 ± 7.5**	**31.4 ± 6.7**
Lean mass (kg)	38.7 ± 8.7	38.8 ± 8.5	**41.6 ± 9.1**	**45.8 ± 7.4**	42.0 ± 10.9	41.7 ± 1.3	**43.3 ± 11.2**	**45.2 ± 10.8**
WC (cm)	86.6 ± 11.0	**82.3 ± 11.4**	84.2 ± 10.8	86.3 ± 9.5	86.2 ± 11.2	**82.4 ± 1.2**	**82.6 ± 12.1**	81.5 ± 9.2
HC (cm)	97.3 ± 11.9	**96.0 ± 10.8**	**98.7 ± 11.2**	**102.4 ± 9.1**	98.9 ± 12.7	**96.2 ± 3.3**	97.7 ± 14.1	**98.2 ± 11.8**
Biochemical								
Glucose mg/dL	<u>91.6 ± 6.0</u>	**86.0 ± 5.2**	88.1 ± 5.1	91.0 ± 6.9	<u>87.8 ± 6.5</u>	**85.7 ± 6.2**	87.0 ± 6.6	87.2 ± 5.6
Insulin μU/mL	20.6 ± 19.9	17.6 ± 13.7	13.7 ± 7.5	20.2 ± 17.7	16.4 ± 8.3	**13.0 ± 5.7**	**13.0 ± 5.7**	13.6 ± 5.8
HOMA-IR	4.6 ± 4.7	3.7 ± 2.9	2.98 ± 1.8	4.59 ± 3.6	3.6 ± 2.0	**2.7 ± 1.3**	**2.8 ± 1.3**	2.93 ± 1.3
Leptin ng/mL	38.5 ± 22.1	**26.8 ± 15.0**	**27.4 ± 22.8**	29.9 ± 24.1	35.1 ± 17.7	**24.6 ± 19.7**	32.6 ± 27.3	25.2 ± 16.8
LDL/HDL	2.2 ± 0.5	2.2 ± 0.8	**1.8 ± 0.5**	1.95 ± 0.4	2.2 ± 0.7	2.1 ± 0.6	**2.1 ± 0.7**	**2.1 ± 0.6**
Triglycerides	95.9 ± 38.2	**82.3 ± 39.0**	**67.8 ± 32.1**	66.7 ± 21.0	90.2 ± 43.9	75.9 ± 32.3	**74.1 ± 31.3**	75.8 ± 29.4
Metabolic Score	2.4 ± 0.5	**2.15 ± 0.6**	**1.9 ± 0.6**	2.2 ± 0.6	2.3 ± 0.6	**2.1 ± 0.5**	**1.9 ± 0.6**	1.9 ± 0.7

Data are expressed as mean ± SD. Timepoint: 1, at baseline; 2, at 2-month of intensive intervention; 3, at 1 year of follow-up; 3, at 2 years of follow-up. <u>Underline values</u> indicated significant differences at baseline between groups ($p < 0.05$). **Bold values** indicated significant changes at follow-up compared to baseline within each group ($p < 0.05$). Abbreviators: M, Male; F, Female BMI-SDS, Body Mass Index Standard Deviation Score; NC, WC, Waist Circumference; HC, Hip Circumference.

higher glucose values at baseline. Insulin levels significantly decreased ($p < 0.05$) in the IG at 2 and 12 months. In the UCG, insulin levels also decreased without reaching statistically significant levels. HOMA-IR index was statistically significantly decreased ($p < 0.05$) from baseline to 2 and 12 months in the IG. No statistically significant differences existed between groups in the HOMA index or insulin level changes.

A significant reduction ($p < 0.001$) in leptin levels was observed in both groups at 2 months of follow-up. However, leptin levels increased significantly at 12 months in the UCG group. Leptin levels changes did not show statistical significant differences between groups at any time of the study. LDL/HDL ratio significantly decreased at 12 months ($p < 0.05$) in both groups and at 24 months ($p < 0.05$) in the IG. The reduction in triglyceride levels was significant during the follow-up period in the UCG. However, in the IG significant decrease was shown at 12 months. The metabolic score showed a significant decrease during follow-up in the IG and at 2 and 12 months in the UCG.

