International Journal of Obesity (2014) **38,** 1120–1125

www.nature.com/ijo

ORIGINAL ARTICLE

Night time sleep macrostructure is altered in otherwise healthy 10-year-old overweight children

R Chamorro[1], C Algarín[1], M Garrido[1], L Causa[2], C Held[2], B Lozoff[3] and P Peirano[1]

OBJECTIVE: Epidemiological evidence shows an inverse relationship between sleep duration and overweight/obesity risk. However, there are few polysomnographic studies that relate the organization of sleep stages to pediatric overweight (OW). We compared sleep organization in otherwise healthy OW and normal-weight (NW) 10-year-old children.
SUBJECTS: Polysomnographic assessments were performed in 37 NW and 59 OW children drawn from a longitudinal study beginning in infancy. Weight and height were used to evaluate body mass index (BMI) according to international criteria. Non-rapid eye movement (NREM) sleep (stages N1, N2 and N3), rapid eye movement (REM) sleep (stage R) and wakefulness (stage W) were visually scored. Sleep parameters were compared in NW and OW groups for the whole sleep period time (SPT) and for each successive third of it using independent Student's *t*-tests or nonparametric tests. The relationship between BMI and sleep variables was evaluated by correlation analyses controlling for relevant covariates.
RESULTS: The groups were similar in timing of sleep onset and offset, and sleep period time. BMI was inversely related to total sleep time (TST) and sleep efficiency. OW children showed reduced TST, sleep efficiency and stage R amount, but higher stage W amount. In analysis by thirds of the SPT, the duration of stage N3 episodes was shorter in the first third and longer in the second third in OW children as compared with NW children.
CONCLUSIONS: Our results show reduced sleep amount and quality in otherwise healthy OW children. The lower stage R amount and changes involving stage N3 throughout the night suggest that OW in childhood is associated with modifications not only in sleep duration, but also in the ongoing night time patterns of NREM sleep and REM sleep stages.

International Journal of Obesity (2014) **38,** 1120–1125; doi:10.1038/ijo.2013.238

Keywords: overweight; sleep duration; NREM sleep; REM sleep; children

INTRODUCTION

Obesity and overweight (OW) in children is a pressing public health problem worldwide.[1] Considering several comorbid conditions and long-term health consequences associated with obesity,[2] there is a need to identify other modifiable factors that may be amenable to therapeutic interventions. Sleep patterns appear to be relevant factors that may contribute to OW.[3] Among disorders/complications seen in OW adult populations, obstructive sleep apnea syndrome and short sleep duration have received the most attention.[4] Respiratory and nonrespiratory sleep disorders are also reported with childhood obesity.[5]

The increases in OW and obesity rates have occurred concurrently with a rise in sleep debt[5,6] and chronic sleep restriction across societies and age groups.[6] This phenomenon appears to be related to social changes, with increasing access and use of electrical technologies, and work demands.[7] In the United States, approximately one-third of adults report sleeping for $\leqslant$7 h per night, with an increasing proportion sleeping for $\leqslant$6 h per night.[8] In the pediatric groups, almost half of 11–17-year-old children sleep for $\leqslant$8 h, with a tendency toward decreasing sleep duration in older adolescents.[9]

The duration of night time sleep and body mass index (BMI) shows an inverse relationship.[10] Sleep curtailment appears to be an independent risk factor for weight gain and obesity risk in children.[11] Meta-analyses and systematic reviews of pediatric studies have consistently concluded that risk estimates for being OW and obese are higher in short sleepers, particularly at young ages.[12,13] These findings have received further support from longitudinal epidemiological studies.[14,15]

Most of the epidemiological evidence, however, is based on maternal or self-reported sleep data. Information of sleep duration is thus likely to be a proxy for the time spent in bed and not necessarily time asleep.[16] Studies based on more objective methods for sleep assessment, such as actigraphy, have also reported a similar tendency.[17] However, little attention has been given to sleep organization throughout the night. Polysomnographic (PSG) evaluation remains the gold standard method for the assessment of sleep organization.

