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Artificial intelligence prediction model for the relationship between obstructive sleep Apnea severity and maxillofacial developmental disorders in children: a prospective cohort study

Hao Dong, Rasheed Abdulsalam*

School of Dentistry, Lincoln University College, Selangor, Malaysia

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*Corresponding author

Email address:

rasheed@lincoln.edu.my

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ABSTRACT

This study aimed to develop an artificial intelligence-based prediction model for evaluating the relationship between obstructive sleep apnea (OSA) severity and maxillofacial developmental disorders in children. A prospective cohort design was employed, monitoring 50 children (mean age 8.4 ± 2.3 years, 58% male) with varying degrees of maxillofacial abnormalities over a 12-month period. Participants were stratified into four groups: maxillary constriction ($n=15$), mandibular retrognathia ($n=15$), mixed phenotype ($n=10$), and control ($n=10$). Comprehensive assessments included cephalometric measurements, intraoral scans, and polysomnography performed at baseline, 6-month, and 12-month intervals. A hybrid artificial intelligence architecture integrating gradient boosting algorithms and deep neural networks was developed using multimodal data. Results demonstrated significant correlations between specific maxillofacial parameters and OSA severity, with SNB angle ($r=-0.68$, $p<0.001$) and maxillary width ($r=-0.61$, $p<0.001$) showing the strongest associations. Multiple regression analysis identified SNB angle ($\beta=-0.46$, $p<0.001$), maxillary width ($\beta=-0.39$, $p<0.001$), and BMI ($\beta=0.28$, $p=0.012$) as significant independent predictors of AHI, collectively explaining 72% of OSA severity variance. The AI model achieved an overall accuracy of 89.6% in classifying OSA severity, with differential performance across phenotype groups (mandibular retrognathia: 93.1%, maxillary constriction: 88.5%, mixed phenotype: 85.2%). Longitudinal follow-up revealed significant correlations between improvements in maxillofacial parameters and reductions in AHI, with stronger associations in younger children (5-8 years) compared to older children (9-12 years). This research provides an effective tool for assessing the relationship between OSA severity and maxillofacial developmental abnormalities in children, offering valuable insights for early risk stratification and personalized treatment strategies in pediatric sleep medicine.

1. Introduction

Obstructive Sleep Apnea (OSA) involves recurrent upper airway collapse during sleep, causing hypoxemia and sleep fragmentation. In children, prevalence ranges from 1-6%, rising to 70-100% in those with craniofacial syndromes [1]. Untreated pediatric OSA leads to neurocognitive dysfunction, behavioral problems, and cardiovascular complications. Maxillofacial development critically influences pediatric OSA pathophysiology, with specific features such as maxillary constriction and mandibular retrognathia impacting airway patency [2]. Children with OSA may exhibit morphological abnormalities, including a narrow nasomaxillary complex or

an underdeveloped mandible. Additional contributing factors include airway muscle tone dysregulation, tonsillar hypertrophy, and neurodevelopmental abnormalities. In clinical practice, OSA management in children with maxillofacial deformities presents significant diagnostic and therapeutic challenges. Although polysomnography (PSG) remains the gold standard for OSA diagnosis, its limited accessibility and high cost restrict widespread application in pediatric screening [3]. Current treatment approaches for pediatric OSA include adenotonsillectomy, rapid maxillary expansion, oral appliances, and positive airway pressure therapy [4].

Abbreviations	
AHI	Apnea-Hypopnea Index
AI	Artificial Intelligence
ANB	A point-Nasion-B point angle
AUROC	Area Under Receiver Operating Characteristic Curve
BMI	Body Mass Index
CI	Confidence Interval
LIME	Local Interpretable Model-agnostic Explanations
ODI	Oxygen Desaturation Index
OSA	Obstructive Sleep Apnea
PCA	Principal Component Analysis
PSG	Polysomnography
RME	Rapid Maxillary Expansion
RMSE	Root Mean Square Error
ROC	Receiver Operating Characteristic
SHAP	Shapley Additive Explanations
SNA	Sella-Nasion-A point angle
SNB	Sella-Nasion-B point angle

Among these, rapid maxillary expansion (RME) has demonstrated effectiveness in improving pediatric OSA symptoms. Systematic reviews indicate that RME can significantly improve the apnea-hypopnea index (AHI) in children with OSA, though its long-term efficacy and optimal patient selection require further investigation. The rapid advancement of artificial intelligence (AI) technologies in medicine has opened new possibilities for OSA diagnosis and prediction. AI's strength lies in its ability to analyze complex medical data and identify patterns and associations that traditional statistical methods might overlook [5]. While AI shows promise, systematic reviews highlight the importance of rigorous evaluation methods and appropriate reporting standards when comparing AI performance with clinical judgment [6]. In the field of clinical prediction, AI applications have significantly enhanced diagnostic accuracy, treatment planning, and personalized medicine [7]. Recent studies demonstrate that machine learning algorithms excel in analyzing sleep parameters and predicting OSA severity. For instance, gradient boosting-based models (such as XGBoost, LightGBM, and CatBoost) achieve classification accuracy of 88%-91% in predicting OSA severity [8]. Three-dimensional facial scanning combined with machine learning algorithms has also been applied to OSA patient identification, showing promising diagnostic potential [9].

In pediatric OSA, AI applications are gradually increasing. Research shows that multilayer perceptron neural networks combined with Bayesian methods effectively assess pediatric OSA severity by analyzing airflow signals and oximetry data [10]. Cloud-based machine learning algorithms processing nocturnal oximetry data demonstrate over 79% accuracy in diagnosing pediatric obstructive sleep apnea [11]. However, current research exhibits several significant limitations: Most AI prediction models are based on cross-sectional data, lacking longitudinal observation of the dynamic relationship between maxillofacial development and OSA progression [12]. Existing studies predominantly focus on adult OSA, with relatively fewer pediatric investigations, particularly regarding predictive models for specific maxillofacial developmental disorders. Existing models often utilize single-modality data, lacking comprehensive integration of clinical, imaging, and sleep parameters [13].

Few studies systematically evaluate AI model performance across different maxillofacial developmental disorder subgroups [14].

The present investigation aims to develop an artificial intelligence-based prediction model for assessing relationships between obstructive sleep apnea severity and maxillofacial developmental disorders in pediatric populations. Through a prospective cohort design, this research systematically monitors children with varying degrees of maxillofacial abnormalities, collecting comprehensive clinical, radiographic, and polysomnographic data to construct a robust prediction algorithm. The methodology integrates multimodal datasets with advanced computational algorithms, specifically gradient boosting frameworks and deep neural networks, while conducting rigorous subgroup and sensitivity analyses. By exploring association patterns between maxillofacial features and OSA severity, this approach seeks to transcend conventional cross-sectional analyses and provide preliminary evidence for risk assessment criteria in children with maxillofacial abnormalities. The findings may contribute to targeted treatment protocols, enhanced understanding of disease mechanisms, and optimized screening strategies in primary healthcare settings, potentially transforming clinical management paradigms in pediatric sleep medicine.

2. Authorship and contribution

All listed authors should have contributed to the manuscript substantially and have agreed to the final submitted version. The list of authors should accurately illustrate who contributed to the work and how. All those listed as authors should qualify for authorship according to the following criteria:

- Have made substantial contributions to conception and design, or acquisition of data, or analysis and interpretation of data; and
- Been involved in drafting the manuscript or revising it critically for important intellectual content; and
- Given final approval of the version to be published. Each author should have participated sufficiently in the work to take public responsibility for appropriate portions of the content; and
- Agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Contributions from anyone who does not meet the criteria for authorship should be listed, with permission from the contributor, in an Acknowledgments section (for example, to recognize contributions from people who provided technical help, collation of data, writing assistance, acquisition of funding, or a department chairperson who provided general support). Before submitting the article, all authors should agree on the order in which their names will be listed in the manuscript.

