The effect of obesity on asthma incidence: Moving past the epidemiologic evidence

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The prevalence of both asthma and obesity has significantly increased over the past 20 years, especially among ethnic minorities, a group disproportionately affected by these 2 disorders. According to the latest National Surveillance for Asthma—United States, 1980-2004, African American adults have higher asthma prevalence (7.6% vs 6.7%) and higher asthma-related morbidity including hospital outpatient visits (10.0 vs 4.3 per 100) and emergency department visits (20.2 vs 6.2 per 100) compared with whites. Although the rate of acute care use for asthma has decreased over the past decade, there is a differential improvement by race, with African Americans having 4 times the rate of asthma-related emergency department use of whites. Obesity has also been found to affect African Americans disproportionately, with black women twice as likely to be obese compared with white women.

Longitudinal studies looking at the relationship between obesity and asthma have shown increasing body mass index (BMI) to be associated with increased asthma incidence. A recent meta-analysis of 7 prospective studies based primarily on white adults shows obesity to be associated with increased risk of asthma with an odds ratio of 1.92 (CI, 1.43-2.59). There is, however, significant heterogeneity among these studies regarding effect size and sex interactions, with odds ratios ranging from 1.4 to 3.5 this association being most consistent in women, and some studies showing no effects of obesity on asthma incidence in males. However, results from the meta-analysis by Beuther and Sutherland found obesity to be associated with increased risk of asthma in both men and women. This is most likely a result of differences in study design, case definition of asthma, and study populations.

Although African Americans are disproportionately affected by both obesity and asthma, the effect of obesity on asthma incidence in that population had not been previously examined. Aside from a direct causal link, the association between obesity and asthma may be mediated via genetics, environment, and psychosocial factors, which may vary among ethnic groups. As such, it is unclear whether the association between obesity and asthma is comparable among blacks and whites. In the current issue, Coogan et al evaluate the association between obesity and asthma incidence in a cohort of African American women. Using a population-based cohort of women enrolled in the Black Women’s Health Study compared with normal weight subjects (BMI 20-24.9 kg/m²), higher BMI was found to be associated with an increased risk of asthma (incidence rate ratio of 1.26-2.85), with higher risk of asthma noted with increasing BMI. This effect persisted after adjusting for potential confounders including smoking status, age, obstructive sleep apnea, BMI at age 18 years, parental history of asthma, and self-reported dietary intake and physical activity. These findings are consistent with those previously reported in the literature and further support an association between obesity and asthma incidence in a group at high risk for both disorders. Specifically, the current study provides strong evidence that the risk of adult-onset asthma among African American women increases as BMI increases.

Has the increase in the US rate of obesity among African Americans (particularly among women) contributed to the higher rates of asthma and asthma morbidity observed in this population? Several investigators have suggested that obesity may be an important causal factor in asthma-related disparities. However, others have proposed that the observed epidemiologic association between obesity and asthma is not causal, but instead is a result of factors such as inaccurate diagnosis of obesity-related respiratory symptoms (pseudo-asthma) as asthma. Clearly, obesity can cause asthmalike symptoms such as shortness of breath and wheeze, as well as gastroesophageal dysfunction (ie, gastroesophageal reflux disease), which may mimic asthma. In addition, studies looking at the association of obesity and cardinal features of asthma pathophysiology, such as hyperresponsiveness and airflow limitation, have yielded conflicting results. Further, even though BMI is a widely accepted measure of obesity, there is controversy regarding whether BMI is the best marker of adiposity. Studies have shown body fat distribution to be more closely associated with lung function and systemic inflammation, thus making it potentially a more accurate measure of obesity when evaluating the effects of obesity on asthma pathophysiology. In addition, the association between obesity and asthma may be explained by shared risk factors including behavioral, environmental, and genetic factors, rather than a causal link. Although Coogan et al attempt to adjust for these possible confounders, this observational study cannot directly answer questions of causation.

Given the overall increases in the prevalence of obesity in the general population, the potential effect of obesity on future asthma incidence may have significant public health impact. According to the meta-analysis by Beuther and Sutherland, approximately 250,000 new adult cases of asthma each year in the
Unites States may be attributable to overweight and obesity. Further, this increased risk of asthma may disproportionally affect African American women, who are at greater risk of obesity. The weight of epidemiologic evidence is now strong and compelling that obesity is an important risk factor for asthma. It is now time to move forward with well-designed, hypothesis-driven mechanistic studies that can clarify the causal effects of obesity on asthma incidence and morbidity in different populations.

REFERENCES