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MISCELLANEOUS

Obstructive sleep apnea in children and adolescents with and without obesity

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Abstract

Purpose To investigate the prevalence of obstructive sleep apnea (OSA) in children referred for obesity treatment, and to compare the prevalence with that of a normal-weight group. Moreover, we examined the association between Body Mass Index Standard Deviation Score (BMI SDS) and the Apnea–Hypopnea Index (AHI).

Methods This cross-sectional study included 139 children aged 7–18 years with overweight/obesity (BMI SDS > 1.28) recruited from an obesity treatment clinic. The normal-weight group consisted of 33 children (BMI SDS ≤ 1.28) aged 7–18 years recruited from schools. Sleep examinations were performed using a type 3 portable sleep monitor (Nox T3). OSA was defined as AHI ≥ 2. Height and weight were measured and the tonsillar size was clinically estimated using the Brodsky scale.

Results The OSA prevalence was 44.6% in children with overweight/obesity compared with 9.1% in the normal-weight group ($p = 0.0002$), and the relative risk of OSA was 4.9 (95% CI 1.6–14.7). In a logistic regression, a one-unit increase in the BMI SDS increased the odds of having OSA by a factor of 1.92 independent of age, sex, tonsillar hypertrophy, and asthma (95% CI 1.33–2.76, $p = 0.0005$). A generalized linear regression adjusted for the same variables revealed an association between BMI SDS and AHI (a one-unit increase in the BMI SDS equaled an average increase in the AHI of 35% (95% CI 19–53%, $p < 0.0001$)).

Conclusions In this study, children with overweight/obesity had a significantly higher prevalence of OSA compared with a normal-weight group. Increased BMI SDS was associated with increased AHI.

Keywords Adolescent · Child · Normal weight · Obesity · Obstructive sleep apnea

Introduction

Obstructive sleep apnea (OSA) is a sleep disorder caused by episodes of partial upper airway obstruction and/or intermittent complete obstruction that disrupt normal breathing during sleep [1]. OSA is associated with cardiovascular complications, neurocognitive problems, and reduced quality of life in childhood [1].

Adenotonsillar hypertrophy is a major risk factor for pediatric OSA [2]. However, several studies have reported that the disorder is more likely to persist in children with overweight/obesity after adenotonsillectomy compared with children with normal weight [3–5], indicating that obesity is key in the pathogenesis of OSA. This is in line with previous studies reporting an OSA prevalence of 24–61% in children and adolescents with overweight/obesity [6–8].

The etiological factors predisposing to the increased risk of OSA in children with obesity are not fully understood.

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Anatomical factors, such as the deposition of fat in the pharyngeal region [9], as well as the physiological effects of obesity on the respiratory system [10] may contribute to the increased risk.

Other risk factors for pediatric OSA reported in cross-sectional studies are premature birth [11, 12], African-American ethnicity [11–13], history of asthma [12, 13], and craniofacial abnormalities [14]. Moreover, there is some evidence that OSA occurs more often in boys than in girls [12].

Although the prevalence of OSA in children with overweight/obesity has been investigated in previous studies, many studies have investigated groups of children referred to sleep clinics on suspicion of OSA. Moreover, only few studies have compared the prevalence of OSA with that of a group of children with normal weight [15–18]. Three of these studies [15, 17, 18] investigated Asian populations. Given the increasing prevalence of childhood obesity, both globally and in Denmark [19, 20], more knowledge about the association between OSA and obesity is important.

We conducted a clinical prospective pragmatic epidemiological study in a Danish population, using a type 3 portable sleep monitor for sleep examinations, to investigate the prevalence of OSA in children referred to an obesity treatment clinic and compared them with a normal-weight group of children. We hypothesized that OSA was more prevalent in children with overweight/obesity, and that the Body Mass Index Standard Deviation Score (BMI SDS) was associated with the Apnea–Hypopnea Index (AHI).

Methods

Design and study population

In this cross-sectional study, children and adolescents with overweight/obesity were recruited at enrolment in the chronic care multidisciplinary treatment program at the Children's Obesity Clinic, Department of Pediatrics, Holbæk University Hospital, Denmark, from June 2015 to June 2016. All newly referred children and adolescents who were eligible according to the inclusion and exclusion criteria were offered participation in the period. The two inclusion criteria for the overweight/obese group were age 7–18 years; and a BMI SDS > 1.28 corresponding to the 90th percentile according to a Danish age- and sex-adjusted reference [21]. For sub-group analysis obesity was defined as a BMI SDS > 2.33 (corresponding to the 99th percentile [21]). The normal-weight group was recruited from different schools on Zealand in Denmark. The 2 inclusion criteria for the normal-weight group were age 7–18 years; and $-1.28 <$ BMI SDS ≤ 1.28 corresponding to a BMI above the 10th percentile and below the 90th percentile [21]. The exclusion criteria for both groups were as follows: neuromuscular disease; craniofacial syndromes/abnormalities; laryngeal and/or tracheal malformations.