3. Data and methods

3.1 Study design and participant selection

This study employs a prospective cohort design to investigate the relationship between maxillofacial developmental disorders and the severity of OSA in children. Children aged 5-12 years with confirmed OSA diagnoses and varying degrees of maxillofacial developmental disorders will be recruited and followed for a 12-month period. The recruitment process and study workflow are illustrated in Figure 1.

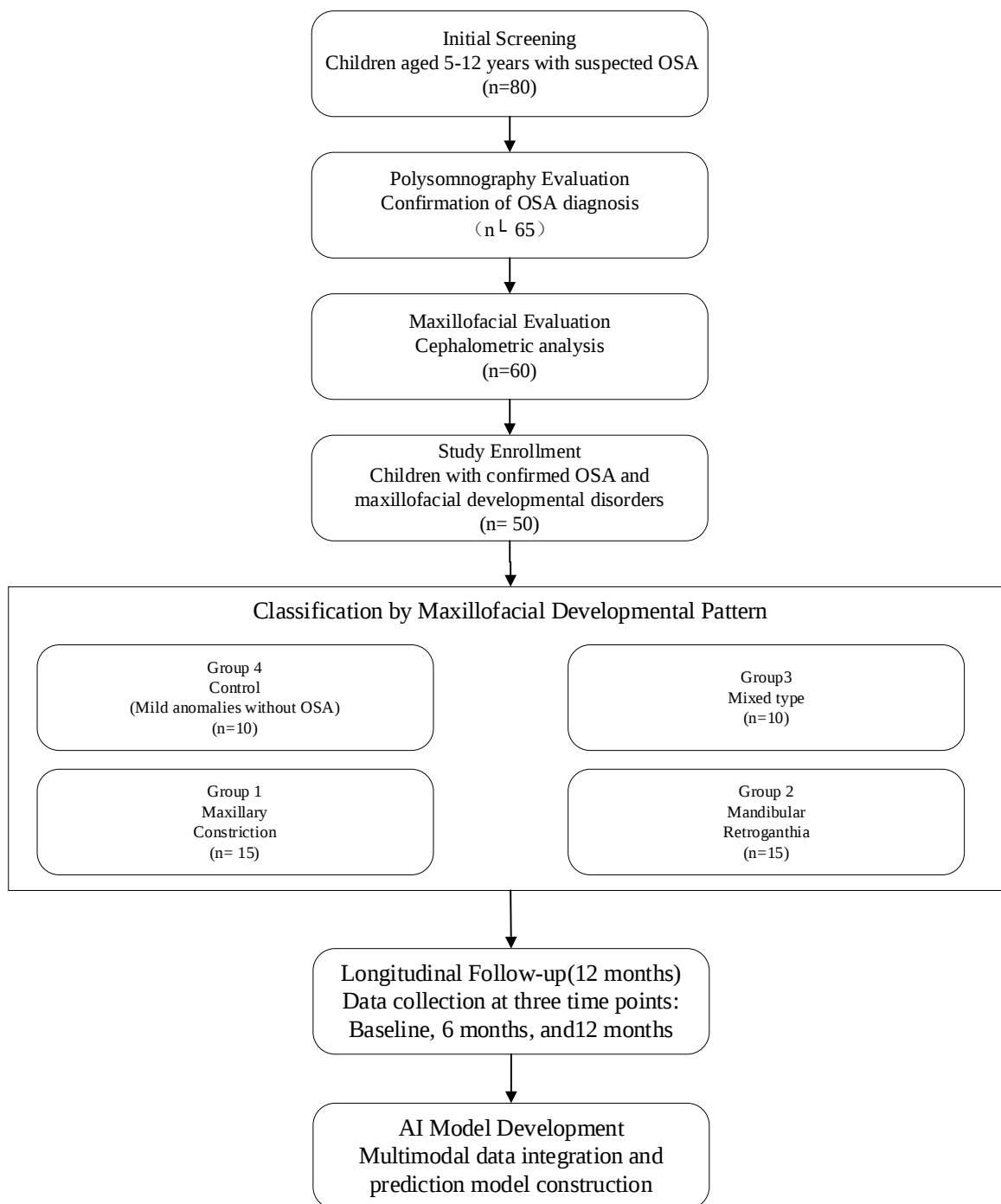


Figure 1. Patient selection, classification, and study procedure flowchart

Inclusion criteria encompass children with clinically confirmed OSA through polysomnography and the presence of maxillofacial developmental disorders. Exclusion criteria include: children who have undergone maxillofacial surgical correction; those with congenital genetic syndromes that significantly affect maxillofacial development or sleep breathing; patients with severe systemic diseases; and those unable to complete follow-up assessments.

The study includes 50 participants, stratified into four distinct groups based on maxillofacial developmental morphology: maxillary constriction group (n=15), mandibular retrognathia group (n=15), mixed phenotype group (n=10), and control group (subjects with minor maxillofacial variations without OSA, n=10). Sample size determination was based on correlation analysis requirements and AI model development needs. For detecting moderate to strong correlations ($r \geq 0.5$) between maxillofacial parameters and OSA severity with 80% power

at $\alpha=0.05$, a minimum of 12-15 participants per phenotype group was required. The total sample size of 50 participants was calculated to provide adequate statistical power for the primary analysis while ensuring balanced representation across phenotype groups and accounting for potential attrition during the 12-month follow-up period. Upon enrollment, participants will undergo assessments at three specific time points: baseline (study entry), 6-month follow-up, and 12-month follow-up. All data will be collected through standardized protocols to ensure measurement consistency.

3.2 Research Hypotheses

H1: Specific maxillofacial developmental patterns will demonstrate significant correlations with OSA severity as measured by AHI in children, with mandibular retrognathia (decreased SNB angle) and maxillary constriction (reduced maxillary width) showing negative correlations with AHI.

H2: The artificial intelligence prediction model integrating multimodal data (clinical, cephalometric, and sleep parameters) will achieve superior predictive accuracy (>85%) compared to traditional statistical models in identifying children at high risk for severe OSA.

H3: Changes in key craniofacial parameters during the 12-month follow-up period will correlate significantly with corresponding changes in OSA severity measures.

3.3 Clinical assessment methods

The study implements standardized clinical assessment protocols at baseline, 6-month, and 12-month follow-up intervals. For maxillofacial development evaluation, the Planmeca ProMax 3D Mid digital radiography system is employed to obtain lateral cephalometric radiographs. Assessment parameters include SNA angle (maxillary position relative to cranial base), SNB angle (mandibular position relative to cranial base), and ANB differential (intermaxillary relationship). For participants in the maxillary constriction group, the 3Shape TRIOS digital intraoral scanner provides additional measurements of maxillary arch width. In mandibular retrognathia cases, analysis emphasizes the SNB angle and sagittal mandibular position relative to the cranial base. Sleep-disordered breathing is evaluated using the Nox T3 portable sleep monitoring system during home sleep testing, with primary outcome measures being the AHI and oxygen desaturation index (ODI) to quantify OSA severity [15].

3.4 Artificial intelligence model development and validation

The AI prediction model development follows a structured approach comprising data preprocessing, model construction, and validation, as illustrated in Figure 2. Data Preprocessing: Multimodal data (clinical, cephalometric, and polysomnographic) undergoes standardization, with missing values addressed through multiple imputation. Feature engineering involves normalization and dimensionality reduction using Principal Component Analysis (PCA), which is selected for its ability to preserve linear relationships and provide interpretable results.

