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# Prevalence of adenoid hypertrophy among 12-year-old children and its association with craniofacial characteristics: a cross-sectional study

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## Abstract

**Background** Identifying the prevalence of adenoid hypertrophy (AH) and craniofacial factors associated with this condition requires studies with random sampling from the general population, and multiple criteria can be used for assessing AH on lateral cephalometric radiograph (LCR). The present analysis represents the first report performed according to these requirements in a large cross-sectional sample of children.

**Methods** LCRs of 517 12-year-old children (286 males, 231 females) randomly selected from the general population were retrospectively retrieved. AH was defined using three criteria (At/Nd, Ad-Ba/PNS-Ba, 1-Npaa/Npa), and twelve craniofacial variables were measured (SNA, SNB, ANB, Wits, Cd-Gn, MnP^SN, MxP^MnP, TPFH/TAFH, OPT^SN, C2ps-C4pi^SN, H-CV, H-FH). Skeletal characteristics were compared between children with and without AH using Mann–Whitney *U* test. Binary logistic regression (adjusted for sex and skeletal growth) was used to independently quantify the association between craniofacial factors and AH.

**Results** The prevalence of children with AH was 17.6% (according to At/Nd), 19.0% (according to Ad-Ba/PNS-Ba), and 13.9% (according to 1-Npaa/Npa). Children with AH presented greater antero-posterior jaw discrepancy (larger ANB, smaller SNB), greater mandibular divergence (larger MnP^SN), forward head posture (larger OPT^SN and C2ps-C4pi^SN), and anteriorly positioned hyoid bone (larger H-CV). Larger SNA (OR = 1.39–1.48), while smaller SNB (OR = 0.77–0.88) and Wits (OR = 0.85–0.87), were associated with greater likelihood of having AH, independently from the assessment method used.

**Conclusions** The prevalence of children with AH ranged from 13.9 to 19.0% based on LCR. Greater antero-posterior maxillo-mandibular discrepancy and mandibular retrusion were independently associated with higher likelihood of having AH.

**Keywords** Adenoids, Lateral cephalometry, Upper airway, Children

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## Background

Adenoids are part of the lymphatic Waldeyer's ring that is located in the superior nasopharynx. They grow after birth until the age of 5–7 years and shrink progressively thereafter [1, 2]. Adenoid hypertrophy (AH) can be physiological, secondary to infections, or a reaction to allergens [3] and, with a prevalence of about 34%, it is a common cause of upper airway obstruction in children [4]. AH is associated with increased nasal resistance to airflow and may lead to mouth-breathing [5], which may negatively impact the quality of life of children [6].

While nasopharyngeal endoscopy is the gold standard for diagnosing AH [7], lateral cephalometric radiograph (LCR) is a useful alternative tool to identify this condition because of its good diagnostic accuracy [7] and reliability [8]. Among numerous methods for assessing AH on LCR, the adenoid/nasopharyngeal ratio (At/Nd) [9], adenoid/retropalatal ratio (Ad-Ba/PNS-Ba) [10], and adenoid/nasopharyngeal area ratio (1-Npaa/Npa) [2] are based on relative measurements that account for individual size variations, leading to improved validity [11]. However, there is no consensus about which should be preferred, as authors reported inconsistent findings regarding the usefulness of both linear [12, 13] and area measurements [12, 14], and clinicians should consider the combination of multiple methods for assessing AH on LCR [15].

Since LCR also allows to assess craniofacial structures [16], it has been previously used for investigating the association between skeletal morphology and AH. However, conflicting results have been reported when children with AH were compared to controls [17, 18]. In fact, previous studies involved heterogeneous age groups that did not account for physiological changes in adenoids size [17, 18], and children were sampled from hospital departments, making them not representative of the general population [17, 18]. Overall, investigating the prevalence of AH and its associations with craniofacial structures requires studies on large samples selected via stratified random sampling from the general population. In addition, a cross-sectional design can reduce the bias related to physiological lymphoid tissue shrinkage [1, 2] and skeletal growth [19]. Twelve-year-old children represent a meaningful target population, as such age corresponds to an appropriate timing for the assessment of orthodontic treatment needs. Especially, given that AH may predispose children to sleep-disordered breathing [1], screening for this condition is advisable during treatment planning [20].

The objective of the present study was to estimate the prevalence of children with AH based on LCR, and to investigate the association between AH and craniofacial characteristics among 12-year-old children. It was

hypothesised that children with increased antero-posterior jaw discrepancy, increased vertical facial height, forward craniocervical posture, and forward hyoid bone position had greater likelihood of having AH.