Consultations

Clinical characteristics were obtained, including age, sex, height, weight, and tonsillar size. The medical history was registered, including self-reported asthma and self-reported adenotonsillar surgery. Asthma was only registered as "yes" if asthma medication was present on the child's medication list. The children and their parents were instructed meticulously in the use of the portable sleep monitor.

Anthropometry

Height was measured to the nearest millimeter using a stadiometer. Weight was measured to the nearest 100 g with the child wearing light indoor clothes and without shoes. BMI was calculated as the weight in kilograms divided by the square of the height in meters (kg/m^2). The BMI SDS was estimated using the LMS method (LMS refers to three smooth age-specific curves called L (lambda), M (mu), and S (sigma) [22]) based on Danish references [21].

Tonsillar hypertrophy

The tonsillar size was clinically assessed and graded from 0 to 4 using the scheme proposed by Brodsky et al. [23]. Tonsillar hypertrophy was defined as a Brodsky score > 2. The assessment was performed by the same medical doctor throughout the study period.

Sleep examinations

Sleep assessments were performed for one night using the Nox T3 device. The device is a type 3 sleep monitor measuring airflow via a nasal cannula; respiratory effort via chest and abdominal belts; body position and activity via an integrated accelerometer; and pulse and oxygen saturation via an oximeter. All sleep examinations were analyzed manually by the same registered polysomnographic technologist (RPSGT) according to the pediatric respiratory rules defined by the American Academy of Sleep Medicine [24]. The RPSGT was blinded to the BMI SDS of the children.

Apneas were identified if there was a ≥ 90% drop in airflow for the duration of at least two breaths. Obstructive apneas were defined as apneas associated with respiratory effort throughout the entire period of the event. Mixed apneas were defined as apneas with absent respiratory effort during one portion of the event and presence of respiratory effort in another portion of the event. Hypopneas were identified if there was a ≥ 30% drop in airflow for the duration of at least two breaths associated with a ≥ 3% oxygen desaturation.

The AHI was defined as the average number of obstructive apneas, mixed apneas, and hypopneas per hour of sleep [i.e., the sum of the Obstructive Apnea Index (OAI), the Mixed Apnea Index (MAI), and the Hypopnea Index (HI)] [25]. Central apneas, defined as apneas with absent respiratory effort, were not included in the AHI [25]. The Oxygen Desaturation Index (ODI) was defined as the average number of oxygen desaturations ≥ 4% per hour of sleep.

Sleep time was estimated as the total recording time minus the episodes of signal artifacts. Only sleep examinations containing ≥ 3.75 h of sleep with a signal quality ≥ 90% were considered eligible for analysis. OSA was defined as AHI ≥ 2. Mild OSA was defined as 2 ≤ AHI < 5, moderate OSA was defined as 5 ≤ AHI < 10, and severe OSA was defined as AHI ≥ 10.

Children with overweight/obesity used the sleep monitor during admission to the obesity clinic, while children in the normal-weight group, due to practical circumstances, used the sleep monitor at home. Both groups of children performed the sleep examination at night.

Statistics

Descriptive characteristics across groups were compared using Chi square test or Fisher's exact test for categorical variables and Mann–Whitney test for continuous variables. The prevalence of OSA in the groups was compared using Chi square test and further described with a relative risk estimate.

A logistic regression model was used to assess the relation between BMI SDS and OSA adjusting for age, sex, tonsillar hypertrophy, and self-reported asthma with medication.

A generalized linear regression, in the negative binomial distribution using log(sleep time) as offset, was performed to describe the association between BMI SDS and AHI, adjusting for age, sex, tonsillar hypertrophy, and self-reported asthma with medication. The same model was used to estimate the difference in AHI between the overweight/obese group and the normal-weight group.

The size of the normal-weight group was calculated a priori based on an expected OSA prevalence of 10% in the normal-weight group and of 45% in the overweight/obese group, power = 0.8, and alfa = 0.05.

The p-values reported are two-tailed and alpha was set at 0.05 for statistical significance. Statistics were performed in SAS Enterprise Guide version 7.1. Graphics were created in GraphPad Prism version 7.04.

