Vision of Pediatrics

Short Communication

Respiration and Growth in Childhood

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Received: March 06th, 2026; **Accepted:** March 17th, 2026; **Published:** March 18th, 2026

Citation: Jeferson Sampaio d'Avila. Respiration and Growth in Childhood. *Vis Pediatr.* 2026; 1(1): 001-004.

Abstract

Child growth represents one of the most sensitive indicators of pediatric health. When children fail to achieve expected growth trajectories, clinicians commonly investigate endocrine, genetic or nutritional causes. However, growing evidence indicates that respiratory disorders and sleep disturbances may significantly influence somatic growth and endocrine regulation.

Conditions such as mouth breathing syndrome, adenotonsillar hypertrophy, allergic rhinitis and pediatric obstructive sleep apnea may disrupt sleep architecture and interfere with the physiological regulation of the growth hormone–insulin-like growth factor-1 (GH–IGF-1) axis. Because growth hormone secretion occurs predominantly during slow-wave sleep, sleep fragmentation and intermittent hypoxia may reduce GH secretion and consequently decrease IGF-1 production.

Clinical studies have demonstrated that treatment of upper airway obstruction, particularly adenotonsillectomy, may restore normal sleep physiology and improve growth patterns in children. The present review discusses the relationship between respiration, sleep physiology and endocrine regulation of growth, highlighting the importance of recognizing sleep-disordered breathing as a potential contributor to growth delay in childhood.

Keywords: Child growth; IGF-1; Adenotonsillectomy; Pediatric sleep apnea; Mouth breathing; Waldeyer's ring

Introduction

Childhood growth reflects a complex interaction between genetic potential, endocrine regulation, nutritional status and overall health. When growth delay occurs, clinicians typically investigate endocrine disorders such as growth hormone deficiency, hypothyroidism or systemic diseases. However, increasing evidence indicates that respiratory disorders and sleep disturbances may also play a significant role in impaired growth during childhood [1-3].

Upper airway obstruction is common in pediatric populations and is frequently associated with adenotonsillar hypertrophy, allergic rhinitis and mouth breathing syndrome. These conditions may produce sleep fragmentation and intermittent

hypoxia, mechanisms capable of altering the physiological secretion of growth hormone and interfering with the GH–IGF-1 axis [3].

Recognition of sleep-disordered breathing as a systemic condition has expanded significantly in recent decades. Pediatric obstructive sleep apnea is now understood to affect not only respiratory function but also metabolic, cardiovascular and neurocognitive regulation [4-6].

Sleep Physiology and the GH–IGF-1 Axis

Growth hormone (GH) is secreted by the anterior pituitary gland in a pulsatile manner, with the highest peaks occurring during slow-wave sleep. This nocturnal secretion stimulates hepatic production of insulin-like growth factor-1 (IGF-1), a peptide responsible for mediating many of the biological effects of GH, including longitudinal bone growth, cellular proliferation and tissue development [7].

Experimental studies have demonstrated that sleep fragmentation significantly reduces the amplitude of GH pulses. Consequently, decreased IGF-1 production may occur in children with chronic sleep disruption, leading to reduced growth velocity [8].

In addition to hormonal mechanisms, inflammatory mediators associated with chronic airway obstruction may contribute to alterations in metabolic regulation. Persistent airway inflammation may increase energy expenditure and reduce the efficiency of growth processes in developing children [9].

Upper Airway Obstruction and Pediatric Sleep Apnea

Hypertrophy of Waldeyer's lymphatic ring, particularly the palatine tonsils and adenoids, represents the most common cause of upper airway obstruction in childhood. Pediatric obstructive sleep apnea associated with adenotonsillar hypertrophy has been widely described and may produce systemic physiological consequences [3,10].

Children with obstructive sleep apnea may present with snoring, mouth breathing, fragmented sleep and behavioral disturbances. In addition to these manifestations, untreated sleep apnea has been associated with cardiovascular alterations, blood pressure variability and neurocognitive impairment [11-13].

Effects of Adenotonsillectomy on Growth

Adenotonsillectomy remains the primary surgical treatment for pediatric obstructive sleep apnea. Evidence from clinical trials and cohort studies indicates that surgical treatment may restore airway patency, normalize sleep architecture and produce systemic benefits [1,14].

Several investigations have reported improvements in height velocity and weight gain following adenotonsillectomy. Restoration of physiological sleep patterns may reestablish GH secretion and IGF-1 production, allowing children to recover normal growth trajectories, a phenomenon frequently described as catch-up growth [2,15].

Clinical research conducted in Brazil also demonstrated significant improvements in height and weight development after adenotonsillectomy, reinforcing the relationship between airway obstruction and impaired growth in children [13].

Systemic Consequences of Pediatric Sleep Apnea

Beyond its impact on sleep quality, pediatric obstructive sleep apnea may produce systemic physiological consequences. Studies evaluating cardiovascular regulation have demonstrated alterations in autonomic nervous system activity and increased blood pressure variability in affected children [11,12].

Other investigations evaluating cardiorespiratory control have shown improvements in thoraco-abdominal synchrony and respiratory mechanics after adenotonsillectomy, reinforcing the systemic nature of sleep-disordered breathing [16,17].

Neurocognitive consequences have also been reported, including deficits in attention, learning and behavioral regulation, which may improve after treatment of airway obstruction [18].

Literature Review

Recent literature increasingly emphasizes the relationship between respiratory disorders and impaired childhood growth. Large clinical trials such as the Childhood Adenotonsillectomy Trial (CHAT) demonstrated that surgical treatment significantly improves sleep quality, behavior and overall quality of life in children with obstructive sleep apnea [1].

Population-based cohort studies have confirmed improvements in height and weight development after adenotonsillectomy, suggesting that airway obstruction may influence metabolic and endocrine growth regulation [2].

Clinical investigations have also demonstrated normalization of IGF-1 levels after surgical treatment of obstructive sleep apnea, supporting the hypothesis that restoration of physiological sleep architecture may reestablish the GH–IGF-1 axis [19].

Recent reviews published in major respiratory and pediatric journals emphasize that persistent pediatric obstructive sleep apnea requires early diagnosis and multidisciplinary management to prevent long-term systemic consequences [3,20]. Taken together, the current evidence suggests that sleep-disordered breathing should be considered an important factor in the evaluation of children presenting with unexplained growth delay.

Conclusion

Growth delay in childhood should not always be interpreted solely as an endocrine disorder. Respiratory obstruction, sleep fragmentation and chronic inflammatory conditions may significantly influence growth patterns.

Recognition of the interaction between sleep physiology, airway obstruction and endocrine regulation is essential for comprehensive pediatric evaluation. Early diagnosis and appropriate management of sleep-disordered breathing may restore normal growth trajectories and improve overall pediatric health.

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