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Snoring toddlers with and without obstructive sleep apnoea differed with regard to snoring time, adenoid size and mouth breathing

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Abstract

Aim: The difficulty of assessing the likelihood of obstructive sleep apnoea (OSA) in children who snore without full-night polysomnography is widely recognised. Our aim was to identify features that were characteristic of two-year-old children with OSA and evaluate whether this information could be used to assess the likelihood of OSA

Methods: The study was carried out as part of the Child-Sleep Project, a longitudinal birth cohort study of children born at Tampere University Hospital, Finland. This part of the study focused on the children in the cohort who snored and was carried out between 2013 and 2015. The primary outcomes were measured using parental questionnaires, polysomnography and clinical examinations.

Results: In total, 52 children participated at a mean age of 27 months (range 23-34). Of these, 32 (44% male) snorers and 20 (70% male) controls. The most significant findings were that children who had OSA demonstrated longer snoring time (P = .003), a greater tendency for mouth breathing (P = .007) and bigger adenoid size (P = .008) than snorers without OSA.

Conclusion: Snoring time, adenoid tissue size and mouth breathing were important features that identified the likelihood of OSA in snoring toddlers.

KEYWORDS

adenoids, obstructive sleep apnoea, snoring time, toddlers, tonsils

Abbreviations: AHI, apnoea/hypopnoea index; OSA, obstructive sleep apnoea.

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1 | INTRODUCTION

It has been estimated that 1.5%-15% of children snore regularly and 1%-5% have obstructive sleep apnoea (OSA).¹⁻⁴ OSA has been associated with many clinical morbidities, such as neurobehavioural problems and increased strain on the metabolism and cardiovascular organs.¹ Indeed, it has been suggested that early treatment of OSA would have a beneficial effect on behavioural changes and quality of life.⁵ The great majority of children with OSA snore, but not all children who snore have OSA. The difficulty of distinguishing children with these two conditions is a widely recognised problem in the field of paediatric sleep medicine.

Full-night polysomnography is still the gold standard for diagnosing OSA.⁶ However, paediatric polysomnography is limited in some centres because of a lack of availability and high cost. Other diagnostic methods, such as nocturnal pulse oximetry, video recording, nap-polysomnography and ambulatory polysomnography, have been estimated to have a poor predictive value when the result was negative.¹ Clinical findings and patient histories are often used as an alternative diagnostic method to assess the likelihood of OSA, although this method has many uncertainties.^{7,8}

Present knowledge of the factors used for assessing the likelihood of paediatric OSA is controversial. The size of the child's tonsils and their snoring history are considered to have high sensitivity, whereas excessive daytime sleepiness and caregiver observed apnoeas during sleep have high specificity.⁹ Nevertheless, it has been demonstrated that a single symptom or sign has poor diagnostic accuracy in predicting paediatric OSA.⁹ The snoring status of children and the presence of OSA can change during childhood, regardless of whether any procedures are carried out.^{10,11} The change in status may be caused be a change in the size of the tonsils and adenoids, with respect to the airway at different ages. Furthermore, the impact of adenotonsillar hypertrophy on paediatric OSA seems to change, as it has been reported to play a more significant role in preschool-aged children than in older children. In addition, the size of the adenoidal tissue seems to be a more important factor than palatine tonsil size in younger children.¹²

The appearance of symptoms and clinical findings of OSA vary at different ages, and paediatric OSA should not be considered as a stable disorder throughout childhood. Therefore, better tools are needed to determine which children need to be examined with polysomnography and whether there are other ways to reliably diagnose or exclude OSA. The aim of present study was to identify the polysomnographic features, patient history and clinical findings that were characteristic of two-year-old children with OSA. This age group was selected because there is scarcity of knowledge of these features in toddlers. The second aim was to determine whether the information that was gathered could be used to assess the likelihood of OSA in toddlers when parents had reported them snoring. It was hypothesised that toddlers with OSA would be more likely to snore more during polysomnography, have larger adenoids and palatine tonsils and demonstrate more mouth breathing than toddlers who snored but did not have OSA.