Results

During the study period, 280 children and adolescents aged 7–18 years with a BMI SDS > 1.28 entered obesity treatment of which 5 fulfilled one of the exclusion criteria and 69 did not wish to participate (Fig. 1). The final analysis included 139 children and adolescents with overweight/obesity. There was no significant difference in age, sex, and BMI SDS between children who did not wish to participate ($n = 69$) and children who were initially included ($n = 206$). Children with unsuccessful sleep examinations ($n = 67$) were significantly younger than children with successful sleep examinations ($n = 139$) (median age 11.4 vs. 12.4 years, $p = 0.02$), but there was no difference with regard to sex or BMI SDS.

A total of 36 children and adolescents aged 7–18 years with − 1.28 < BMI SDS ≤ 1.28 volunteered to participate in the normal-weight group. None of these children fulfilled any of the exclusion criteria, but 3 were not included in the final analysis because of inadequate signal quality/sleep time < 3.75 h.

None of the children in any of the groups reported known sleep disorders at the time of inclusion in the study.

Comparison of children with and without overweight/obesity

Demographic and anthropometric characteristics of the groups are presented in Table 1. By design, the groups differed significantly in BMI SDS (median 3.02 vs. 0.44, $p = 0.0001$).

Sleep parameters are presented in Table 1. AHI, HI, MAI, and ODI were significantly higher in children with overweight/obesity, whereas the sleep time and the oxygen saturation were significantly lower compared with the normal-weight group.

The OSA prevalence in children with overweight/obesity was 44.6% compared with 9.1% in the normal-weight group ($p = 0.0002$), and the relative risk of OSA was 4.9 [95% confidence interval (CI) 1.6–14.7]. The prevalence of OSA in the groups, using different AHI cutoff values for defining OSA, is shown in Table 1.

Mild OSA was the most common form of OSA occurring in 32% of the children with overweight/obesity and in 3% of the children in the normal-weight group ($p = 0.0008$). The distribution of the OSA severity in each group is illustrated in Fig. 2.

The median AHI of children with OSA and overweight/obesity ($n = 62$) was 3.3 (interquartile range 2.7–5.7). In this group, hypopneas represented 84% of all respiratory events; obstructive apneas accounted for 14%; and mixed apneas for 1% of all respiratory events.

Risk of OSA

Results from the logistic regression analysis, including a total of 172 children, are shown in Table 2. Increasing BMI SDS significantly increased the risk of OSA independently

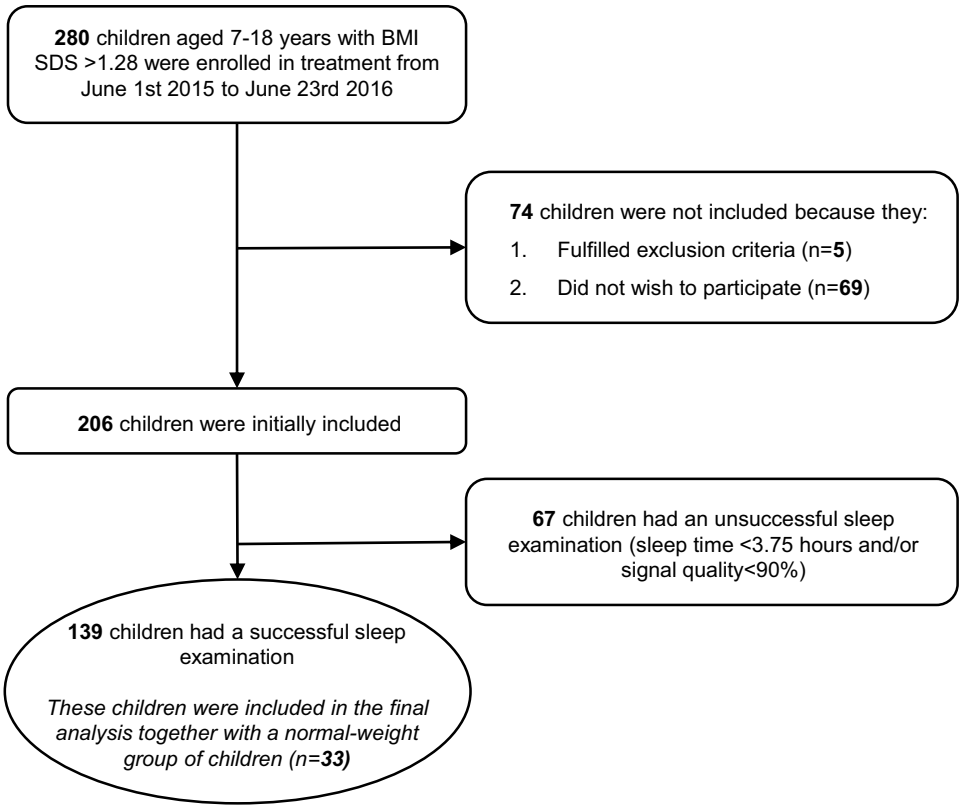

Fig. 1 Flow chart demonstrating the inclusion of children and adolescents in the study

of age, sex, tonsillar hypertrophy, and self-reported asthma with medication [adjusted odds ratio (OR) 1.92, $p=0.0005$].

