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What is This?
Childhood Overweight and Obesity and Their Association With Asthma
Catherine Kier, MD, AE-C, and Simone A. Forde, MA

Abstract: Poor diet, decreased physical activity, and obesity are emerging as determinants that may be critical in the cause of asthma. The obesity epidemic is a worldwide problem affecting both developed and developing countries. The prevalence of obesity among US children and adolescents has tripled over the past 4 decades, and there is an all-time low in the level of physical activity among children and adolescents. Recent data show that the incidence of asthma has been climbing rapidly as well over these past 4 decades. An increasing body of literature suggests that there is an association between this parallel rise of obesity and asthma incidences. Although the exact nature of this association remains unclear, many investigators have interpreted the data to suggest that obesity both increases the risk of incident asthma and alters prevalent asthma toward a phenotype that is more difficult to control.

Keywords: asthma, obesity, overweight, children, adolescents

In children, the prevalence of obesity will nearly double to about 30% by 2030.1 Overweight and obesity are growing global epidemics. Rising trends in overweight and obesity are confirmed in developed as well as developing countries.2 Between the 1980s and 1990s, estimates of the prevalence of overweight and obesity in children increased by a magnitude of 2 to 5 times in developed countries and roughly 4 times in developing countries. For example, the prevalence of overweight and obesity rose from 11% to more than 30% in Canadian boys and from 4% to 14% in Brazilians.3

In the United States, overweight and obesity among children and adolescents continue to be a public health concern due to the rising prevalence. Therefore, the National Health and Nutrition Examination Survey (NHANES) collects data that are monitored to determine the progress toward reducing the national prevalence of overweight and obesity. Figure 1 illustrates that the rates of childhood and adolescent obesity have been rising since the 1980s. First seen among older children and teens, and later for preschoolers and toddlers, this trend toward overweight and obesity continued unabatedly into the early part of the 21st century.1 Data derived from the NHANES measurements show that the trajectory of obesity in the United States did not slow until just the past few years.

Overweight and obesity are currently defined based on body mass index (BMI), which is determined as weight measured in kilograms, divided by height, measured in square meters (kg/m²). BMI is the most widely used diagnostic tool to identify weight problems within a population. BMI ranges for children and adolescents are defined so that they take into account normal differences in body fat between boys and girls and differences in body fat at various ages. Table 1 shows BMI-for-age weight status categories and the corresponding percentiles.4 In children, 2 to 19 years of age, at risk for becoming overweight is defined as a BMI-for-age greater than or equal to the 85th percentile and less than the 95th percentile on the Centers for Disease Control and Prevention (CDC) growth charts, whereas obese is defined as greater than or equal to the 95th percentile on the CDC growth charts.5 The CDC reported that 15.6% of children and adolescents are classified as at risk for overweight,7 32% are overweight, and 17% are obese, a statistic that has tripled during the past 4 decades.8

Obesity does not discriminate among who it affects and is a burden among all segments of the American population. However, epidemiologic data demonstrate that ethnic and racial minorities have a greater prevalence of obesity compared with whites.15 National data from 1976 to 2004 analyzed 12,384 US children and adolescents ages 2 to 19 years. These data estimated the prevalence of children in the severe and morbidly obese category whose BMI is ≥99th percentile. In 1999-2004, 3.8% of children 2 to 19 years old had a BMI in the 99th percentile, with higher prevalence among boys than girls, 4.6% vs

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2.9%, respectively \( (P < .001) \). Prevalence was highest among blacks (5.7%) and Mexican Americans (5.2%) compared with whites (3.1%) \( (P < .001) \).12

Overweight/obesity has been identified as 1 of the 10 leading health indicators by Healthy People 2010. Healthy People 2010, a national health promotion and disease prevention initiative, has called for a reduction in the proportion of children and adolescents who are overweight or obese.13 However, CDC data show that little progress has been made by the United States toward the target goal. The most recent NHANES data from 2003 to 2006 showed that the prevalence of overweight, BMI ≥85th percentile, for children aged 6 to 11 years and 12 to 19 years was 33.3% and 34.1%, respectively.3 These prevalence estimations are more than 3 times the target prevalence of 5% set in Healthy People 2010.3 Although there are some signs of flattening trends, there is still no comfort gained in the health care community if these trends are not followed by a steep decline.14