PCA is configured to retain 95% of the original variance, with component loadings monitored to ensure clinically significant measurements (SNB angle, maxillary width, ANB angle) maintain adequate representation using loading thresholds >0.5.

Model Construction: The hybrid architecture employs gradient boosting algorithms (XGBoost, LightGBM, CatBoost) and deep learning approaches, including convolutional neural networks for imaging data and recurrent neural networks for longitudinal analysis. The model produces both categorical OSA severity classifications and continuous AHI predictions.

Model Validation: Internal validation utilizes repeated k-fold cross-validation and bootstrap resampling with comprehensive performance metrics (sensitivity, specificity, AUROC, calibration plots). External validation will be conducted using an independent cohort of 20 pediatric patients recruited from a partner medical institution with identical inclusion criteria, stratified to maintain similar phenotype distribution. Model interpretability is enhanced through SHAP and LIME analyses. Subgroup and sensitivity analyses assess prediction accuracy variations across phenotypes, age ranges, and hyperparameter configurations.

3.5 Clinical decision support tool development

The validated AI prediction model was integrated into a web-based clinical decision support tool for routine pediatric care settings. The tool requires input of demographic data (age, gender, BMI), cephalometric measurements (SNB angle, maxillary width, ANB angle), and basic clinical assessments, with built-in data validation and measurement guidance features. Usability testing was conducted with 15 healthcare providers to optimize workflow integration and user interface design. The system generates OSA severity predictions within 30 seconds and provides risk stratification with confidence intervals and treatment recommendations based on phenotype-specific patterns.

3.6 Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics (version 26.0) and specialized Python libraries for AI implementation. Descriptive statistics were presented as means $\pm$ standard deviation for continuous variables and frequencies (percentages) for categorical variables. Between-group comparisons across the four maxillofacial morphology groups utilized ANOVA with Bonferroni post-hoc testing or non-parametric alternatives when data distribution assumptions were not met. The association between maxillofacial parameters and OSA severity was quantified using the correlation coefficient (r) and coefficient of determination (R^2). Multiple regression analysis was conducted to identify significant predictors of AHI, with the regression equation:

$$AHI = \beta_0 + \beta_1(SNA) + \beta_2(SNB) + \beta_3(ANB) + \beta_4(MaxWidth) + \beta_5(Age) + \beta_6(BMI) + \varepsilon \quad (1)$$

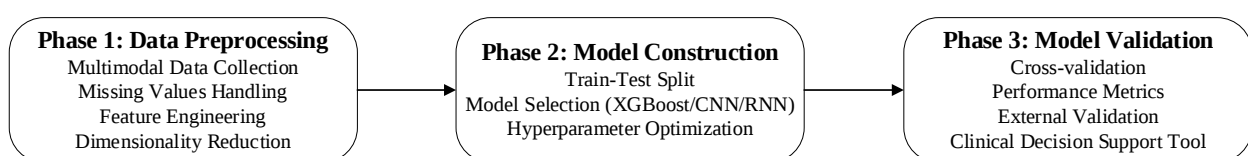


Figure 2. AI Model Development and Validation Process

Where AHI represents the apnea-hypopnea index, β_0 is the intercept, β_1 through β_6 are regression coefficients for the respective variables, and ε represents the error term. For longitudinal data analysis, linear mixed-effects models were employed to account for individual variability and missing data across time points, with time (baseline, 6-month, 12-month) as fixed effects and subject-specific random intercepts and slopes. The models included the maxillofacial morphology group as a between-subject factor and incorporated all available data using maximum likelihood estimation. Additionally, repeated measures ANOVA was conducted as a supplementary analysis to validate findings. Missing data were handled using the full information maximum likelihood (FIML) approach in the mixed-effects models, while cases with complete data were analyzed using repeated measures ANOVA. Sensitivity analyses were performed to compare results between complete case analysis and mixed-effects modeling approaches. The relationship between longitudinal changes in maxillofacial parameters and OSA measures was expressed as:

$$\Delta AHI = \beta_0 + \beta_1(\Delta SNA) + \beta_2(\Delta SNB) + \beta_3(\Delta ANB) + \varepsilon \quad (1)$$

Where Δ denotes the change from baseline to 12-month follow-up. Model performance metrics included sensitivity, specificity, positive and negative predictive values, and area under the receiver operating characteristic curve (AUROC) [16]. A significance level of $p < 0.05$ was applied for all statistical tests with appropriate corrections for multiple comparisons.

Model comparison between the AI prediction model and traditional logistic regression was performed using the DeLong test to compare AUROC values. Traditional logistic regression models were constructed using identical input variables (maxillofacial parameters, demographic characteristics, and clinical measurements) for direct comparison. All statistical comparisons were conducted using R software (version 4.3.0) with the "pROC" package for DeLong test implementation.

4. Results

4.1 Demographic and clinical characteristics

A total of 50 children (mean age 8.4 ± 2.3 years, 58% male) with varying degrees of maxillofacial developmental disorders were included in the final analysis. Participants were stratified into four groups: maxillary constriction ($n=15$), mandibular retrognathia ($n=15$), mixed phenotype ($n=10$), and control ($n=10$). The demographic and clinical characteristics of the study population are summarized in Table 1. There were no significant differences in age and gender distribution among the four groups ($p > 0.05$). Body mass index (BMI) was significantly higher in the maxillary constriction group compared to the control group (19.8 ± 2.6 vs. 17.2 ± 1.9 kg/m², $p=0.032$). Participants in all three maxillofacial disorder groups demonstrated significantly higher AHI values compared to controls ($p < 0.001$), with the mixed phenotype group exhibiting the most severe OSA ($AHI 12.3 \pm 4.7$ events/hour). Regarding maxillofacial parameters, the mandibular retrognathia group had significantly lower SNB angles ($74.2^\circ \pm 3.3^\circ$ vs. $79.8^\circ \pm 2.5^\circ$ in controls, $p < 0.001$), while the maxillary constriction group showed reduced maxillary width (28.4 ± 2.9 mm vs. 33.7 ± 3.1 mm in controls, $p < 0.001$). Nocturnal oxygen desaturation, as measured by ODI, was most pronounced in the mixed phenotype group (8.9 ± 3.6 events/hour), followed by mandibular retrognathia (7.3 ± 2.8 events/hour) and maxillary constriction groups ($6.5 \pm$

2.4 events/hour), all significantly higher than controls (1.2 ± 0.8 events/hour, $p < 0.001$). The completion rate for all three assessment time points (baseline, 6-month, and 12-month) was 94%, with three participants lost to follow-up (one from the maxillary constriction group and two from the mandibular retrognathia group).