As evident from Table 2, age also significantly increased the risk of OSA (adjusted OR 1.25, $p=0.0008$). Sex, tonsillar hypertrophy, and self-reported asthma with medication were not related to the risk of OSA in the analysis (Table 2).

Association between BMI SDS and AHI

The scatter plot with a simple linear regression line (Fig. 3) illustrates a positive relation between BMI SDS and AHI.

A generalized linear regression analysis on a total of 172 children was performed to describe the association between BMI SDS and AHI, adjusting for age, sex, tonsillar hypertrophy, and self-reported asthma with medication. An increase in the BMI SDS of one unit equaled an average increase in the AHI of 35% (95% CI 19–53%, $p<0.0001$).

Including only the group with overweight/obesity ($n=139$) in the analysis, the result was still significant after adjusting for the same variables [i.e., an increase in the BMI SDS of one unit equaled an average increase in the AHI of 57% (95% CI 10–123%, $p=0.01$)].

Average difference in AHI between groups

To estimate the average difference in AHI between the groups, a generalized linear regression analysis was performed, adjusting for age, sex, tonsillar hypertrophy, and self-reported asthma with medication. On average, the AHI was 3.2 times higher in children with overweight/obesity compared with children in the normal-weight group (95% CI 2.1–5.0, $p<0.0001$).

Additional analyses: sleep time

Because sleep time was significantly shorter in children with overweight/obesity (Table 1), the results were verified by controlling for sleep time as a possible confounder in the logistic regression analysis and in the generalized linear regression models. Controlling for sleep time did not change the results of any of the analyses, and sleep time was not associated with the risk of OSA.

Discussion

In this cross-sectional study, we found that the prevalence of OSA in children and adolescents referred for obesity treatment was significantly higher compared with a normal-weight group of children (44.6% vs. 9.1%). The result is consistent with similar studies [16, 18]. However, a direct comparison with other studies is difficult due to the inconsistency in the definition of respiratory events included in the AHI and because there is no consensus

Table 1 Demographic, anthropometric, and sleep parameters in 139 children and adolescents with overweight/obesity and in 33 children and adolescents with normal weight

	Overweight or obesity ($n=139$)	Normal weight ($n=33$)	p-Value
Demographic/anthropometric parameters			
Male, *n* (%)	59/139 (42.5)	12/33 (36.4)	0.52
Age, years	12.3 (10.2–14.5)	11.8 (10.5–13.3)	0.38
Caucasians, *n* (%)	113/139 (81.3)	30/33 (90.9)	0.18
BMI SDS	3.02 (2.49–3.28)	0.44 (−0.53 to 0.72)	**< 0.0001**
BMI SDS > 2.33, *n* (%)	116/139 (83.5)	0/33 (0)	**< 0.0001**
Tonsillar hypertrophy, *n* (%)	21/139 (15.1)	2/33 (6.1)	0.26
Tonsillectomy, *n* (%)	14/138[a] (10.1)	1/33 (3.0)	0.31
Adenoidectomy, *n* (%)	18/137[b] (13.1)	2/32[a] (6.3)	0.37
Asthma, *n* (%)	16/139 (11.5)	2/33 (6.1)	0.53
Sleep parameters			
AHI, events/hour	1.6 (0.8–3.2)	0.5 (0.2–0.8)	**< 0.0001**
HI, events/hour	1.5 (0.7–2.7)	0.4 (0.2–0.8)	**< 0.0001**
OAI, events/hour	0.0 (0.0–0.2)	0.0 (0.0–0.1)	0.07
MAI, events/hour	0.0 (0.0–0.1)	0.0 (0.0–0.0)	**0.01**
Sleep time, hours	7.7 (6.1–8.5)	8.6 (7.0 − 9.1)	**0.009**
Oxygen saturation	95.5 (94.4–96.1)	95.7 (95.3–96.7)	**0.04**
ODI, events/hour	1.6 (0.9–2.7)	1.0 (0.5–1.5)	**0.003**
AHI ≥ 1, *n* (%)	96/139 (69.1)	8/33 (24.2)	**< 0.0001**
AHI ≥ 2, *n* (%)	62/139 (44.6)	3/33 (9.1)	**0.0002**
AHI ≥ 5, *n* (%)	18/139 (13.0)	2/33 (6.1)	0.27

