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RESEARCH ARTICLE

Insights into Childhood Obesity: Sleep Patterns, Vitamin D, and Metabolic Dynamics

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ABSTRACT

Objective: The high prevalence of childhood obesity necessitates a deeper understanding of its underlying pathophysiological mechanisms and associated conditions to effectively address this public health concern. This study investigated the association between vitamin D levels, insulin resistance, dyslipidemia, and sleep patterns in the context of childhood obesity.

Methods: This study used data from 115 obese children and adolescents aged 9.9–18 years, identified by body mass index (BMI, kg/m^2) >95th percentile for the children receiving care at a Pediatric Endocrinology Outpatient Clinic. We collected sleep-related information, including onset time and duration, through direct parental questioning and compared these sleep patterns with key health indicators, such as vitamin D levels, insulin resistance, and dyslipidemia.

Results: Children who went to bed before 21:30 had the lowest mean HOMA-IR value (4.8±2.6), whereas children who slept between 21:30 and 23:00 (5.4±2.5) and after 23:00 (5.0±2.5) exhibited slightly higher values (p=0.374 and p=0.789, respectively). Similarly, children who went to bed earlier had lower mean levels of TC (158.7 mg/dl vs. 161.2 mg/dl), LDL (92.3 mg/dl vs. 95.6 mg/dl), and TG (84.1 mg/dl vs. 106.3 mg/dl) than those who slept later (p=0.743, p=0.619 and p=0.067, respectively). However, children who went to bed before 21:30 had significantly higher HDL levels (49.7 mg/dl vs. 44.3 mg/dl, p=0.019). Regarding sleep duration and 25(OH)D levels, 58.0 % of children with 25(OH)D levels <20 µg/L slept ≤7 hours, whereas 42.0 % slept ≥8 hours. In contrast, 73.5 % of children with 25(OH)D levels ≥20 µg/L slept ≥8 hours, whereas only 26.5 % slept ≤7 hours (p=0.003).

Conclusions: Earlier bedtimes and sufficient sleep duration are associated with reduced insulin resistance, more favorable lipid profiles, and higher vitamin D levels in obese children.

Keywords: Obese children, sleep time, sleep duration, vitamin D, insulin resistance

INTRODUCTION

Childhood obesity is a global health concern with lasting implications because it increases the risk of obesity-related complications in adulthood. (1–3). Addressing the roots of childhood obesity is crucial in combating this health challenge (4). Beyond factors like poor dietary habits and reduced physical activity, attention is turning towards the influence of sleep disorders and vitamin D on metabolic imbalances (5). Investigating the complex interplay of these elements holds promise for comprehending the triggers of childhood obesity and advancing effective interventions.

Vitamin D [25(OH)D], known for its effects on calcium, phosphorus, and bone health, is closely linked to obesity and body mass index (BMI) (6). Studies have shown that adolescents with higher BMIs tend to have lower 25(OH)D levels (7,8). Moreover, vitamin D deficiency (<12 µg/L) is prevalent among obese individuals (9). Low levels of 25(OH)D may trigger hormonal responses that promote lipogenesis and obesity (10).

Vitamin D deficiency is also associated with sleep disorders because brain regions containing 25(OH)D receptors are involved in sleep regulation. (11). Low 25(OH)D levels have been linked to poor sleep quality and duration (12). Shortened sleep duration during childhood has been associated with higher rates of childhood obesity (13,14). These findings indicate a strong relationship between vitamin D insufficiency or deficiency, obesity, and sleep disorders.

Consistent with this view, recent studies have emphasized the potential influence of sleep and 25(OH)D on metabolic

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health. (15). Evidence suggests that inadequate sleep and 25(OH)D levels may negatively impact metabolism and insulin sensitivity. However, existing literature has mainly focused on adults, highlighting the need for more research to understand the interplay between sleep duration, timing, and obesity, especially in childhood obesity.

The present study investigated the potential associations between sleep patterns (i.e., sleep timing and duration), insulin resistance, dyslipidemia, and 25(OH)D levels in obese children. The findings of this study may contribute to the development of preventive strategies against obesity within the field of preventive medicine.

