# HEALTH EFFECTS

# Environmental Risk Factors for Asthma in Children

#### Introduction

Asthma is the leading chronic disease of children in industrialized countries [1]. Although the disease process is not completely known, an essential feature is the characteristic inflammation of the bronchial tube. Common factors that contribute to the occurrence of asthma are exposure to outdoor air pollutants, tobacco smoke, and allergens. Symptoms of asthma include wheezing, constricted breathing, coughing, increased heart rate, and sweating [2]. There is no cure for asthma, but it can be controlled with medication and proper management.

Asthma in children results from the complex interaction of different factors. Genetic predisposition to allergic reaction leaves children more susceptible to asthma. Allergens such as pet dander and dust mites may sensitize the airway, leading to inflammation. Lastly, contributing factors such as cigarette smoke, respiratory infections, and air pollutants may cause irritation of the bronchial tubes and compound the effects of allergens.

With the rates of asthma steadily increasing each year, asthma has become the most common childhood illness after the common cold [3]. In Canada, asthma is one of the most prevalent chronic conditions in children, affecting 12% of Canadian children [3, 4]. Approximately 20 children in Canada die each year from asthma. Asthma is the leading cause of respiratory-related hospital admissions among children aged 0-14 years. The condition is also the leading cause of absenteeism from school.

In the U.S., self-reported asthma prevalence increased 74% among children ages 5 to 14 from 1980 to 1995 [5]. The number of children dying from asthma almost tripled from 93 in 1979 to 266 in 1996 [1]. Asthma affects almost 5 million school-aged children, resulting in an estimated loss of 14 million school days annually. Higher morbidity and mortality are generally observed in low-income and minority populations [1, 5]. The increase in prevalence has been largely attributed to environmental factors [6]. This paper will describe in detail the factors that contribute to asthma in children and discuss measures to prevent exposure to environmental triggers.

### Allergens

Common household allergens include dust mites, molds, cockroaches, and pet dander. Allergens in the lungs trigger bronchial hyperresponsiveness and airway inflammation [6]. Briefly, detection of the allergen

by lymphocyte cells in the lung initiates the allergy-induced asthma reaction. This triggers the production of proinflammatory products, which narrow the airway, and the release of specific IgE antibodies [7]. The tendency to develop these antibodies, referred to as sensitization to allergens, is associated with most asthmatic attacks [6]. More than 80% of asthmatic children in the U.S. are allergic to one or more indoor or outdoor allergens [7].

#### **Dust Mites**

Dust mite allergen sources include bedding, carpets, upholstered furniture, stuffed toys, and fabric-covered items. Dust mites are uncommon at high altitudes and in cool dry environments. Studies have shown that relocating asthmatic children to high altitudes for five weeks reduces hyperresponsiveness and IgE antibody production [7]. These symptoms quickly returned after two weeks at lower altitudes.

#### Molds

The presence of molds is a growing health concern. Schools have delayed the start of the school year in order to remove excessive mold allergen. Molds can grow on a variety of surfaces, including wood, paper, carpet, and food [8]. Children allergic to molds can experience severe asthma attacks.

#### Cockroaches

Cockroaches are attracted to homes by food and water. Their diet includes human food, vegetable fibers, and animal-based glue. Cockroach allergen is often associated with asthma in low-income and minority children. In a study of urban asthmatic children, 85% of the bedrooms tested positive for cockroach allergens [8].

#### **Outdoor Air Pollutants**

The Clean Air Act of 1971 designated six criteria pollutants known or believed to be adversely associated with human health. This category of pollutants includes lead, carbon monoxide, nitrogen dioxide, sulfur dioxide, ozone, and particulates with diameters less than 10 micrometers. Because people with asthma are more sensitive to pollutants than the general population, the National Ambient Air Quality Standard (NAAQS) established under the Clean Air Act has set standards for these pollutants at levels low enough to not exacerbate their condition. Although all but lead have been significantly linked to asthma, ozone and particulates pose greater health risks because of their high prevalence in the US and Canada [9]. Unlike

allergic reactions, these pollutants are believed to trigger an asthmatic response by causing an acute irritation-induced inflammatory reaction in the bronchial passage [10].