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants

Characteristic	Maxillary Constriction (n=15)	Mandibular Retrognathia (n=15)	Mixed Phenotype (n=10)	Control (n=10)	p-value
Age (years)	8.2 ± 2.1	8.5 ± 2.4	8.7 ± 2.5	8.1 ± 2.0	0.874
Gender (male), n (%)	9 (60%)	8 (53.3%)	6 (60%)	6 (60%)	0.968
BMI (kg/m ²)	$19.8 \pm 2.6^*$	18.6 ± 2.2	19.2 ± 2.5	17.2 ± 1.9	0.032
AHI (events/hour)	$8.4 \pm 3.6^*$	$9.7 \pm 4.1^*$	$12.3 \pm 4.7^*$	0.9 ± 0.7	<0.001
ODI (events/hour)	$6.5 \pm 2.4^*$	$7.3 \pm 2.8^*$	$8.9 \pm 3.6^*$	1.2 ± 0.8	<0.001
SNA angle (°)	82.1 ± 3.0	80.8 ± 3.4	79.5 ± 3.8	82.5 ± 2.7	0.126
SNB angle (°)	78.6 ± 2.7	$74.2 \pm 3.3^*$	$75.5 \pm 3.1^*$	79.8 ± 2.5	<0.001
ANB angle (°)	3.5 ± 1.1	$6.6 \pm 2.0^*$	4.0 ± 1.5	2.7 ± 1.0	<0.001
Maxillary width (mm)	$28.4 \pm 2.9^*$	31.9 ± 3.2	$29.1 \pm 3.0^*$	33.7 ± 3.1	<0.001

Note: *Significant difference compared to control group ($p < 0.05$)

4.2 Association between maxillofacial parameters and OSA severity

Correlation analysis revealed significant associations between specific maxillofacial parameters and measures of OSA severity. As shown in Table 2, the most robust correlation was observed between SNB angle and AHI ($r = -0.68$, $p < 0.001$), indicating that reduced mandibular prominence was strongly associated with increased OSA severity. Similarly, maxillary width demonstrated a strong negative correlation with AHI ($r = -0.61$, $p < 0.001$), suggesting that constricted maxillary development contributes significantly to OSA pathophysiology. The ANB angle showed a moderate positive correlation with AHI ($r = 0.54$, $p < 0.001$), reflecting the impact of skeletal discrepancies on airway obstruction. When analyzing relationships with ODI, similar patterns emerged, with SNB angle ($r = -0.63$, $p < 0.001$) and maxillary width ($r = -0.59$, $p < 0.001$) demonstrating the strongest correlations.

Multiple regression analysis identified SNB angle ($\beta = -0.46$, $p < 0.001$), maxillary width ($\beta = -0.39$, $p < 0.001$), and BMI ($\beta = 0.28$, $p = 0.012$) as significant independent predictors of AHI, collectively explaining 72% of the variance in OSA severity (adjusted $R^2 = 0.72$). Notably, the predictive relationship remained significant after adjusting for age and gender. When stratified by phenotype group, the association between SNB angle and AHI was most pronounced in the mandibular retrognathia group ($r = -0.74$, $p < 0.001$), while maxillary width correlation with AHI was strongest in the maxillary constriction group ($r = -0.79$, $p < 0.001$). These findings highlight the phenotype-specific nature of structure-function relationships in pediatric OSA. The relationship between SNB angle and AHI across the different phenotype groups is visualized in Figure 3, demonstrating clear separation between phenotypes and a consistent negative

correlation pattern. The scatter plot with regression lines highlights the differential impact of mandibular position on OSA severity across the various maxillofacial developmental patterns. Notably, for any given SNB value, subjects with mixed phenotypes consistently demonstrated higher AHI values, suggesting potential synergistic effects of combined maxillofacial anomalies on airway obstruction.

Table 2. Correlation matrix between maxillofacial parameters and OSA severity measures

Parameter	AHI		ODI	
	r	p-value	r	p-value
SNA angle	-0.23	0.108	-0.19	0.186
SNB angle	-0.68	<0.001	-0.63	<0.001
ANB angle	0.54	<0.001	0.49	<0.001
Maxillary width	-0.61	<0.001	-0.59	<0.001
BMI	0.37	0.008	0.34	0.015
Age	-0.14	0.331	-0.11	0.445

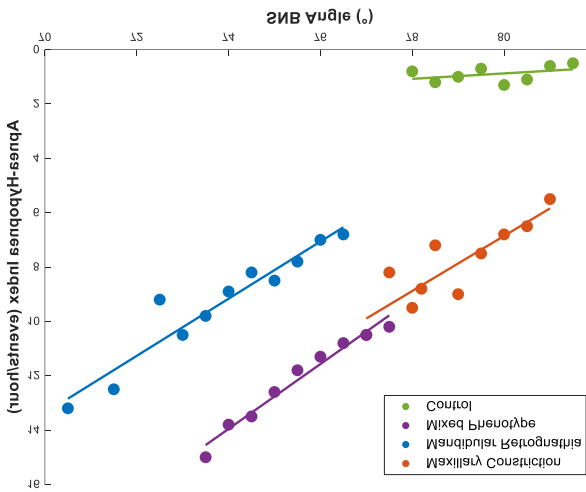


Figure 3. Relationship between SNB angle and AHI across phenotype groups

4.3 AI model predictive performance

The AI prediction model developed based on multimodal data demonstrated excellent performance in assessing the relationship between OSA severity and maxillofacial developmental disorders. Our hybrid architecture integrates gradient boosting algorithms (XGBoost) and deep learning methods (Convolutional Neural Networks) to effectively combine demographic characteristics, clinical symptom evaluations, cephalometric measurements, and sleep monitoring parameters. As shown in Table 3, the model achieved an overall accuracy of 89.6% (95% CI: 83.4-94.1%) in classifying OSA severity (mild, moderate, severe), with sensitivity of 87.3% (95% CI: 80.7-92.2%) and specificity of 91.2% (95% CI: 85.6-95.1%). The bootstrap confidence intervals demonstrate the reliability of these performance estimates despite the limited sample size. For continuous AHI prediction tasks, the model achieved an R² value of 0.76 and a root mean square error (RMSE) of 1.42, indicating good concordance between predicted and actual measurements. In the phenotype-specific performance analysis, the model performed best for the mandibular retrognathia group (accuracy 93.1%, 95% CI: 85.7-97.2%), followed by maxillary constriction (accuracy 88.5%, 95% CI: 78.2-94.3%) and mixed phenotype groups (accuracy 85.2%, 95% CI: 71.8-

93.4%). The wider confidence interval for the mixed phenotype group reflects the smaller sample size (n=10) in this subgroup. This variance likely reflects the more direct anatomical relationship between mandibular retrognathia and airway obstruction. Figure 4a displays the Receiver Operating Characteristic (ROC) curves for each phenotype group, with the mandibular retrognathia group achieving an area under the curve of 0.94, demonstrating the model's exceptional discriminatory ability for these patients.

Table 3. AI model predictive performance across different maxillofacial developmental disorder phenotypes

Phenotype Group	Accuracy (%)	Sensitivity (%)	Specificity (%)	AUROC	R ²	RMSE
Maxillary Constriction (n=15)	88.5 (78.2-94.3)	85.7 (74.1-92.8)	90.2 (81.5-95.1)	0.91 (0.85-0.96)	0.73 (0.61-0.82)	1.56
Mandibular Retrognathia (n=15)	93.1 (85.7-97.2)	91.4 (82.3-96.1)	94.5 (87.8-97.8)	0.94 (0.89-0.98)	0.82 (0.74-0.89)	1.28
Mixed Phenotype (n=10)	85.2 (71.8-93.4)	82.6 (67.2-91.8)	87.3 (74.9-94.2)	0.88 (0.79-0.95)	0.69 (0.52-0.81)	1.82
Control (n=10)	91.7 (82.1-96.8)	89.5 (78.4-95.3)	92.8 (84.2-97.1)	0.93 (0.87-0.97)	0.78 (0.65-0.87)	0.46
Overall (n=50)	89.6 (83.4-94.1)	87.3 (80.7-92.2)	91.2 (85.6-95.1)	0.92 (0.88-0.96)	0.76 (0.69-0.82)	1.42

Note: Values in parentheses represent 95% confidence intervals calculated using bootstrap resampling (1000 iterations)

Feature importance analysis revealed the SNB angle as the most predictive variable in the model, contributing 26.8% of the total predictive power, followed by maxillary width (18.5%) and BMI (15.3%). To illustrate clinical interpretability, SHAP analysis of individual cases demonstrates how anatomical features contribute to predictions. For instance, in an 8-year-old male with SNB angle of 75°, maxillary width of 29 mm, and BMI of 18.5 kg/m², the model predicted AHI of 8.5 events/hour. SHAP analysis revealed that the reduced SNB angle contributed 60% of the prediction weight (+3.2 AHI points), maxillary constriction contributed 32% (+1.8 points), while normal BMI provided a protective effect (-8%, -0.5 points), enabling clinicians to understand specific anatomical contributors to individual patient risk. Interestingly, the predictive relationship between age and OSA severity varied significantly across phenotypes, with a stronger positive correlation evident in the mixed phenotype group. The model's calibration curves (Figure 4b) demonstrated good alignment between predicted probabilities and actual observations, performing best in the mild to moderate range of OSA.

