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 photochemical 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].

Particulates 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 µm (PM$_{2.5}$) 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 µm (PM$_{10}$) 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 µg/m$^3$ for PM2.5 and 150 µg/m$^3$ 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$_{2.5}$. 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 15µg/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 µg/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 µg/m$^3$ 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 same-day 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$_{2.5}$ exceeded EPA standards.

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 elevated 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 smokers who smoked was significantly higher than those in children of non-smoking 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:


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