INTRODUCTION
The increasing prevalence of and mortality from asthma, especially among minority urban populations, has made it a renewed focus of public health attention. Asthma is considered a chronic condition that is marked by both bronchial hyperresponsiveness and inflammation in the tissues in the airways.

Asthma hospitalizations among U.S. children increased by 4.5% per year between 1979 and 1987, and the death rate from asthma increased 31% in the same time period. From 1978 through 1989, asthma mortality in the U.S. increased with a near doubling in the mortality rates in both nonwhites and whites; the mortality rates for nonwhites were 4 times those of whites.

In contrast, a survey of hospital admissions and death from asthma in Canada found no nationwide increase in death rate from asthma between 1980 and 1989; though the rate of hospitalization for asthma increased greatly in that time period. A large cross-sectional study of asthma prevalence in children in Canada indicates that higher asthma rates are found on the east coast. Surveyed parents reported that 4.7% of children overall had physician-diagnosed asthma. Asthma was most common in the two Maritime provinces (7.6%) and least common in British Columbia (3.3%) and Quebec (3.4%).

Asthma prevalence has been found to be higher in urban areas. A survey of inner-city children in New York City showed that 14.3% reported ever having asthma, and 8.6% reported current asthma, a rate twice that of the general U.S. population (4.3%). A study of asthma mortality in Chicago found an overall asthma mortality rate of 16.42 deaths per million from 1980 to 1988 for persons aged 5 to 34 years; this is approximately three times the rate for the general U.S. population. Rates were highest among poor black persons. Targonski et al. studied asthma mortality among persons aged 5 to 34 years in Chicago from 1968 to 1991, and found a 337% increase in mortality for African Americans, while there was no significant increase among Whites. A study of asthma hospitalization among 5- to 14-year-old Medicaid patients in Michigan also found much larger increases among urban children, from 3.2 per 1000 in 1980 to 7.1 per 1000 in 1984.

The root causes for the continued increases in asthma prevalence and severity are under debate. The environmental factors that have been associated with increased asthma symptoms have generally not increased during recent decades; in the case of outdoor air pollutants, concentrations have decreased in the developed nations. Martinez et al. studied a cohort of children from infancy to six years of age, and found that, for most children, wheezing does not increase the risk of asthma later in life. However, for a group of children with elevated immunoglobulin (IgE) levels and a family history of asthma, wheezing appears to be related to a predisposition to asthma. The authors could not determine whether the increased allergic sensitization was due to exposure at an early age or a predisposition to allergic sensitization.

CRITERIA POLLUTANTS
The U.S. Clean Air Act of 1970 established six pollutants as "criteria" pollutants -- sulfur dioxide, nitrogen oxides, carbon monoxide, ozone, particulate matter and lead. The resulting network of monitoring stations has provided a substantial database of pollution levels in many U.S. and Canadian cities which has been used in numerous studies of airborne pollution effects on respiratory function. There is little evidence of associations between asthma and either lead or carbon monoxide; evidence for associations with the remaining four pollutants is summarized below.

Although air pollution levels tend to be higher in urban centers, there is also substantial transboundary movement of pollutants in the Great Lakes region. Researchers in New York and Ontario have estimated that regional air pollution transported across the Great Lakes from the U.S. contributed strongly to the high acid aerosol concentrations measured in the city of Toronto.

Particulate Matter. There is considerable evidence that increases in particulate matter are associated with increased respiratory mortality and hospital admissions for asthma. The current U.S. standard for PM<sub>10</sub> is 150 µg/m<sup>3</sup> (24-hour average); a recent review of epidemiological studies indicates that the lowest observable effect level for asthma is above 50 µg/m<sup>3</sup> for PM<sub>10</sub> and about 25-75 µg/m<sup>3</sup> for PM<sub>2.5</sub>. A review by researchers at Harvard combined results from several studies and concluded that each 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> was associated with an approximate 3% increase in asthma attacks, bronchodilator use, and lower respiratory symptoms. A recent study at Harvard School of Public Health found that both increases in "particle-strong acidity" and a 20 ng/m<sup>3</sup> increase in respirable particle (PM<sub>10</sub>) concentration on the previous day were associated with a 0.5 l/min decrease in peak flow rate. However, a study of respiratory effects in 18 U.S. and 6 Canada cities found an increase in bronchitis, but not asthma or wheeze, in cities with higher levels of particle-strong acidity.

