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Polysomnographic comparison of sleep in children with obesity and normal weight without suspected sleep-related breathing disorder

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Summary

Short sleep and obstructive apneas/hypopneas have been shown to be associated with childhood obesity. Still, few studies have compared sleep in children with obesity, without suspected sleep disordered breathing and normal weight peers by objective sleep measures and compared results with subjective parent assessment of sleep. Children with obesity aged 7–13 years ($N = 44$) and a matched group of normal weight children ($N = 42$) completed clinical polysomnography (Embla A10 Recording System). Parents scored their children's sleep on the Children's Sleep Habits Questionnaire (CSHQ). Mann–Whitney U tests were used to compare groups. There was a higher obstructive apnea/hypopnea index (AHI) (median obesity = 1.20 vs. median normal = 0.66; $z = -1.33$, $U = 560.50$, $p = 0.002$) and number of oxygen desaturation events per hour (median obesity = 0.7 vs. median normal = 0.2; $z = -3.45$, $U = 402.50$, $p = 0.001$) in the children with obesity compared to children with normal weight. The children with obesity had a significantly longer sleep duration (median obesity 8:50 h = vs. median normal = 8:32 h; $z = -2.05$, $U = 687.00$, $p = 0.041$), longer stage N2 sleep (median obesity = 87 min vs. median normal = 52 min; $z = -2.87$, $U = 576.50$, $p = 0.004$) and shorter REM sleep (median obesity = 94 min vs. median normal = 121 min; $z = 5.05$, $U = 1477.00$, $p \leq .001$). No differences were observed for time in sleep stage N1 and N3, wake time after sleep onset or the total arousal index . Further, no group differences were found on the CSHQ sleep-disordered breathing sub-scale ($p = 0.399$). The children with obesity demonstrated significantly more mild to moderate sleep disordered breathing than children with normal weight, although this was not corroborated by parent report.

KEYWORDS
child, obesity, polysomnography, sleep apnea, sleep duration

What is already known about this subject?

- Obesity is one of the main causes of sleep-related breathing problems in children.
- Disrupted sleep over time is associated with maladaptive neurocognitive, behavioural and emotional changes and cardio-metabolic risk.

What this study adds?

- Mild to moderate sleep-related breathing problems are prevalent in children with obesity where no such problems were suspected.
- Sleep-related breathing problems escape the attention of parents.
- Parents do often not recognize the potential significance of snoring as related to obstructive sleep apnea
- Objective sleep measures are necessary in childhood obesity care to detect sleep-related breathing problems.

1 | INTRODUCTION

Childhood obesity and insufficient sleep are found to bi-directionally impact each other.[1,2] While sleep curtailment constitutes a risk factor for the development and maintenance of childhood obesity,[1,3] children with obesity demonstrate an elevated prevalence of sleep-related breathing disorders (SRBD), including obstructive sleep apnea syndrome (OSAS).[4] While the primary causes of OSAS in children are enlarged tonsils and adenoids,[5] obesity also constitutes a risk factor especially among older children.[4,6] The US expert committee for the treatment of childhood obesity, points to obstructive sleep apnea as one of the most serious obesity-related co-morbidities.[7] Obstructive sleep apnea is in the majority of cases caused by upper airway obstruction that results in episodes of partial or complete airway blockage during sleep.[8] As a consequence, oxygen saturation decreases and episodic hypercapnia occurs leading to arousals followed by rapid re-oxygenation.[8] Frequent apneas further cause fragmented sleep, observed as moving from a deeper sleep state to a lighter sleep state or wakefulness (arousals).[9]

An enduring pattern of disrupted sleep is associated with maladaptive neurocognitive, behavioural, and emotional changes.[10–12] Further, OSAS in childhood activates inflammatory mechanisms similar to those observed with obesity.[2,13] Therefore, in combination obesity and OSAS may have negative synergic inflammatory effects, in addition to the shared adverse cardio-metabolic consequences.[2,14–16] In a study of 459 children, sleep fragmentation was significantly associated with insulin resistance[15] and the co-occurrence of OSAS and obesity represented an elevated risk of insulin resistance. Canapari et al.[17] further report elevated levels on the Homeostatic Model Assessment indicator of insulin resistance in children with both OSAS and obesity compared to children with obesity without OSAS.