## Methods

### Participants

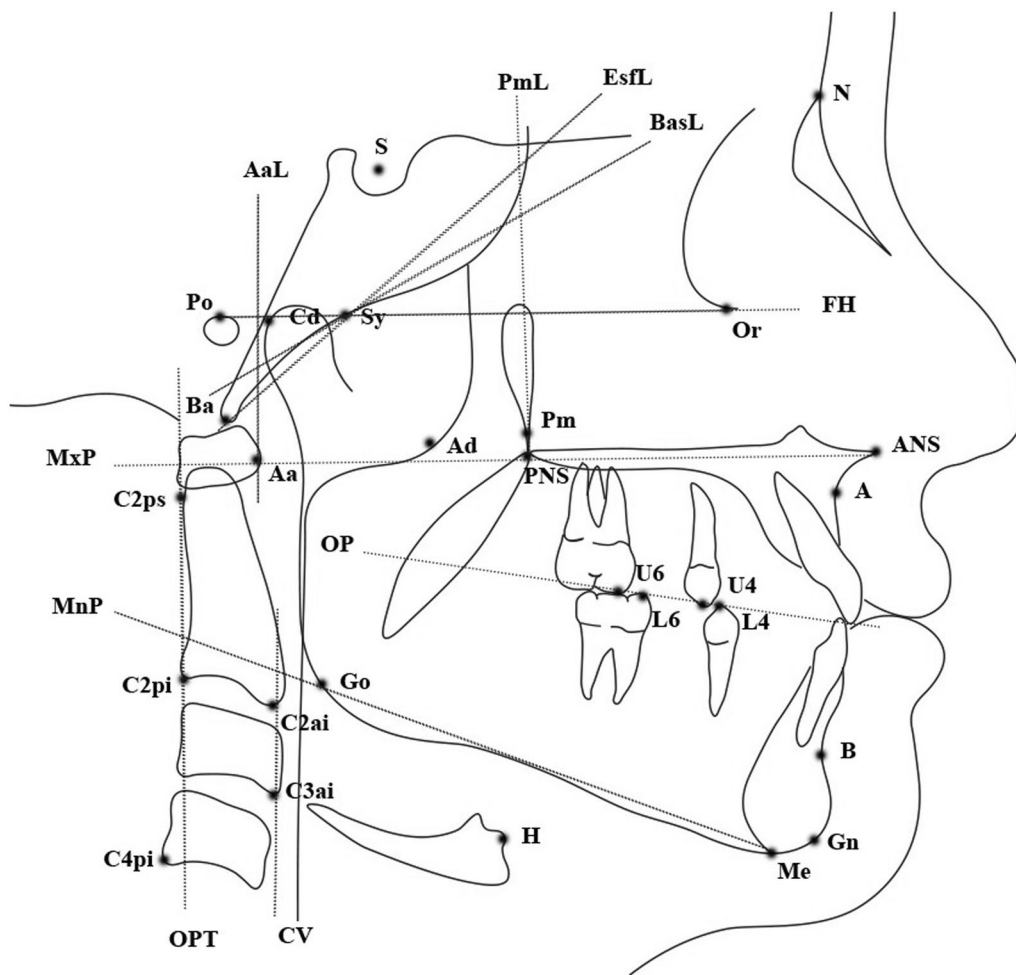
LCRs of 517 children were retrieved from a previous cross-sectional study that performed stratified sampling on 11 randomly selected schools in Hong Kong in the 1980s [21] and referred participants to Prince Philip Dental Hospital (Hong Kong SAR) for taking the X-ray. 12-year-old children, who had not received orthodontic treatment, and whose LCR was taken with similar methods were included. The study was approved by the Institutional Review Board of The University of Hong Kong / Hospital Authority Hong Kong West Cluster (UW12-405).

### Lateral cephalograms acquisition and analysis

One X-ray machine (GE1000, General Electric, Milwaukee, WI, USA) was used to obtain all LCRs, which were acquired in natural head posture [21]. Cephalometric analysis was carried out with computer software (CASOS, SoftEnable Technology, Hong Kong SAR), while the nasopharyngeal areas were measured with graphical software (Image) [22]. Linear and area measurements were adjusted according to a magnification of 8.75% and 18.27%, respectively. Cephalometric points and lines were identified (Fig. 1 and Table 1), and variables were measured (Table 2). AH was defined according to three parameters: the adenoid/nasopharyngeal ratio (At/Nd) [9], adenoid/retropalatal ratio (Ad-Ba/PNS-Ba) [10], and adenoid/nasopharyngeal area ratio (1-Npaa/Npa) [2] (Fig. 2). Children were classified as either having AH or not having AH according to cut-off values of 0.62 for At/Nd (>0.62 indicating AH) [9], 0.60 for Ad-Ba/PNS-Ba (>0.60 indicating AH) [10], and 0.35 for 1-Npaa/Npa (<0.35 indicating AH) [2]. Skeletal maturity was assessed using the cervical vertebral maturation (CVM) method and growth stages were defined as pre-pubertal (CS1-CS2), pubertal (CS3-CS4), and post-pubertal (CS5-CS6) [19].

### Calibration and method error

The primary assessor (KLT) carried out all measurements after calibration with a secondary assessor (GM). The calibration consisted of tracing and discussing 10 LCR and repeating the procedure until an intraclass correlation coefficient (ICC) > 0.75 was achieved. For intra-assessor reliability, 20% of the LCRs ( $n=104$ ) were re-measured by the primary assessor after a wash-out period of about 2 weeks.



**Fig. 1** Cephalometric landmarks and lines

### Sample size calculation

The sample size was calculated based on a binary logistic regression model with the presence of AH as dependent variable and 14 independent variables (2 covariates to adjust the model for confounding effects, and 12 factors to investigate their effect on the dependent variable). According to a reported prevalence of AH of 31.76% in children and adolescents [17], and requiring at least 10 subjects for each independent variable [23], the sample size was calculated as  $N = (14 \times 10) / 0.3176 = 441$ . Given the retrospective nature of the study, all the 517 records fulfilling the inclusion criteria were included.

### Data analysis

The measurement error was calculated with Dahlberg's formula [24]. ICC was used to calculate the intra-assessor agreement of continuous variables (<0.50 "poor", 0.50–0.74 "moderate", 0.75–0.90 "good", >0.90 "excellent" [25]). Cohen's kappa ( $K$ ) was used to calculate the

intra-assessor agreement of growth stages and the agreement among the three assessment methods of AH ( $\leq 0.20$  "poor", 0.21–0.40 "slight", 0.41–0.60 "fair", 0.61–0.80 "good", 0.81–0.92 "very good",  $\geq 0.93$  "excellent" [26]). Chi-square test was used to determine variations in the distribution of children by sex and growth stages with respect to the presence of AH, independently for each of the three diagnostic methods. Normal distribution of continuous data was assessed using Shapiro–Wilk test, and Mann–Whitney  $U$  test was used to compare the craniofacial variables between children with and without AH, independently for each of the three diagnostic methods. Binary logistic regression (with covariates adjustment for growth stage and sex) was used to assess the independent contribution of each craniofacial factor to the presence of AH. Three binary logistic regression models, one for each diagnostic method, were developed. Statistical analysis was performed using SPSS 27.0 (IBM, New York, USA), at significance  $\alpha = 0.05$ .