MATERIAL AND METHODS

Study population

This study included 115 obese children and adolescents aged 9.9–18 years who were under the care of the Pediatric Endocrinology Outpatient Clinic. To identify children with obesity, we relied on the body mass index (BMI). BMI was calculated using the formula body weight (kg) / (height)(m))^2, and patients with a BMI percentile above the 95th percentile for age and sex were included in the study. Moreover, patients with chronic systemic diseases, ongoing medication use, endocrinopathy, psychological disorders, or syndromic-monogenic obesity were excluded from the analysis.

The research was conducted during a specific period, from September 2018 to November 2018, which coincided with the autumn season in Turkey. During this period, data related to 25(OH)D levels were systematically collected. The collection procedures were carried out in the morning. Due to the patients' treatment schedules following sample collection, only a single sample per patient was obtained as part of the data collection process.

Subsequently, we assessed sleep-related data, including sleep onset time and sleep duration for the participants by direct questioning from their parents. The acquired data were meticulously documented on designated forms designed for this specific inquiry. All aspects of this study were executed in full compliance with ethical guidelines and regulations. Prior to the commencement of the study, an official endorsement was obtained from the ethics committee, which was in line with the principles outlined in the Declaration of Helsinki. Active efforts were made to inform the families of the participants about the study purposes and methods. Subsequently, formal informed consent was obtained from the families, thereby reinforcing ethical considerations in research practices.

Measures

Anthropometric measurements including weight, weight z-score, height, height z-score, BMI, BMI percentile (BMI %), and BMI z-score were calculated and recorded during physical examinations, along with a comprehensive medical history. The patients' puberty status was determined according to the Tanner-Marshall scoring system, categorizing patients as P1 (prepubertal), P2-P4 (peripubertal), or P5 (postpubertal).

Various biochemical parameters were assessed in the study. Fasting blood glucose (FBG), fasting insulin, lipid profile [serum total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG)], alanine aminotransferase (ALT), aspartate aminotransferase (AST), and 25(OH)D (ng/ml) levels] were analyzed. Vitamin D deficiency was defined as <12 µg/L, insufficiency as <20 µg/L, and sufficiency as ≥20 µg/L, according to established criteria. (16).

FBG levels were measured using the glucose hexokinase method. Serum TC was quantified enzymatically using the oxidase method, whereas HDL and TG levels were measured using a homogenic enzymatic method on Roche Modular (Germany) automatic biochemistry analyzers. LDL-C was calculated using the Friedewald formula [LDL-C = (TC – HDL-C) - TG/5] for triglyceride levels ≤400 mg/dl. For samples with TG levels >400 mg/dl, LDL-C was measured using a specific colorimetric enzymatic assay kit. Fasting insulin levels were assessed using the radioimmunoassay (RIA) method. ALT and AST were measured using the enzymatic UV colorimetric method on serum samples from blood specimens using Roche Cobas c 503 and Roche Cobas c 702 automated analyzers. Plasma samples collected in purple-top EDTA tubes were used to measure 25(OH)D using high-performance liquid chromatography (HPLC) method (Thermo Fisher Scientific, USA).

To evaluate insulin resistance, serum fasting insulin levels and the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) index were assessed. The HOMA-IR index was calculated using the formula [Fasting blood glucose (mmol/L) x Fasting Insulin (mIU/L)] / 22.5 (17). Given that HOMA-IR values can be influenced by factors such as ethnicity, age, and pubertal status, the study utilized established thresholds to determine insulin resistance: HOMA-IR values >2.22 for prepubertal girls, >2.67 for prepubertal boys, >3.82 for pubertal girls, and >5.22 for pubertal boys, as determined by Kurtoğlu et al. in the context of Turkish children (18). ALT and AST levels were measured in patients to assess the absence of liver enzyme dysfunction.

To assess the participants' sleeping habits in terms of both sleeping time and duration, we employed two distinct items. In our study cohort, three distinct peaks in sleep timing were observed. Accordingly, to measure sleep onset time, we used a 3-category variable that included the following periods: (i) before 21:30, (ii) between 21:30 and 23:00, and (iii) after 23:00. Based on the National Sleep Foundation's recommendations, which advise against less than 7 hours of sleep for school-aged children and teenagers, we divided sleep duration into two groups: (i) 4-7 hours and (ii) 8 hours or more. (19).