The study of sleep macrostructure includes characteristics such as sleep duration, sleep efficiency and the organization of rapid eye movement (REM) sleep (stage R) and non-REM (NREM) sleep stages 1 (N1), 2 (N2) and 3 (N3).[18] These sleep stages cycle throughout the sleep period time (SPT), with the deepest stage of NREM sleep (stage N3) prevailing in the first part and stage R in the last part of the SPT. Consequently, analyzing the number, amount and mean duration of NREM sleep stages and stage R episodes according to thirds of the SPT may contribute to a better understanding of the temporal distribution of sleep stages.[19] This approach is in line with studies suggesting that in addition to sleep amount other sleep

[1]Sleep Laboratory, Institute of Nutrition and Food Technology (INTA), University of Chile, Santiago, Chile; [2]Electrical Engineering Department, University of Chile, Santiago, Chile and [3]Center for Human Growth and Development and Department of Pediatrics and Communicable Diseases, University of Michigan, Ann Arbor, MI, USA. Correspondence: R Chamorro, Sleep Laboratory, INTA, University of Chile, Avda. El líBano 5524, Macul, Santiago, 138-11, Chile.
E-mail: rchamorro@inta.uchile.cl
Received 2 June 2013; revised 21 November 2013; accepted 8 December 2013; accepted article preview online 19 December 2013; advance online publication, 21 January 2014

characteristics may be related to obesity, including sleep timing and sleep regularity.[20]

Sleep organization appears relevant to the issue of OW and obesity, as stage R and NREM sleep stages are critically involved in endocrine and metabolic regulation.[5,21] PSG-based studies in obese adults have shown reduced sleep efficiency and stage R amount.[22] Similar findings have been shown in the few available studies of obese children and adolescents, with emphasis on stage R modifications.[23,24] However, previous studies with objective assessment of sleep organization in OW children have been conducted in small sample sizes or in subjects with affective disorders that typically alter stage R features.[24–26] The amount of stage R and stage N3 are increased after surgical interventions for weight reduction in obese adults,[27] but results in adolescents are less consistent.[26]

The aim of the present study was to evaluate sleep macrostructure characteristics (sleep duration and sleep stages organization) in otherwise healthy OW and normal-weight (NW) 10-year-old children. We hypothesized that total sleep duration, sleep quality and the amount of both stage N3 and stage R would be diminished in OW children.

SUBJECTS AND METHODS

Subjects

A total of 96 children who had neurophysiological evaluations at 10 years were included in the present study. All children were participants in an ongoing longitudinal study of the behavioral and developmental effects of iron-deficiency anemia in infancy. As detailed previously,[28] inclusion criteria for enrollment in the infancy phase of the study were healthy full-term birth, with birth weight $\geqslant 3$ kg, without perinatal complications, and the absence of acute or chronic illnesses. Iron status was assessed, and infants with iron-deficiency anemia at 6, 12 or 18 months were considered for neurophysiological evaluations. The control group consisted of randomly chosen infants who were clearly nonanemic (venous Hb $\geqslant 115\,g\,l^{-1}$). All participants were treated with oral iron for at least 6 months and had normal hemoglobin concentrations after treatment. No participant has been iron-deficient anemic at subsequent follow-ups.

Parents provided signed informed consent and children signed an informed assent at 10 years. The original and follow-up research protocols were approved and reviewed annually by the Institutional Review Boards of the University of Michigan Medical Center, Ann Arbor, and INTA, University of Chile, Santiago.

Anthropometric measurements

Weight and height were measured wearing light clothes and no shoes using a Seca scale (model 700, Seca, Hamburg, Germany) at the Sleep Laboratory before the PSG recording. Weight was measured with an accuracy of 100 g and height was measured with a fixed tallimeter with an accuracy of 1 mm. BMI (weight (kg)/(height (m))^2) was calculated and evaluated by age and sex BMI *z*-score according to the World Health Organization (WHO) charts. The following categories were used:[29] NW ($-1 <$ BMI *z*-score < 1; $n = 37$), OW ($1 \leqslant$ BMI *z*-score < 2, $n = 29$) and obese (BMI *z*-score $\geqslant 2$, $n = 30$). Given that OW and obese children were pooled together in the same group (OW group), the groups comprised 37 NW and 59 OW children.

