It is recognized that children are at increased risk of adverse health effects from exposures to a myriad of air pollutants. Recent evidence has demonstrated that acute or prolonged exposures to even moderate levels of ambient air pollution cause low grade airway inflammation \(^1\)\(^2\) and possibly permanent lung tissue remodeling \(^3\) in laboratory animals. It is generally agreed that the internationally observed increases in asthma diagnosis is not the result of diagnostic changes nor of significant changes in genetic factors related to asthma.\(^1\)\(^4\) Despite the fact that ambient air pollution has been reported to have decreased in the past two decades,\(^5\)\(^7\) air pollution in general continues to be advanced as a contributing factor to increased respiratory morbidity in children, adolescents and adults. Asthma continues to be recognized as a difficult disease to diagnose mainly because of the large variation in the presentation, non-specificity and severity of symptoms associated with the disease. There is an emerging concern that undiagnosed or "silent" asthma may be more prevalent than expected and that contemporary exposures to indoor and outdoor air contaminants may elicit asthma-like symptoms in a large segment of the population.

Exposures to air pollutants and to naturally occurring environmental factors relevant to this overview paper can be considered to be derived from outdoor or ambient air pollutants and/or indoor air pollutants. Many outdoor air pollutants penetrate significantly into the indoor environment and are expected to contribute markedly to the total and cumulative exposure individuals receive on a daily and lifetime basis. Recent research findings have revealed that Canadian children (<12 years old) spend on average more than 70% of their time indoors at home, 10% indoors at school and 8.5% outdoors.\(^9\) It is therefore important that we consider and assess the individual and joint role each environment has on the health of children.

This overview paper will attempt to summarize the converging evidence that many air pollutants and environmental factors act independently, but more importantly interactively, to contribute to the increased manifestation and severity of asthma and asthma-like symptoms in healthy children and in asthmatic children and adolescents.

**Air pollution**

In this brief review it is not possible to identify all air pollutants known to affect health. Table I lists the current Canadian National Ambient Air Quality Objectives for specific ambient air pollutants. Table II provides a reference list for commonly encountered indoor air pollutants. The terms "air pollutant" and "air pollution" are commonly used and encompass all types of air contaminants. These may vary markedly by location and region; thus "air pollution" in Vancouver, B.C. may be very different from "air pollution" in an industrial city such as Steubenville, Ohio. Similarly, the air pollution associated with transportation for Toronto, Montreal and Vancouver may vary if the fuel used and the transportation fleet varies across the cities.

**Outdoor air pollution**

Outdoor or "ambient" air pollution primarily results from the combustion of fos-
Sil fuels from “stationary sources” such as power plants and from “mobile sources” such as motor vehicles. The emissions from these sources vary in that the primary emissions from stationary sources are sulphur dioxide (SO₂) and particulate matter (PM) while motor vehicle emissions include carbon monoxide (CO), nitrogen oxides (NOₓ), hydrocarbons and, until recently, lead. Emissions from specific industrial processes such as smelting, paper production, refining, and solid waste disposal have unique emission profiles and these emissions vary by the characteristics of the specific industry. Air pollutants are present in the atmosphere either in gaseous form or as suspended aerosols or what is referred to as particulate matter (PMₙ, where n refers to the particle size in microns (µm), i.e., PM₁₀ includes particles with a theoretical average aerodynamic diameter of 10 µm or less). The size of the particle determines if the particle can enter and deposit in the airways and lungs. Larger particles (i.e., PM₁₀) are filtered out by the nose and upper airway while smaller particles penetrate and deposit on the lining of the airways and lungs.

The ambient concentrations of sulphur dioxide (SO₂), ozone (O₃) and PM₁₀ have remained constant or increased in the past decade⁵⁻⁷ in many regions across Canada. Furthermore, the composition of the pollution mixtures has changed recognizably and given rise to such problems as acid rain and enhanced global climate change. Children’s exposures to outdoor air pollution are enhanced when children are outdoors and engaging in physical activities which increase ventilation. Notwithstanding the reported improvement in air quality in general, significant associations continue to be observed between adverse health outcomes in children and adults at current air pollution concentrations in Canada.

Indoor air pollution

Indoor air quality is influenced by outdoor air pollution concentrations, indoor “sources” of pollution, the building itself and the habits of the residents. It is well recognized that many outdoor pollutants penetrate indoors.¹¹ SO₂ indoors are estimated to be about 10-20% of outdoor levels in winter months. Ozone, because of its highly reactive nature, absorbs to surfaces and is found indoors at 30-60% of outdoor concentrations. Coarse particles

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Exposure Period</th>
<th>Maximum Desirable* Concentration (ppm)</th>
<th>Maximum Acceptable† Concentration (ppm)</th>
<th>Maximum Tolerable‡ Concentration (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Suspended</td>
<td>1 hr</td>
<td>120</td>
<td>400</td>
<td></td>
</tr>
<tr>
<td>Particulate Matter (TSP)</td>
<td>24 hrs</td>
<td>60</td>
<td>70</td>
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<tr>
<td>Carbon Monoxide (CO)</td>
<td>1 hr</td>
<td>13</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>8 hrs</td>
<td>5</td>
<td>13</td>
<td></td>
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</tr>
<tr>
<td>1 year</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxidants / Ozone (O₃)</td>
<td>1 hr</td>
<td>50</td>
<td>82</td>
<td>150</td>
</tr>
<tr>
<td>24 hrs</td>
<td>15</td>
<td>25</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>1 year</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitrogen Dioxide (NO₂)</td>
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<tr>
<td>24 hrs</td>
<td>110</td>
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</tr>
<tr>
<td>1 year</td>
<td>30</td>
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<td></td>
</tr>
<tr>
<td>Sulphur Dioxide (SO₂)</td>
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<td>340</td>
<td></td>
</tr>
<tr>
<td>24 hrs</td>
<td>60</td>
<td>110</td>
<td>310</td>
<td></td>
</tr>
</tbody>
</table>