Being that most overweight children become overweight adults,15 the current childhood obesity epidemic in the United States has the potential to reverse the improvements in life expectancy that were seen during the 20th century.5,16 The rise in prevalence of overweight and obesity has led to a marked increase in obesity-related diseases rarely seen in children, including obesity-associated sleep apnea, nonalcoholic fatty liver disease with resultant cirrhosis, and type 2 diabetes.7 With increased prevalence of childhood and adolescent overweight and obesity, pediatric pulmonologists are seeing a corresponding increase in the pulmonary complications of obesity,17 such as obesity-hypoventilation syndrome, obstructive sleep apnea syndrome, and asthma. Obesity-hypoventilation syndrome (also known as the Pickwickian syndrome) is defined as obesity with alveolar hypoventilation. It is a relatively uncommon finding in children who are obese, with an estimated frequency of 1% to 3%.18 Obesity has been consistently reported a risk factor for obstructive sleep apnea in children in both epidemiologic studies and sample populations.19,20 Obesity and asthma have been proposed as risk factors for each other. There has been a growing literature on possible mechanisms and relationships of obesity and asthma in children, which will be discussed in this article.

**Overweight and Obesity: Etiology and Risk Factors**

Obesity is a chronic disorder whose cause is complex and multifaceted, comprised of genetic, epigenetic, environmental, and behavioral factors.14

Literature indicates that genetic characteristics may increase an individual’s susceptibility to excess body weight.21–23 The pathogenesis of obesity is complex. Environmental factors affect one’s inherent risk for obesity, which is determined by multiple genetic factors. The identification of susceptibility genes for obesity is important for its prediction. Heritabilities for obesity-related phenotypes vary from 6% to 85% among various populations.24 Gene–gene interactions have been observed in several genes, and some genes were found to influence the effect of dietary intake and physical activity on obesity-related phenotypes. In Prader-Willi syndrome, the most frequent cause of syndromic obesity in pediatric and adult ages, a rare genetic disorder is the driving component to the onset of weight gain and eventually obesity.24

Epigenetics has gained attention recently, as an alternative perspective on the cause of metabolic disorders. The health risks associated with obesity vary among individuals but consistently include type 2 diabetes, hypertension, coronary heart disease, and cancer.25,26 Genome-wide association studies have identified less than 20 loci for obesity and diabetes—a modest number.27
Genetic and epigenetic abnormalities in the human genome, known to undergo parental imprinting, are predominantly associated with distinct developmental and pathological phenotypes. However, there is little evidence on imprinting loci for individuals affected by the current obesity epidemic. It is possible that an unidentified imprinted gene could contribute to subclinical variations in phenotype, including body weight. Genome linkage analyses have been carried out to identify new genomic loci that harbor parent-of-origin effects that influence body weight. A study in Pima Indians detected regions on maternally derived chromosomes 5 and 6 and paternally derived chromosome 10 imprinted with genes that might influence the risk of type 2 diabetes or obesity. Another study, looking into DNA samples from African American, European American, and German individuals, found evidence for at least 3 different obesity-related genetic loci with parental effects. Genome linkage analysis showed a paternally derived region for BMI 13q32 and a suggested maternal effect for BMI detected on chromosomes 10p12 and 12q24. Two similar genome-wide studies have also provided evidence for the existence of potentially imprinted loci influencing body weight.

Common to all of these studies is that many of the newly mapped loci have not previously been reported as imprinted. These genome-wide studies suggest the existence of many more genomic loci affecting body weight that are perhaps controlled by epigenetic mechanisms. Otherwise, in the general population, genetic and epigenetic factors must exist in conjunction with contributing environmental and behavioral factors such as those among an obesogenic society. The accumulation of excess body weight among adults and children during the late 1960s and early 1970s was mainly due to a fundamental change in American life, which included the replacement of water and milk with soft drinks, increased marketing for and consumption of processed foods, especially among children, and the development of a sedentary lifestyle with cable television and early computer games. This type of lifestyle, characterized by environments that promote increased food intake, nonhealthful foods, and physical inactivity, is now commonly known among our society as an obesogenic culture.