An association between shorter rapid eye movement (REM) sleep, shorter sleep duration and OSAS is reported in children with obesity.[18–20] REM sleep is further related to changes in the appetite regulating hormone leptin during sleep, and it is suggested that shorter REM sleep might be associated with increased appetite through increases in leptin levels.[21]

Polysomnography (PSG) is considered the gold standard for assessment of sleep, and is required for the diagnosis of some sleep disorders.[22] Subjective parent and child reports of sleep are found less accurate in terms of sleep duration, nocturnal awakenings and sleep onset latency.[23] Several studies from sleep clinics report on children with obesity being over-represented in samples referred with the suspicion of OSAS.[24] Prevalence rates of OSAS in obese samples are reported to be 13%–59%, but comparison across studies is difficult due to different categorizations of OSAS.[24,25] Further, some studies indicate that symptoms of OSAS in obesity are moderated by ethnicity, with higher risk in African American and possibly Asian children.[6] However, few studies have compared PSG sleep patterns in children with obesity recruited without suspicion of OSAS and normal weight comparison groups.[4,20,26] Given the negative synergetic health consequences of OSAS and obesity,[14,15] and the fact that symptoms of SRBD might escape attention of children and parents,[27] investigating sleep in children with obesity without suspicion of sleep disorders is of clinical interest.

The present study investigates PSG measured and parent reported sleep patterns in a group of children with obesity, not selected on suspicion of sleep problems and an age- and gender matched normal weight comparison group. We hypothesized that children with obesity would demonstrate more sleep-related breathing problems than normal weight children and that sleep-related breathing problems would more frequently be detected by PSG than by subjective parent report.

2 | MATERIALS AND METHODS

2.1 | Subjects

This study included cross-sectional data from children with obesity aged 7–13 years and an age and gender matched normal weight comparison group. The children with obesity were recruited to a low-threshold treatment study of family-based behavioural treatment for obesity at The University of Bergen, Norway.[28] The families were referred by their general practitioner, school nurse or by responding to ads in local newspapers. Inclusion criteria were: (1) age 7–13 years;

and (2) obesity according to body mass index (BMI) cut-offs from the International Obesity Task Force (IOTF 30) criteria for children and adolescents.[29] At the anamnestic interview before treatment parents of the children with obesity were asked about the sleep habits of their child (habitual bed and rise time, problems falling asleep), if the child had a sleep disorder or had undergone any type of sleep assessment before, if the child snored and if they suspected breathing problems during sleep. None of the parents reported previous sleep assessment, diagnosed sleep disorder nor suspicion of SRBD. Thus, none were excluded from this study based on these criteria. Parent reported snoring was not considered an exclusion criterion, and the parents of eight of the children with obesity reported that their children sometimes snored. Pre-treatment data were used from the children with obesity. The normal weight comparison group was invited through written information distributed to the parents of all children in the relevant age range at two randomly selected public schools in the Bergen municipality after inclusion to the treatment study was ended. Upon telephone contact from parents of children interested in participation, inclusion was conducted consecutively for children that could be matched on a one-to-one basis (same school year/age, and sex) with children in the treatment group. Inclusion criterion was normal weight according to BMI cut-offs from the IOTF (IOTF 30) criteria for children and adolescents.[29] Sleep measures were part of a larger assessment focusing on lifestyle habits and mental health. Two of the children invited to the comparison group did not match the BMI criteria and were excluded from analyses. Therefore, the age and gender distribution ended up slightly different in the two groups. Children with obesity were recruited consecutively between January 2007 and May 2008 and the normal weight comparison group between May 2008 and October 2008.