PSG assessment

Subjects underwent an overnight PSG recording at the Sleep Laboratory, INTA, University of Chile. The protocol followed the individual's routine time schedule for food intake and sleep. Accompanied by a parent, children arrived at the laboratory 2 h before they usually fell asleep, so that they could become familiar with the personnel and the laboratory setting. All recordings started at the child's usual bedtime and continued until spontaneous awakening the next morning. Recordings were performed in a special, quiet and comfortable room with controlled temperature, light and humidity. PSG recordings were performed using Cadwell Easy EEG II system (Cadwell Lab., Kennewick, WA, USA) and included the following signals: electroencephalogram with electrode placement according to the 10–20 system[30] (F3, F4, C3, C4, O1, O2) referenced to the contralateral mastoid, left and right electrooculogram, chin electromyogram, left and right tibialis electromyogram, electrocardiogram, thermistor and nasal pressure cannula, thoracic and abdominal effort, peripheral oxygen saturation, snoring and position sensors. Data were acquired and stored in digital format for subsequent analyses. Each PSG recording was transformed off-line to the European Data Format.[31] Throughout the night, any meaningful behavior, such as general body movements and/or body position, was noted by trained personnel.

Scoring and processing of sleep–wake stages

The duration of daytime waking episode was obtained by asking mothers when her child woke up on the morning of the PSG evaluation. Based on PSG sleep onset time, we calculated the duration of the waking episode.

Sleep and waking stages were visually scored in 30-s epochs and defined as stages N1, N2 and N3, stage R and wakefulness (stage W), according to international standard criteria.[32,33] Scoring was performed without knowledge of the children's NW or OW status or background characteristics. The resulting sleep data were processed using tools provided by the Sleep-Analyzer system.[34]

The following conventional sleep parameters were evaluated:

- Time in bed;
- SPT: time from sleep onset to sleep end;
- Total sleep time (TST): time from sleep onset to the end of the final sleep epoch minus time awake;
- Sleep latency (in min): time from lights out to sleep onset, defined as the first epoch of any sleep stage;
- REM latency: time from sleep onset to the first stage R epoch;
- Sleep efficiency: the percentage ratio between TST and SPT (TST/SPT $\times$ 100);
- Stage shifts: number of transitions between sleep and wake stages during the SPT;
- Sleep cycle: the time elapsed between the first epoch of any NREM sleep stage to the last epoch of the succeeding stage R of at least 1 min duration;
- Time spent in stage W after sleep onset (WASO), that is, the time spent awake between sleep onset and end of sleep, in min;
- Percentage and total duration of TST spent in each NREM sleep stage and stage R, and percentage and total duration of SPT spent in WASO.

In addition, for each sleep–wake stage the number and duration of episodes was assessed for the whole SPT and for each successive third of it.[19]

Analysis of respiratory events

Respiratory events were detected by automated processing using Cadwell Easy EEG II software (Cadwell Lab). The definitions of obstructive apnea and hypopnea were based on the American Academy of Sleep Medicine criteria.[35] Respiratory events lasting two or more respiratory cycles were scored. After automated detection, a visual editing of the whole recording was performed to add, confirm or reject the respiratory events before computing a final result. The obstructive apnea–hypopnea index per hour of sleep was then calculated.