Ozone. As reviewed by Koren, ozone has been associated with increased hospitalization rates for asthma, but it has been difficult to separate the effects of ozone from those of other pollutants such as acid aerosols. Compared with the current U.S. standard of 0.12 ppm (24-hour average), the recent review by Lebowitz indicates that the lowest effect level for asthma occurs at 0.08 ppm. In a recent clinical study, asthmatic subjects exposed to 0.16 ppm ozone were significantly more sensitive to ozone exposure than nonasthmatics.

Sulfur dioxide. Koren summarized clinical studies which have indicated that asthmatics may develop significant increases in airway resistance with brief exposures to sulfur dioxide at concentrations as low as 0.25 ppm; the U.S. standard is 0.14 ppm (24-hour). A recent study of 14 asthmatics exposed to SO<sub>2</sub> while exercising found increasing symptoms and lung function responses in concentrations above 0.5 ppm. However, Lebowitz found that the lowest effect level from two epidemiological studies was 200 µg/m<sup>3</sup> (0.08 ppm).

Nitrogen dioxide. Clinical studies, summarized by Koren, have indicated that some asthmatics are inherently more responsive to NO<sub>2</sub> than others. NO<sub>2</sub> differs from the other three pollutants in that it may be found in higher concentration indoors, due in part to NO<sub>2</sub> production from gas stoves or heaters. A meta-analysis of data from 11 studies showed a significant association between estimated NO<sub>2</sub> exposure and illness. Lebowitz reviewed epidemiological studies, and found the results too conflicting to determine a lowest observable effect level.

A monthly review and summary of the scientific literature on human health effects and environmental pollutants, with an emphasis on pollutants of the Great Lakes ecosystem. Prepared under the direction of the Health Professionals Task Force of the International Joint Commission. This does not represent the official position of the International Joint Commission.

Health Professionals Task Force Secretary: Jim Houston International Joint Commission Canada Section 100 Metcalfe Street Ottawa, Ontario K1P 5M1 phone (613) 955-0200 fax (613) 963-5583

Volume 1 Issue 5 April 1996

ASTHMA AND AIR POLLUTION

Ozone. As reviewed by Koren, ozone has been associated with increased hospitalization rates for asthma, but it has been difficult to separate the effects of ozone from those of other pollutants such as acid aerosols. Compared with the current U.S. standard of 0.12 ppm (24-hour average), the recent review by Lebowitz indicates that the lowest effect level for asthma occurs at 0.08 ppm. In a recent clinical study, asthmatic subjects exposed to 0.16 ppm ozone were significantly more sensitive to ozone exposure than nonasthmatics.

Sulfur dioxide. Koren summarized clinical studies which have indicated that asthmatics may develop significant increases in airway resistance with brief exposures to sulfur dioxide at concentrations as low as 0.25 ppm; the U.S. standard is 0.14 ppm (24-hour). A recent study of 14 asthmatics exposed to SO<sub>2</sub> while exercising found increasing symptoms and lung function responses in concentrations above 0.5 ppm. However, Lebowitz found that the lowest effect level from two epidemiological studies was 200 µg/m<sup>3</sup> (0.08 ppm).

Nitrogen dioxide. Clinical studies, summarized by Koren, have indicated that some asthmatics are inherently more responsive to NO<sub>2</sub> than others. NO<sub>2</sub> differs from the other three pollutants in that it may be found in higher concentration indoors, due in part to NO<sub>2</sub> production from gas stoves or heaters. A meta-analysis of data from 11 studies showed a significant association between estimated NO<sub>2</sub> exposure and illness. Lebowitz reviewed epidemiological studies, and found the results too conflicting to determine a lowest observable effect level.

A monthly review and summary of the scientific literature on human health effects and environmental pollutants, with an emphasis on pollutants of the Great Lakes ecosystem. Prepared under the direction of the Health Professionals Task Force of the International Joint Commission. This does not represent the official position of the International Joint Commission.

Health Professionals Task Force Secretary: Jim Houston International Joint Commission Canada Section 100 Metcalfe Street Ottawa, Ontario K1P 5M1 phone (613) 955-0200 fax (613) 963-5583

Volume 1 Issue 5 April 1996

ASTHMA AND AIR POLLUTION

Ozone. As reviewed by Koren, ozone has been associated with increased hospitalization rates for asthma, but it has been difficult to separate the effects of ozone from those of other pollutants such as acid aerosols. Compared with the current U.S. standard of 0.12 ppm (24-hour average), the recent review by Lebowitz indicates that the lowest effect level for asthma occurs at 0.08 ppm. In a recent clinical study, asthmatic subjects exposed to 0.16 ppm ozone were significantly more sensitive to ozone exposure than nonasthmatics.