# Ozone

Ozone is formed in the atmosphere through a series of photochemcial reactions involving nitrogen oxides and volatile organic compounds (VOCs). Although generally associated with harmful ultraviolet radiation, ground-level ozone makes up the majority of urban smog [9]. Ozone impairs normal pulmonary function, resulting in decreased lung volume. Sensitive populations such as children could experience a 10% decrease in lung function because of ozone exposure [9]. The US Environmental Protection Agency has set air quality standard of 80 ppb for an 8-hour ozone average [11].

The adverse effect of ozone air pollution is often more extreme during hot humid conditions and has been associated with 10-20% of all summertime respiratory hospital visits in the US [10]. Burnett et al. found similar associations between ozone concentrations and respiratory admissions for 16 Canadian cities over a 10-year period (1981-1991) after controlling for other pollutants and weather conditions [12].

#### Particulate Matter

Particulates in the air are comprised of various pollutants in a range of sizes. Fine particulate matter have a diameter less than or equal to 2.5  $\mu$ m (PM<sub>2.5</sub>) and are a product of combustion. Sulfates and nitrates are fine particulates produced by wood stoves or automobiles [13]. As such, they are found both indoors and outdoors. Course particles with diameters between 2.5 and 10  $\mu$ m (PM<sub>10</sub>) are formed physically by the crushing or grinding of surfaces. Aluminosilicates and oxides comprise the majority of PM10, which can be transported by wind or with traffic movement [9]. The US Environmental Protection Agency has set 24-hour air quality standards of 65  $\mu$ g/m<sup>3</sup> for PM2.5 and 150  $\mu$ g/m<sup>3</sup> for PM10 [11].

Because of the mixture of pollutants, associations between outdoor particulate matter and asthma are inconsistent. In general, stronger associations have been seen with PM<sub>2.5</sub>. Norris et al. found significant associations between emergency department visits for asthma in children and fine particulate matter in Seattle [13]. Similar results were observed in studies of other cities throughout the U.S. and Canada [9].

Concentration of particulate matter in indoor air can differ substantially from outdoor concentrations. With low air exchange rates, contributions from indoor particulate sources cooking like appliances and heater can accumulate to dangerous levels [9]. Based on evidence linking particulates to adverse health outcomes like asthma, a revised annual standard of  $15\mu\,/m^3$  was set for PM $_{2.5}$  [10]. This means that the three-year average of the 24-hour concentrations must not exceed 15.0  $\mu g/m^3$  [11].

A recent study by Gent et al. examined the simultaneous effects of particulate matter and ozone on childhood asthma using parent-reported respiratory symptoms and rescue medication [14]. The cohort of 271 children living in Connecticut and Massachusetts were followed from April to September and air and meteorological measurements were collected during this time period. In the single-pollutant models, same-day  $PM_{2.5}$  concentrations of 12.1-18.9  $\mu$ g/m3 and chest tightness were significantly associated with an odds ratio of 1.24. Significant odds ratios were observed for ozone exposures at the highest level for both sameday and previous-day models and wheeze, persistent cough, chest tightness and shortness of breath. Odds ratios increased with increasing exposure. In the copollutant model, ozone but not particulates significantly predicted increased risk of asthma symptoms and rescue medication. The lack of association with particulate matter may be due to the low concentrations measured during the study time period; there were no days where the level of PM<sub>2.5</sub> exceeded EPA

#### Environmental Tobacco Smoke

The most significant indoor particulate source is cigarette smoke. In infants, prenatal exposure to second hand smoke, or environmental tobacco smoke (ETS), reduces respiratory flow possibly because of altered lung structure [15]. Similar to other particulate matter, postnatal exposure to ETS causes a proinflammatory lung irritant [16]. In addition, ETS has been associated with elevate serum IgE antibodies, an indication of allergy development.