Statistical analysis

Statistical analyses used the independent samples *t*-test or a nonparametric test depending on a variable's distribution. Categorical variables were analyzed using χ^2-test or Fisher's exact test. The relationship between sleep and growth variables was tested using bivariate Pearson's or Spearman's correlation depending on the distribution, considering the combined data of the NW and OW groups. Relevant background factors associated with sleep time and sleep stages variables were also assessed by Pearson's or Spearman's correlations. Birth weight was associated with some sleep variables and therefore was included as a control variable in a partial correlation analysis, together with age, gender, iron status (both in infancy and at 10 years), sleep onset time and obstructive apnea–hypopnea index. All analyses were done using SPSS (v.15.0, Chicago, IL, USA). Statistical significance was set at α-level of $\leqslant 0.05$.

RESULTS

Background characteristics

Groups were similar regarding age, gender, gestational age, iron status in infancy and cow milk consumption during the first year of life (Table 1). The groups differed in anthropometric variables at 10 years by design. In addition, the OW group had higher birth weight and percentage of maternal obesity, and lower percentage of iron sufficiency at 10 years (see Table 1).

Conventional sleep parameters

Morning wakeup and sleep onset times and the resulting length of the previous diurnal waking episode were similar in both groups (Table 2). This was also the case for bed-time and wake-up time and SPT. However, compared with NW children, OW children showed shorter TST ($P<0.03$), higher WASO ($P<0.01$) and consequently reduced sleep efficiency ($P<0.01$). The number of sleep cycles was lower ($P<0.05$) and stage R latency tended to be longer in the OW group ($P<0.057$, Table 2). The groups were similar regarding the obstructive apnea–hypopnea index.

Sleep–wake stages

The OW group had a higher amount of stage W ($P<0.02$), a lower amount of stage R ($P<0.05$) and a suggestive tendency for a lower amount of stage N2 ($P<0.06$) relative to the NW group; stage N3 total amount was similar in both groups (Table 2). The proportion of sleep stages within TST was the same in both groups, but WASO was higher in the OW group: 8.5% vs 5.7% ($P<0.04$).

In the initial third of the SPT, the first stage N3 episode was shorter in the OW group ($P<0.04$) with a suggestive trend for reduced duration of N3 episodes ($P<0.07$). In the middle third of the SPT, the OW group showed longer episodes and higher percentage of stage N3 ($P<0.02$) compared with the NW group. In the last third, the groups were similar in stage N3.

Sleep and anthropometric variables

Anthropometric parameters did not correlate with sleep onset, sleep offset, time in bed or SPT. BMI was negatively correlated with sleep efficiency ($r=-0.22$, $P<0.03$) and mean duration of N2 episodes (rho $=-0.28$, $P<0.004$). After controlling for potential covariates, there was a negative correlation between BMI and TST ($r=-0.28$, $P<0.01$) and sleep efficiency ($r=-0.24$, $P<0.03$) (Table 3). Regarding sleep–wake stages, BMI was positively related to stage W amount (rho $=0.20$, $P<0.05$) and percentage (rho $=0.21$, $P<0.04$) and negatively to the amounts of stage N2 ($r=-0.24$, $P<0.03$) and stage R ($r=-0.25$, $P<0.03$, Figure 1 and Table 3). There were no statistically significant relationships between BMI and the amounts of stages N1 or N3.

Table 1. Background characteristics

Background variable	*Normal weight (n = 37)*	*Overweight (n = 59)*	P-*value*[a]
Age, years	10.3 ± 0.2	10.2 ± 0.2	NS
Gender, male, *n* (%)	24 (64.8)	39 (66.1)	NS
Weight, kg	32.6 ± 4.0	46.3 ± 9.1	0.0001
Weight *z*-score[b]	−0.15 ± 0.86	1.2 ± 0.83	0.0001
Height, m	1.38 ± 0.6	1.42 ± 0.1	0.005
Height *z*-score[b]	−0.19 ± 0.95	0.36 ± 0.96	0.007
BMI (kg m^{-2})	16.9 ± 1.2	22.7 ± 3.3	0.0001
BMI *z*-score[c]	0.09 ± 0.6	2.04 ± 0.7	0.0001
Birth weight, g	3402.7 ± 336.5	3598.1 ± 394.4	0.01
Birth height, cm	50.5 ± 2.1	50.8 ± 1.7	NS
Gestational age, weeks	39.4 ± 0.96	39.2 ± 0.98	NS
Cow milk/formula consumption, ml per day[d]	396.4 ± 180.7	308.7 ± 214.9	NS
Maternal obesity, *n* (%)	8 (22.2)	27 (50)	0.008
IDA in infancy, %	56.7	50.8	NS
Iron sufficient at 10 years, *n* (%)	36 (97.2)	48 (87.3)	0.02