KEY NOTES

- The difficulty of assessing the likelihood of obstructive sleep apnoea (OSA) in children who snore without a fullnight polysomnography is a widely recognised problem.
- Our study found that children with OSA snored for longer at night than children who snored, but did not have OSA.
- Adenoid tissue size and mouth breathing were important clinical factors when assessing the likelihood of OSA in toddlers at a mean age of 27 months.

2 | METHODS

2.1 | Study design and participants

The present study was carried out as part of the Child-Sleep Project, which is a longitudinal birth cohort study that comprises 1673 children born between April 2011 and February 2013 at Tampere University Hospital, Finland. The study protocol was approved by the Ethical Committee of the Pirkanmaa Hospital District in March 2011. The inclusion criteria were families who spoke Finnish and lived in the Pirkanmaa Hospital District. They were recruited by local maternity clinics during the prenatal period, once the pregnancy had reached 32 weeks. The first of a series of questionnaires that concentrated on sleep, somatic and mental health, behaviour, temperament and family relationships were filled out at this stage (n = 1673). The other were completed at three (n = 1427), eight (n = 1291), 18 (n = 1163) and 24 months (n = 947) after the birth.¹³ The present study concentrated on children who snored in the Child-Sleep cohort.

The Sleep Disturbance Scale for Children,¹⁴ which evaluates sleeps disorders in children, was used to estimate snoring frequency at the age of eight and 24 months. The question from the sleep-disordered breathing subscale, which asks how often the child snore, was used to select the patients. The children were included if their parents indicated they snored always or often, namely daily or three to five times a week. They were excluded if they snored sometimes or occasionally, defined as once or twice a week or once or twice a month, or never. Snoring for a minimum of three nights per week has been used to indicate a significant amount of snoring in children in previous studies.¹⁵ The exclusion criteria also included children who only snored during respiratory infections or had severe illnesses and disabilities. Children who did not snore regularly were recruited as controls. They were recruited among the children whose parent indicated they snored never, occasionally or sometimes in the Sleep Disturbance Scale for Children at the age of eight or 24 months. The families were contacted and asked to participate in the study, and a parent was interviewed on the telephone to confirm the occurrence of snoring or lack of it. The parents provided written, informed consent so that their children and themselves could take part in the

study. All research was performed in accordance with the relevant guidelines and regulations.

2.2 | Outcome measures

Some of the Sleep Disturbance Scale for Children questions were repeated at the time of the clinical examinations. These dealt with how frequently the child gasped for breath or was unable to breathe during sleep, how often the child snored and how often the child was tired during the daytime. Moreover, the parents were asked about the child's breathing habits and nasal symptoms. The questions, the possible answers and how the answers were grouped for the analysis are presented in Table 1.

The examination carried out by the paediatrician included measurements of weight and height and a general paediatric and neurological assessment.

The otorhinolaryngological examination included inspection of the oral cavity and oropharynx, anterior rhinoscopy and nasal flexible fibreoptic endoscopy of the nasopharynx, hypopharynx and larynx. Nasal flexible fibreoptic endoscopy has previously been determined to be a reliable method for assessing paediatric adenoid size, when compared to radiographic assessment methods.¹⁶ A four-class scale was used to determine the adenoid size with respect to the nasopharyngeal volume, in which class one indicated that tissue filled 0%-25% of the nasopharyngeal volume, class two was 26%-50%, class three was 51%-75% and class four was 76%-100%. Palatine tonsil size was assessed using Friedman's tonsil classification.¹⁷ Zero indicated no tonsils, one indicated that the tonsils were hidden between the palatoglossal and palatopharyngeal arch, two indicated that the tonsils extended to the arches, three indicated that the tonsils extended beyond the arches, but not to the midline, and four indicated that the tonsils extended to the midline. Furthermore, the breathing habit of the child, mainly through mouth or nose, was observed.