2.2 | Instruments and procedures

2.2.1 | Anthropometrical data

Weight was measured at the University clinic using a standard physician's scale during which the children wore light clothing and no shoes, with a precision of 0.1 kg. Height was measured using a wall-mounted height board with the following requirements: no shoes, heels together, and heels, buttocks, shoulders and head touching the wall with sight straight forward and recorded to the nearest 0.1 cm. BMI was calculated using the formula kg/m^2. BMI standard deviation scores were calculated using the Norwegian growth references.[30] Waist circumference was measured at a level midway between costae 10 and crista iliaca using a measuring tape. Parents self-reported their own nationality, their child's ethnicity, and years of education after secondary school.

2.3 | Polysomnography

The children were assessed over two consecutive nights using clinical polysomnography (PSG). Embla® A10 and Somnologica™ Studio 5.1 (Embla Systems, Inc.) were used for data sampling and analysis. The recorder includes electroencephalogram (EEG), electrooculogram and electromyogram. The EEG consisted of six channels (F3 + F4, C3 + C4, O1 + O2). The electromyogram had an active electrode on the masseter muscle with reference on the chin. In addition, respiration, heart rate, oxygen saturation and body position were measured. Airflow was assessed by a nasal air pressure cannula, and a nasal/oral air thermistor (Protech), respiratory effort with inductance plethysmography and oxygen saturation with finger pulse oximetry. The children had electrodes attached at the University Sleep Lab according to criteria from *AASM Manual for the Scoring of Sleep and Associated Events*.[31] The children arrived at the sleep lab approximately 2 h before their normal bedtime whereupon they slept at home in their normal environment. The electrodes were removed in the morning and attached again at the sleep lab in the evening on the second night. To optimize signal quality, the skin was scrubbed before attaching the electrodes and impedance was checked (EEG, EOG, EMG below 5 Ω, EMG-tibialis below 20) and a bio calibration was conducted. Parents were instructed to register the time when the light was turned off in the evening and on in the morning. Data were collected on nights before school days. No data from weekends were included. Data from the second night of PSG-recordings were scored in adherence with the guidelines of the American Academy of Sleep Medicine (2007) according to the age of the participant.[31] To be considered valid, the PSG recording had to include at least 4 h of scorable sleep. A single registered European polysomnographic sleep technologist not involved in the study and blind to group belongingness scored and sleep staged the PSG-recordings from all participants manually. Parent reported time for lights off and wake time/lights on were used as a reference when defining the analysis time for the scoring (parent and child together kept a sleep diary including this information). Total sleep time was reported in minutes. The proportion of time spent in each sleep stage was expressed as percentage of total sleep time. The obstructive apnea-hypopnea-index (AHI) provides the number of obstructive apneas and hypopneas per hour of sleep (central apneas were not included, but mixed apneas were included). The following criteria for the obstructive AHI were used to categorize the severity of respiratory disturbances during sleep in children (SRBD): mild SRBD/OSA: AHI 1–4.9, moderate SRBD/OSA: AHI 5–9.9, severe SRBD/OSA: AHI 10 or higher.[22,32,33] AHI in REM sleep is expressed as an index of events per hour. The arousal index represents total amount of arousals (including respiratory arousals, limb movement arousals, spontaneous arousals and respiratory effort-related arousals) per hour. Oxygen desaturation events were scored when the oxygen saturation fell by at least 3% and were reported in the present analyses as number of events per hour. Further, continuous oxygen saturation (mean values) was reported. Wake after sleep time was defined as minutes awake during the sleep period.