**Table 1** Cephalometric landmarks and lines used to identify craniofacial structures and adenoids

Abbreviation	Name	Description
<i>Cephalometric points</i>		
A	A-point	The deepest point of the concavity of the anterior maxilla between ANS and the alveolar crest
Aa	Anterior atlas	The most anterior point of the atlas
Ad	Adenoids	The intersection between PNS-Ba and the anterior border of the adenoids
H	Hyoid	The most anterior–superior point on the body of the hyoid bone
ANS	Anterior nasal spine	The anterior tip of the bony apex of the maxillary complex
B	B-point	The deepest point in the concavity of the anterior mandible between the alveolar crest and the bony chin
Ba	Basion	The most inferior point of the anterior border of the foramen magnum
C2ai	2nd cervical vertebra A	The most anterior–inferior point of the second cervical vertebra
C2pi	2nd cervical vertebra B	The most posterior–inferior point of the second cervical vertebra
C2ps	2nd cervical vertebra C	The most posterior–superior point of the second cervical vertebra
C3ai	3rd cervical vertebra	The most anterior–inferior point of the third cervical vertebra
C4pi	4th cervical vertebra	The most posterior–inferior point of the fourth cervical vertebra
Cd	Condylion	The most posterior–superior point on the mandibular condyle
Gn	Gnathion	The most anterior–inferior point on the bony chin
Go	Gonion	The intersection of the tangents to the inferior and posterior borders of the mandible
U4	Maxillary first premolar cusp	The cusp tip of the maxillary first premolar
L4	Mandibular first premolar cusp	The cusp tip of the mandibular first premolar
U6	Maxillary first molar cusp	The mesial cusp tip of the maxillary first permanent molar
L6	Mandibular first molar cusp	The mesial cusp tip of the mandibular first permanent molar
Me	Menton	The most inferior point on the body chin
N	Nasion	The most anterior point of the fronto-nasal suture
Sy	Spheno-occipital synchondrosis	The anterior border of the spheno-occipital synchondrosis
Or	Orbitale	The most inferior point of the inferior borders of the bony orbit
Pm	Pterygo-maxillare	The most inferior point of the pterygomaxillary fissure
PNS	Posterior nasal spine	The posterior tip of the bony apex of the maxillary complex
Po	Porion	The most superior point of the external auditory meatus
S	Sella	The geometric centre of the sella turcica
<i>Cephalometric lines</i>		
CV	Cervical vertebrae	The line joining C2ai and C3ai
AaL	Anterior atlas line	The line perpendicular to MxP and passing through Aa
Esfl	Sphenoid line	The line tangent to the inferior border of the sphenoid bone and passing through Ba
FH	Frankfort horizontal plane	The line joining Or and Po
MnP	Mandibular plane	The line joining Me and Go
MxP	Maxillary plane	The line joining ANS and PNS
OP	Occlusal plane	The line joining the midpoint between U4 and L4 with the midpoint between U6 and L6
OPT	Odontoid process tangent	The line joining C2ps and C2pi
PmL	Pterygomaxillary line	The line perpendicular to MxP and passing through Pm
BasL	Basioccipital line	The line representing the anterior margin of the basioccipital bone

All bilateral points were marked by choosing the average between the left and right side

## Results

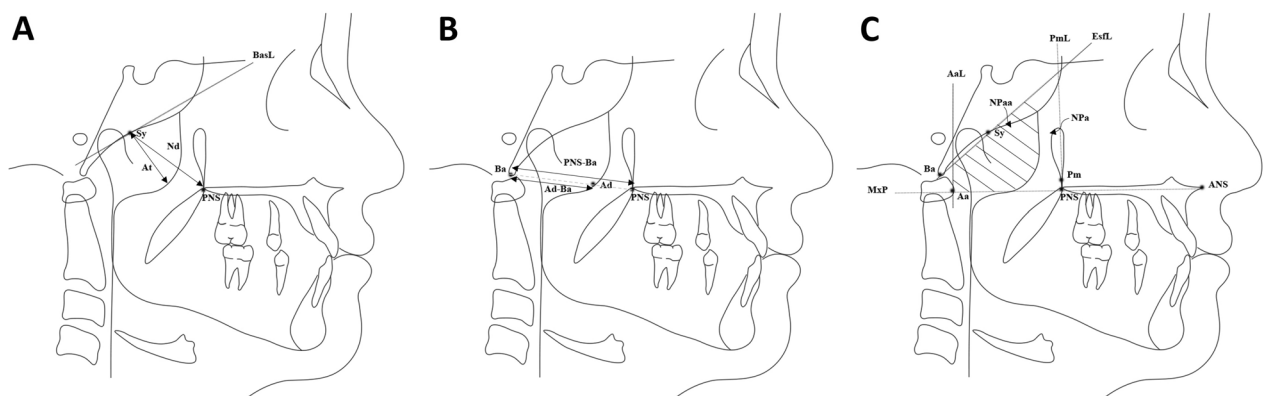
### Sample characteristics

A total of 517 children, 286 males (55.3%) and 231 females (44.7%), were included. Regarding skeletal

growth, 121 males (92.4%) and 10 females (7.6%) were in pre-pubertal stage, 157 males (55.7%) and 125 females (44.3%) were in pubertal stage, while 8 males (7.7%) and 96 females (92.3%) were in post-pubertal stage.

**Table 2** Cephalometric variables used for craniofacial structures and adenoids

Abbreviation	Name	Description
<i>Measured variables (for calculation of variables included in the analysis)</i>		
At	mm	Nasopharyngeal soft tissue thicknesses of adenoid
Ad-Ba	mm	Retropalatal soft tissue thickness of adenoid
Nd	mm	Nasopharyngeal depth
Npa	mm <sup>2</sup>	Nasopharyngeal area
Npaa	mm <sup>2</sup>	Nasopharyngeal adenoid area
PNS-Ad	mm	Airway sagittal depth
TAFH	mm	Total anterior facial height
TPFH	mm	Total posterior facial height
<i>Measured variables (directly included in the analysis)</i>		
ANB	°	ANB angle
C2ps-C4pi^SN	°	Total craniocervical angle
Cd-Gn	mm	Mandibular length
H-CV	mm	Horizontal position of hyoid bone
H-FH	mm	Vertical position of hyoid bone
MnP^SN	°	Mandibular plane angle
MxP^MnP	°	Maxillo-mandibular planes angle
OPT^SN	°	Upper craniocervical angle
SNA	°	SNA angle
SNB	°	SNB angle
Wits	mm	Wits appraisal
<i>Calculated variables (included in the analysis)</i>		
At/Nd	%	Adenoid/nasopharyngeal ratio
Ad-Ba/PNS-Ba	%	Adenoid/retropalatal ratio
1-Npaa/Npa	%	Adenoid/nasopharyngeal area ratio
TPFH/TAFH	%	Jarabak ratio