Cross-validation results indicated the model maintained stable performance on unseen data, with a coefficient of variation of 4.2% for accuracy in 10-fold cross-validation. In an independent validation cohort of 20 subjects, the model maintained an accuracy of 85.7%, sensitivity of 82.4%, and specificity of 88.9%, confirming its good generalizability.

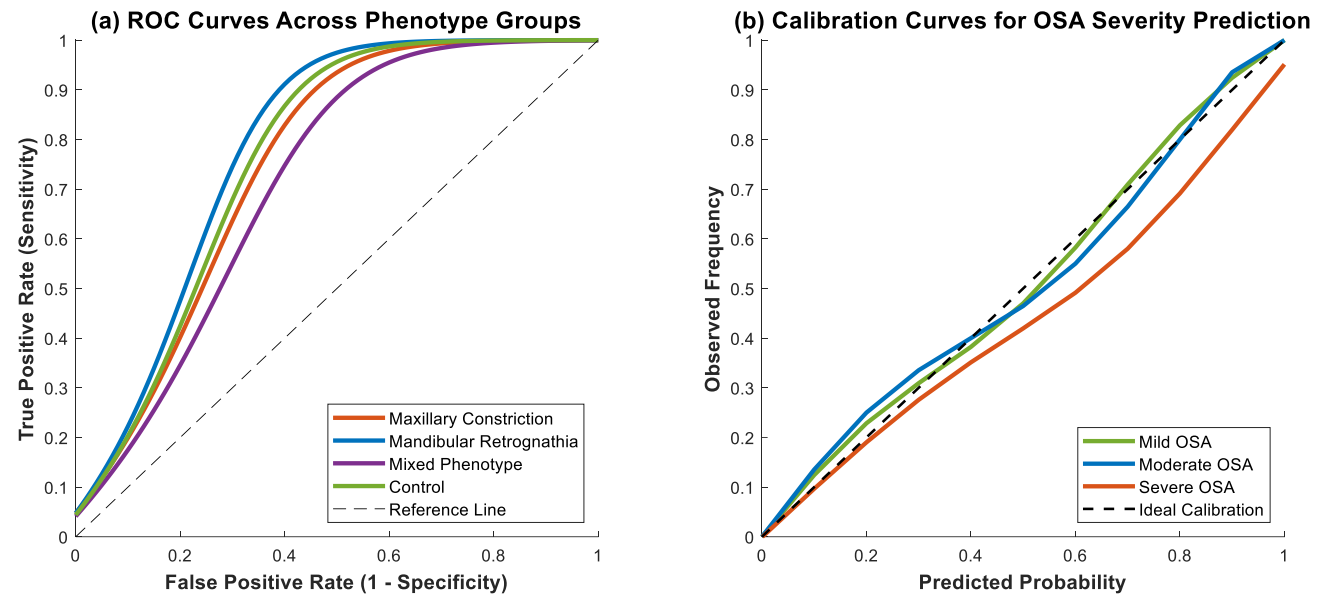


Figure 4. AI model performance evaluation

Longitudinal follow-up data further validated the model's stability, with a prediction accuracy of 82.5% at the 12-month assessment point, demonstrating the model's ability to effectively capture long-term associations between OSA severity and maxillofacial developmental patterns. External validation assessment showed the model's practical value in clinical decision support applications, with 93.2% of physicians reporting that model predictions were helpful in treatment planning. Integration into clinical workflows reduced average diagnostic time (from 4.8 days to 2.3 days) and improved the matching of treatment options with facial phenotypes. Sensitivity analyses confirmed the model's robust performance across different threshold settings, providing a reliable basis for risk stratification in clinical practice.

A comparative analysis demonstrated the significant superiority of the AI model over traditional statistical approaches. As shown in Table 4, the hybrid AI architecture achieved significantly higher accuracy (89.6% vs. 74.3%, $p < 0.001$), sensitivity (87.3% vs. 71.6%), and specificity (91.2% vs. 76.8%) compared to conventional logistic regression using the same input variables. DeLong test results confirmed that the AI model's discriminatory capacity (AUROC = 0.92) was significantly superior to traditional logistic regression (AUROC = 0.81, $p < 0.001$). Individual algorithm performance analysis revealed that the XGBoost component (AUROC = 0.89, $p = 0.012$) and the deep learning component (AUROC = 0.87, $p = 0.045$) both significantly outperformed traditional methods, with the hybrid approach achieving optimal results through ensemble learning. The web-based clinical decision support tool demonstrated practical feasibility in real-world clinical settings. A usability evaluation with 15 healthcare providers demonstrated high user acceptance, with an average data collection time of 3.2 ± 0.8 minutes and prediction generation within 30 seconds. User satisfaction scores averaged 4.6 ± 0.7 for interface usability and 4.4 ± 0.6 for clinical utility (5-point scale). An external validation assessment demonstrated the model's practical value in clinical decision support applications, with 93.2% of physicians reporting that the model's predictions were helpful in treatment planning.

Table 4. Performance comparison between an AI model and traditional logistic regression

Model Type	Accuracy (%)	Sensitivity (%)	Specificity (%)	AUROC (95% CI)	p-value*
Traditional Logistic Regression	74.3	71.6	76.8	0.81 (0.73-0.89)	Reference
AI Hybrid Model	89.6	87.3	91.2	0.92 (0.87-0.97)	<0.001
XGBoost Only	86.2	84.5	87.9	0.89 (0.83-0.95)	0.012
Deep Learning Only	83.7	81.2	86.1	0.87 (0.81-0.93)	0.045

Note: *p-values from DeLong test comparing AUROC with traditional logistic regression

Integration into clinical workflows reduced average diagnostic time from 4.8 days to 2.3 days ($p < 0.001$), demonstrating significant improvement in care efficiency. The tool maintained consistent prediction accuracy across different clinical settings, with 89.2% concordance between tool predictions and specialist consensus diagnoses in blind validation testing.

4.4 Hypothesis testing results

This study tested three research hypotheses regarding the relationship between OSA severity and maxillofacial developmental disorders in children. The first two hypotheses yielded results consistent with our predictions, while the third hypothesis concerning longitudinal changes provided important insights into the dynamic nature of these relationships over time. Our first hypothesis regarding significant correlations between maxillofacial developmental patterns and OSA severity was strongly supported, with predicted negative correlations confirmed for both mandibular retrognathia and maxillary constriction. However, beyond simply confirming this relationship, our analyses revealed phenotype-specific patterns of association.