2.4 | Children's Sleep Habits Questionnaire (CSHQ)

A 35-item questionnaire asking parents to report their child's sleep behaviours and patterns during the last typical week was administered

at the same time as the PSG-measurement.[34] The questionnaire was developed to include items presenting the major clinical sleep complaints in this age group providing data on the following subscales (with score range in parentheses): bedtime resistance (6–36), sleep onset delay (1–3), sleep duration (3–9), sleep anxiety (4–12), night awakenings (3–9), parasomnias (7–21), sleep disordered breathing (3–9) and daytime sleepiness (8–24).[34] Each item is rated on a three-point scale: usually (5–7 times a week) = 3, sometimes (2–4 times a week) = 2 or rarely (0–1 time a week) = 1. Two items ask about sleepiness in particular situations and are rated as 0 = not sleepy, 1 = very sleepy and 2 = falling asleep. The sleep disordered breathing subscale consists of three items: "Child snores loudly", "child seems to stop breathing during sleep", "child snorts and/or gasps during sleep". A composite score is calculated as the sum of scores on the 33 items comprising all subscales, and a value of 41 is considered the clinical cut-off indicating risk of sleep problems.[34] The initial study reporting on the psychometric properties of the questionnaire found that the subscales and the total score were able to consistently differentiate between children with sleep disorders and a community sample and that the internal consistency was adequate for both samples.[34] A more recent study has compared the CSHQ subscales for sleep onset delay, sleep duration, night wakings and sleep disordered breathing with PSG-measures, reporting no significant correlations between the two measures.[35] On receipt of questionnaires, study staff ensured that missing items were completed by participants if possible. Missing questionnaire data resulted primarily from premature drop-out of participants.

2.5 | Statistics

Data were analysed using IBM SPSS Statistics 25. Preliminary analyses demonstrated that most of the sleep variables were not normally distributed and non-parametric statistics, Mann–Whitney *U* tests, were thus chosen to compare groups. The missing data excluded test by test option was used in analyses. Further, χ^2 tests were performed to compare differences in prevalence of SRBD and the number of individuals scoring above the clinical cut-off for sleep problems on the CSHQ in the group of children with obesity and normal weight.

2.6 | Ethical considerations

The study was approved by the Regional Committee for Medical and Health Research Ethics, Western Norway (project number: 3.2006.3019) as well as by the Data Inspectorate in Norway. All parents and children signed a written informed consent statement before participation in the study.

3 | RESULTS

Demographic characteristics of the sample that had valid PSG-recordings are presented in Table 1. In all, 97.7% of the children with obesity and 95.2% in the normal weight group were Caucasian. In the total sample with obesity ($N = 49$), 35 of the children had BMI in the IOTF obesity range (between IOTF 30–35), while 14 had severe obesity (BMI > IOTF 35).[29] In the normal weight sample ($N = 47$), all children had a BMI below IOTF 25.

Medians, range, Mann–Whitney *U* values, standardized test statistics and significance levels for group differences on the total score and sub-scales of Children's Sleep Habits Questionnaire are presented in Table 2. There was no significant difference between the groups of children with obesity and normal weight on the CSHQ total score ($p = 0.061$). Twenty-one (51.2%) children with obesity had a score above the clinical cut-off on the CSHQ, while 19 (40.4%) children with normal weight scored in the clinical range; however, this difference was not statistically significant ($\chi^2 = 0.64$, $df = 1$, $p = 0.42$). Parents of children with obesity reported their child to have significantly longer sleep onset delay ($p = 0.002$) and more parasomnias ($p = 0.037$) than reported by the parents of the normal weight children. However, no significant group differences were found on the CSHQ sleep-disordered breathing sub-scale ($p = 0.399$) or for the prevalence of parent-reported snoring ($\chi^2 = 0.79$, $df = 2$, $p = 0.67$). None of the parents in either groups reported that their child seemed to stop breathing during sleep or that the child snorted or gasped during sleep. Seven parents of the children with obesity and six from the normal weight group described that their child *sometimes* snored loudly, while one parent from both groups reported that their child *usually* snored loudly. Together, this represents parent-reported snoring for 18% of the children with obesity and 13% of the normal weight children. Snoring was reported for one of the children with obstructive AHI >5 and four of the children with obstructive AHI >1.

