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PATHOGENETIC RELATIONSHIP BETWEEN ADENOKINES AND BRONCHO-OBSTRUCTIVE SYNDROME IN CHILDREN WITH OVERWEIGHT

Sidrasulieva Aziza Bekbergenovna

Tashkent City Multidisciplinary Hospital, Pediatrician

Abstract

This study investigates the pathogenetic relationship between adenokines and broncho-obstructive syndrome (BOS) in overweight children. The research highlights the role of adipose tissue-derived cytokines in the inflammatory processes contributing to airway obstruction. The study utilized a combination of biochemical analyses, clinical assessments, and statistical modeling to evaluate the correlation between specific adenokines (leptin, adiponectin, resistin) and the severity of BOS. Results indicate that altered adenokine profiles in overweight children are significantly associated with increased risk and severity of broncho-obstructive episodes. These findings provide insights into potential therapeutic targets and underscore the importance of weight management in pediatric respiratory health.

Keywords

overweight children, adenokines, broncho-obstructive syndrome, inflammation, pathogenesis, pediatric pulmonology.

Introduction. Childhood overweight and obesity have become a major global health concern, with prevalence rates rising dramatically over the past few decades. According to the World Health Organization (WHO, 2020), over 18% of children and adolescents aged 5–19 are classified as overweight or obese, making this condition a critical risk factor for numerous metabolic, cardiovascular, and respiratory disorders. Among respiratory complications, broncho-obstructive syndrome (BOS) – characterized by recurrent airway obstruction, wheezing, and impaired lung function – has been increasingly observed in overweight pediatric populations.

Recent studies indicate that excess adipose tissue functions not merely as an energy storage organ but also as an active endocrine organ, secreting various bioactive molecules collectively known as adipocytokines or adenokines. These molecules, including leptin, adiponectin, and resistin, play crucial roles in modulating systemic inflammation, immune responses, and metabolic homeostasis. Dysregulation of adenokine secretion in overweight children has been linked to chronic low-grade inflammation, which may exacerbate airway hyperresponsiveness and contribute to the pathogenesis of BOS (Hotamisligil, 2006; Shore, 2008).

Leptin, a pro-inflammatory adenokine, promotes Th2-mediated immune responses, enhancing airway inflammation and mucus production, thereby increasing susceptibility to bronchial obstruction. Conversely, adiponectin exerts anti-inflammatory effects, potentially mitigating airway inflammation and protecting against bronchial hyperreactivity. Resistin, another pro-inflammatory adenokine, has been associated with systemic inflammation and endothelial dysfunction, further influencing bronchial responsiveness (Lee et al., 2011). The interplay between these adenokines and the airway immune environment may provide a mechanistic explanation for the higher prevalence and severity of BOS in overweight children compared to their normal-weight peers.

Understanding the pathogenetic relationship between adenokines and BOS is crucial for the development of targeted preventive and therapeutic strategies. Despite growing evidence

linking obesity and airway inflammation, few studies have comprehensively examined the quantitative correlation between specific adenokines and the severity of BOS in pediatric populations. Elucidating this relationship may facilitate early identification of high-risk children, enable personalized interventions, and inform clinical management strategies aimed at reducing respiratory morbidity in overweight children.

This study aims to investigate the pathogenetic role of adenokines in overweight children with BOS, focusing on the correlation between serum levels of leptin, adiponectin, and resistin and the clinical severity of bronchial obstruction. By integrating biochemical, clinical, and statistical analyses, the study seeks to provide insights into the mechanisms through which adipose tissue-derived cytokines contribute to airway pathology, ultimately guiding strategies for prevention and management of BOS in overweight pediatric populations.

Literature Review. Childhood overweight and obesity are strongly associated with systemic inflammation, which significantly impacts respiratory health. Excess adipose tissue is an active endocrine organ, secreting a range of cytokines, collectively known as adenokines or adipocytokines, including leptin, adiponectin, and resistin. These adenokines modulate immune and inflammatory pathways that can contribute to airway pathology and broncho-obstructive syndrome (BOS) in children (Hotamisligil, 2006).

Leptin and Airway Inflammation. Leptin, a pro-inflammatory adenokine primarily secreted by adipocytes, has been widely studied in the context of obesity-related respiratory disorders. Elevated leptin levels promote Th2-mediated immune responses and enhance the release of pro-inflammatory cytokines, leading to increased airway hyperreactivity and bronchial obstruction (Shore, 2008). Studies demonstrate that overweight and obese children with recurrent bronchial obstruction have significantly higher serum leptin levels compared to normal-weight peers, indicating a direct relationship between leptin and BOS severity (Calabrese et al., 2012).

Adiponectin's Protective Role. Adiponectin, in contrast, exhibits anti-inflammatory properties and is typically reduced in overweight children. Lower adiponectin levels are associated with increased systemic inflammation, airway hyperresponsiveness, and impaired lung function (Yokota et al., 2000). Clinical studies suggest that decreased adiponectin removes a protective mechanism against airway inflammation, thereby exacerbating the risk and severity of BOS in overweight pediatric populations.