**Fig. 2** Cephalometric landmarks and measurements used to identify AH according to the three methods: At/Nd (A), Ad-Ba/PNS-Ba (B), and 1-Npaa/Npa (C)

**Measurements error and agreement**

The measurement error was <1.0° for angular measurements, <1.0 mm for linear measurements, and <2.0% for ratios. Regarding the independent variables, the

intra-assessor agreement was very good for skeletal growth ( $K=0.823$ ), and very good to excellent for craniofacial structures ( $ICC=0.908-0.990$ ). Regarding the methods used for identifying children with AH, the

intra-assessor agreement of each method was excellent ( $ICC=0.956-0.999$ ), and the agreement among the methods was very good between At/Nd and Ad-Ba/PNS-Ba ( $K=0.877$ ), very good between At/Nd and 1-Npaa/Npa ( $K=0.818$ ), and good between Ad-Ba/PNS-Ba and 1-Npaa/Npa ( $K=0.790$ ) (Appendix Tables 6, 7).

**Prevalence of adenoid hypertrophy**

Ninety-one children (17.6%, 49 males and 42 females) were classified as having AH according to At/Nd, 98 (19.0%, 52 males and 46 females) according to Ad-Ba/PNS-Ba, and 72 (13.9%, 35 males and 37 females) based on 1-Npaa/Npa. There was no difference in sex distribution or skeletal growth stage between children with and without AH, independently from the assessment method used (Table 3).

**Association between adenoid hypertrophy and craniofacial characteristics**

Regarding horizontal measurements, ANB of children with AH was larger (for all methods) and SNB was smaller (for At/Nd and 1-Npaa/Npa). The logistic regression showed that ANB, SNB, and Wits were significant predictors in all models (for each degree of increase of ANB, children were 38.8–47.5% more likely of having AH; for each degree of decrease in SNB, they were 13.6–29.3% more likely to have AH; and for each millimetre of decrease in Wits, they were 15.6–18.0% more likely of having AH). Larger Cd-Gn was also significant (for At/Nd and 1-Npaa/Npa). For vertical measurements, MnP^SN was larger in children with AH (for At/Nd). However, no vertical parameter was significant in the logistic regression. Regarding craniocervical posture, both OPT^SN (for At/Nd) and C2ps-C4pi^SN (for At/

Nd and Ad-Ba/PNS-Ba) were larger in children with AH. However, no craniocervical parameter was significant in the logistic regression. For the hyoid bone position, H-CV was larger among children with AH (for At/Nd). The logistic regression showed that children with more anteriorly positioned hyoid bone were more likely of having AH (for At/Nd) (Tables 4, 5).

**Discussion**

Adenoids have been assessed on LCR since 1946 [27], and numerous parameters have been proposed to identify children with AH. The present study adopted three methods, with the first based on the ratio between the maximum thicknesses of the adenoids (measured from the basilar part of the occipital bone) and the width of the nasopharynx (measured along the line connecting the posterior nasal spine with the spheno-occipital synchondrosis). This parameter, named At/Nd, was first described by Fujioka et al. [9]. An advantage of using ratio measurements is that the adenoidal size is considered with respect to the individual nasopharyngeal capacity. In children, At/Nd has excellent specificity (95%) and positive predictive value (94%), but low sensitivity (41%) and negative predictive value (39%) [28]. At/Nd has also been shown to correlate well with clinical symptoms and weight of surgically removed adenoids [29]. The second parameter consisted of the ratio between the thickness of the adenoids and the width of the nasopharynx (both measured along the line connecting the posterior nasal spine with Basion). This ratio, named Ad-Ba/PNS-Ba and proposed by Kemaloglu et al. [10], is similar to At/Nd but measured along a different line that is more representative of the retropalatal area. Among children, it has excellent specificity (97%) and positive predictive

**Table 3** Distribution of sex and growth stages based on adenoid hypertrophy identified with three different methods

	At/Nd		Ad-Ba/PNS-Ba		p value	1-Npaa/Npa		p value	n	%	n	%	p value	n	%	p value
	Non-hypertrophic	Hypertrophic	Non-hypertrophic	Hypertrophic		Non-hypertrophic	Hypertrophic									
n	426	91	419	98		445	72									
%	82.4	17.6	81.0	19.0		86.1	13.9									
	n	%	n	%	p value	n	%	n	%	p value	n	%	n	%	p value	
Sex																
Male	237	55.6	49	53.8	0.816	234	55.8	52	53.1	0.652	251	56.4	35	48.6	0.250	
Female	189	44.4	42	46.2		185	44.2	46	46.9		194	43.6	37	51.4		
Growth stage																
Pre-pubertal	108	25.4	23	25.3	0.995	108	25.8	23	23.5	0.840	115	25.8	16	22.2	0.806	
Pubertal	232	54.5	50	54.9		226	53.9	56	57.1		241	54.2	41	56.9		
Post-pubertal	86	20.2	18	19.8		85	20.3	19	19.4		89	20.0	15	20.8		

Chi-square test was used to compare either sex or growth stage distribution within non-hypertrophic and hypertrophic patients

**Table 4** Comparison of craniofacial characteristics between children with and without adenoid hypertrophy, for each of the three assessment methods

