# Childhood Asthma Management and Environmental Triggers

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**KEYWORDS**

• Childhood asthma • Asthma exacerbations • Prevention • Management
• Environmental triggers • Trigger remediation

**KEY POINTS**

- The causes of asthma development are not known and thus asthma cannot be prevented based on the current level of understanding.
- Home environmental intervention strategies can reduce asthma morbidity in sensitized children but are probably not effective in children not sensitized to the specific allergen.
- Interventions tailored to the allergen sensitization of the child coupled with education and appropriate severity-specific asthma medication may be the most effective strategy to reduce asthma morbidity.
- Efforts to prevent environmental tobacco smoke exposure are important for everyone in the household.

## INTRODUCTION

Asthma is the most common chronic disease of children, affecting more than 6.8 million children in the United States (9.3% of all children).[1] Asthma cannot be cured. Asthma cannot be prevented. However, asthma can be controlled. This article first reviews asthma epidemiology and the current understanding of the genetics of asthma. It then discusses the various proposed hypotheses for the continuing increase in asthma prevalence and risk factors for asthma. In addition, it discusses emerging work on asthma prevention and strategies to prevent asthma exacerbations.

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## EPIDEMIOLOGY

In industrialized countries with Western lifestyles, lifetime asthma prevalence is high and has increased approximately 2.7 percentage points per year since 1997.[2] In the United States, the lifetime reported asthma prevalence in individuals of all ages is 13.0% (2012). Lifetime prevalence is higher in children (14%) than in adults and increases from early childhood (7.0% at 0–4 years of age) to adolescence (18.4% at 15–19 years of age), with current asthma prevalence showing a similar trend.[1] Asthma is a major public health problem costing the health care system nearly $56 billion annually, with direct health care costs estimated at $50.1 billion and indirect costs (lost productivity) contributing an additional $5.9 billion.[2]

Asthma in childhood disproportionately affects more boys than girls and underrepresented minority populations with African Americans and (some) Hispanic people having higher rates than other racial and ethnic groups. Children of Puerto Rican origin have the highest asthma prevalence of all ethnic and racial groups, whereas Mexican children have one of the lowest reported rates, making it difficult to examine prevalence when all individuals of Hispanic origin are grouped together.[2–4]

Asthma morbidity is high but hospitalization rates for asthma decreased 24% between 2003 and 2010 (National Health Interview Survey, 2001–2011).[2] Compared with adults, children are disproportionately hospitalized for asthma. Approximately 29% of the asthma hospital discharges in 2010 occurred in children less than 15 years of age even though only 21% of the US population is less than 15 years of age.[2] In 2010, asthma was responsible for 2.1 million emergency room visits, 10.6 million physician office visits, and 1.2 million hospital visits.[2]

Asthma deaths are rare in children but increase with age. In 2009, 157 children less than 15 years of age (0.2 per 100,000) died of asthma compared with 617 adults older than 85 years.[2] Black adult women have the highest mortality from asthma (2.5 per 100,000), with an age-adjusted death rate in black people that is 3.1 times higher than the rate in the white population.[2]

### *Genetics of Asthma*

Asthma is a complex and chronic disease that depends on the interplay of genetic and environmental triggers. More than 100 candidate genetic loci have been identified using epidemiologic linkage studies.[5] This finding suggests that individuals with asthma may have many susceptibility loci, each of small effect. Multiple genome-wide association studies using population cohorts have identified several regions associated with asthma, including the 17q21 locus (ORMDL3/GSDML), IL33 on chromosome 9p24, HLA-DR/DQ on 6p21, IL1RL1/IL18R1 on 2q12, WDR36/TSLP on 5q22, and IL13 on 5q31.[5] Many of the associations have not been replicated in other populations, suggesting a role of population-specific variants in the causation of asthma and showing an important role for regulatory genes. Genomic studies using human fetal tissue suggest a complex interaction between environmental factors such as in utero smoke exposure, maternal diet, folate and vitamin D, lung structural genes, and lung function growth trajectory.[6]

African ancestry has been found to affect lung function and asthma severity. Using ancestry informative markers, lung function is inversely correlated with the percentage of African ancestry in 3 independent African American populations.[7] Genetic variation leading to an increased risk for exacerbations is also more common in African individuals.[8] Results from the GALA (Genetics of Asthma in Latino Americans) study show that individuals who are Puerto Rican have a higher degree of African and European ancestry than Mexican American individuals. It has been suggested that this