Abbreviations: BMI, body mass index; IDA, iron-deficiency anemia; NS, not significant. [a]*P*-values are from *t*-tests for continuous variables and χ^2-test for categorical variables. [b]Number of subjects varied slightly because of occasional missing data in some measures. [c]BMI *z*-score adjusted by age and gender according to the World Health Organization (WHO) reference growth standards. [d]Cow milk/formula consumption during the first year of life.

Table 2. Sleep macrostructure characteristics

PSG parameters	*Normal weight (n = 37)*	*Overweight (n = 59)*	P-*value*
Diurnal waking duration, min	805.0 ± 101.6	809.9 ± 102.3	NS
OAHI (*n* events/h)[a]	0.4 (0.1–0.8)	0.6 (0.3–0.9)	NS
Sleep onset, hh:mm	23:10 ± 0:41	23:20 ± 0:48	NS
End of sleep, hh:mm	7:23 ± 1:01	7:18 ± 0:47	NS
Time in bed, min	614.3 ± 64.7	605.2 ± 48.0	NS
Sleep period time, min	500.6 ± 49.4	478.2 ± 61.2	0.05
Total sleep time, min	467.1 ± 63.4	426.8 ± 79.0	0.008
Sleep efficiency, %[b]	93.3 ± 6.9	89.4 ± 9.5	0.02
Sleep cycles, *n*	4.6 ± 1.1	4.1 ± 1.5	0.05
Stage shift/h, *n*	39.2 ± 9.6	41.0 ± 9.2	NS
Sleep latency, min[a]	9.9 (4.8–27.5)	13.5 (4.5 34.0)	NS
Stage R latency, min	120.0 ± 50.2	138.7 ± 55.2	0.05
WASO, min[a]	18.0 (5–63.7)	38.5 (12.5–76.9)	0.03
Stage N1, min[a,c]	40.9 (25.2–52.7)	36.5 (24.9–53.5)	NS
Stage N2, min[c]	236.2 ± 50.7	216.6 ± 47.4	0.06
Stage N3, min[c]	94.7 ± 24. 3	94.6 ± 21.8	NS
Stage R, min[d]	87.3 ± 27.9	75.6 ± 29.6	0.05

Abbreviations: NS, not significant; OAHI, obstructive apnea–hypopnea index; PSG, polysomnographic; WASO, wake after sleep onset. [a]Data are presented as median (interquartile range). [b]Sleep efficiency = (total sleep time/sleep period time) × 100. [c]Stages N1 N2, and N3 = non-rapid eye movement sleep stages. [d]Stage R = rapid eye movement sleep.

DISCUSSION

We compared the macrostructure of night time sleep in a sample of otherwise healthy 10-year-old OW and NW children. OW children showed shorter TST and higher WASO, indicating lower sleep efficiency. Differences between groups were also apparent regarding sleep stages, with OW children having decreased stage R amount and altered duration of stage N3 episodes during the first two-thirds of the SPT. Even though both groups showed similar night time stage N3 amount, the duration of the episodes in the OW group was shorter in the first third and longer in the second third of the SPT relative to the NW group. These findings extend the epidemiological evidence relating sleep duration and OW in children and suggest that, in addition to sleep amount, the organization of sleep stages and sleep efficiency are also altered.