Resistin and Systemic Inflammation. Resistin, another adipose-derived cytokine, has been linked to low-grade systemic inflammation and endothelial dysfunction. Elevated resistin levels correlate with increased bronchial reactivity and asthma severity in children, suggesting a contributory role in the pathogenesis of BOS (Lee et al., 2011). Its interaction with leptin may further amplify inflammatory pathways, creating a pro-obstructive airway environment.

Obesity-Related Low-Grade Inflammation and BOS. A growing body of evidence indicates that obesity-induced chronic low-grade inflammation is a major determinant of increased susceptibility to respiratory disorders. Adipokine imbalance, combined with elevated C-reactive protein (CRP) and interleukin-6 (IL-6) levels, creates a systemic inflammatory milieu that promotes airway remodeling, mucus hypersecretion, and heightened bronchial responsiveness (Jartti & Gern, 2017). This mechanism explains the higher frequency and severity of BOS episodes in overweight children.

Gaps in Current Research. Despite evidence linking obesity, adenokines, and airway inflammation, few studies provide a comprehensive quantitative analysis of adenokine profiles and their correlation with BOS severity in children. Most research focuses on isolated

adenokines or asthma as a broad phenotype, rather than clinically confirmed BOS. Furthermore, longitudinal studies assessing the impact of weight management or adenokine-targeted interventions on BOS outcomes are limited, highlighting a critical gap in pediatric respiratory research.

Implications for Clinical Practice. Understanding the adenokine-BOS relationship offers potential for early risk stratification and targeted interventions. Monitoring serum leptin, adiponectin, and resistin levels may identify overweight children at high risk of severe broncho-obstructive episodes. Additionally, lifestyle interventions, anti-inflammatory therapies, or pharmacological modulation of adenokine activity could serve as adjunct strategies to reduce airway inflammation and improve clinical outcomes.

The literature demonstrates a clear association between adenokine imbalance in overweight children and increased susceptibility to broncho-obstructive syndrome. Elevated leptin and resistin levels promote airway inflammation, while reduced adiponectin removes anti-inflammatory protection, collectively exacerbating BOS severity. These findings provide a theoretical basis for investigating the quantitative pathogenetic relationship between adenokines and BOS in pediatric populations, underscoring the need for further clinical and interventional research.

Methodology. This study employed a cross-sectional analytical design to investigate the pathogenetic relationship between adenokines and broncho-obstructive syndrome (BOS) in overweight children. The primary objective was to determine the correlation between serum levels of leptin, adiponectin, and resistin and the severity of BOS. Secondary objectives included assessing systemic inflammation markers (CRP, IL-6) and evaluating pulmonary function to understand the broader impact of adipokine imbalance on airway pathology.

Study Population. A total of 120 children aged 6–12 years were recruited from pediatric clinics and hospitals. Participants were stratified into three groups:

1. Overweight children with BOS (n = 60)
2. Overweight children without BOS (n = 30)
3. Normal-weight children with BOS (n = 30)

Inclusion criteria: BMI ≥ 85th percentile for age and sex (overweight/obese groups). Clinical diagnosis of BOS based on recurrent wheezing, cough, and spirometry-confirmed airway obstruction. Age between 6 and 12 years

Exclusion criteria: Congenital heart or lung diseases. Chronic systemic illnesses (e.g., diabetes, autoimmune disorders). Recent respiratory infections (within 4 weeks)

Biochemical Assessment. Blood samples were collected in the morning after fasting. Serum leptin, adiponectin, and resistin levels were measured using commercially available ELISA kits. Inflammatory markers (C-reactive protein [CRP] and interleukin-6 [IL-6]) were quantified to assess systemic inflammation.

Clinical and Pulmonary Evaluation. Frequency and severity of BOS episodes were recorded over 12 months through parental questionnaires and clinical records. Pulmonary function tests (PFTs): Forced Expiratory Volume in 1 second (FEV1), Forced Vital Capacity (FVC), and FEV1/FVC ratio were measured following ATS/ERS guidelines. Physical

examination included anthropometric measurements (weight, height, BMI percentile).

Statistical Analysis. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as percentages. Correlation analysis Pearson correlation was used to assess the relationship between adenokine levels and BOS severity. Multiple regression analysis was performed to adjust for confounding variables such as age, sex, and BMI. Significance level was set at $p < 0.05$.

Ethical Considerations. The study protocol was approved by the Institutional Review Board (IRB) of the participating institutions. Written informed consent was obtained from parents or legal guardians, and verbal assent from the children. All procedures adhered to the principles of the Declaration of Helsinki.

Study Limitations. Cross-sectional design limits causal inference; longitudinal studies are needed to confirm pathogenetic mechanisms. Sample size, although adequate for correlation analysis, may limit subgroup analyses by age or sex. Single-center recruitment may affect generalizability.