A review of recent studies strongly supports the association between environmental tobacco smoke and asthma [7]. Asthmatic children of mothers who smoked had reduced lung function compared to asthmatic children of mothers who did not smoke. A study of 415 children in Canada revealed that the severity of asthma attacks in children of mothers who smoked was significantly higher than those in children of nonsmoking mothers. ETS has also been linked to greater susceptibility to viral or bacterial infection, another contributing cause of asthma.

## Conclusion

The personal choices we make in our lives can have monumental effects on the lives of the children around us. One of the most obvious things we can do to prevent predisposing asthmatic children to contributing factors such as smoke is by refraining from smoking when pregnant or near small children. Also, a clean and well-ventilated home reduces a child's exposure to house dust mites, cockroaches, and molds. Outdoor air quality should be monitored, especially on hot humid days, to minimize exposure of children who possess a genetic tendency to be asthmatic. Proper prevention may help reduce asthma in children.



#### References Cited:

- CDC. Asthma's impact on children and adolescents. National Center for Environmental Health. July 2003. www.cdc.gov/nceh/airpollution/asthma/children.htm.
- 2. Children's Health Canada. Asthma in Children. www.childrenshealth
- 3. Asthma in Canada. Asthma statistics. Aug 2003. www.asthmaincanada.com/stats/
- 4. Health Canada. Chronic respiratory diseases Centre for chronic prevention and control. July 2003.
  - www.hc-sc.gc.ca/pphb-dgspsp/ccdpc-cpcmc/crd-mrc/asthma\_e.html.
- CDC. Surveillance for Asthma-U.S., 1980-1999.
  MMWR. 51(SS01): 1-13. March 29, 2002.
  www.cdc.gov/mmwr/
- Holt PG. 1998. Program responsiveness to environmental antigens that trigger allergic respiratory disease in adulthood is initiated during the prenatal period. Environmental Health Perspectives 106(Suppl 3): 795-802.
- Gold DR. 2000. Environmental tobacco smoke, indoor allergens, and childhood asthma. Environmental Health Perspectives 108:643-651 Supp.4.
- U.S.EPA. Asthma and indoor environments.
  Asthma triggers Molds. July 2003.
  www.epa.gov/iaq/asthma/ triggers/molds/html
- Rosenstreich DL, Eggleston P, Lattan M, et al. 1997. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. New England Journal of Medicine 336:1356-1363.
- Suh HH, Bahadori T, Vallarino J, and JD Spengler. 2000. Criteria air pollutants and toxic air pollutants. Environmental Health Perspectives 108:625-633 Supp.4.
- U.S.EPA. Final revisions to the ozone and particulate matter air quality standard Current and revised standards.
  www.epa.gov/oar/oaqps/ozpmbro/current.htm
- U.S.EPA. Asthma and indoor environments.
  Asthma triggers Ozone. July 2003.
  www.epa.gov/iaq/asthma/ triggers/ozone/html
- 13. Burnett RT, Brook JR, Yung WT et al. 1997. Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. Environmental Research 72:24-31.

- 14. Gent JF, Triche EW, Holford TR et al. 2003. Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. JAMA 290: 1859-67.
- Norris G, YoungPong S, Koenig J, et al. 1999. An association between fine particles and asthma emergency department visits for children in Seattle. Environmental Health Perspectives 107:489-493.
- U.S.EPA. Asthma and indoor environments.
  Asthma triggers Second Hand Smoke. July 2003. www.epa.gov/iaq/asthma/triggers/shs/html
- 17. Platts-Mills T, Carter M, and P Heymann. 2002. Specific and nonspecific obstructive lung disease in childhood: Causes of changes in the prevalence of asthma. Environmental Health Perspectives 108(Suppl 4): 725-73

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