A full-night polysomnography was performed at the sleep laboratory at the Tampere University Hospital using an Embla N7000 device (Natus Medical Incorporated, Pleasanton, California, USA). The following signals were recorded: eight channels of electroencephalography, two channels of electro-oculography, submental electromyography, airflow by oronasal thermistor and nasal pressure transducer signal, oxygen saturation by two pulse oximeters, thoracoabdominal inductance plethysmography, diaphragmatic and abdominal electromyography. We also recorded electrocardiography, end-tidal partial pressure of carbon dioxide, sleeping position and snoring. An Emfit mattress sensor (Emfit Ltd, Finland) was used to detect breathing and body movements. The children were also video recorded. All of the recordings were manually analysed by two independent clinical neurophysiologists. The sleep stages and respiratory events were scored according to the paediatric rules in the American Academy of Sleep Medicine manual.¹⁸ Snoring episodes lasting more than three consecutive breathing cycles were selected, based on piezo and nasal pressure signal and verified by listening. The percentage of time with snoring, referred to as total sleep time, was calculated for each child. A diagnosis of OSA was made if the obstructive AHI was more than one per hour.¹⁹

2.3 | Statistical analysis

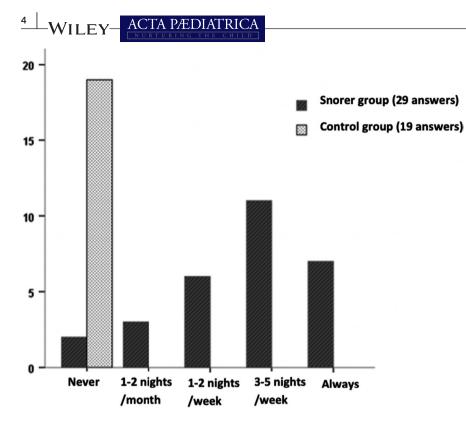
The statistical analysis was performed using IBM SPSS Statistics, version 22 or newer (IBM Corp). Cross-tabulation was used to compare qualitative variables, and the strength of the association was evaluated with Fisher's exact test. The Mann-Whitney *U* test and Kruskal-Wallis test were used to compare quantitative variables between the groups. Bonferroni correction was used when the Mann-Whitney *U* test was used to compare differences pairwise after the Kruskal-Wallis test. *P* < .05 was considered statistically significant.

3 | RESULTS

The prevalence of snoring in the cohort based on the questionnaire completed at 8 months after the birth was 3.2%, 41 snorers among 1291 children whose parents replied. At the timepoint of the

tions	How often does your child gasp for breath or is unable to breath during sleep? How often does your child snore? How often does your child experience daytime somnolence? Never. Occasionally (1-2 times per month or less). Sometimes (1-2 times per week). Often (3-5 times per week). Always (daily)	Breathing difficulties during sleep: Yes or no. Snoring frequency: Maximum of two nights per week or minimum of three nights per week. Frequency of daytime sleepiness: Maximum of two days per week or minimum of three days per week
	Does your child breathe through the nose or mouth when relaxed during the daytime? Mainly through nose. Mainly through mouth. Don't know.	Breathing habit: Mainly through nose. Mainly through mouth. Don't know.
	Does your child have constant rhinitis or nasal stuffiness? Yes or no	Nasal rhinitis and stuffiness: Yes or no.

TABLE 1Questions parents wereasked during the clinical examinations



24-month questionnaire, the prevalence was lower, 2.5%, 24 snorers among 947 children,

The total number of children who took part in the study at approximately 24 months was 52. Of these, 32 (44% male and 56% female) were snorers and 20 (70% male and 30% female) were controls. At the time of otorhinolaryngological examination, 19 of the parents of 20 children in the control group reported that their children currently never snore, and one did not answer the question. Current reported snoring was distinctly more common (P < .001) in the snorer group and the frequency varied within the group (Figure 1). The parents of three children from the snorer group did not answer the question. Consequently, the snorer group represents those children who had had remarkable snoring between the ages of 8 months and 24 months, although current snoring at the time of the clinical examinations varied. This is a common situation, because the snoring is not a stable feature in children.