The inverse relationship between sleep duration and BMI is in line with previous studies in pediatric and adults groups based on sleep questionnaires and self- or parental-report.[36,37] In our study, the relationship between sleep amount and BMI was apparent even after adjusting for several covariates, in agreement with the few other PSG studies of OW children.[23,24] Sleep efficiency was also inversely associated with BMI, suggesting that sleep consolidation is diminished in this group. A recent study of children and adolescents based on self-report data found that poorer sleep quality and higher sleep disturbances, as well as shorter sleep duration, were associated with higher adiposity, supporting the idea that both sleep duration and quality are related to childhood obesity.[38] As all participants in our study were healthy and most children in the OW group were not severely, the shorter sleep amount and lower sleep efficiency in

Table 3. Partial correlation analysis[a] between sleep features and sleep stages and anthropometric parameters

PSG variable	*Weight z-score*	*Height z-score*	*BMI*	*BMI z-score*
Sleep period time, min	− 0.11 (NS)	− 0.04 (NS)	− 0.17 (NS)	− 0.14 (NS)
Total sleep time, min	− 0.14 (NS)	0.02 (NS)	− **0.28** (0.01)	− **0.25** (0.03)
Sleep efficiency, %[b]	− 0.07 (NS)	0.1 (NS)	− **0.24** (0.03)	− **0.22** (0.05)
Stage shift/h, *n*	0.14 (NS)	0.16 (NS)	0.07 (NS)	0.09 (NS)
WASO, min	0.09 (NS)	− 0.05 (NS)	**0.22** (0.05)	0.21 (0.06)
Stage N1, min[c]	0.07 (NS)	0.09 (NS)	− 0.03 (NS)	− 0.03 (NS)
Stage N2, min[c]	− 0.11 (NS)	0.05 (NS)	− **0.24** (0.03)	− **0.22** (0.05)
Stage N3, min[c]	− 0.01 (NS)	− 0.07 (NS)	0.02 (NS)	0.03 (NS)
Stage R, min[d]	− 0.09 (NS)	0.03 (NS)	− **0.25** (0.03)	− 0.21 (0.06)

Abbreviations: BMI, body mass index; NS, not significant; PSG, polysomnographic; WASO, wake after sleep onset. Values are *r* coefficients, and level of statistical significance is shown in brackets; *r* values in bold indicate statistically significant correlation result. [a]Partial correlation analysis controlling for age, gender, iron status in infancy, iron status at 10 years, birth weight, maternal BMI, sleep onset time and obstructive apnea–hypopnea index (OAHI). [b]Sleep efficiency = (total sleep time/sleep period time) × 100. [c]Stages N1, N2 and N3 = non-rapid eye movement sleep stages. [d]Stage R = rapid eye movement sleep.