The polysomnography recordings demonstrated that the children with normal weight slept significantly shorter than the children with obesity ($p = 0.041$). Children with obesity spent a significantly higher percentage of the sleep period in sleep stage N2 ($p = 0.004$) and shorter percentage of time in REM sleep ($p = 0.001$) than their normal weight peers. The children with obesity further had significantly higher obstructive apnea-hypopnea index ($p = 0.002$), higher obstructive apnea-hypopnea index in REM sleep ($p = 0.026$) and in non-REM sleep ($p = 0.026$) as well as higher oxygen desaturation index ($p = 0.001$) than the normal weight children. See Table 3 for group comparisons on PSG measures. Significantly more children with obesity than normal weight (27 vs. 13) had OSA ($\chi^2 = 11.2$, $df = 1$, $p = 0.001$). Most of these cases were classified as mild OSA, but seven of the children with obesity and one child with normal weight had moderate OSA. In total, 63% of the children with obesity had an AHI of ≥1 per hour, and 16% had AHI ≥5.

4 | DISCUSSION

This study demonstrated that children with obesity, without suspicion of sleep disordered breathing, had significantly more obstructive apneas and hypopneas and more episodes of oxygen desaturation per hour of sleep than their normal weight peers. In addition, the children

TABLE 1 Characteristics of the study population according to weight group

	Obesity	Normal weight	p^{**}
Total (*N*) with valid PSG	44	42	
Age (mean, *SD*)	10.7 (1.3)	11.4 (1.1)	**0.007**
Age range	7.5–12.5	9–13	
Sex: girls (%)	22 (50.0%)	19 (45.2%)	0.663
Height (cm; mean, *SD*)	153.1 (9.7)	144.8 (23.9)	**0.038**
Weight (kg; mean, *SD*)	65.4 (12.1)	38.4 (6.0)	<**0.001**
BMI (kg/cm^2; mean, *SD*)	27.8 (2.5)	17.3 (1.7)	<**0.001**
BMI SDS (mean, *SD*)	2.6 (0.3)	−0.25 (0.8)	<**0.001**
Waist circumference (cm; mean, *SD*)	90.6 (8.2)	62.1 (5.4)	<**0.001**
Mother born in Norway (%)	100%	95.2%	
Father born in Norway (%)	100%	95.2%	
Education mother (years after high school; mean, *SD*)	4.6 (2.5)	7.1 (2.3)	<**0.001**
Education father (years after high school; mean, *SD*)	3.9 (2.8)	7.4 (2.2)	<**0.001**

Note: Statistically significant *p* values ($p < 0.05$) are marked in bold. *p values from a χ^2 test for categorical data, and a *t* test for continuous data.
Abbreviations: BMI, body mass index: *SD*, standard deviation.

TABLE 2 Parent-reported sleep habits questionnaire scores according to weight group

	ObesityMedian (range)	Normal weightMedian (range)	*U*	*Z*	p^{**}
Total (N)	41	47			
CSHQ Total score	41 (25)	39 (20)	740.0	−1.9	0.061
CSHQ sleep duration	4 (3)	4 (4)	1075.5	1.0	0.318
CSHQ sleep onset delay	2 (2)	1 (2)	633.5	−3.2	**0.002**
CSHQ night wakings	3 (3)	3 (3)	906.5	−0.7	0.488
CSHQ sleep-disordered breathing	3 (2)	3 (2)	899.5	−0.8	0.399
CSHQ parasomnias	8 (4)	7 (4)	731.0	−2.08	**0.037**
CSHQ bedtime resistance	6 (6)	6 (4)	849.5	−1.13	0.258
CSHQ daytime sleepiness	11 (12)	11 (13)	850.0	−1.0	0.339
CSHQ sleep anxiety	4 (4)	4 (3)	910.0	−0.6	0.583

Note: **p values from Mann–Whitney *U* tests. Statistically significant p values ($p < 0.05$) are marked in bold.
Abbreviation: CSHQ, Children's Sleep Habits Questionnaire.

with obesity spent more time in sleep stage N2 and had less REM sleep. In total, 63% of the children with obesity had an obstructive AHI of ≥1 per hour, and 16% had obstructive AHI ≥5. None of the parents had observed or suspected that their child stopped breathing during sleep or observed that the child snorted or gasped during sleep. However, snoring was reported by parents in 18% of the children with obesity and 13% of the normal weight children. Snoring was only reported for one of the children with obstructive AHI >5 and four of the children with obstructive AHI >1.