At the time of clinical examination, the children had a mean age of 27 months (range 23-34). Their mean body mass index was 16.5 kg/m² (range 14.6-20.2), and their mean body mass index z-score was -0.02 (range -1.4-2.2). The body measurements of six children were missing. Three children had previously undergone an adenoidectomy, and one had received an adenoidectomy and partial tonsillectomy.

A full-night polysomnography was successfully performed on 49 children: 31 snorers and 18 controls. The time period between the otorhinolaryngological examination and the polysomnography was 19 days on average (range 0-99). Two children, one snorer and one control, did not arrive for the polysomnography and one registration failed in the control group because the child had a respiratory infection. The polysomnography findings of the snorers and controls are presented in Table 2. Significant differences were found between

obstructive AHI and snoring time, but not between the sleep parameters. OSA was diagnosed in nine children (56% male) in the snorer group and in none in the control group. Consequently, 29% (9 of 31) of snorers examined with polysomnography had OSA.

The snorer group was further divided into two groups, according to the diagnosis of OSA. The polysomnography findings of the OSA group and the no OSA group are presented in Table 3. Significant differences were found between AHI, obstructive AHI, snoring time, total sleep time and sleep stage N1, which is the transition period from being awake to falling asleep. In the OSA group, obstructive AHI was 1.2 to 6.3 per hour (median 1.5 per hour) and the snoring time was 16.0%-94.7% (median 63.6%) of the total sleep time. In the no OSA group, obstructive AHI was 0.0-0.4 per hour (median 0.1 per hour) and snoring time was 0.0%-87.8% (median 6.9%) of total sleep time. In contrast, obstructive AHI was 0.0-0.9 per hour (median 0.0 per hour) in the control group and snoring time was 0.0%-34.5% (median 0.0%). The differences in snoring time between these three groups were statistically significant (Figure 2).

The parental reports (Table 4) and the clinical findings (Table 5) were analysed to find differences between the OSA group and the no OSA group. Mouth breathing, as reported by parents, was more common (P = .015) in the OSA group (4/9 children) than in the no OSA group (1/22 children). The prevalence of breathing difficulties while sleeping was not associated with the OSA diagnosis. In addition, the parent-reported frequency of current snoring, daytime tiredness and nasal stuffiness did not differ between the OSA group and the no OSA group.

During the clinical examination, mouth breathing observed by otorhinolaryngologist was more common in the OSA group (6/9 children) than in the no OSA group (3/22) (P = .007). Adenoid size was found to be larger in the OSA group (P = .008). However, not all

FIGURE 1 Frequency of snoring reported by parents at the time of the clinical examination in the snorer and control groups
 TABLE 2
 Sleep and respiratory
parameters between the snorer and control groups

	Snorer group (n = 31)	No snoring control group (n = 18)	P values
Total recording time (minutes)	598.0 (570.5-630.5)	617.9 (575.8-640.7)	.206
Total sleep time (TST) (minutes)	505.5 (464.0-551.5)	533.5 (487.4-593.0)	.084
Sleep stage N1 (%/TST)	4.1 (2.6-7.2)	3.1 (1.4-6.3)	.195
Sleep stage N2 (%/TST)	32.5 (28.3-39.3)	36.4 (34.2-41.1)	0.081
Sleep stage N3 (%/TST)	31.4 (26.2-38.8)	30.3 (24.3-36.0)	.431
Rapid eye movement (REM) sleep (%/TST)	28.8 (25.9-32.2)	27.2 (25.5-31.9)	.548
REM latency(minutes)	71.0 (57.5-117.0)	63.5 (50.3-92.4)	.238
Arousal index (number/hour)	9.6 (8.7-11.2)	9.9 (8.3-11.3)	.947
Apnoea/hypopnoea index (number/hour)	1.7 (1.1-3.0)	1.8 (1.1-3.4)	.841
Obstructive apnoea/ hypopnoea index (number/ hour)	0.3 (0.0-1.3)	0.0 (0.0-0.1)	.003
Oxygen desaturation index 3% (number/hour)	1.0 (0.4-2.2)	1.6 (0.7-3.0)	.367
Snoring time percentage (%/ TST)	19.5 (2.3-58.6)	0.0 (0.0-12.2)	<.001