For continuous variables data are presented with medians and interquartile ranges

Bold values indicate $p < 0.05$

BMI SDS Body Mass Index Standard Deviation Score, *AHI* Apnea–Hypopnea Index, *HI* Hypopnea Index, *OAI* Obstructive Apnea Index, *MAI* Mixed Apnea Index, *ODI* Oxygen Desaturation Index

[a]One did not know the answer

[b]Two did not know the answer

regarding the AHI cutoff value defining OSA. Moreover, the definition of overweight/obesity varies between studies. We chose to define OSA as AHI ≥ 2 because this is the most commonly used definition [26]. With an AHI cutoff value ≥ 5 we could not detect a statistically significant difference in the OSA prevalence. This is possibly explained by the fact that our study population in general demonstrated mild OSA.

Children with overweight/obesity had significantly higher AHI, HI, MAI, and ODI compared with children in the normal-weight group. Overall the OAI accounted for a relatively small percentage of the AHI, which might explain why we could not detect a statistically significant difference in OAI between the groups.

In accordance with our hypothesis, the generalized linear regression showed an association between BMI SDS and AHI. This is supported by Xu et al. [17] and Kohler et al. [27]. The finding indicates that weight reduction is seemingly important in the treatment of OSA in children and adolescents with overweight/obesity. Few studies have investigated the impact of weight-loss intervention on OSA in children and adolescents, but the existing studies indicate that the AHI is reduced during obesity treatment [6–8].

The relation between BMI SDS and OSA was confirmed in the logistic regression analysis. In line with some other studies [15, 16, 18], the model demonstrated that increasing BMI SDS significantly increased the risk of having OSA. In both the generalized linear regression and in the logistic regression we chose to adjust for possible confounders such as sex, age, tonsillar hypertrophy, and self-reported asthma with medication.

Interestingly, we found that older age increased the risk of OSA. The reason for this finding is not completely clear. A likely explanation is the developmental changes in the upper airway. For instance, the upper airway neuromuscular tone is reported to decrease with age [28] possibly impacting on the collapsibility of the upper airway during sleep.

In our analysis, tonsillar hypertrophy was not identified as a significant independent risk factor for OSA. Su et al. [18] compared risk factors for OSA in pre-school children with school-aged children, and they found that adenotonsillar hypertrophy was a risk factor for OSA in only

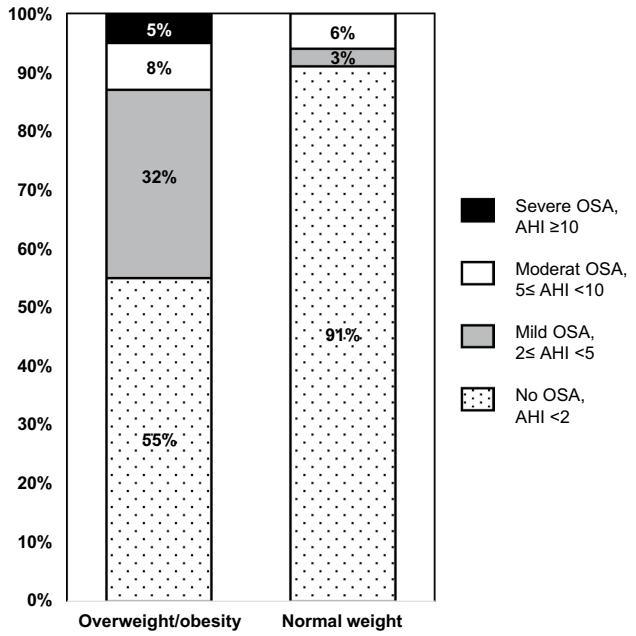

Fig. 2 Diagram showing the distribution of the severity of obstructive sleep apnea (OSA) in children and adolescents with overweight/obesity ($n = 139$) and in children and adolescents with normal weight ($n = 33$)