Statistics

All data were analyzed using the STATA V.16 program. Descriptive statistics were expressed as mean ± standard deviation for normally distributed variables, median (min-max) for non-normally distributed variables, and number of cases and (%) for nominal variables. The significance of the difference between the groups was evaluated using Mann–Whitney U

test, Pearson's chi-square and Kruskal-Wallis test. A p value of less than 0.05 was considered statistically significant.

RESULTS

The descriptive statistics are summarized in Table 1 (Panels A and B), and the comparative statistics are presented in Table 2 (Panels C, D, E, F). Panel A of Table 1 provides a comprehensive overview of the demographic, anthropometric, and biochemical parameters under examination. A total of 115 obese children were included in the study. The mean values of parameters including age, weight (kg), weight z-score, height (cm), height z-score, BMI (kg/m^2), BMI%, and BMI z-score for the study cohort are provided in Panel A of Table 1.

Additionally, Table 1 Panel A presents the biochemical parameters of the 115 obese children who participated in the study. In the study group, 7.8% of the participants were classified as prepubertal, 39.1% as peripubertal (P2-P4), and 53.0% as postpubertal. These parameters include FBG, fasting insulin, ALT, AST, TC, LDL, HDL, TG, and 25(OH)D levels, along with the means and standard deviations of HOMA-IR values calculated according to pubertal status.

Furthermore, Panel B of Table 1 presents the percentage distribution of patients according to sleep timing and sleep duration.

The mean HOMA-IR values within distinct sleep timing categories are shown in the three groups in Panel C of Table 2. The group that slept before 21:30 (the reference group) exhibited the lowest mean HOMA-IR value at 4.8 ± 2.6. Comparatively, those who slept between 21:30 and 23:00 h demonstrated a slightly higher mean HOMA-IR value of 5.4 ± 2.5 (p=0.374), whereas those who slept after 23:00 h displayed a mean HOMA-IR value of 5.0 ±2.5 (p=0.789).

In order to investigate the relationship between sleep timing and dyslipidemia parameters, a two-group analysis was undertaken for enhanced comparability, as shown in Panel D

Table 1. Descriptive Statistics of Demographic, Anthropometric, Biochemical Parameters, and Sleep Patterns

Panel A: Demographic, Anthropometric, and Biochemical Parameters		
Parameter	**Mean (± Standard deviation in parentheses)**	
Age (decimal year)	13.2 (±2.2)	
Weight (kg)	72.3 (±16.6)	
Weight z score	2.2 (±1.05)	
Height (cm)	158.4 (±9.3)	
Height z score	0.5 (±1.0)	
Body mass index (BMI, kg/m^2)	28.5 (±4.3)	
Body mass index % (BMI %)	97.1 (±3.0)	
Body mass index z score (BMI z score)	3.0 (±1.0)	
Fasting blood glucose (FBG, mg/dl)	86.4 (±7.1)	
Fasting insulin (mIU/ml)	23.6 (±10.7)	
Alanine aminotransferase (ALT, U/L)	18.2 (±9.3)	
Aspartate aminotransferase (AST, U/L)	20.5 (±5.0)	
Total cholesterol (TC, mg/dl)	160.8 (±27.4)	
Low-density lipoprotein (LDL, mg/dl)	95.1 (±23.2)	
High-density lipoprotein (HDL, mg/dl)	45.0 (±8.2)	
Triglycerides (TG, mg/dl)	103.4 (±43.8)	
HOMA-IR	5.2 (±2.5)	
Vitamin D (25(OH)D) (µg/L)	16.6 (±8.5)	
Panel B: Descriptive statistics of sleeping patterns (timing and duration)		
Sleep timing	**N**	**Percentage (%)**
Before 21:30	15	13.0
21:30-23:00	53	46.1
After 23:00	47	40.9
Sleep duration	**N**	**Percentage (%)**
4-7 hours	56	48.7
7 hours or more	59	51.3

Table 2. Comparative Analysis of Sleep Patterns and Metabolic Parameters (Mean ± Standard Deviation in parentheses)