Note: Median and interquartile ranges (0.25-0.75) are presented. P values <.05 bolded.

TABLE 3 The sleep and respiratory parameters between the OSA and no OSA groups

	OSA group (n = 9)	No OSA group (n = 22)	P values
Total recording time (minutes)	602.0 (591.0-634.0)	585.5 (563.0-615.5)	.132
Total sleep time (TST) (minutes)	555.5 (504.0-569.5)	477.8 (458.4-516.4)	.005
Sleep stage N1 (%/TST)	3.0 (1.4-3.6)	5.6 (3.2-8.4)	.005
Sleep stage N2 (%/TST)	32.5 (26.7-38.7)	32.7 (28.7-39.8)	.660
Sleep stage N3 (%/TST)	35.6 (26.5-41.3)	29.8 (25.8-35.3)	.374
Rapid eye movement (REM) sleep (%/TST)	31.0 (27.2-32.8)	27.8 (25.9-32.1)	.249
REM latency (minutes)	69.5 (61.8-96.8)	75.8 (54.0-117.0)	.773
Arousal index (number/hour)	10.0 (9.2-12.7)	9.4 (8.4-11.1)	.223
Apnoea/hypopnoea index (number/hour)	3.6 (2.2-5.0)	1.2 (0.7-1.9)	<.001
Obstructive apnoea/hypopnoea index (number/hour)	1.5 (1.3-3.1)	0.1 (0.0-0.4)	<.001
Oxygen desaturation index 3%(number/hour)	2.2 (0.2-6.3)	0.9 (0.5-0.19)	.513
Snoring time percentage (%/TST)	63.6 (30.0-84.0)	6.9 (0.6-27.7)	.001

Note: Median and interquartile ranges (0.25-0.75) are presented. *P* values <.05 bolded.

children with OSA had large adenoids. The size of the palatine tonsil did not differ significantly between the groups. None of the children had nasal septum deviations or significant deformities in the upper respiratory tract.

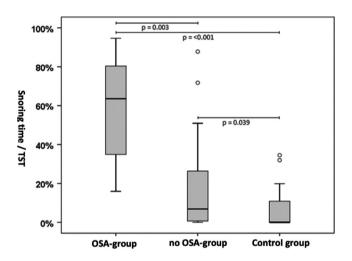
4 DISCUSSION

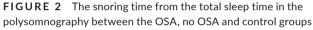
Paediatric OSA is a relatively common disorder. Its epidemiology, symptoms and clinical features differ from those of adult OSA. Furthermore, it has also been demonstrated that there is a difference in clinical findings and risk factors between children of different ages with OSA ^{12,20} and that disorder can spontaneously resolve.¹¹ For this reason, we should consider paediatric OSA as a disorder that transforms as the child grows up, rather than a stable entity.

The polysomnography results showed that children with OSA had longer snoring time from the total sleep time than children who snored but do not have OSA. In future, it would be beneficial to consider whether snoring time could be a useful marker when distinguishing children with OSA from children who snore without OSA, ACTA PÆDIATRICA

and whether this might enable the development of a simple and valid screening method for paediatric OSA.