3.2. Sleep changes after lifestyle intervention

At baseline, participants slept 521.81 min (SD: 52.14) daily with an efficiency of 94.3% (SD: 2.8). Sleep duration was assessed in children considering the recommended sleep hours, (more than 9 h in the 6–12 years group and more than 8 h in the 13–16 years group). At the onset of the study, 31.3% of children in the UCG and 42.9% in the IG fulfilled the recommendation. At the end of the study, the percentage of patients who fulfilled the recommendation changed to 31.6% in the UCG and 47.2% in the IG. McNemar test did not detect statistically significant differences between the changes. In the whole group total sleep time increased without statistical significance, from 521.81min (SD: 52.14) to 526.95 (SD: 93.93). There were not statistically significant differences regarding the age group.

There were no differences in sleep parameters between weekdays and weekend days (data not shown). Therefore, we only analyzed the weighing data for all the sleep variables described previously in the methods. Sleep parameters before and after lifestyle intervention are described in Fig. 2. During the lifestyle intervention, children and adolescents slept 8.5 h per day at basal time without significant changes in the total time in bed or total sleep time. Interestingly, sleep latency significantly decreased ($p < 0.001$) at 12 and 24 months in the IG. In the same group, sleep efficiency was significantly improved at all times, with a significant difference at 2 month-change compared to UCG ($p = 0.030$). Moreover, WASO and awakenings duration parameters significantly decreased in the IG at 12 and 24 months.

Additional analyses were performed to evaluate changes in sleep parameters regarding BMI decreased or not among the participants. In the group of patients whose BMI did not decrease, only sleep latency decreased significantly ($p < 0.001$). In the group of patients whose BMI diminished, sleep efficiency significantly increased ($p < 0.001$), whereas latency, WASO and awakening duration decreased significantly ($p < 0.01$). There were no statistically significant differences in the sleep duration whether the BMI decreased or not.

3.3. Association between sleep parameters and anthropometrical or biochemical outcomes

We performed a multivariable-adjusted model in all participants to evaluate the association between sleep parameters with anthropometric and biochemical variables (see Table 2). At baseline, a significant positive association was found between WASO and leptin (r = 0.180, p = 0.016). Moreover, after 2 months of therapy, a significant inverse association was found between total time in bed with triglycerides and metabolic score and between total sleep time and leptin, triglycerides, and metabolic score. One year after therapy, triglyceride levels were inversely associated with total time in bed and sleep time. In addition, in the same period, the metabolic score was directly associated with WASO and the number of awakenings and inversely associated with efficiency.

4. Discussion

The present study objectively measured sleep characteristics before and after a 24-month lifestyle intervention and investigated the associations between sleep variables and adiposity and metabolic parameters in children and adolescents with abdominal obesity. To our knowledge, this study is the first control-randomized trial that has studied the effect of a lifestyle intervention on sleep habits in a pediatric population with obesity.