Panel C: HOMA-IR Levels according to Sleep Timing (Three Groups)			
Group	**HOMA-IR* (Mean ±SD)**	**P-value**	
Before 21:30	4.8 (± 2.6)	Reference	
21:30–23:00	5.4 (± 2.5)	0.374	
After 23:00	5.0 (± 2.5)	0.789	
Panel D: Lipid Profile Parameters according to Sleep Timing (Two Groups)			
Parameter	**Before 21:30 (Mean ± SD)**	**After 21:30 (Mean ± SD)**	**P-value**
Total cholesterol level (mg/dL)	158.7 (± 23.8)	161.2 (± 28.1)	0.743
LDL* (mg/dL)	92.3 (± 22.2)	95.6 (± 23.5)	0.619
TG* (mg/dL)	84.1 (± 39.4)	106.3 (± 43.8)	0.067
HDL* (mg/dL)	49.7 (± 10.4)	44.3 (± 7.7)	0.019
Panel E: 25(OH)D* Levels according to Sleep Timing (Three Groups)			
Group	**25(OH)D (µg/L) (Mean±SD)**	**P-value**	
Before 21:30	20.3 (± 8.2)	Reference	
21:30–23:00	15.7 (± 9.2)	0.06	
After 23:00	16.5 (± 7.5)	0.122	
Panel F: 25(OH)D Status according to Sleep Duration (Two Groups)			
Group	**25(OH)D < 20 µg/L (%)**	**25(OH)D ≥ 20 µg/L (%)**	**P-value**
4–7 hours	58	26.5	Reference
8 hours or more	42	73.5	0.003

*HOMA-IR: Homeostatic Model Assessment of Insulin Resistance, LDL: Low-density lipoprotein, TG: Triglycerides, HDL: High-density lipoprotein, 25(OH)D: Vitamin D

of Table 2 (findings based on three groups are also available upon request). We grouped the participants based on their sleep onset time: those who slept before 21:30 and those who slept after 21:30. Children who went to bed before 21:30 exhibited the following mean values for lipid profiles: TC 158.7± 23.8 mg/dl, LDL 92.3± 22.2 mg/dl, TG 84.1± 39.4 mg/dl; and HDL 49.7 ±10.4 mg/dl. In contrast, children who went to bed after 21:30 had higher mean values: TC 161.2 ±28.1 mg/dl, LDL 95.6 ±23.5 mg/dl, TG 106.3 ±43.8 mg/dl, and HDL 44.3± 7.7 mg/dl. Although the differences in TC, LDL, and TG levels were not statistically significant (p=0.743, p=0.619, p=0.067), the HDL levels were significantly higher in those who slept earlier (p=0.019).

Panel E of Table 2 examines the relationship between sleep timing and 25(OH)D levels. Participants who went to bed between before 21:30 had the highest mean 25(OH)D level at 20.3 µg/L, followed by those who went to bed after 23:00 at 16.5 µg/L and those who went to bed between 21:30 and 23:00 at 15.7 µg/L. These findings show that earlier bedtimes (before 21:30) are associated with higher 25(OH)D levels compared to later sleep timings. Although the mean 25(OH)D levels were higher in the earlier bedtime group, the differences did not reach statistical significance (p=0.06, p=0,122).

The analysis of the relationship between sleep duration and 25(OH)D levels (see Panel F of Table 2) revealed notable findings. Among individuals with 25(OH)D levels below 20 µg/L, 58.0% (n=47) reported a sleep duration of 7 hours or less, whereas 42.0 % (n=34) slept for 8 hours or more. In contrast, among individuals with 25(OH)D levels of 20 µg/L or higher, a lower proportion (26.5 % (n=9)) had a sleep duration of 7 hours or less, whereas the majority (73.5% (n=25)) slept for 8 hours or more. This difference was statistically significant (p=0.003).

DISCUSSION

This study investigated the relationships between sleep timing, duration, and key metabolic markers, including insulin resistance (HOMA-IR), lipid profiles (TC, LDL, HDL, TG), and vitamin D levels [25(OH)D]. Given the growing evidence of sleep's role in regulating metabolic and endocrine functions, we hypothesized that sleep timing and duration may influence these markers.