We clearly showed that parents could reliably evaluate whether the child snored when the questionnaire and polysomnography data were compared. However, there was no statistical difference between the parents' assessment of snoring frequency in the OSA group and in the no OSA group. None of the parents of the children with OSA reported their child having breathing difficulties while sleeping. Nevertheless, all of the children who were diagnosed with OSA were





part of the snorer group. These findings confirm that patient histories on snoring frequency or remarkable breathing pauses cannot be reliably used to distinguish children with OSA from children who snore without OSA.⁸ However, it seems to be beneficial to target diagnostics of OSA to those children whose parents report they snore.²¹

We observed that at the age of 2 years adenoid size and mouth breathing seemed to be important factors when assessing the likelihood of OSA, but palatine tonsil size was not. It has previously been reported that adenoid size seemed to influence the probability of OSA in younger children.^{12,20} However, palatine tonsil size has not been seen as a significant factor. This raises the question about whether an adenoidectomy without tonsillectomy might be a sufficient procedure for young paediatric patients with OSA. Having an adenoidectomy on its own is not a well-researched procedure for treating paediatric OSA.⁶ However, there is a evidence that it could be enough for young patients.²² Moreover, adenoidectomy has been shown to have fewer complications than adenotonsillectomy.²³ Conversely, it has been suggested that between 2% and 29% of children who have undergone an adenoidectomy because of airway obstruction will need a tonsillectomy in the future.²⁴ Further research is needed on adenoidectomy alone as a treatment for paediatric OSA in young children. Perhaps, an adenoidectomy on its own, or combined with later myofunctional therapy, could be used to treat young children with OSA. Furthermore, myofunctional therapy has already been shown to reduce residual symptoms and mouth breathing after an adenotonsillectomy.^{25,26}

TABLE 4The anamnestic informationreported at the time of the clinicalexaminations between the OSA and noOSA groups

Anamnestic information reported by parents	OSA group n = 9 (%)	no OSA- group n = 22 (%)	P values
Breathing difficulties during sleep			
No	9 (100)	17 (77)	.287
Yes	0 (0)	4 (18)	
Missing answers	O (O)	1 (5)	
Snoring frequency			
Maximum of two nights per week	1 (11)	10 (45)	.110
Minimum of three nights per week	7 (78)	11 (50)	
Missing answers	1 (11)	1 (5)	
Frequency of daytime sleepiness			
Maximum of two days per week	9 (100)	15 (68)	.141
Minimum of three days per week	0 (0)	6 (27)	
Missing answers	0 (0)	1 (5)	
Breathing habit			
Mainly through nose	2 (22)	15 (68)	.015
Mainly through mouth	4 (44)	1 (5)	
Don't know	3 (33)	6 (27)	
Missing answers	0 (0)	0 (0)	
Nasal rhinitis or stuffiness			
No	7 (78)	18 (82)	1.000
Yes	2 (22)	4 (18)	
Missing answers	0 (0)	0 (0)	
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Note: P values < .05 bolded.

TABLE 5 The clinical findings betweenthe OSA and no OSA groups

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	OSA group n = 9	no OSA group n = 22	P values
Sex			
Male	5 (56%)	8 (36%)	.433
Female	4 (44%)	14 (64%)	
Age (months, median, IQR 0.25-0.75)	27.5 (25.5 -29.5)	25.0 (23.0 -27.0)	.108
Body mass index (kg/m ² , median, IQR 0.25-0.75)	15.8 (15.4 - 16.4) Missing 1	15.7 (15.4 - 17.0) Missing 3	.969
Body mass index z-score (median, IQR 0.25-0.75)	-0.5 (-1.00.2) Missing 1	-0.6 (-1.0 -0.4) Missing 3	1.000
Mouth breathing			
No	3 (33%)	19 (86%)	.007
Yes	6 (66%)	3 (14%)	
Palatine tonsil size(Friedman o	lass)		
1	2 (22%)	6 (27%)	.531
2	3 (33%)	11 (50%)	
3	4 (44%)	5 (23%)	
4	0 (0%)	0 (0%)	
Adenoid size			
0%-25%	2 (22%)	13 (59%)	.008
26%-50%	3 (33%)	9 (41%)	
51%-75%	2 (22%)	0 (0%)	
76%-100%	2 (22%)	0 (0%)	
Secretion at nasal mucosa			
No	4 (44%)	5 (23%)	.385
Yes	5 (56%)	17 (77%)	
Swelling at nasal mucosa			
No or slight	9 (100%)	19 (86%)	.537
Moderate or severe	0 (0%)	3 (14%)	

Note: P values <.05 bolded.