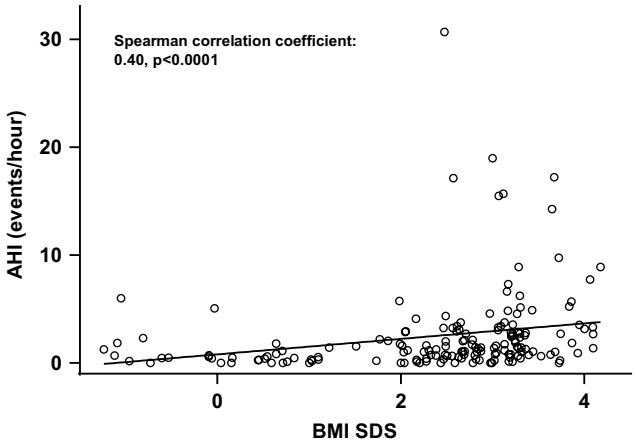

Fig. 3 Scatter plot with a simple linear regression line illustrating the relation between Body Mass Index Standard Deviation Score (BMI SDS) and the Apnea–Hypopnea Index (AHI) in 172 children and adolescents with and without obesity

Table 2 Logistic regression analysis including a total of 172 children and adolescents with and without overweight/obesity assessing the risk of OSA in relation to different independent variables

Independent variables	Adjusted OR	95% CI	*p*-Value
BMI SDS, one-unit increase	1.92	1.33–2.76	**0.0005**
Age, one-year increase	1.25	1.10–1.42	**0.0008**
Sex, male vs. female	0.85	0.41–1.75	0.66
Tonsillar hypertrophy, grade > 2 vs. grade ≤ 2	2.39	0.90–6.37	0.08
Self-reported asthma, yes vs. no	0.51	0.16–1.69	0.27

Bold values indicate $p < 0.05$

BMI SDS Body Mass Index Standard Deviation Score, *OR* odds ratio, *CI* confidence intervals

pre-school children. In this study, all children were school-aged (7–18 years). Moreover, only 13% of the children had tonsillar hypertrophy, and none of the children had a tonsillar grade > 3. Therefore, it seems plausible that tonsillar hypertrophy was not a major risk factor for OSA in our study population.

We assessed the tonsillar size clinically by the Brodsky scale, and to prevent inter-observer variability the same medical doctor assessed the tonsils throughout the entire study period. The use of fiber endoscopy or magnetic resonance imaging could provide a more accurate measure of both the adenoids and the tonsils and should be considered in future studies.

There is some evidence that asthma is associated with OSA [12, 13]. The relation between asthma and OSA has not been fully understood, but airway inflammation and nocturnal bronchoconstriction may predispose to the increased risk of OSA [13]. In this study, presence of self-reported asthma with medication did not significantly increase the risk of OSA. However, further studies are required in larger populations of children with a diagnosis of asthma to more meticulously investigate the link between asthma and OSA.

African-American ethnicity and Asian ethnicity are reported as possible risk factor for OSA [11–13, 29]. Our study population consisted primarily of Caucasians, and the majority of the remaining children were of Middle-Eastern origin. Adjusting for ethnicity (Caucasian yes vs. no) did not change the results of our analyses.

Sleep time was significantly shorter in children with obesity compared with the normal-weight group. This may be due to the fact that the medical staff woke the children in the obesity clinic up in the morning to participate in other investigations as part of their medical evaluation. We took this into account by adjusting for sleep time in the additional analyses. This did not change the results of the logistic regression analysis or the generalized linear regression models.

The study has some limitations. First, 33% of the children and adolescents with overweight/obesity had an unsuccessful sleep examination. This could have introduced a degree of selection bias which could potentially affect the prevalence and severity of OSA. These children were significantly younger than children with successful sleep examinations, reflecting that younger children had more difficulties sleeping with portable sleep monitor. However, they did not differ significantly in sex or BMI SDS.

Second, the children with overweight/obesity used the portable sleep monitor during admission to the obesity clinic while the normal-weight group used the device at home. Whether or not this has had any impact on the AHI is unknown. Sleeping in an unfamiliar environment can contribute to the so-called first-night effect, characterized by reduced sleep quality in individuals sleeping with polysomnography (PSG) for two or more consecutive nights. However, Scholle et al. investigated the first-night effect in children, and they found no night-to-night variability in the respiratory parameters such as the AHI [30].

Third, the generalizability of the normal-weight group to the general pediatric population may be questioned. However, none of the children included in this group were known with sleep disturbances before inclusion in the study and none of them were overweight. We have therefore no reason to believe that the children in this group were biased due to selection.