Variable	Unit	Ad-Ba/PNS-Ba						1-Npaa/Npa																	
		Non-hypertrophic			Hypertrophic			Non-hypertrophic			Hypertrophic														
		Median	LQ	UQ	Median	LQ	UQ	Median	LQ	UQ	Median	LQ	UQ												
		n = 426	n = 91	n = 419	n = 98	n = 445	n = 72	p value	Δ	p value	Δ	p value	Δ												
<i>Horizontal</i>																									
SNA	°	85.5	82.6	88.3	86.0	82.8	88.2	0.5	0.959	85.5	82.5	88.1	86.1	82.9	88.6	0.5	0.336	85.5	82.8	88.3	85.1	82.6	88.1	-0.4	0.506
SNB	°	81.3	78.9	83.9	80.1	77.2	82.5	-1.2	<b>0.005</b>	81.2	78.8	83.7	80.4	77.4	83.6	-0.9	0.132	81.2	78.8	83.9	79.9	77.3	82.5	-1.4	<b>0.008</b>
ANB	°	4.2	2.9	5.6	5.8	3.7	7.1	1.6	< <b>0.001</b>	4.2	2.9	5.6	5.8	3.6	6.7	1.6	< <b>0.001</b>	4.2	2.9	5.7	5.8	3.7	6.6	1.6	<b>0.001</b>
Wits	mm	-3.8	-6.4	-1.1	-3.1	-6.2	-0.6	0.7	0.337	-3.8	-6.3	-1.0	-3.4	-7.0	-0.7	0.5	0.774	-3.8	-6.4	-1.0	-3.4	-6.2	-0.8	0.5	0.624
Cd-Gn	mm	110.6	107.2	115.1	112.0	108.8	115.3	1.5	0.104	110.7	107.2	115.1	111.6	108.3	115.3	0.9	0.176	110.7	107.2	115.1	111.6	108.7	115.3	0.9	0.201
<i>Vertical</i>																									
MnPa^SN	°	33.8	30.3	36.5	35.0	30.4	39.5	1.2	<b>0.030</b>	33.9	30.3	36.6	34.3	30.4	39.2	0.4	0.131	33.9	30.2	36.8	34.8	30.8	39.1	0.9	0.083
MxPa^MnP	°	26.5	23.4	29.1	27.8	23.6	31.2	1.3	0.052	26.5	23.3	29.1	27.6	23.8	31.1	1.1	0.054	26.6	23.3	29.3	27.9	24.0	31.1	1.3	0.070
TPFH/TA FH %	%	0.7	0.7	0.7	0.7	0.6	0.7	0.0	0.097	0.7	0.7	0.7	0.7	0.6	0.7	0.0	0.191	0.7	0.7	0.7	0.7	0.6	0.7	0.0	0.124
<i>Cranio-cervical</i>																									
OPT^SN	°	97.1	91.6	101.8	99.4	93.0	105.1	2.4	<b>0.014</b>	97.2	91.7	101.9	98.7	93.0	105.1	1.5	0.084	97.2	91.8	102.1	99.2	93.0	105.1	2.0	0.097
C2ps^-	°	99.5	94.5	104.2	102.1	95.4	108.5	2.6	<b>0.008</b>	99.5	94.5	104.2	101.4	94.7	107.5	1.8	<b>0.042</b>	99.7	94.5	104.2	101.9	94.8	107.7	2.1	0.052
<i>Hyoid bone</i>																									
H-CV	mm	32.4	30.1	35.3	33.9	30.4	36.0	1.5	<b>0.022</b>	32.4	30.1	35.3	33.6	30.4	36.1	1.2	0.058	32.4	30.3	35.3	33.2	30.2	35.9	0.8	0.234
H-FH	mm	81.8	76.6	85.9	82.8	77.2	89.5	1.0	0.072	81.9	76.8	86.0	82.1	76.8	89.3	0.2	0.255	81.9	76.9	86.2	81.7	75.3	89.5	-0.3	0.577

LQ lower quartile (25th percentile), UQ upper quartile (75th percentile)

Significant p values are reported in bold

Mann-Whitney U test was used to compare non-hypertrophic and hypertrophic patients

Δ = difference between hypertrophic and non-hypertrophic patients

**Table 5** Binary logistic regression models showing the relationship between the dependent factors and adenoid hypertrophy, for each of the three assessment methods

		At/Nd			Ad-Ba/PNS-Ba			1-Npaa/Npa		
		AOR	p value	Pseudo R <sup>2</sup>	AOR	p value	Pseudo R <sup>2</sup>	AOR	p value	Pseudo R <sup>2</sup>
<i>Covariates adjustments</i>										
		0.148			0.103			0.121		
Sex	Male	1.000	0.303		1.000	0.445		1.000	0.071	
	Female	1.397			1.263			1.871		
Growth stage	Pre-pubertal	1.000	0.921		1.000	0.727		1.000	0.788	
	Pubertal	1.002			0.906			1.074		
	Postpubertal	1.108			1.139			1.247		
<i>Craniofacial factors</i>										
SNB		0.818	<b>0.003</b>		0.880	<b>0.044</b>		0.773	<b>&lt; 0.001</b>	
ANB		1.475	<b>&lt; 0.001</b>		1.404	<b>&lt; 0.001</b>		1.388	<b>&lt; 0.001</b>	
Wits		0.853	<b>0.003</b>		0.865	<b>0.005</b>		0.847	<b>0.005</b>	
Cd-Gn		1.075	<b>0.014</b>		1.052	0.064		1.083	<b>0.011</b>	
MnP^SN		0.943	0.532		0.897	0.226		0.869	0.171	
MxP^MnP		1.025	0.729		1.083	0.249		1.124	0.142	
TPFH/TAFH		7.119	0.779		0.372	0.882		12.907	0.737	
OPT^SN		0.947	0.312		0.927	0.141		0.917	0.137	
C2ps-C4pi^SN		1.064	0.259		1.093	0.089		1.083	0.179	
H-CV		1.073	<b>0.042</b>		1.058	0.094		1.064	0.093	
H-FH		0.980	0.349		0.972	0.183		0.965	0.139	

AOR adjusted odd ratio, R<sup>2</sup> coefficient of determination

Significant p values are reported in bold

SNA was removed from the model because of redundancy with SNB and ANB (and it showed no statistical significance in the group comparison)

value (97%), but moderate sensitivity (71%) and negative predictive value (70%) [11]. The third parameter represented the proportion of the nasopharyngeal area that is not occupied by the adenoids (1-Npaa/Npa), as proposed by Handelman and Osborne [2]. It has excellent specificity (94%) and positive predictive value (95%), but moderate sensitivity (75%) and negative predictive value (72%) [11]. In general, the present study showed a prevalence of children with AH between 13.9% and 19.0%, with good agreement among the three measurements methods. Of note, the calculated prevalence was lower than the 34.46% reported by a previous meta-analysis of studies using nasoendoscopy in children [4]. The reason for this difference could be due to the lower sensitivity of LCR compared to nasoendoscopy, with a reported pooled sensitivity of 86% in children [7]. In particular, the only two identified studies using nasoendoscopy in randomly selected samples included children between 5- and 14-year-old [4], further justifying differences between previously reported values and the present cross-sectional study among 12-year-old children. No difference

was present in the prevalence of AH between males and females, in agreement with a former investigation [30].