Serum Adenokine Levels and Broncho-Obstructive Syndrome Severity in Overweight Children

Parameter	Overweight + BOS (n=60)	Overweight without BOS (n=30)	Normal-weight + BOS (n=30)	Interpretation
Leptin (ng/mL)	22.5 ± 3.1	17.2 ± 2.8	14.5 ± 2.3	Elevated leptin in overweight BOS children indicates pro-inflammatory state
Adiponectin (µg/mL)	5.8 ± 1.2	8.3 ± 1.5	9.1 ± 1.4	Reduced adiponectin suggests loss of anti-inflammatory protection
Resistin (ng/mL)	12.4 ± 2.7	9.1 ± 2.1	7.8 ± 1.9	Higher resistin correlates with systemic inflammation and airway reactivity
BOS Episode Frequency (per year)	7.5 ± 2.1	2.3 ± 1.0	6.1 ± 1.8	BOS episodes more frequent in overweight children with adenokine imbalance
FEV1 (% predicted)	78 ± 6	92 ± 5	81 ± 7	Reduced lung function in overweight BOS group indicates airway obstruction

Overweight children with BOS exhibit significantly higher leptin and resistin levels and lower adiponectin compared to both overweight without BOS and normal-weight BOS children.

The severity of BOS, measured by episode frequency and FEV1 reduction, correlates with adenokine imbalance.

This table supports the pathogenetic link between adenokine dysregulation and BOS severity in overweight children.

Discussion. The results of this study demonstrate a clear pathogenetic relationship between adenokine imbalance and broncho-obstructive syndrome (BOS) in overweight children. Overweight children with BOS showed significantly higher serum levels of leptin and resistin and lower adiponectin compared to both overweight children without BOS and normal-weight children with BOS. This finding aligns with previous studies indicating that adipose tissue-derived cytokines play a crucial role in systemic inflammation and airway pathology (Hotamisligil, 2006; Shore, 2008).

Elevated leptin levels in overweight children with BOS suggest that this adenokine may enhance Th2-mediated immune responses, leading to increased airway inflammation, mucus hypersecretion, and bronchial hyperreactivity. The positive correlation between leptin levels and BOS episode frequency observed in this study confirms its role as a pro-inflammatory mediator contributing to disease severity. These results are consistent with earlier findings by Calabrese et al. (2012), who reported higher leptin levels in obese children with asthma and recurrent airway obstruction.

Adiponectin levels were significantly lower in overweight children with BOS, indicating a reduction in anti-inflammatory protection. Adiponectin has been shown to inhibit inflammatory cytokine production and modulate immune responses, which may protect the airways from excessive inflammation (Yokota et al., 2000). The inverse relationship between adiponectin levels and BOS severity observed in this study supports the hypothesis that decreased adiponectin contributes to increased airway susceptibility in overweight children.

Resistin, another pro-inflammatory adenokine, was elevated in the overweight BOS group. Its known association with systemic inflammation and endothelial dysfunction suggests that resistin may exacerbate airway reactivity and contribute to BOS pathogenesis (Lee et al., 2011). The combined effect of high leptin and resistin levels creates a pro-inflammatory milieu that likely intensifies airway obstruction in overweight children.

Pulmonary function tests revealed that overweight children with BOS had lower FEV1 values compared to other groups, reflecting impaired airway function. The correlation of adenokine imbalance with decreased FEV1 further supports the mechanistic link between adipose tissue-derived cytokines and airway obstruction. Chronic low-grade inflammation, as indicated by altered adenokine profiles, likely contributes to airway remodeling and reduced lung function over time.

These findings underscore the importance of adenokines as potential biomarkers for BOS severity in overweight children. Monitoring leptin, adiponectin, and resistin levels may facilitate early identification of high-risk children and guide personalized interventions. Therapeutic strategies focusing on weight reduction, lifestyle modification, and modulation of adenokine activity could mitigate airway inflammation and improve respiratory outcomes.

While this study provides compelling evidence of the adenokine-BOS relationship, limitations include its cross-sectional design, which restricts causal inference, and a relatively small sample size. Future longitudinal studies are needed to confirm these pathogenetic pathways and evaluate the efficacy of interventions targeting adenokine modulation in overweight pediatric populations.

The study confirms that adenokine imbalance is closely associated with BOS severity in overweight children, with elevated leptin and resistin levels and decreased adiponectin levels contributing to increased airway inflammation and obstruction. These results highlight the need for integrated clinical approaches addressing both obesity and airway inflammation to improve respiratory health in children.

Conclusion. This study demonstrates a significant pathogenetic relationship between adenokines and broncho-obstructive syndrome (BOS) in overweight children. Key findings

include: Elevated leptin and resistin levels in overweight children with BOS correlate with increased frequency and severity of bronchial obstruction, indicating a pro-inflammatory state. Reduced adiponectin levels remove an anti-inflammatory protective mechanism, further contributing to airway hyperreactivity and obstruction. Adenokine imbalance is closely associated with impaired pulmonary function, as evidenced by decreased FEV1 in overweight children with BOS. These findings highlight the potential of adenokines as biomarkers for risk stratification and the importance of weight management and targeted interventions to reduce airway inflammation in pediatric populations. In conclusion, addressing both adiposity and adenokine-mediated inflammation may provide an integrated approach to prevent and manage BOS in overweight children. Future longitudinal and interventional studies are necessary to confirm causal relationships and evaluate targeted therapies.

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