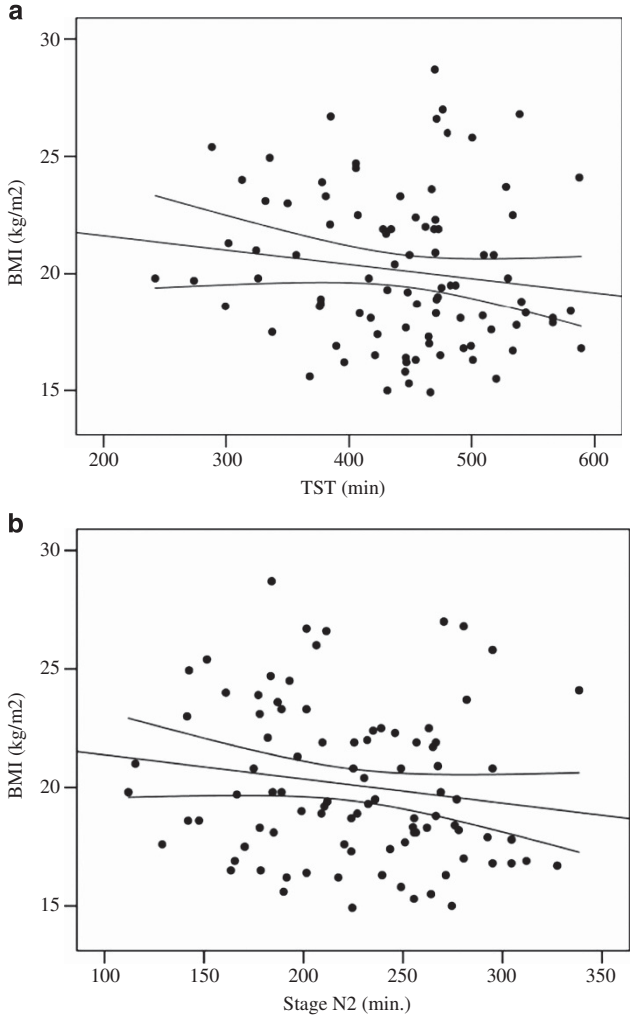

Figure 1. Correlation between total sleep time and stage N2 (non-REM sleep stage 2 (in min)) and BMI (in kg m^{-2}). Scatter plot of correlations between TST (in min) and BMI ($r = -0.28$) and stage N2 and BMI ($r = -0.24$) are shown in (**a**) and (**b**), respectively. Central and curve lines within the graph indicate tendency line and 95% confidence interval.

this group cannot be attributed to medical conditions that are known to alter sleep patterns.[39]

The features of reduced sleep amount and sleep efficiency in the OW group are in agreement with PSG characteristics reported in severely obese adults and adolescents[22,27] that are even more pronounced in obese adolescents with polycystic ovarian syndrome.[40] Weight loss (through bariatric surgery) leads to the improvement not only in respiratory abnormalities and sleep quality, but also in sleep architecture in adults, with an increase in both stage N3 and stage R.[27] These findings of sleep alterations corrected by weight loss add support to the close relationship between sleep macrostructure changes and weight excess.

The whole spectrum of sleep breathing disorders is exacerbated by increased body weight,[41] but sleep modifications relate to energy balance in OW adults even in the absence of respiratory or motor sleep-related abnormalities. Sleep restriction[42] and sleep fragmentation[43] reduced stage R amount. Stage R is negatively related to food intake and feelings of hunger/appetite, changes that were associated with higher insulin and lower glucose-like peptide 1 profiles the following day.[43] In depressive/anxious children and adolescents without sleep respiratory abnormalities, Liu *et al.*[23] reported altered stage R patterns—longer latency and lower amount—in OW subjects compared with NW subjects. In line with these results, Wojnar *et al.*[24] reported a greater reduction of stage R in OW children with major depressive disorder relative to OW children without depressive disorder. In our study, the longer REM latency and lower amount of stage R in OW children agree with the above mentioned results, supporting the association between weight excess and stage R organization.

It is well known that the latency and amount of stage R are influenced by several factors, including age, sleep restriction/deprivation, circadian phase, pharmacological effects and pathological conditions.[44] As age was almost identical in our study groups and recordings were performed during naturally occurring sleep, these factors are unlikely to explain group differences in stage R organization. Moreover, our findings come from a group of healthy children, in contrast to studies involving children and adolescents with affective disorders,[23,24] which are conditions known for stage R modifications.[25]

Earlier studies in adults have also reported that stage R relates to weight and associated parameters. For instance, Adam[45] observed a positive correlation between the amount and percentage of stage R and body weight in healthy adults, but others have reported contradictory data.[46] In pediatric populations, decreased night time stage R has been reported in obese children,[47] suggesting that stage R relates to body weight and could be modified by OW. Our observations that OW children showed reduced stage R and stage R amount was inversely associated with BMI add support to the hypothesis that stage R is a key player in weight gain in humans.[23]