Sulfur dioxide. Koren summarized clinical studies which have indicated that asthmatics may develop significant increases in airway resistance with brief exposures to sulfur dioxide at concentrations as low as 0.25 ppm; the U.S. standard is 0.14 ppm (24-hour). A recent study of 14 asthmatics exposed to SO<sub>2</sub> while exercising found increasing symptoms and lung function responses in concentrations above 0.5 ppm. However, Lebowitz found that the lowest effect level from two epidemiological studies was 200 µg/m<sup>3</sup> (0.08 ppm).

Nitrogen dioxide. Clinical studies, summarized by Koren, have indicated that some asthmatics are inherently more responsive to NO<sub>2</sub> than others. NO<sub>2</sub> differs from the other three pollutants in that it may be found in higher concentration indoors, due in part to NO<sub>2</sub> production from gas stoves or heaters. A meta-analysis of data from 11 studies showed a significant association between estimated NO<sub>2</sub> exposure and illness. Lebowitz reviewed epidemiological studies, and found the results too conflicting to determine a lowest observable effect level.
AIRBORNE ALLERGENS
A number of studies have found that asthma symptoms can be triggered by increases in levels of allergens such as pollens or mold spores.\(^1\) In one recent panel study, researchers at Harvard\(^2\) collected daily peak flow rates and asthma symptoms from a group of children, along with daily outdoor pollutant and aerosol levels. They found, in addition to significant associations between particulate pollutants and decreased respiratory function, evidence that mold spore concentration independently reduces lung function. A 10,000 spores/m\(^3\) increase in Cladosporium was found to be associated with a 1.0 L/min decrease in morning peak flow rate, while a 60 spores/m\(^3\) increase in Epicoccum was associated with a 1.5 L/min decrease in morning peak flow rate.

TOXIC AIR POLLUTANTS
As described earlier, most research on air pollution and asthma has focused on the criteria pollutants. A team of researchers at Cincinnati\(^3\), University of Cincinnati and asthma have focused on the criteria pollutants. A team of researchers at Cincinnati\(^3\), University of Cincinnati and asthma presentations to the emergency room. A change in pollen concentration (both rise and fall) was also found to be a significant predictor. It has been proposed that rainfall or moisture in the air exacerbates the effects of pollen. As summarized by Knox\(^4\), pollen grains rupture by osmotic shock in rainwater, releasing hundreds of starch granules from each grain, and inhalation challenge tests have shown that starch granule suspensions are capable of eliciting immune responses in allergic people.

REFERENCES:

INTERACTIVE EFFECTS
Pollutant interactions. The concentrations of criteria pollutants are often correlated, and ambient air nearly always contains a mixture of pollutants. Some recent clinical studies have indicated that pollutants in combination have additive effects. Frampton et al.\(^22\) found that asthmatics were significantly more likely to be hospitalized following exposure to sulfuric acid aerosol followed by ozone exposure. Liewa et al.\(^27\) suggest that ozone measurements can be used in estimating exposure to toxic air pollutants, especially reactive hydrocarbons.

Pollution/allergens: Gilmour\(^28\) reviewed animal and human studies indicating that exposure to pollutants may heighten sensitization to allergens. Increased allergic sensitivity has been found in animal studies with exposure to ozone, nitrogen dioxide or sulfur dioxide; the pollutant exposure levels were higher than those found in ambient conditions. In one clinical study on humans with exposure to 0.12 ppm ozone (the U.S. air quality standard) some subjects showed increased bronchial responsiveness following antigen challenge in longer residence time for allergens in the lung, or oxidant-induced release of inflammatory substances in lung tissue resulting in heightened sensitization to allergens.

Rain/Allergens: A recent report from London\(^29\) supports previous studies in noting the occurrence of an “epidemic” of emergency room admissions for asthma following a thunderstorm, with a sudden drop in air temperature. Number of lightning strikes, increase in rainfall, fall in temperature, rise in air pressure and relative humidity were associated with increased asthma presentations to the emergency room. A change in pollen concentration (both rise and fall) was also found to be a significant predictor. It has been proposed that rainfall or moisture in the air exacerbates the effects of pollen. As summarized by Knox, pollen grains rupture by osmotic shock in rainwater, releasing hundreds of starch granules from each grain, and inhalation challenge tests have shown that starch granule suspensions are capable of eliciting immune responses in allergic people.