Analyses of the relationship between sleeping time and HOMA-IR showed that going to bed earlier in childhood was associated with lower HOMA-IR levels. Although we did not find statistically significant differences, the trend of slightly lower insulin resistance values among those sleeping before 21:30 suggests that an earlier sleeping time has a potential positive impact on insulin resistance. This result is consistent with the findings of Reutrakul and Van Cauter. (20), who argued that the circadian system is linked to glucose metabolism. Although further research is required to fully elucidate the underlying mechanisms, given that many hormones exhibit circadian regulation, sleep disturbances can disrupt this rhythm. As a result, circadian rhythm misalignment is believed to contribute

to alterations in glucose metabolism, primarily through a reduction in insulin sensitivity (21,22). Likewise, individuals who go to bed earlier might have better alignment with their natural circadian rhythm, which could positively influence their metabolic processes (23,24).

The relationship between sleep timing and dyslipidemia parameters revealed significant trends. Specifically, individuals who retired to bed before 21:30 demonstrated lower mean levels of TC, LDL, and TG compared with those who slept later. However, the observed differences in TC, LDL, and TG did not reach statistical significance (p=0.743, p=0.619, p=0.067). Conversely, those who went to bed earlier exhibited significantly higher mean levels of HDL (p=0.019). These findings show that earlier sleep onset is associated with a more favorable lipid profile, which is characterized by reduced TC, LDL, and TG levels, alongside elevated HDL. Furthermore, these results agree with the existing literature highlighting the critical role of sleep timing, duration, and quality in modulating lipid metabolism. In a recent study, which examined the relationship between sleep timing, night sleep duration, and dyslipidemia, it was observed that individuals with later sleep timing had a higher risk of dyslipidemia. (25). A relevant study conducted by Smiley et al. (26) exemplified this connection, highlighting that prolonged sleep duration correlated with improved lipid profiles in adolescents.

The final phase of our analysis examined the relationship between sleep timing, sleep duration, and 25(OH)D levels. Although the differences between sleep timing groups were not statistically significant, our findings indicate a partial association, with earlier bedtimes higher 25(OH)D levels. Additionally, children who likely experienced insufficient sleep (i.e., 7 hours or less) tended to have lower 25(OH)D levels. These findings align with the existing literature examining the relationship between sleep duration and 25(OH)D levels, suggesting that increased sleep duration is associated with higher 25(OH)D concentrations (12,27,28). However, only few studies have investigated the relationship between sleep timing (bedtime) and 25(OH)D levels. For instance, a recent study by Al-Shawwa et al. (29) examined the relationship between 25(OH)D and sleep patterns in children. Their findings indicated that lower 25(OH)D levels were associated with adverse sleep outcomes, such as reduced sleep duration and delayed sleep onset. The observed relationship between sleep and 25(OH) D levels improves our understanding of how sleep habits influence broader health outcomes. Specifically, the findings suggest that sleep patterns may not only affect metabolic factors, such as insulin resistance, and have implications for other aspects of health.

Limitations of the Study

This study has several important potential limitations. First, sleep timing and duration were based on parental reports, which may have introduced bias. Parents may not know the exact bedtime. Second, the study did not collect information regarding the presence of sleep disorders, which could have influenced the results. In addition, some of our analyses' results were not statistically significant. We hypothesized that significant findings could be obtained using a larger sample size. Therefore, future research should involve a larger cohort to further explore these relationships. Furthermore, the observed relationships should be interpreted with caution because they are correlational rather than causal and may be influenced by confounding factors. For instance, sleep patterns may correlate with lifestyle factors such as diet and exercise, complicating efforts to isolate the direct effects.

CONCLUSION

Our analysis, which included both statistically significant and non-significant findings, consistently supports the hypothesis that earlier sleep onset is associated with improved metabolic health. Specifically, earlier bedtimes were linked to reduced insulin resistance, more favorable lipid profiles, and potentially higher vitamin D levels. Additionally, children who experienced insufficient sleep (defined as ≤7 hours per night) tended to have lower vitamin D levels, emphasizing the broader implications of sleep duration on metabolic and endocrine health. These findings underscore the critical role of sleep in maintaining metabolic homeostasis.

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