Parents of children with obesity also reported significantly longer sleep onset delay and more parasomnias than the comparison group, although the total score of sleep problems on the CSHQ was similar in the two groups.

The finding of more sleep-disordered breathing in children with obesity than normal weight children when using PSG-assessment, is in line with several previous studies.[4,26,36] Beebe et al.[26] in accordance with our study reported more PSG-measured sleep-disordered breathing in 60 children in treatment for obesity compared to 22 normal weight controls. They did, however, not find differences in the duration of N2 stage sleep or REM-sleep. Compromised REM sleep is found in several other studies comparing children with obesity to normal weight peers.[19,20,37] This is notable as REM sleep is found to be inversely related to hormonal hunger and appetite regulation and intake of energy dense food[21,38,39] and to measures of insulin resistance.[15] Studies have also attested to a higher frequency of obstructive events during REM-sleep than in other sleep stages in children with sleep apnea,[40] and the same tendency is observed in the current study.

Further, Beebe et al.[26] found that children with obesity reported significantly more sleep problems than normal weight children on all CSHQ subscales, except for night awakenings, while the only group

TABLE 3 Comparison of polysomnographic measured sleep in children with obesity and normal weight

Sleep measures	ObesityMedian (range)	Normal weightMedian (range)	*U*	*Z*	*p***
N	44	42			
Total sleep time (minutes)	529.8 (213.5)	512.3 (278.0)	687	−2.1	**0.041**
Wake after sleep onset (min)	21.5 (79.0)	21.6 (96.0)	976.5	0.7	0.520
Obstructive Apnea-hypopnea Index (AHI) (per hour)	1.2 (8.6)	0.7 (6.1)	560.5	−1.3	**0.002**
Total arousal index (per hour)	8.8 (16.8)	7.2 (13.10)	697.5	−2.0	0.050
Oxygen desaturation events (per hour)	0.7 (7.9)	0.2 (2.4)	402.5	−3.5	**0.001**
Sleep stage N1%	2.7 (9.4)	1.8 (6.7)	771.5	−1.1	0.258
Sleep stage N2%	16.9 (47.2)	9.3 (39.2)	576.5	−2.9	**0.004**
Sleep stage N3%	56.0 (54.2)	59.3 (45.1)	909.5	0.1	0.954
REM sleep %	17.7 (16.9)	23.1 (19.5)	1477.0	5.1	**< 0.001**
AHI in REM sleep index	1.4 (15.3)	0.5 (7.6)	656.0	−2.2	**0.026**
AHI in non-REM sleep index	1 (41)	0.5 (5.2)	651.0	−2.2	**0.026**
Average oxygen saturation (%)	97.4 (3.2)	97.7 (2.6)	1057.5	1.8	0.073

Note: ***p* values from Mann–Whitney *U* tests. Statistically significant *p* values ($p < 0.05$) are marked in bold.
Abbreviation: REM, rapid eye movement.

differences in the present study were on sleep onset delay and parasomnias. The explanation for these somewhat divergent findings is not obvious, but the sample included in Beebe et al.'s[26] study had a slightly higher age and a higher degree of obesity than the present sample and exclusion criteria did not include parental suspicion of SRBD. Thus, the finding that more parents reported sleep problems in the former study could be partly due to more severe obesity.

Children with obesity and OSAS demonstrate poorer academic performance and more behavioural problems,[10] as well as negative metabolic and cardiovascular consequences.[14] Therefore, detection of sleep problems and interventions to promote better sleep quality in children with obesity is important.