In this study (IGENOI), a lifestyle intervention improved most sleep parameters in children and adolescents with abdominal obesity. Participants slept an average of 8.5 h per day at baseline, which aligns with other studies that objectively measured sleep duration [11,36]. Based on the American Sleep and Pediatric Societies, the recommended sleep duration range was 9–12 h for children 6–12 years old, close to our results (8.5 h). Similarly, our 13–16 year group slept 8.5 h, within the recommended 8–10 h per night [37]. Moreover, we did not find changes in the sleep quantity (total sleep time and total time in bed) due to the lifestyle intervention. However, there were some interesting findings
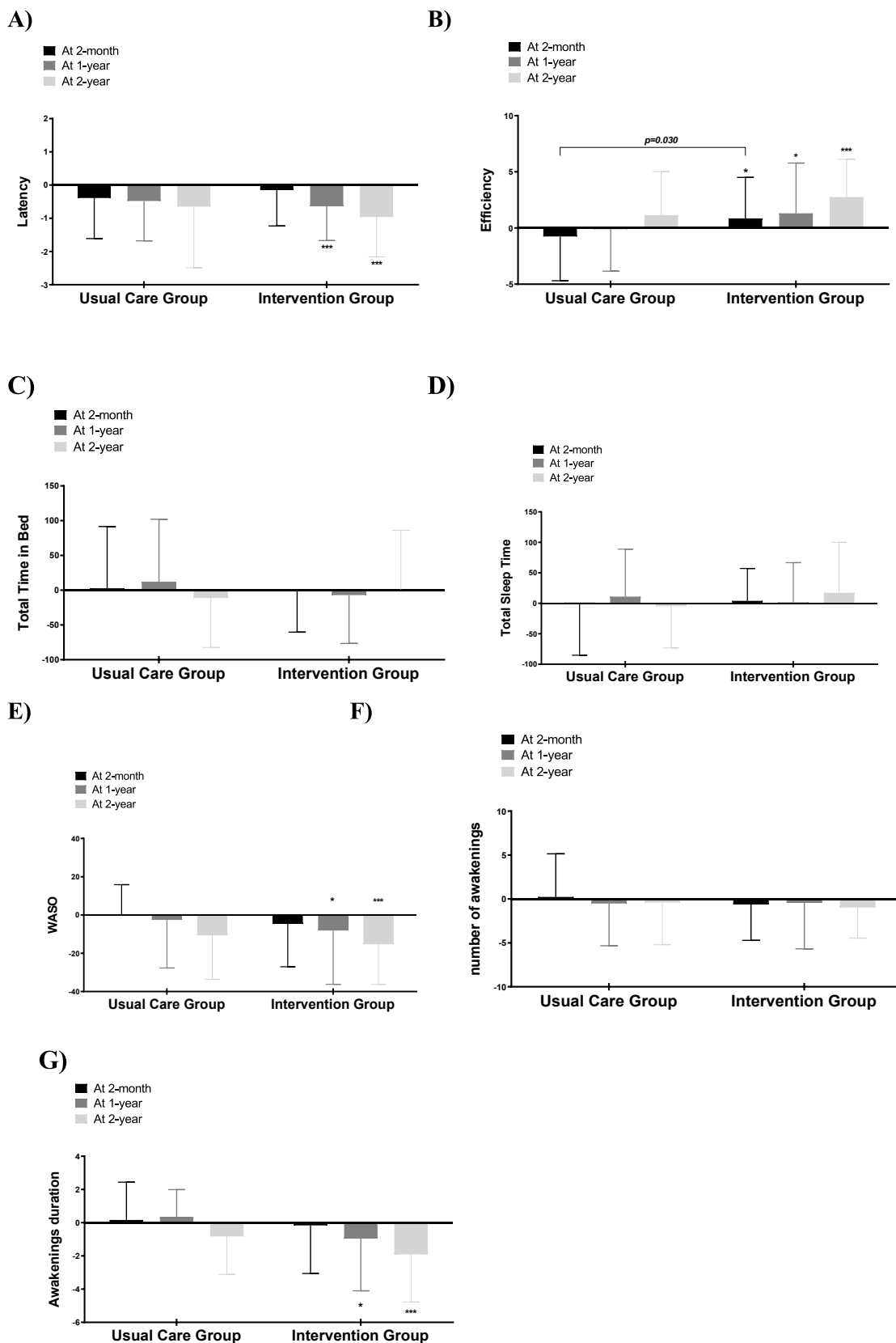
Fig. 2. Objectively measured sleep parameters before and after lifestyle intervention.
Data are expressed in mean and SD of the weighted average workout for each sleep variable. *p < 0.05, ***p < 0.001. According to a Bonferroni correction, a p < 0.0167 was considered statistically significant for baseline comparisons.

Table 2
Associations between changes in sleep variables with anthropometric and biochemical changes. Multiple linear regression analyses adjusted by age, sex, and Tanner stage.

	Leptin	Triglycerides	Metabolic Score
At Baseline			
Wake After Sleep Onset	0.180, *0.016*		
At 2-month			
Wake After Sleep Onset			
Total Time in Bed		−0.143, *0.017*	−0.001, *0.043*
Total Sleep Time	−0.070, *0.043*	−0.160, *0.009*	
At 1-year			
Wake After Sleep Onset			0.005, *0.045*
Total Time in Bed		−0.141, *0.029*	
Total Sleep Time		−0.149, *0.033*	
Efficiency			−0.050, *0.006*
Number of Awakenings			0.037, *0.012*

Significant associations are represented as β-coefficients and *p-value*.

related to sleep quality (sleep latency, sleep efficiency, WASO, and awakening duration). The decrease in sleep latency, awakenings duration, and WASO in the IG appears to be linked to the lifestyle intervention, as it was not observed in the UCG, resulting in higher sleep efficiency. These findings suggest that a healthier lifestyle behavior in the IG positively affects various aspects of sleep quality in children and adolescents with abdominal obesity.