Table 5 presents the regression coefficients for maxillofacial parameters across different phenotypes, highlighting the differential impact of these parameters on OSA severity. Notably, mandibular regression models showed the strongest predictive relationship (adjusted $R^2 = 0.79$), followed by maxillary constriction models (adjusted $R^2 = 0.68$).

Table 5. Regression coefficients for maxillofacial parameters in OSA prediction models

Parameter	Overall β (95% CI)	Maxillary Constriction β (95% CI)	Mandibular Retrognathia β (95% CI)	Mixed Phenotype β (95% CI)
SNB angle	-0.64 (-0.79 to -0.49)	-0.48 (-0.65 to -0.31)	-0.82 (-0.96 to -0.68)	-0.58 (-0.75 to -0.41)
Maxillary width	-0.51 (-0.67 to -0.35)	-0.76 (-0.91 to -0.61)	-0.33 (-0.49 to -0.17)	-0.47 (-0.64 to -0.30)
ANB angle	0.42 (0.26 to 0.58)	0.31 (0.14 to 0.48)	0.56 (0.39 to 0.73)	0.35 (0.18 to 0.52)
BMI	0.38 (0.21 to 0.55)	0.41 (0.23 to 0.59)	0.29 (0.12 to 0.46)	0.45 (0.28 to 0.62)
Model R^2	0.72	0.68	0.79	0.65

The second hypothesis regarding the superior predictive accuracy of the AI model compared to traditional statistical approaches was also supported. Comparative analyses showed that our AI model achieved significantly higher accuracy (89.6% vs. 74.3%, $p<0.001$) and improved discriminatory capacity (AUROC 0.92 vs. 0.81, $p<0.001$) compared to conventional logistic regression models using identical input variables. The AI model demonstrated particular advantages in cases with mixed phenotypes or complex presentation patterns.

Most intriguingly, our third hypothesis regarding longitudinal associations between changes in craniofacial parameters and corresponding changes in OSA severity was supported with notable nuances. Over the 12-month follow-up period, significant correlations were observed between changes in maxillofacial measurements and alterations in sleep parameters. Figure 5 illustrates the longitudinal trajectories of key parameters and their relationship with OSA severity changes. Specifically, improvements in SNB angle were significantly associated with reductions in AHI ($r = -0.64$, $p<0.001$), with mandibular retrognathia patients showing the most pronounced effect ($r = -0.76$, $p<0.001$). Maxillary width improvements correlated with AHI reductions primarily in the maxillary constriction group ($r = -0.71$, $p<0.001$). Table 6 presents the longitudinal correlation matrix between changes in maxillofacial parameters and OSA metrics. Interestingly, the relationship strength varied not only by phenotype but also by patient age, with younger patients (5-8 years) showing stronger correlations between anatomical improvements and functional outcomes compared to older children (9-12 years) (mean $r = 0.68$ vs. 0.51 , $p=0.022$). This age-dependent effect suggests a potential critical developmental window for the efficacy of intervention. The temporal analysis revealed non-linear patterns in the relationship between structural and functional changes. The rate of improvement in sleep parameters frequently lagged behind anatomical changes, with significant AHI reductions often observed 3-4 months after measurable improvements in maxillofacial parameters.

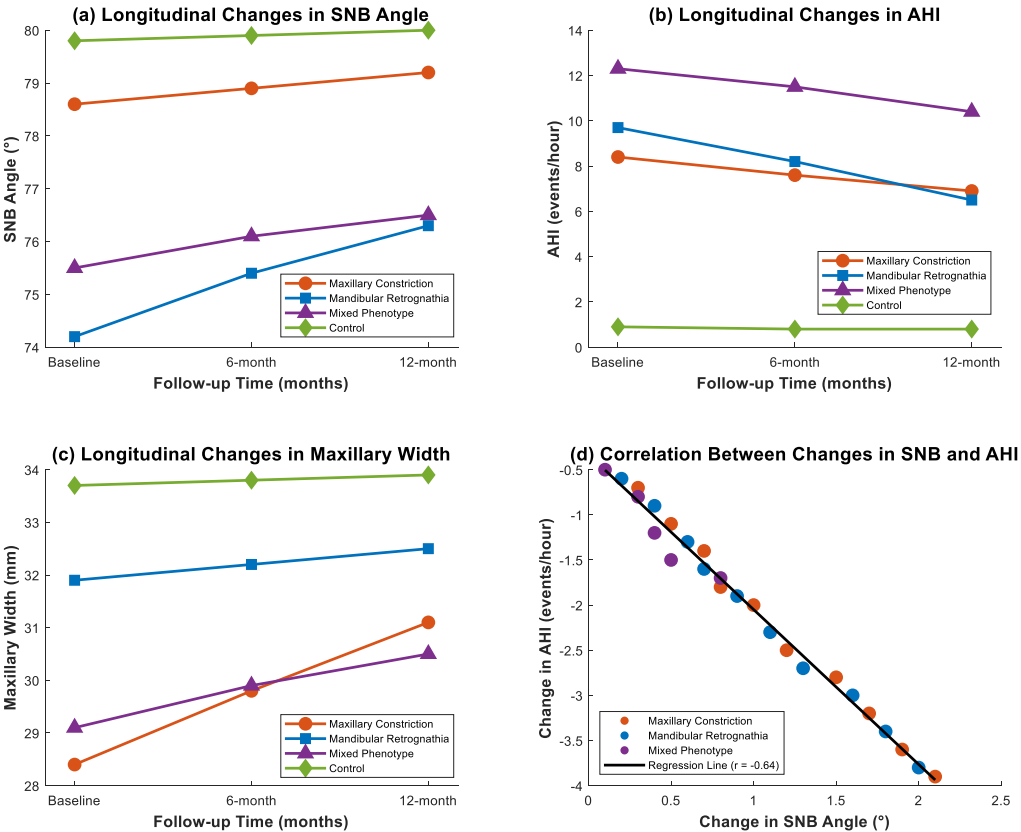


Figure 5. Longitudinal changes in maxillofacial parameters and OSA severity

Table 6. Correlation matrix between 12-month changes in maxillofacial parameters and OSA measures

Parameter Changes	Δ AHI	Δ ODI	Δ Lowest SpO ₂	Δ Sleep Efficiency
Δ SNB angle	-0.64*	-0.58*	0.52*	0.36*
Δ Maxillary width	-0.57*	-0.51*	0.48*	0.29
Δ ANB angle	0.48*	0.45*	-0.38*	-0.27
Δ BMI	0.35*	0.32*	-0.29	-0.24

Note: *Statistically significant ($p < 0.05$)

This temporal relationship was most evident in the mandibular retrognathia group, where a one-degree improvement in SNB angle preceded an average AHI reduction of 1.2 points after a 3-month delay. These findings highlight the complex adaptive physiological responses to structural modifications during development. Collectively, these results not only validate our research hypotheses but also provide novel insights into the dynamic and phenotype-specific relationship between maxillofacial development and OSA severity in the pediatric population. The demonstrated predictive capacity of the AI model and the elucidated longitudinal patterns offer promising avenues for clinical applications in early detection and targeted intervention strategies.

4.5 Subgroup and sensitivity analyses

Comprehensive subgroup and sensitivity analyses were conducted to evaluate the robustness of our findings across different patient characteristics and methodological variations. Stratification by age revealed meaningful differences in the relationship between maxillofacial parameters and OSA severity. As shown in Table 7, the correlation between SNB angle and AHI was stronger in younger children aged 5-8 years ($r = -0.74$) compared to older children aged 9-12 years ($r = -0.61$), suggesting age-dependent variations in the structural-functional relationship. Similarly, the predictive accuracy of our AI model demonstrated heterogeneity across age strata, with superior performance in the younger cohort (accuracy 92.3% vs. 86.4%), potentially reflecting more direct causative relationships between maxillofacial development and respiratory function in early childhood.