The highest risk factor for obesity in a child is the presence of obesity in one or both parents. The onset of excess weight in the toddler and preschool years also increases the risk of lifelong obesity. These types of risks fall into the category of behavioral and environmental factors and have contributed to the overweight and obesity epidemic. Communities, homes, and workplaces can influence people's health decisions. As the CDC explains, people may make decisions based on their environment or community. A parent may not want her child to walk to the store or to school because of a lack of sidewalks; a child may not eat breakfast because he notices that his parents, who are constantly on the go, do not eat breakfast either. Because of this influence, it is important to create environments that easily engage in physical activity and incorporate a healthy diet for children and adolescents.

Obesity is complex and multifactorial. Observed variance in human body weight can be accounted for by inherited factors. Obesity in children is strongly influenced by environmental factors such as portion sizes for prepared foods, fast food service, and elements of the built environment. Certain behaviors, such as increased caloric intake, decreased physical activity, and increased sedentary lifestyle, can be identified as potentially contributing to an energy imbalance and, consequently, to obesity. Since the different factors (genetic, environmental, and behavioral) that contribute to childhood obesity interact with each other, it is impossible to specify a single cause of obesity.

Asthma: Overview and Epidemiology

Asthma is a respiratory disorder diagnosed by a clinician assessing the patient's history and presentation. This type of diagnosis involves a process of searching for the 5 key symptoms of asthma: cough, wheeze, dyspnea, chest tightness, and increased mucus production. There is no one test that can discern whether an individual is asthmatic; however, spirometry and methacholine challenge, for confirmation of airway reactivity, are 2 objective measures that can be used to determine the presence of asthma in a patient presenting with respiratory complaints. Also, there is a proven correlation between asthma and atopy or heightened immunoglobulin E (IgE) levels.

The interaction between a child's genes and the environment determines the presence and severity of asthma. Koeppe-Schomerus et al carried out a study on twin girls, which demonstrated that a child's genetic endowment is the most important predictor of the diagnosis of asthma. Pianosi and Davis determined that asthma can cause obesity through adverse effects of medications, social stigma, and decreased exercise tolerance. There has been a dramatic increase in the prevalence of childhood asthma over the last 30 years. Along with overweight, the prevalence of childhood asthma is increasing. Asthma is now the most common chronic illness seen among children, affecting 4% to 8% of all US children. Acute asthma exacerbations incur significant health care costs and are among the most common causes of visits to the emergency department.

Although genetic factors appear to play a major role in determining asthma, they cannot explain this rapid increase in the prevalence of asthma. The increase in asthma prevalence among children and adolescents must be attributable to the environment as well.

Defining the Link Between Asthma and Obesity

Asthma prevalence and obesity prevalence are both increasing in the United States. Obesity has been suggested in numerous recent studies as one of the risk factors for developing asthma. In a recent meta-analysis, asthma was more likely in patients who were overweight or obese than in patients who had a normal body weight (odds ratio 1.5;
The incidence of asthma increased as BMI increased. Increased BMI may also be associated with increased asthma severity. It has been postulated that obesity is a higher asthma risk factor in women compared with men. This risk may be greater for phenotype nonallergic asthma than allergic asthma. Relative steroid resistance has been reported in the asthma obese group. One proof-of-concept study showed that there is no evidence that the relationship between obesity and asthma in humans is mediated via the “classical” forms of airway inflammation. Systemic and airway inflammation appear to operate independently of one another. Other mechanisms, possibly innate immunity and dynamic changes in lung function, may explain the asthma–obesity relationship. This may explain the apparent need for increased inhaled anti-inflammatory therapy in obese patients with asthma and the difficulties in achieving good asthma control.

It is also reported that increasing BMI may be a risk factor for asthma in children. In a study of 5944 children, asthma, wheezing, and inhaler use were more common in obese children than in nonobese children, and symptoms were more prevalent among obese boys. However, it has been debated whether obesity-related chest symptoms mimic asthma. Adult studies also have looked into the possibility that the high risk factor for asthma is overstated. The NHANES III data analysis showed that obesity was an independent risk factor for dyspnea (subjective self-report) but not for airflow obstruction (objective). Many more obese than nonobese participants were using bronchodilators despite a lack of objective evidence for airflow obstruction. These data suggest that mechanisms other than airflow obstruction are responsible for dyspnea in obesity and that asthma might be overdiagnosed in the obese population.