Hypertrophic adenoids may obstruct the nasopharynx, forcing the child to breathe through the mouth, which may lead to the characteristic “adenoid facies” [31]. However, the relationship between AH and craniofacial morphology should be considered with caution, as patients may not show the expected “mouth-breathing dental stereotype” [18], and it is time for scientific studies to critically analyse confounders and statistical findings. In fact, multiple genetic and epigenetic factors may affect craniofacial growth [32]. In this complex scenario, despite the comparison of the median values of a variable (e.g. SNA) between two groups (e.g. children with AH versus without AH) provides information about the presence of an association, it does not quantify its strength and it does not account for confounders [33]. The present study showed that increased maxillo-mandibular sagittal discrepancy with retruded and hyperdivergent mandible was associated with AH, which was in partial agreement with the findings of a previous study in children with sleep-disordered breathing



[34]. In addition, in the present work, forward head posture and forward position of the hyoid bone were present in children with AH. Although these findings are in contrast to previous works that found no significant evidence of such associations [17, 35], the former studies involved small samples (i.e. small statistical power), participants were recruited from hospitals (i.e. not representative of the general population), and they involved patients of a wide age-range (i.e. confounding factor due to changes in adenoids size). This said, a simple group comparison does not allow proper investigation of the association between AH and craniofacial characteristics. While “risk” is the probability of occurrence of an event (e.g. the chance of having AH), “odds” is the ratio of the probability of occurrence of an event to the probability of that event not occurring (e.g. the chance of having AH with respect to the chance of not having it). The “odds ratio” (OR) is the ratio of odds of an event (e.g. odds of having AH) in one group (e.g. children with retruded mandible) versus the odds of that event in another group (e.g. children without retruded mandible) [36]. Furthermore, multiple logistic regression calculates the “adjusted OR”, which provides the independent effect of a variable (e.g. SNA) while holding all other variables fixed (e.g. SNB, Wits) on a binomial outcome (e.g. having versus not having AH) [37]. The present study showed increased adjusted OR of having AH among children with skeletal Class II tendency characterised by retruded mandible and decreased Wits appraisal. Despite a former study performing a regression analysis found no significant relationship between craniofacial proportions and adenoid size, of note no horizontal skeletal parameter was included [38].

Overall, LCR accompanied by medical history examination may be useful for the screening of patients with suspected AH, providing indications of the need for referral to otorhinolaryngologist [39]. Given the excellent specificity but low/moderate sensitivity of the proposed methods (i.e. non-negligible chance to miss a patient truly having AH), orthodontists may not rely on a single parameter for identifying children with AH, and a combined assessment using linear and area measurements may be advisable. Since the onset of AH in children is multifactorial, with asthma, allergic rhinitis, and atopic dermatitis among the risk factors [30], orthodontic options should be integrated in a multidisciplinary treatment planning [40]. Appropriate timing for orthodontic intervention, with respect to medical and/or surgical options, should be discussed with other specialists for optimising clinical outcomes while minimising the burden to patients (Additional file 1).

### Limitations

Although the use of a retrospective archive from the 1980s allowed for analyses that would be impossible today for ethical reasons, the prevalence of AH may have changed over the past forty years. A recent meta-analysis [4] showed that only one 2005 study from Brazil [41] and another 2008 study from Turkey [42] included random samples from the general population. Even broadening the inclusion criteria, no study on AH prevalence was performed in Hong Kong and high heterogeneity was found between studies [4]. Furthermore, the incidence of adenotonsillectomy may be determined by factors other than the prevalence of AH [43] and local policies may influence environmental factors [44], limiting the use of adenotonsillectomy as a proxy for AH. Therefore, currently available data may not allow speculating on possible trends of changes in prevalence of AH over time in Hong Kong.

Further longitudinal studies are necessary to investigate possible causal relationships and the direction of such cause-effects between the development of altered craniofacial features and the onset of AH (e.g. whether a genetically small nasal cavity may lead to mouth-breathing and frequent upper respiratory tract infections causing AH [45, 46], or whether AH may lead to mouth-breathing and altered craniofacial development [5, 31]).

### Conclusions

- The prevalence of AH among 12-year-old children ranged between 13.9 and 19.0%, based on the method used for its assessment on LCR.
- In this population, greater antero-posterior maxillo-mandibular discrepancy and mandibular retrusion were associated with higher likelihood of having AH, which should be considered during orthodontic treatment planning.
- LCR is commonly used in orthodontic patients and, despite being a static 2D assessment with biohazard related to ionising radiations, it may help in the identification of those with AH for further consideration with other specialists.

### Appendix

See Tables 6 and 7.

**Table 6** Intra-examiner agreement estimated with intraclass correlation coefficient (ICC), and respective measurement errors estimated with Dahlberg's formula

	Unit	ICC		p value	Dahlberg's error
		Value	95% CI		Value
<i>Assessment methods (continuous)</i>					
At/Nd	%	0.992	0.988–0.995	< 0.001	0.86
Ad-Ba/PNS-Ba	%	0.956	0.936–0.970	< 0.001	1.53
1-Npaa/Npa	%	0.999	0.999–1.000	< 0.001	0.16
<i>Independent variables (continuous)</i>					
SNA	°	0.961	0.899–0.981	< 0.001	0.63
SNB	°	0.968	0.946–0.980	< 0.001	0.58
ANB	°	0.908	0.867–0.937	< 0.001	0.75
Wits	mm	0.945	0.898–0.968	< 0.001	0.94
Cd-Gn	mm	0.967	0.943–0.980	< 0.001	0.96
MnP^SN	°	0.982	0.967–0.990	< 0.001	0.69
MxP^MnP	°	0.962	0.943–0.975	< 0.001	0.89
TPFH/TAFH	%	0.950	0.927–0.966	< 0.001	0.86
OPT^SN	°	0.990	0.982–0.994	< 0.001	0.73
C2ps-C4pi^SN	°	0.990	0.983–0.993	< 0.001	0.74
H-CV	mm	0.913	0.873–0.940	< 0.001	0.95
H-FH	mm	0.982	0.974–0.988	< 0.001	0.91

