Quantifying the Contribution of Obesity to Incident Childhood Asthma: It’s About Time

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Several studies in the past 15 years have reported a link between obesity and asthma, 2 of the most common chronic pediatric diseases. As we have understood these associations better, there has been recognition of 2 mechanistically distinct types of pediatric obesity–related asthma, one in which asthma precedes obesity and the other in which obesity precedes asthma. However, the incidences of these types of pediatric obesity–related asthma are not known. In the current issue of Pediatrics, Lang et al address this gap in knowledge and report a risk of 23% to 28% of new asthma cases attributable to obesity among children residing in the United States.

This study has many strengths. Access to detailed data in the PEDSnet system, which includes clinical and medical claims data collected longitudinally on a quarterly basis from 8 large pediatric health systems in the United States, allowed the authors to conduct a stringent analysis of asthma diagnosis using different definitions routinely used in asthma research. They included children that were diagnosed in 1 clinical encounter, in 2 or more clinical encounters, who were prescribed a controller medication, or who had evidence of airway obstruction or reactivity on spirometry as an objective measure of airway disease. They excluded those on asthma medications without a formal diagnosis and those who were diagnosed with asthma within a short period of clinic follow-up. An inclusion of large health systems across the country allowed representation of all races and ethnicities. Matching children for demographic characteristics and insurance status addressed the confounding effect of these variables. In addition, the analytic approach of modeling with and without comorbid health conditions including allergic rhinitis, food allergy, and gastroesophageal reflux disease clarified the minimal influence of these comorbidities on the relative risk of incident asthma among children who are overweight and obese. Together, this study highlights the substantial independent contribution of obesity to incident asthma in a nationally representative sample of US children.

The findings by Lang et al are important for many reasons. Childhood asthma prevalence has remained steady at ~10% for the past several years despite the implementation of programs for early diagnosis, proper management, and the mitigation of environmental exposures. On the basis of this study, with the prevalence of obesity at ~20% among children in the United States and rising in certain subsets and with obesity increasing the risk of asthma by 23% to 28%, we are looking at an ~5% increase in childhood asthma due to obesity, suggesting that over time obesity-induced asthma will become a major type of childhood asthma. Moreover, the onset of disease in childhood adds several more years of morbidity when compared with obesity-induced adult-onset asthma. With this daunting burden of disease, there is a silver
lining. Obesity may be the first modifiable risk factor for asthma, offering an opportunity for primary prevention of asthma. There has long been awareness of the cardiovascular and endocrine implications of obesity in children. It is time we recognize the effects of obesity on the pulmonary system.

However, it is important to recognize that not all children with obesity develop asthma. Researchers, in initial investigations into the mechanisms underlying pediatric obesity-related asthma, have identified a role for truncal adiposity, metabolic abnormalities including insulin resistance and dyslipidemia, and obesity-mediated systemic inflammation. All these mechanisms are notable for the lack of a role of allergy. There is consistent evidence that obesity-related asthma in children is nonallergic, severe, and poorly responsive to asthma medications as compared with normal-weight asthma. Unlike allergic asthma, there are no targeted therapies for nonallergic asthma.

Putting the findings of Lang et al in context of the potential mechanisms underlying obesity-related asthma, we suggest that clinicians consider measurement of waist circumference and quantification of metabolic abnormalities as part of their evaluation of children who are obese in an attempt to identify those at risk of developing pulmonary complications of obesity. Because normative values exist for waist circumference and for the classification of metabolic abnormalities in children, those with evidence of ≥1 of these obesity-related complications should be the ones actively screened for asthma. In addition, in light of the poor response that children with obesity and asthma have to medications, there is an urgent need for continued investigations of mechanisms that differentiate children with obesity who develop asthma from those who do not and differentiate children who become obese because of asthma from those who develop asthma because of obesity. Together, this knowledge will guide the development of novel targeted therapy for obesity-related asthma, given its poor responsiveness to current asthma medications.

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