There is growing evidence about the impact of lifestyle factors on sleep quality in children. In a recent systematic review, Fonseca et al. concluded that higher physical fitness levels were associated with more extended periods of sleep and better sleep quality in children and adolescents aged 6–19 years [38]. Regarding the diet, using objective sleep measures, a higher intake of fruit and vegetables accompanied by low consumption of fast food and soda was associated with healthier sleep quality in children [39]. Other factors such as screen time (Whiting et al., 2021), stress, and emotional well-being (Thumann et al., 2019) have also been shown to negatively influence sleep quality in children. Overall, these studies suggest that lifestyle factors such as physical activity, diet, screen time, and emotional well-being can modify sleep quality in children, highlighting the importance of a holistic approach to encouraging healthy sleep habits in this population.

Sleep duration has been extensively studied regarding weight status and metabolic risk factors, with solid evidence of a negative association with overweight and obesity in children (Epel et al., 2004), similar to our results of a negative association with triglycerides and metabolic score. However, few studies examined other sleep dimensions. Along with our lifestyle intervention, we observed that WASO was directly associated with leptin levels and metabolic scores. In a case-control study, children with obesity associated with metabolic syndrome presented increased WASO, measured by polysomnography, compared to matched children with obesity but without metabolic syndrome [40]. Regarding sleep efficiency, a negative association with the metabolic score was found. Other studies used accelerometry to measure sleep efficiency objectively and reported a significant negative association between higher sleep efficiency and poorer anthropometric outcomes [41–44]. Two studies in children showed that the number of awakenings was a significant predictor of obesity. Liu et al. found a significant difference between children with overweight/obesity and normal-weight children regarding sleep maintenance, specifically concerning night awakening problems reported by parents [45]. Bagley and El-Sheikh reported that the number of awakenings, measured by accelerometry, was only significant if the episodes lasted at least 5 min each [41].

Up to our knowledge, there are no studies, exploring the relationship between leptin and objectively measured sleep parameters in children before and after an integral lifestyle intervention. In our study, we found a positive association between WASO and leptin at the onset of the study, and we observed an inverse significant association between total sleep time and leptin after 2-month intervention. Martínez et al.

described in American Mexican children a decrease in leptin levels if sleep duration increases [46]. The control of diurnal blood leptin is regulated by multiple factors such as sex, feeding, fasting, sleep and endocrine alterations [47]. The evidence suggests a bidirectional relationship between sleep duration and leptin [48]. In our study, the decrease in leptin can be mainly attributed to the loss of fat mass. It would be interesting to measure melatonin level in children with obesity. There is a study which measures melatonin levels in saliva in children with obesity and found and increase 1 h after sleep more than two fold compared to controls [49].

This study provides important insight into the potential benefits of lifestyle interventions on sleep quality in children and adolescents with obesity. These findings add to the growing body of research on the relationship between sleep and metabolic health outcomes in children, emphasizing the need to consider multiple dimensions of sleep beyond just sleep duration. However, we acknowledge that the present study has several limitations. First, the age range of the participants is wide, as well as the pubertal stages. In order to control these potential confounders, sex, age, and pubertal stage were included in the statistical models. Another limitation of the study could be the lack of statistical power for sleep parameters analysis between the two groups during the follow-up due to a reduction in sample size (dropout). Also, the sleep values may be interfered with by the subject and device characteristics [50]. The main strengths of our study are the following: a) the study is longitudinal and therefore allows for paired comparisons with baseline data used as control, b) registered nutritionists collect dietary data, and c) participants' response was good, which resulted in substantial weight loss and notable changes in lifestyle factors and sleep d) several dimensions of sleep have been objectively measured.

5. Conclusion

Our lifestyle intervention shows a reduction in anthropometric indexes and several biochemical parameters, and improved sleep quality at 2, 12, and 24 months of follow-up, mainly decreasing sleep latency, awakenings duration, and WASO accompanied by improved sleep efficiency. To our knowledge, the present study is the first in which we investigated the effect of a lifestyle intervention on sleep parameters in a pediatric population with abdominal obesity.

CRediT authorship contribution statement

Ana Catalán-Lambán: Carried it out, Performed data analyses and assisted with interpretation of the study findings, Contributed to writing and revising the manuscript. **Ana Ojeda-Rodríguez:** Carried it out, Performed data analyses and assisted with interpretation of the study findings, Contributed to writing and revising the manuscript. **Amelia Marti del Moral:** Conceived and designed the study, Contributed to writing and revising the manuscript. **Cristina Azcona-Sanjulian:** Conceived and designed the study, Performed data analyses and assisted with interpretation of the study findings, Contributed to writing and revising the manuscript.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

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