ICC intraclass correlation coefficient, CI confidence interval

**Table 7** Intra-examiner and inter-method agreement estimated with Cohen's kappa (K)

	Unit	K		
		Value	SE	p value
<i>Agreement within assessment methods (binary)</i>				
At/Nd	0/1	0.886	0.064	< 0.001
Ad-Ba/PNS-Ba	0/1	0.892	0.061	< 0.001
1-Npaa/Npa	0/1	0.960	0.040	< 0.001
<i>Agreement between assessment methods (binary)</i>				
At/Nd versus Ad-Ba/PNS-Ba	0/1	0.877	0.028	< 0.001
At/Nd versus 1-Npaa/Npa	0/1	0.818	0.035	< 0.001
Ad-Ba/PNS-Ba versus 1-Npaa/Npa	0/1	0.790	0.037	< 0.001
<i>Independent variables (categorical)</i>				
Growth stage	1/2/3	0.823	0.049	< 0.001

K = Cohen's kappa, SE = standard error

**Abbreviations**

1-Npaa/Npa	Adenoid/nasopharyngeal area ratio
2D	Two-dimensional
Ad-Ba/PNS-Ba	Adenoid/retropalatal ratio
AH	Adenoid hypertrophy
At/Nd	Adenoid/nasopharyngeal ratio
CVM	Cervical vertebral maturation
ICC	Intraclass correlation coefficient
K	Kohen's Kappa coefficient
LCR	Lateral cephalometric radiograph
OR	Odds ratio

**Supplementary Information**

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**Additional file 1.** Complete raw dataset of the measurements used for the analysis presented in the study.

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**Author contributions**

KLT, FS, and MG were involved in conception and design; KLT and MG helped in data acquisition; KLT, FS, and KYL contributed to data analysis; KLT and FS were involved in preparation of figures and tables; KLT, FS, and MG helped in data interpretation; KLT and FS contributed to drafting of the manuscript; FS was involved in writing of the final manuscript; FS, MG, CPM, and YY helped in critical revision. All authors approved the final version submitted for publication.

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**Availability of data and materials**

All data generated or analysed during this study are included in this published article and its supplementary information files (Additional file 1: Raw dataset).

**Declarations**

**Ethics approval and consent to participate**

The present study was approved by the Institutional Review Board of the University of Hong Kong/Hospital Authority Hong Kong West Cluster (UW12-405)

**Consent for publication**

Not applicable.

**Competing interests**

The authors declare that they have no competing interests.

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**References**

- Papaioannou G, Kambas I, Tsaousoglou M, Panaghiotopoulou-Gartaganis P, Chrousos G, Kaditis AG. Age-dependent changes in the size of adenotonsillar tissue in childhood: implications for sleep-disordered breathing. *J Pediatr*. 2013;162(2):269-74.e4.
- Handelman CS, Osborne G. Growth of the nasopharynx and adenoid development from one to eighteen years. *Angle Orthod*. 1976;46(3):243-59.
- Huang SW, Giannoni C. The risk of adenoid hypertrophy in children with allergic rhinitis. *Ann Allergy Asthma Immunol*. 2001;87(4):350-5.