In this study, we chose a pragmatic approach using the Nox T3, reflecting that portable sleep monitoring is often the realistic first choice because of the costs and inaccessibility PSG. The Nox T3 device has demonstrated good measurement agreement compared with PSG in adults [31]. Similar type 3 sleep monitors, with the same number of channels, have been validated in children, and a recent meta-analysis by Certal et al. concluded that unattended sleep studies, including type 3 devices, are generally valid tools for predicting both the presence and the severity of OSA in children (pooled area under the curve = 0.88) [32]. According to the most recent European Respiratory Society Statement, on the diagnosis and management of obstructive sleep-disordered breathing in children, unattended sleep studies may be used as an alternative tool for diagnosing OSA if PSG is not available [33]. To avoid inter-observer variability all sleep examinations were analyzed manually by the same RPSGT. Because we did not perform full PSG, information about arousals was not acquired. This could potentially result in underestimation of hypopneas. Furthermore, full PSG gives information about sleep architecture, providing a more accurate detection of the total sleep time.

The primary strength of this study is that we focused on children referred to an obesity treatment clinic and not on symptomatic children referred to a sleep clinic for suspected OSA. Furthermore, it was a strength that we compared the OSA prevalence with a normal-weight group of children and that the study population was relatively large in comparison with prior studies.

Conclusions

In conclusion, our study supports our hypothesis that the prevalence of OSA in children and adolescents with overweight/obesity referred to an obesity treatment clinic is significantly higher compared with children in a normal-weight group and that there is an association between increased BMI SDS and increased AHI.

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Author Contributions IGA, PH, and JCH designed the study. IGA collected and analyzed the data and drafted the manuscript. PH and JCH critically revised and approved the manuscript.

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Compliance with ethical standards

Conflict of interest Author IGA received a study-related grant from ResMed Maribo, Denmark. Author JCH declares he has no conflict of interest. Author PH declares he has no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors. Approvals were obtained from the Regional Danish Ethics Committee (Protocol ID SJ-404) and the Danish Data Protection Agency (ID no. REG-111-2014). The study was registered in Clinicaltrials.gov (ID no.: NCT02463201).

Informed consent Informed consent was obtained from all individual participants included in the study.

References

1. Marcus CL, Brooks LJ, Draper KA et al (2012) Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 130:e714–e755. https://doi.org/10.1542/peds.2012-1672
2. Kang K-T, Chou C-H, Weng W-C et al (2013) Associations between adenotonsillar hypertrophy, age, and obesity in children with obstructive sleep apnea. PLoS One 8:e78666. https://doi.org/10.1371/journal.pone.0078666
3. Mitchell RB, Kelly J (2007) Outcome of adenotonsillectomy for obstructive sleep apnea in obese and normal-weight children. Otolaryngol Head Neck Surg 137:43–48. https://doi.org/10.1016/j.otohns.2007.03.028
4. O'Brien LM, Sitha S, Baur LA, Waters KA (2006) Obesity increases the risk for persisting obstructive sleep apnea after treatment in children. Int J Pediatr Otorhinolaryngol 70:1555–1560. https://doi.org/10.1016/j.ijporl.2006.04.003
5. Marcus CL, Moore RH, Rosen CL et al (2013) A randomized trial of adenotonsillectomy for childhood sleep apnea. N Engl J Med 368:2366–2376. https://doi.org/10.1056/NEJMoa1215881