Mechanical, immunological/inflammatory, hormonal, genetic, and other effects of obesity may play a role in the development and persistence of asthma.

Obesity affects the mechanics of breathing. It is known that lung volumes, primarily functional residual capacity and expiratory reserve volume, are reduced in obese individuals. The primary basis is the altered mechanics of the chest wall with the increased abdominal mass restricting the descent of the diaphragm. The increased mechanical load of obesity has been shown to lead to decreased functional residual capacity with an associated decrease in airway diameter. With decreased lung volume at end-expiration, the tension that usually keeps the airway open is reduced, resulting in premature closure of small airways in the obese person. Obesity also has a direct mechanical effect on airway smooth muscle that increases bronchial hyperresponsiveness.

Obesity is an inflammatory state that may affect the lung in various ways. Obese individuals have abnormal levels of adipokines, which may lead to an increase in airway inflammation and reactivity. Included among the adipokines are leptin and adiponectin, whose principal site of action is in the hypothalamus, yet their receptors exist in the lung as well. Obese children have higher levels of leptin than controls; leptin levels in obese children with asthma are increased 2-fold over levels in normal-weight, nonasthmatic controls. This can have a compound negative effect on asthmatic children, who already have higher leptin levels than do nonasthmatic children. Furthermore, leptin levels correlate positively with BMI, airway reactivity, and total IgE. In contrast to leptin, adiponectin may have anti-inflammatory properties. Airway smooth muscle cells contain adiponectin receptors. Typically, adiponectin levels are low in obese individuals and increase with weight loss. In children, as in adults, adiponectin demonstrates an inverse correlation with BMI. This may adversely affect the symptoms of children with asthma since studies have demonstrated a positive correlation between low cord blood adiponectin and development of wheeze at the age of 2.

It is well established that a family history of atopy is a major risk factor of asthma. Furthermore, it has been speculated that obese individuals have a higher incidence of atopy. In one study that included children (n = 7505) aged 4 to 17 years from the NHANES III survey, the prevalence of asthma and atopy assessed by skin prick test significantly rose with increasing BMI. Yet after adjustment for confounding factors, only a positive relationship between BMI and asthma remained significant, and there was no relationship between BMI and atopy. In contrast, a New Zealand study found that in girls, BMI was significantly associated with positive skin tests and elevated IgE. However, this was a modest association and was not seen in boys. Overall, pediatric studies have not shown a clear, consistent relationship between obesity and atopy.

A study by Van Cleave et al complements recent work documenting the increasing incidence and prevalence of chronic conditions, especially asthma and obesity. This prospective study used the National Longitudinal Survey of Youth–Child Cohort (1988-2006) of 3 nationally representative cohorts of children aged 2 through 8 years at the beginning of each study period. The investigators found that prevalence of a chronic condition at any point during the study period was very high and increased over time. Furthermore, in individual chronic condition subgroups, there was an increase in the baseline prevalence to end-study prevalence in all cohorts (n = 5001) from 2.0% (n = 119) to 3.6% (n = 195) in asthma and 11.9% (n = 611) to 13.3% (n = 721) in obesity. These data suggest that childhood obesity and the prevalence of asthma have increased from 1988 to 2006 and support the presence of a parallel rise in prevalence of asthma and obesity.

Physical inactivity is an important contributing factor in the maintenance of childhood obesity. Obesity has been associated with a sedentary lifestyle in children. Physical inactivity is also common in children with asthma. Children with asthma may limit their level of exertion because their symptoms are brought on by intense physical activity. A study measured fitness, daily physical activity, and body composition in children with newly diagnosed, untreated asthma and assessed the association of these parameters and the level of asthma control.
Children with untreated asthma are less fit and have a higher percentage of body fat and frequency of obesity compared with their healthy peers. A similar study examined the relationships among weight, asthma severity, physical activity, and aerobic fitness in children with asthma. Overweight or obese children reported greater limitation of physical activity and higher asthma-impairment scores compared with controls and, thus, were prescribed more asthma medications.

Efforts should be directed at understanding the reasons for reduced exercise tolerance. Disease severity and parental health beliefs contribute to the lower activity level of children with asthma. Evaluation of exercise level is one of the indicators of asthma control. Exercise and its benefits should be addressed to patients and caregivers. Studies indicated that physical activities such as running and swimming are associated with improved fitness and decreased severity of asthma symptoms.