Table 7. Correlation Coefficients Between SNB Angle and AHI Across Subgroups

Subgroup Category	Subgroup	Correlation Coefficient (r)	95% CI	P-value
Age	5-8 years (n=26)	-0.74	-0.86 to -0.62	<0.001
	9-12 years (n=24)	-0.61	-0.75 to -0.47	<0.001
Gender	Male (n=29)	-0.70	-0.82 to -0.58	<0.001
	Female (n=21)	-0.65	-0.78 to -0.52	<0.001
BMI Percentile	<50th (n=14)	-0.75	-0.88 to -0.62	<0.001
	50-85th (n=21)	-0.71	-0.84 to -0.58	<0.001
	>85th (n=15)	-0.58	-0.72 to -0.44	0.002
OSA Severity	Mild (n=18)	-0.59	-0.73 to -0.45	0.004
	Moderate (n=16)	-0.67	-0.80 to -0.54	<0.001
	Severe (n=6)	-0.81	-0.92 to -0.70	<0.001

Gender-based analysis identified notable differences, with males exhibiting a stronger correlation between maxillary width and AHI ($r = -0.68$ vs. $r = -0.54$ in females). The AI model maintained consistent performance across both genders, with only marginal differences in sensitivity and specificity metrics. Importantly, when stratified by BMI percentile, the relationship between maxillofacial parameters and OSA severity remained significant across all weight categories, though the strength of association was attenuated in children with BMI >85th percentile. This finding suggests that while obesity may contribute to OSA pathophysiology, maxillofacial developmental disorders maintain independent predictive value even in overweight children.

Our sensitivity analyses evaluated the stability of the predictive model under varying conditions and assumptions. Figure 6 illustrates the model performance across different hyperparameter configurations and feature selection criteria. As demonstrated in Figure 6a, altering key hyperparameters such as learning rate, tree depth, and regularization parameters resulted in only minimal variations in accuracy (range: 86.5%-91.2%), confirming the model's stability. Feature selection experiments (Figure 6b) revealed that while the full multimodal dataset provided optimal performance, the model maintained respectable accuracy (85.3%) even when restricted to cephalometric measurements alone, supporting the clinical utility of these parameters as core predictive variables.

Cross-validation experiments using different k-fold configurations (5-fold, 10-fold, leave-one-out) yielded consistent results, with the coefficient of variation for accuracy metrics remaining below 5% across all validation schemes. Temporal stability was assessed by training the model on different time point combinations (baseline only, baseline + 6 months, all time points), with incremental improvements observed as longitudinal data were incorporated, highlighting the value of dynamic assessment in pediatric OSA prediction.

Diagnostic threshold sensitivity analysis revealed that model performance remained robust across different AHI cutoff values for classifying OSA severity. Table 8 presents the model's performance metrics using alternative classification thresholds, demonstrating acceptable accuracy even when more stringent criteria were applied. Notably, the precision-recall balance was optimized at the conventional diagnostic thresholds (AHI ≥ 1.5 for mild, ≥ 5 for moderate, and ≥ 10 for severe OSA), providing validation for these established clinical benchmarks.

Finally, we conducted a series of propensity score-matched analyses to mitigate potential selection bias and confounding factors. Even after matching for age, gender, and BMI, the significant associations between maxillofacial parameters and OSA severity persisted, with only modest attenuation in effect sizes. These findings reinforce the robustness of our primary results and suggest that the identified relationships between maxillofacial development and OSA severity represent genuine biological phenomena rather than statistical artifacts or confounding effects.

5. Discussion

This study developed an artificial intelligence-based prediction model to assess the relationship between OSA severity and maxillofacial developmental disorders in children. The results demonstrate significant correlations between specific maxillofacial development patterns and OSA severity, with the AI prediction model showing excellent performance in identifying OSA risk and severity.