In this respect, there is evidence that both circadian and ultradian stage R regulation mechanisms appear to be affected by OW.[48] Patients with narcolepsy/cataplexy are characterized by higher BMI and altered stage R patterns.[49] Rodent models of

obesity show reduced stage R during the light (rest) phase and increased stage R during the dark (active) phase, suggesting altered stage R circadian regulation.[47,50] Our findings of reduced number of sleep cycles, lower stage R amount and a suggestive tendency for longer stage R latency in OW children suggest an altered ultradian regulation of stage R in humans. Experimental studies in rodent models of obesity showing longer duration of NREM sleep episodes and lower frequency of transitions between NREM sleep and stage R might support this interpretation.[51]

With acute sleep restriction in adults, the amounts of both stage R and stage N2 are reduced[52] and relate to BMI, hunger perception and energy intake.[42] These findings may be pertinent to our observation that stage N2 and stage R amounts were inversely associated with BMI, whereas stage W amount was positively associated with it.

For the whole night, we did not observe differences in the total amount or proportion of stage N3 between groups. However, the OW group showed lower and higher proportions of this stage during the first and second thirds of the SPT, respectively. It is well accepted that stage N3 relates to the homeostatic component of sleep organization, with the length of the preceding waking episode acting as a key factor.[53] Given that the timing and length of the diurnal waking episode were similar in both groups, it is unlikely that this could explain our findings. Differences in the organization of stage N3 could suggest a slower (or more extended) process of fulfilling the restorative function of sleep in OW children. However, it was not within the scope of this study to assess whether this pattern relates to altered homeostatic sleep regulation in OW children.

Chronic sleep restriction could modify body weight and BMI by several mechanisms.[54] For instance, altered endocrine pathways result in increased food intake, reduced daytime physical activity, disturbed carbohydrate metabolism and/or autonomic nervous system activity.[54] These changes could modify the balance between energy intake and energy expenditure, leading to weight gain over time. Although experimental data support increased energy intake and reduced energy expenditure arising from sleep loss,[52] some studies indicate no effect.[55] Nonetheless, if shorter and more fragmented sleep exposes OW children to adverse changes in metabolic regulation as in healthy adults under experimental conditions,[56] then OW children could be more prone to metabolic disruptions.

Some experimental evidence has already shown that not only sleep restriction but also changes in stage R and stage N3 are involved in hunger/appetite regulation and nutritional parameters.[42,43] In animal models, rats with stage R deprivation increased their body weight and food intake (especially for rich-carbohydrate diets).[57] Adults who were habitual short sleepers and increased their sleep amount over a 6-year period had slower increase in BMI and fat mass gain than those who did not.[58] In addition, both sleep duration and sleep quality were positively related to fat mass loss in OW adults submitted to a low-caloric dietary intake.[59] These findings further support the interpretation that a sustained pattern of sleep restriction could lead to increased weight gain and OW risk over time at the population level, and that interventions to improve sleep in OW subjects may have beneficial effects on BMI and fat mass.

Our study has several limitations. A single night recording in the laboratory may alter sleep organization in some children more than others. Additional nights would be needed to evaluate this issue. Bedtime and sleep onset time were established following each child's routine. Although this approach may seem more susceptible to uncontrolled factors than using a fixed bedtime, we considered it important to increase child comfort by respecting the usual timing of sleep. Anthropometric measurements of weight and height were only measured once. Although more than one measure would be desirable, all measures were performed by trained personnel at the same circadian time. We could not ascertain the influence of physical activity and food intake on the observed differences in sleep organization, as we did not have objective measures of these factors. Finally, although groups consisted of the same percentage of children with or without iron deficiency anemia in infancy, we cannot rule out its potential effect on sleep organization in some children more than others.

In conclusion, otherwise healthy OW children showed a reduced amount of sleep and lower sleep efficiency than NW children. The lower amount of stage R and altered distribution of stage N3 in OW children indicate that the ongoing night time pattern of sleep–waking stages is also disrupted.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGEMENTS

We are grateful to the children and their parents who have made this research possible and the technicians and personnel for their valuable collaboration. This study was supported by grants from Chilean Agency for Funding in Science and Technology (CONICYT, Fondecyt 1110513 and 1120319) and the US National Institutes of Health (NIH R01 HD33487).

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