6. Verhulst SL, Franckx H, Van Gaal L et al (2009) The effect of weight loss on sleep-disordered breathing in obese teenagers. Obesity (Silver Spring) 17:1178–1183. https://doi.org/10.1038/oby.2008.673
7. Siegfried W, Siegfried A, Rabenbauer M, Hebebrand J (1999) Snoring and sleep apnea in obese adolescents: effect of long-term weight loss-rehabilitation. Sleep Breath 3:83–88. https://doi.org/10.1007/s11325-999-0083-7
8. Van Hoorenbeeck K, Franckx H, Debode P et al (2012) Weight loss and sleep-disordered breathing in childhood obesity: effects on inflammation and uric acid. Obesity 20:172–177. https://doi.org/10.1038/oby.2011.282
9. Arens R, Sin S, Nandalike K et al (2011) Upper airway structure and body fat composition in obese children with obstructive sleep apnea syndrome. Am J Respir Crit Care Med 183:782–787. https://doi.org/10.1164/rccm.201008-1249OC
10. Koenig SM (2001) Pulmonary complications of obesity. Am J Med Sci 321:249–279
11. Rosen CL, Larkin EK, Kirchner HL et al (2003) Prevalence and risk factors for sleep-disordered breathing in 8- to 11-year-old children: association with race and prematurity. J Pediatr 142:383–389. https://doi.org/10.1067/mpd.2003.28
12. Spilsbury JC, Storfer-Isser A, Rosen CL, Redline S (2015) Remission and incidence of obstructive sleep apnea from middle childhood to late adolescence. Sleep 38:23–29. https://doi.org/10.5665/sleep.4318
13. Redline S, Tishler PV, Schluchter M et al (1999) Risk factors for sleep-disordered breathing in children. Am J Respir Crit Care Med 159:1527–1532. https://doi.org/10.1164/ajrccm.159.5.9809079
14. Cakirer B, Hans MG, Graham G et al (2001) The relationship between craniofacial morphology and obstructive sleep apnea in whites and in African–Americans. Am J Respir Crit Care Med 163:947–950. https://doi.org/10.1164/ajrccm.163.4.2005136
15. Wing YK, Hui SH, Pak WM et al (2003) A controlled study of sleep related disordered breathing in obese children. Arch Dis Child 88:1043–1047
16. Beebe DW, Lewin D, Zeller M et al (2007) Sleep in overweight adolescents: shorter sleep, poorer sleep quality, sleepiness, and sleep-disordered breathing. J Pediatr Psychol 32:69–79. https://doi.org/10.1093/jpepsy/jsj104
17. Xu Z, Jiaqing A, Yuchuan L, Shen K (2008) A case-control study of obstructive sleep apnea–hypopnea syndrome in obese and nonobese Chinese children. Chest 133:684–689. https://doi.org/10.1378/chest.07-1611
18. Su M-S, Zhang H-L, Cai X-H et al (2016) Obesity in children with different risk factors for obstructive sleep apnea: a community-based study. Eur J Pediatr 175:211–220. https://doi.org/10.1007/s00431-015-2613-6
19. Ng M, Fleming T, Robinson M et al (2014) Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet 384:766–781. https://doi.org/10.1016/S0140-6736(14)60460-8
20. Pearson S, Olsen LW, Hansen B, Sørensen TIA (2005) [Increase in overweight and obesity amongst Copenhagen schoolchildren, 1947–2003]. Ugeskr Laeger 167:158–162
21. Nysom K, Mølgaard C, Hutchings B, Michaelsen KF (2001) Body mass index of 0 to 45-year-old Danes: reference values and comparison with published European reference values. Int J Obes Relat Metab Disord 25:177–184. https://doi.org/10.1038/sj.ijo.0801515
22. Cole T, Green P (1992) Smoothing reference centile curves: the LMS method and penalized likelihood. Stat Med 11:1305–1319
23. Brodsky L (1989) Modern assessment of tonsils and adenoids. Pediatr Clin North Am 36:1551–1569
24. Berry R, Brooks R, Gamaldo C et al (2014) The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications, Version 2.1. American Academy of Sleep Medicine, Darien
25. American Academy of Sleep Medicine (2014) international classification of sleep disorders, 3rd edn. American Academy of Sleep Medicine, Darien
26. Andersen IG, Holm J-C, Homøe P (2016) Obstructive sleep apnea in obese children and adolescents, treatment methods and outcome of treatment—a systematic review. Int J Pediatr Otorhinolaryngol 87:190–197. https://doi.org/10.1016/j.ijporl.2016.06.017
27. Kohler M, Lushington K, Couper R et al (2008) Obesity and risk of sleep related upper airway obstruction in caucasian children. J Clin Sleep Med 4:129–136
28. Marcus CL, Fernandes Do Prado LB, Lutz J et al (2004) Developmental changes in upper airway dynamics. J Appl Physiol 97:98–108. https://doi.org/10.1152/japplphysiol.00462.2003
29. Kohler MJ, van den Heuvel CJ (2008) Is there a clear link between overweight/obesity and sleep disordered breathing in children? Sleep Med Rev 12:347–361. https://doi.org/10.1016/j.smrv.2008.04.008
30. Scholle S, Scholle HC, Kemper A et al (2003) First night effect in children and adolescents undergoing polysomnography for sleep-disordered breathing. Clin Neurophysiol 114:2138–2145. https://doi.org/10.1016/S1388-2457(03)00209-8
31. Cairns A, Wickwire E, Schaefer E, Nyanjom D (2014) A pilot validation study for the NOX T3TM portable monitor for the detection of OSA. Sleep Breath 18:609–614. https://doi.org/10.1007/s11325-013-0924-2
32. Certal V, Camacho M, Winck JC et al (2015) Unattended sleep studies in pediatric OSA: a systematic review and meta-analysis. Laryngoscope 125:255–262. https://doi.org/10.1002/lary.24662
33. Kaditis AG, Alonso Alvarez ML, Boudewyns A et al (2016) Obstructive sleep disordered breathing in 2- to 18-year-old children: diagnosis and management. Eur Respir J 47:69–94. https://doi.org/10.1183/13993003.00385-2015