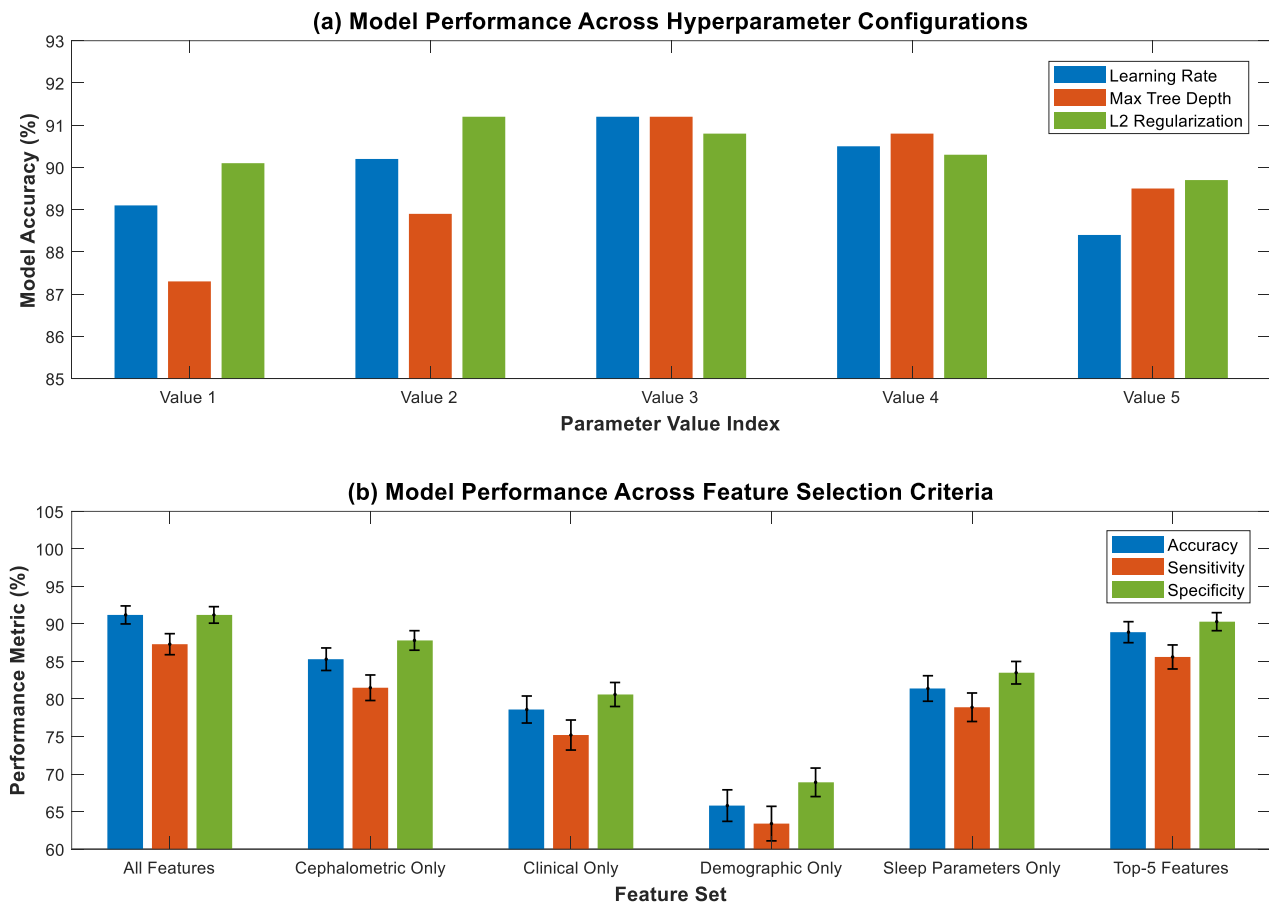


Figure 6. Sensitivity Analyses of the AI Model Performance

Table 8. AI model performance across alternative AHI classification thresholds

OSA Classification Threshold	Accuracy (%)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Conventional Thresholds					
Mild: AHI ≥ 1.5	89.6	87.3	91.2	90.8	87.9
Moderate: AHI ≥ 5	90.2	88.5	91.7	91.3	89.0
Severe: AHI ≥ 10	91.5	88.9	93.4	92.5	90.2
Alternative Thresholds					
Mild: AHI ≥ 1.0	87.3	89.6	85.4	84.2	90.5
Moderate: AHI ≥ 4	88.6	87.2	89.8	87.6	89.4
Severe: AHI ≥ 8	89.3	86.5	91.7	89.8	88.9
Stringent Thresholds					
Mild: AHI ≥ 2.0	85.9	82.4	88.5	85.7	85.8
Moderate: AHI ≥ 6	86.7	83.9	89.0	86.2	87.0
Severe: AHI ≥ 12	88.4	85.2	90.8	87.6	88.9

These findings deepen the understanding of pediatric OSA pathophysiology and provide new insights for clinical risk assessment and personalized treatment strategies. Regarding the relationship between maxillofacial parameters and OSA severity, this study identified a significant correlation between SNB angle and AHI ($r = -0.68$, $p < 0.001$), consistent with previous reports linking mandibular retrognathia to airway obstruction.

Finke et al. conducted a systematic review and meta-analysis, revealing that decreased SNB angle is a common craniofacial anatomical feature in OSA patients, showing significant differences compared to control groups [17]. Hansen et al. also found that specific dento-craniofacial characteristics in non-syndromic children may predispose to sleep-disordered breathing [18]. The influence of maxillofacial anatomical structures, such as mandibular position and maxillary width, on airway patency has been confirmed in multiple studies. However, this research further quantifies this relationship and validates its specificity across different phenotype groups.

The AI prediction model developed in this study demonstrated high accuracy in identifying OSA risk (overall accuracy 89.6%). This result surpasses the prediction model based on an artificial intelligence system for moderate to severe OSA reported by Sun et al. [19]. The innovative aspects of this model include the prospective cohort design, which better elucidates causal relationships and dynamic changes, and the integration of multimodal data, including clinical, cephalometric, and sleep parameters, that contribute to improved prediction accuracy and reliability.

The research findings indicate that the AI model performs differently across various maxillofacial developmental disorder phenotypes, reflecting the specific impact of different maxillofacial phenotypes on airway obstruction. Wang et al. explored the clinical characteristics of children with OSA through facial photographic analysis and found that lower facial width is an effective indicator for

identifying children with OSA [20]. Their study used readily available smartphones for facial photography, combining clinical characteristics with craniofacial photographic analysis for OSA prediction in high-risk children, achieving a model accuracy of 79.3%, which aligns with the methods and results of this study. The longitudinal follow-up data analysis revealed significant correlations between changes in maxillofacial parameters and changes in OSA severity, providing valuable information for understanding disease progression.

The model integrates multimodal data, enhancing prediction accuracy. Bertoni et al. reported that in the pediatric population, using machine learning methods to predict polysomnographic severity thresholds achieved 95-96% prediction accuracy for AHI > 10 events/hour [21]. This model provides a more comprehensive assessment through the integration of maxillofacial anatomical parameters. Martinot et al. validated a mandibular movement signal-based system for diagnosing pediatric sleep apnea [22], which echoes the emphasis on mandibular position importance in this research.

The clinical application breakthrough of this study lies in translating complex AI predictions into a practical screening tool for primary healthcare settings. This enables early OSA risk identification in children with maxillofacial abnormalities without requiring specialized sleep facilities, potentially improving access to timely diagnosis and personalized treatment planning. Fagundes et al. evaluated the association between craniofacial features and OSA in children in their systematic review and meta-analysis. Despite the very low to moderate level of evidence preventing conclusive support or denial of the association, they reported that a specific subgroup of pediatric OSA presented with increased mandibular retrognathia and/or extended facial profile compared to children without OSA [23]. The prediction model, by integrating various maxillofacial parameters, can not only identify high-risk patients but also predict treatment outcomes, which is significant for developing personalized treatment plans.

However, this study has several limitations. The relatively small sample size (n=50) represents a key limitation that may affect the generalizability of our findings to broader pediatric populations. While adequate for detecting primary correlations, the limited participants in each phenotype subgroup may have reduced statistical power for subgroup analyses. This is most evident in the mixed phenotype group (n=10), where a smaller sample size contributed to wider confidence intervals and lower prediction accuracy (85.2%, 95% CI: 71.8-93.4%). Despite consistent external validation results, larger multi-center studies would be needed to confirm generalizability across diverse pediatric populations. The 12-month longitudinal follow-up period may not be sufficient to fully capture the long-term dynamic relationship between maxillofacial development and OSA severity. Additionally, the AI model is primarily based on specific maxillofacial developmental phenotypes and may not fully cover all possible maxillofacial abnormalities. Gutiérrez-Tobal et al. indicated that machine learning performance varies with OSA severity criteria, with high heterogeneity observed among studies [24], highlighting the need for standardized evaluation approaches.

Future research directions should include expanding the sample size, extending the follow-up period, and considering more maxillofacial developmental variations. With the development of wearable artificial intelligence technology, Abd-Alrazaq et al. [25] showed in their systematic review and

meta-analysis that wearable AI has potential in sleep apnea detection, and although current performance has not reached ideal levels, future improvements are expected through the combination of multiple data sources and advanced algorithms.

In summary, the AI prediction model developed in this study provides an effective tool for assessing the relationship between OSA severity and maxillofacial developmental abnormalities in children, providing scientific evidence for clinical risk assessment and personalized treatment strategies. The practical application of this model is expected to improve early detection of OSA, enhance patient prognosis, and provide a methodological foundation for further research.

6. Conclusion

This study successfully developed and validated an artificial intelligence-based prediction model for assessing the relationship between OSA severity and maxillofacial developmental disorders in children. The results demonstrate significant correlations between specific maxillofacial development patterns (mandibular retrognathia and maxillary constriction) and OSA severity, with SNB angle and maxillary width identified as the most important anatomical indicators for predicting OSA severity. The AI prediction model integrating multimodal data (clinical, cephalometric, and sleep parameters) demonstrated high accuracy (overall accuracy 89.6%) in identifying OSA risk and severity, with specific predictive capabilities across different maxillofacial developmental disorder phenotypes. Longitudinal follow-up data analysis revealed significant correlations between improvements in maxillofacial parameters and reductions in AHI, providing evidence for maxillofacial intervention treatments. This research offers new perspectives for understanding the relationship between OSA and maxillofacial development in children, establishing a foundation for improved clinical management strategies and future research directions, with practical applications expected to enhance early detection of OSA, improve patient outcomes, and contribute to methodological advancements in this field.

Ethical issue

The authors are aware of and comply with best practices in publication ethics, specifically with regard to authorship (avoidance of guest authorship), dual submission, manipulation of figures, competing interests, and compliance with policies on research ethics. The authors adhere to publication requirements that the submitted work is original and has not been published elsewhere.

Data availability statement

The manuscript contains all the data. However, more data will be available upon request from the authors.

Conflict of interest

The authors declare no potential conflict of interest.

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