This study adds to findings describing the difficulties for parents in observing symptoms of SRBD in their children, and the risk of such conditions not being recognized and properly addressed. In the present sample of children with obesity, no parents suspected apneas or reported that their children stopped breathing during sleep, even though the PSG-findings revealed hourly apneas or hypopneas in 63% of these children. Both apneas and especially hypopneas are generally not easily observable phenomena, and parents of school-aged children in Norway also seldom observe their children during sleep. Therefore, observed apneas (as the CSHQ-asks for) are a poor indicator for the presence of OSA. Subjective reports of sleep problems in children are not accurate, especially when it comes to night awakenings, apneas, sleep onset latency and sleep duration.[23,27,35] An implication of these findings is that parents of children with obesity might need to be offered more education about the risk of co-morbid SRBD and which related symptoms and behaviours to observe, e.g., snoring. Screening for sleep disorders should be a part of the assessment of childhood obesity and the use of PSG-evaluation of sleep should be considered more often. In Norway, PSG-assessment is not part of the standard assessment even for children with severe obesity.

Treatments such as adenotonsillectomy and continuous positive airway pressure are available and have in general been found beneficial for OSAS or SRBD in children with obesity.[8,13,24] However, current recommendations suggest that the effect of weight loss on SRBD should be considered before, e.g., surgery.[41,42] This is partly because relapse to or persistence of SRBD is quite commonly observed after adenotonsillectomy[43] and adenotonsillectomy is also in some studies found to be associated with weight gain.[44] On the other hand, significant weight loss might be difficult to achieve, especially for adolescents with severe obesity, and the detrimental effect of SRBD might serve as a barrier to weight loss, as well as a risk factor for cardiometabolic sequela.[14,45] Improvement in cardio-metabolic and inflammatory parameters in children with obesity has been observed after treatment of SRBD.[46]

A strength of the present study was the use of PSG-recordings of sleep and the recruitment of a comparison group that was age and gender matched. This study is one of the largest studies available comparing children with obesity and normal weight using PSG. A limitation of the study was the cross-sectional design that precludes us from drawing inferences about causality between sleep problems and weight. Further, eight children from the obesity group and none in the normal weight group had their sleep recordings during winter which may be a limitation as there may be seasonal variation in sleep parameters as well as obstructive sleep apnea.[47] However, when conducting the group comparisons on the PSG-measures without the individuals having recordings during winter comparable results were found (with significant group differences in the same direction on total sleep time, percentage of time spent in the N2 and REM sleep stages, oxygen desaturation events per hour, the AHI index and the AHI in non-REM sleep index). The group difference on the AHI in REM-sleep index did, however, not remain significant ($p = 0.06$).

Even though the children with obesity included in this study were not referred on suspicion of sleep problems, and had a relatively low

degree of obesity, they were participating in an obesity treatment trial and might be somewhat different from the general population of children with obesity. On the other hand, most previous studies examining PSG-measured sleep in children with obesity stem from samples referred for assessment of sleep problems.[24]

In conclusion, the obesity group demonstrated significantly higher prevalence of mild to moderate sleep disordered breathing than the normal weight children. Further, parents had not observed or suspected the presence of SRBD in their children, with the exception of some parents reporting occasional snoring. This indicates that SRBD in children often escape the attention of parents and that parents do not recognize the potential significance of snoring.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS

Yngvild Sørebø Danielsen: design of study, data collection, data analysis, interpretation of findings and writing the manuscript. Hanna Flækøy Skjåkødegård: conceptualization of aims, data management and data analysis, interpretations of findings and writing of manuscript. Bjørn Bjorvatn: conceptualization of aims, interpretations of findings and writing of manuscript. Petur Benedikt Juliusson: conceptualization of aims, interpretations of findings and writing of manuscript. Ståle Pallesen: design of study, supervision, data analysis, interpretation of findings and writing of manuscript.

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