4. Pereira L, Monyror J, Almeida FT, Almeida FR, Guerra E, Flores-Mir C, et al. Prevalence of adenoid hypertrophy: a systematic review and meta-analysis. *Sleep Med Rev.* 2018;38:101–12.
5. Dinis PB, Haider H, Gomes A. The effects of adenoid hypertrophy and subsequent adenoidectomy on pediatric nasal airway resistance. *Am J Rhinol.* 1999;13(5):363–9.
6. Jiang X, Ren X, Liu H, Tian J, Du C, Luo H, et al. Health-related quality of life among children with adenoid hypertrophy in Xi'an, China. *Int J Pediatr Otorhinolaryngol.* 2015;79(12):2321–6.
7. Duan H, Xia L, He W, Lin Y, Lu Z, Lan Q. Accuracy of lateral cephalogram for diagnosis of adenoid hypertrophy and posterior upper airway obstruction: a meta-analysis. *Int J Pediatr Otorhinolaryngol.* 2019;119:1–9.
8. Savoldi F, Xinyue G, McGrath CP, Yang Y, Chow SC, Tsoi JKH, et al. Reliability of lateral cephalometric radiographs in the assessment of the upper airway in children: a retrospective study. *Angle Orthod.* 2020;90(1):47–55.
9. Fujjoka M, Young LW, Girdany BR. Radiographic evaluation of adenoidal size in children: adenoidal-nasopharyngeal ratio. *AJR Am J Roentgenol.* 1979;133(3):401–4.
10. Kemaloglu YK, Goksu N, Inal E, Akyildiz N. Radiographic evaluation of children with nasopharyngeal obstruction due to the adenoid. *Ann Otol Rhinol Laryngol.* 1999;108(1):67–72.
11. Souki MQ, Souki BQ, Franco LP, Becker HM, Araujo EA. Reliability of subjective, linear, ratio and area cephalometric measurements in assessing adenoid hypertrophy among different age groups. *Angle Orthod.* 2012;82(6):1001–7.
12. Hibbert J, Whitehouse GH. The assessment of adenoidal size by radiological means. *Clin Otolaryngol Allied Sci.* 1978;3(1):43–7.
13. Maw AR, Jeans WD, Fernando DC. Inter-observer variability in the clinical and radiological assessment of adenoid size, and the correlation with adenoid volume. *Clin Otolaryngol Allied Sci.* 1981;6(5):317–22.
14. Holmberg H, Linder-Aronson S. Cephalometric radiographs as a means of evaluating the capacity of the nasal and nasopharyngeal airway. *Am J Orthod.* 1979;76(5):479–90.
15. Major MP, Flores-Mir C, Major PW. Assessment of lateral cephalometric diagnosis of adenoid hypertrophy and posterior upper airway obstruction: a systematic review. *Am J Orthod Dentofacial Orthop.* 2006;130(6):700–8.
16. Gu M, McGrath CP, Wong RW, Hagg U, Yang Y. Cephalometric norms for the upper airway of 12-year-old Chinese children. *Head Face Med.* 2014;10:38.
17. Wysocki J, Krasny M, Skarzynski PH. Patency of nasopharynx and a cephalometric image in the children with orthodontic problems. *Int J Pediatr Otorhinolaryngol.* 2009;73(12):1803–9.
18. Souki BQ, Pimenta GB, Souki MQ, Franco LP, Becker HM, Pinto JA. Prevalence of malocclusion among mouth breathing children: do expectations meet reality? *Int J Pediatr Otorhinolaryngol.* 2009;73(5):767–73.
19. Baccetti T, Franchi L, McNamara JA. The cervical vertebral maturation (CVM) method for the assessment of optimal treatment timing in dentofacial orthopedics. *Semin Orthod.* 2005;11(3):119–29.
20. Behrents RG, Shelgikar AV, Conley RS, Flores-Mir C, Hans M, Levine M, et al. Obstructive sleep apnea and orthodontics: an American Association of Orthodontists White Paper. *Am J Orthod Dentofacial Orthop.* 2019;156(1):13–28.
21. Michael Stephen C. Cephalometric analyses based on natural head posture of Chinese children in Hong Kong: 香港大學; 1986.
22. Schneider CA, Rasband WS, Eliceiri KW. NIH Image to ImageJ: 25 years of image analysis. *Nat Methods.* 2012;9(7):671–5.
23. Peduzzi P, Concato J, Kemper E, Holford TR, Feinstein AR. A simulation study of the number of events per variable in logistic regression analysis. *J Clin Epidemiol.* 1996;49(12):1373–9.
24. Dahlberg G. Statistical methods for medical and biological students. London: George Allen and Unwin; 1940.
25. Koo TK, Li MY. A guideline of selecting and reporting intraclass correlation coefficients for reliability research. *J Chiropr Med.* 2016;15(2):155–63.
26. Byrt T. How good is that agreement? *Epidemiology.* 1996;7(5):561.
27. Weitz HL. Roentgenography of adenoids. *Radiology.* 1946;47:66–70.
28. Wormald PJ, Prescott CA. Adenoids: comparison of radiological assessment methods with clinical and endoscopic findings. *J Laryngol Otol.* 1992;106(4):342–4.
29. Elwany S. The adenoidal-nasopharyngeal ratio (AN ratio). Its validity in selecting children for adenoidectomy. *J Laryngol Otol.* 1987;101(6):569–73.
30. Evcimik MF, Dogru M, Cirik AA, Nepesov MI. Adenoid hypertrophy in children with allergic disease and influential factors. *Int J Pediatr Otorhinolaryngol.* 2015;79(5):694–7.
31. Linder-Aronson S. Respiratory function in relation to facial morphology and the dentition. *Br J Orthod.* 1979;6(2):59–71.
32. Ho ACH, Savoldi F, Wong RWK, Fung SC, Li SKY, Yang Y, et al. Prevalence and risk factors for obstructive sleep apnea syndrome among children and adolescents with cleft lip and palate: a survey study in Hong Kong. *Cleft Palate Craniofac J.* 2023;60(4):421–9.
33. Altman N, Krzywinski M. Association, correlation and causation. *Nat Methods.* 2015;12(10):899–900.
34. Kawashima S, Peltomaki T, Sakata H, Mori K, Happonen RP, Ronning O. Craniofacial morphology in preschool children with sleep-related breathing disorder and hypertrophy of tonsils. *Acta Paediatr.* 2002;91(1):71–7.
35. Baroni M, Ballanti F, Franchi L, Cozza P. Craniofacial features of subjects with adenoid, tonsillar, or adenotonsillar hypertrophy. *Prog Orthod.* 2011;12(1):38–44.
36. Bland JM, Altman DG. The odds ratio. *BMJ.* 2000;320:1468.
37. LaValley MP. Logistic regression. *Circulation.* 2008;117(18):2395–9.
38. Pawlowska-Seredynska K, Umlawska W, Resler K, Morawska-Kochman M, Pazdro-Zastawny K, Krecicki T. Craniofacial proportions in children with adenoid or adenotonsillar hypertrophy are related to disease duration and nasopharyngeal obstruction. *Int J Pediatr Otorhinolaryngol.* 2020;132: 109911.
39. Major MP, Saltaji H, El-Hakim H, Witmans M, Major P, Flores-Mir C. The accuracy of diagnostic tests for adenoid hypertrophy: a systematic review. *J Am Dent Assoc.* 2014;145(3):247–54.
40. Gu M, Savoldi F, Chan EYL, Tse CSK, Lau MTW, Wey MC, et al. Changes in the upper airway, hyoid bone and craniofacial morphology between patients treated with headgear activator and Herbst appliance: a retrospective study on lateral cephalometry. *Orthod Craniofac Res.* 2021;24(3):360–9.
41. Santos RS, Cipolotti R, D'Avila JS, Gurgel RQ. Schoolchildren submitted to video nasopharyngoscopy examination at school: findings and tolerance. *J Pediatr (Rio J).* 2005;81(6):443–6.
42. Aydin S, Sanli A, Celebi O, Tasdemir O, Paksoy M, Eken M, et al. Prevalence of adenoid hypertrophy and nocturnal enuresis in primary school children in Istanbul, Turkey. *Int J Pediatr Otorhinolaryngol.* 2008;72(5):665–8.
43. Rosenfeld RM, Green RP. Tonsillectomy and adenoidectomy: changing trends. *Ann Otol Rhinol Laryngol.* 1990;99(3 Pt 1):187–91.
44. Mason TG, Schooling CM, Chan KP, Tian L. An evaluation of the air quality health index program on respiratory diseases in Hong Kong: an interrupted time series analysis. *Atmos Environ.* 2019;211:151–8.
45. Kukwa W, Guilleminault C, Tomaszewska M, Kukwa A, Krzeski A, Migacz E. Prevalence of upper respiratory tract infections in habitually snoring and mouth breathing children. *Int J Pediatr Otorhinolaryngol.* 2018;107:37–41.
46. Warren DW, Hairfield WM, Seaton DL, Hinton VA. The relationship between nasal airway cross-sectional area and nasal resistance. *Am J Orthod Dentofacial Orthop.* 1987;92(5):390–5.

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