4.1 | Strengths and limitations

The strength of the present study was that it focused on a homogeneous group of two-year-old children. In previous studies, the study populations have been heterogeneous with regard to the age of the children and few studies have focused on toddlers. We acknowledge the limitations of our study, including the low number of patients. A higher number could have strengthened our results. The prevalence of snoring was generally lower in our cohort, at 2.5%-3.2%, than previous studies of infants and toddlers, where the prevalence of snoring a minimum of three nights per week ranged from 5.3%-11%.^{3,4} In addition, the study protocol was timeconsuming for the families, and this reduced the number of families willing to participate in the study. There were only nine children with OSA and the severity of their OSA was generally mild, with obstructive AHI of 1.2-6.3. Despite this mild severity, we were still able to find detectable changes between the children with OSA and the children who snored without OSA. This strengthened the

presumption that features that help assess the likelihood of OSA can be detected.

Three children had previously undergone an adenoidectomy and one had an adenoidectomy and partial tonsillectomy, which could be interpreted as a confounder. However, these children were not excluded because the regrowth of adenoid tissue after surgery is possible and the status of the tissue was evaluated during otorhinolaryngological examinations. Nevertheless, all the statistical analyses between the OSA and no OSA groups were repeated without the children who had received surgery and the significance of the results did not change when P < .05 was statistically significant.

5 | CONCLUSION

This study had two main findings that helped to assess the likelihood of OSA in toddlers. First, snoring time from total sleep time was longer in children with OSA than children who snored but did ACTA PÆDIATRICA

not have OSA. In future, it is possible that snoring time could be used as a diagnostic tool when distinguishing children who snore with and without OSA. Second, children with OSA breathed through their mouth more often and had bigger adenoid tissues than children who snored without OSA. In this age group, the size of the palatine tonsil did not seem to have a significant influence.

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CONFLICTS OF INTEREST

The authors have no conflicts of interest to declare.

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REFERENCES

- Marcus CL, Brooks LJ, Ward SD, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics*. 2012;130:e714-e755.
- Lumeng JC, Chervin RD. Epidemiology of pediatric obstructive sleep apnea. Proc Am Thorac Soc. 2008;5:242-252.
- Montgomery-Downs HE, Gozal D. Sleep habits and risk factors for sleep-disordered breathing in infants and young toddlers in Louisville, Kentucky. *Sleep Med.* 2006;7:211-219.
- Li AM, Sadeh A, Au CT, Goh DYT, Mindell JA. Prevalence of habitual snoring and its correlates in young children across the Asia Pacific. J Paediatr Child Health. 2013;49:153-159.
- Marcus CL, Moore RH, Rosen CL, et al. A randomized trial of adenotonsillectomy for childhood sleep apnea for the childhood adenotonsillectomy trial (CHAT). N Engl J Med. 2013;368:2366-2376.
- Kaditis AG, Alonso Alvarez ML, Boudewyns AN, et al. Obstructive sleep disordered breathing in 2- to 18-year-old children: Diagnosis and management. *Eur Respir J.* 2016;47:69-94.
- Gozal D, Kheirandish-Gozal L. New approaches to the diagnosis of sleep-disordered breathing in children. Sleep Med. 2010;11:708-713.
- Brietzke SE, Katz ES, Roberson DW. Can history and physical examination reliably diagnose pediatric obstructive sleep apnea/hypopnea syndrome? A systematic review of the literature. *Otolaryngol Head Neck Surg.* 2004;131:827-832.
- Certal V, Catumbela E, Winck JC, Azevedo I, Teixeira-Pinto A, Costa-Pereira A. Clinical assessment of pediatric obstructive sleep apnea: a systematic review and meta-analysis. *Laryngoscope*. 2012;122:2105-2114.
- Luo R, Schaughency E, Gill AI, Dawes PJD, Galland BC. Natural history of snoring and other sleep-disordered breathing (SDB) symptoms in 7-year-old New Zealand children: a follow-up from age 3. *Sleep Breath.* 2015;19:977-985.
- 11. Chervin RD, Ellenberg SS, Hou X, et al. Prognosis for spontaneous resolution of OSA in children. *Chest*. 2015;148:1204-1213.