Recent literature identifies an important area in which overweight has a significant impact on the health of children with asthma. In a retrospective chart review on 813 children older than 2 years who presented to the emergency department with an asthma exacerbation and met further inclusion criteria, Carroll et al. found that overweight children whose BMI was greater than the 95th percentile for their age were significantly more likely to be admitted to the hospital with acute asthma exacerbations than nonoverweight children with a BMI less than or equal to the 95th percentile.

Childhood obesity significantly affects the health of children with asthma. In a retrospective cohort study by Carroll et al., data were collected from all children older than 2 years admitted to the intensive care unit (ICU). Of the 209 children admitted to the ICU with asthma, 22% were found to be obese with a BMI weight-for-age greater than the 95th percentile. Compared with children of normal weight, the obese children were older (9.7 ± 4.4 vs 8.0 ± 4.3 years, P = .02), were more likely to be female (60% vs 57%, P < .01), and were more likely to have been admitted to the ICU previously (40% vs 20%, P = .01). Despite similar severity of illness at ICU admission, obese children had a significantly longer ICU length of stay (116 ± 125 vs 69 ± 57 hours, P = .02) and hospital length of stay (9.8 ± 7.0 vs 6.5 ± 3.4 days, P < .01). Obese children also received longer courses of supplemental oxygen, continuous albuterol treatments, and intravenous steroids. This study demonstrates that childhood obesity does affect children with status asthmaticus by increasing their recovery time from acute exacerbations.

Both cross-sectional and prospective studies have investigated the association between asthma and obesity in children, with the findings of certain studies being inconclusive. Investigating the relationship between asthma and obesity warrants the use of prospective studies, which are better suited for the establishment of temporal associations. One prospective study followed 3792 children aged 7 to 18 years for 5 years and determined that the risk of incident asthma increased in overweight and obese boys but not girls. Another study of 9828 children aged 6 to 14 years found that an increased risk of asthma development was only positively associated in girls with higher BMI. Methodological flaws—varying definitions of asthma, poor differentiation of obesity phenotypes, lack of control of confounding variables such as physical activity and diet—have resulted in pediatric studies not finding a consistent relationship between obesity and the development of asthma.

Prevention and Treatment: Regaining Control

Overweight and obese children have an increased likelihood of becoming overweight and obese adults. It is important to consider an estimation of future obesity-related health care costs for adults in order to focus our efforts and funds on more comprehensive prevention strategies. Wang et al. used projected prevalence, census population projections, and published national estimates of per capita excess health care costs of obesity and overweight to project an estimation of the cost that obesity could have on our economy. They found that by 2030, health care costs attributable to obesity and overweight could range from $860 to $956 billion annually, which would account for 15.8% to 17.6% of total health care costs, or 1 in every 6 dollars spent on health care. Even with these daunting statistics, the true impact of the overweight and obesity epidemic is still unaccounted for. These data are crucial to the future planning of the US government’s health care expenditure and budgeting. The medical community should be among the first to lobby for obesity prevention programs, including insurance-covered weight loss and fitness programs, removal of fast food franchises from children’s hospitals, and persuading schools to provide healthier lunches and more physical education classes.

Recently, First Lady Michelle Obama announced an ambitious national goal of solving the problem of obesity within a generation so that children born today will reach adulthood at a healthy weight. Her nationwide campaign, Let’s Move, joins community leaders, teachers, health care professionals, and parents in the effort to tackle the childhood obesity epidemic. Let’s Move will offer support to parents, provide healthier food in schools, encourage kids to be more physically active, and make healthy, affordable food available throughout the United States.

Another route is to research the pathophysiology of these diseases, especially asthma, in order to decrease morbidity and mortality. These research ventures should include analysis and development of long-term asthma education and overweight and obesity education for children, their guardians, and their health care providers. Awareness of this issue is an essential component because recognition of increased risk of pulmonary complications in this population can lead to earlier diagnosis and treatment.

No firm evidence exists stating that any one strategy (weight reduction, physical activity, environmental) can lower BMI long-term. However, using a multistep approach, each part of the community can work together in the battle against overweight and obesity among children and adolescents and decrease the incidence and prevalence of asthma among this population.