- Valera FCP, Avelino MAG, Pettermann MB, et al. OSAS in children: correlation between endoscopic and polysomnographic findings. Otolaryngol - Head Neck Surg. 2005;132:268-272.
- Paavonen JE, Saarenpää-Heikkilä O, Pölkki P, Kylliäinen A, Porkka-Heiskanen T, Paunio T. Maternal and paternal sleep during pregnancy in the Child-sleep birth cohort. *Sleep Med.* 2017;29:47-56.
- 14. Bruni O, Ottaviano S, Guidetti V, et al. The sleep disturbance scale for children (SDSC) construct ion and validation of an instrument to evaluate sleep disturbances in childhood and adolescence. *J Sleep Res.* 1996;5:251-261.
- Kaditis AG, Alonso Alvarez ML, Boudewyns AN, et al. ERS statement on obstructive sleep disordered breathing in 1- to 23-monthold children. *Eur Respir J.* 2017;50:1700985.
- Kindermann CA, Roithmann R, Neto JFL. Sensitivity and specificity of nasal flexible fiberoptic endoscopy in the diagnosis of adenoid hypertrophy in children. Int J Pediatr Otorhinolaryngol. 2008;72:63-67.
- 17. Friedman M, Tanyeri H, La Rosa M, et al. Clinical predictors of obstructive sleep apnea. *Laryngoscope*. 1999;109:1901-1907.
- Berry RB, Budhiraja R, Gottlieb DJ, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events. J Clin Sleep Med. 2012;8:597-619.
- American Academy of Sleep Medicine. In: Sateia MJ, ed. International Classification of Sleep Disorders 3rd ed, Darien, IL: American Academy of Sleep Medicine; 2014:63.
- Kang K-T, Weng W-C, Lee C-H, Hsiao T-Y, Lee P-L, Hsu W-C. Clinical risk assessment model for pediatric obstructive sleep apnea. *Laryngoscope*. 2016;126:2403-2409.
- Nieminen P, Tolonen U, Löppönen H. Snoring and obstructive sleep apnea in children. Arch Otolaryngol Head Neck Surg. 2000;126:481-486.
- Reckley LK, Song SA, Chang ET, Cable BB, Certal V, Camacho M. Adenoidectomy can improve obstructive sleep apnoea in young children: systematic review and meta-analysis. J Laryngol Otol. 2016;130:990-994.
- Gallagher TQ, Wilcox L, McGuire E, Derkay CS. Analyzing factors associated with major complications after adenotonsillectomy in 4776 patients: Comparing three tonsillectomy techniques. Otolaryngol - Head Neck Surg. 2010;142:886-892.
- 24. Black AP, Shott SR. Is adenoidectomy alone sufficient for the treatment of airway obstruction in children? *Laryngoscope*. 2014;124:6-7.
- 25. Villa MP, Evangelisti M, Martella S, Barreto M, Del Pozzo M. Can myofunctional therapy increase tongue tone and reduce symptoms in children with sleep-disordered breathing? *Sleep Breath*. 2017;21:1025-1032.
- Guilleminault C, Huang YS, Monteyrol PJ, Sato R, Quo S, Lin CH. Critical role of myofascial reeducation in pediatric sleep-disordered breathing. *Sleep Med.* 2013;14:518-525.

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