* Maximal Desirable Level: the long-term goal for air quality
† Maximal Acceptable Level: intended to provide adequate protection against effects on soil, water, vegetation materials, animals, visibility and personal comfort and well-being
‡ Maximal Tolerable Level: concentrations beyond which, due to diminishing margin of safety, appropriate action is required without delay to protect the health of the general population.

**Figure 1.** Schematic of observed adverse health responses in children and adolescents exposed to air pollutants.
(PM₁₀), because of their large aerodynamic size, penetrate poorly into sealed buildings, unlike ambient fine particles (≤ PM₁₀) which are found indoors at 80% of the outdoor concentrations. Carbon monoxide (CO) and NO₂ from outdoor sources also are known to penetrate indoor environments, with little or no reduction in concentrations. The indoor concentrations of outdoor sources of pollens and fungi are reduced by closing the windows and using air conditioning in the summer.

### Indoor sources

Tobacco smoke is the most important combustion product and the major source of fine particles indoors. PM₂.₅ are increased by 1 μg/m³ per cigarette smoked averaged over a 24-hour period. Poorly installed, vented or maintained gas and wood-burning units can significantly increase indoor levels of carbon monoxide, nitrogen dioxide and particles. One study found that indoor CO levels were less than 5 ppm for airtight stoves and 8 ppm for non-airtight models. Maximum particle concentrations ranged from 71 μg/m³ for airtight stoves to 650 μg/m³ for non-airtight. In homes with attached garages, there is a risk of automobile fumes entering as a pollutant. Volatile organic compounds (VOCs) are gaseous chemicals released by numerous sources; paints, lacquers, glues, personal grooming agents (perfumes, hair sprays), cleaning agents and building materials. Formaldehyde, a component of particle board, can irritate the eyes, nose and throat. Ott and Roberts have recently published a provocative review of indoor air contaminants and personal exposures to various compounds.

The most important sources of indoor allergens are dust mites and furry and feathered pets. In one study, of 66% of patients with poorly controlled asthma who said that they were allergic to animals, 48% had furry pets. Bacterial endotoxins and fungi with allergic and mycotoxic potential are also found indoors.

Dust mites, fungi, and bacteria require moisture to proliferate. Permeation of rain or ground water into the building envelope and condensation on cold interior surfaces can promote microbiological contamination. Water vapour produced by people and pets, and cooking and showering requires a sufficient air exchange rate to prevent moisture problems. Increased air exchange may also dilute the concentrations of some indoor airborne pollutants, probably explaining why environmental tobacco smoke has less effect on asthma in the summer than in the winter. Mattresses, upholstered furniture and carpets are the most important reservoirs for dust mites and difficult, if not impossible, to deeply clean.

### Health effects

Figure 1 illustrates the types of outcomes which have been observed to occur in children and adolescents as a result of exposures to outdoor and indoor air pollutants. The type and range of adverse effects of air pollution exposures in children are not dissimilar to those reported for adults. Sub-clinical and clinically significant increases in symptoms such as occasional cough, wheeze, bronchitis and small transient changes in lung function have been noted.
in numerous epidemiologic studies. These studies included asthmatic and non-asthmatic children acutely and chronically exposed to various air pollutants. In children, with and without asthma, cough is being interpreted increasingly as a sign of heightened bronchial responsiveness and a sign of inadequate treatment if the child is asthmatic. Equivalently it can be used as a marker of non-specific increased airway hyper-reactivity which is consistent with a predisposition for response to air pollution exposures. Recent data have indicated that cough receptors are more highly stimulated by pollutant exposures in asthmatic children. Pronounced and persistent cough and/or wheeze, chest tightness or pain with inspiration have been reported in community studies assessing the effect of urban smog pollution in children. Increases in the use of asthma medication in children have been reported\(^{17,19}\) in response to episodes of ambient air pollution consisting of particulate matter and ozone. Controlled exposure studies in asthmatic and non-asthmatic children have reported increases in cough and wheeze symptoms when ambient polluted air is introduced into the chamber.\(^{20-22}\) Furthermore, studies have demonstrated decreases in lung function\(^{23,26}\) and increases in symptoms and medication use\(^{17,18}\) in children and adolescents resulting from acute exposures to pollutants. Increased respiratory hospitalizations in very young children (<2 years old) have been reported to occur in Canada and to be associated with ambient concentrations of pollutants to a greater degree than in adults.\(^{27}\)

Children's airways and lungs develop significantly in the early years and well into adolescence, and the additional pathophysiologic burden of responding to air pollution effects may overwhelm normal mechanisms and promote symptom development, manifestation and respiratory disease progression.

In summary, the observed adverse health effects of exposures to air pollution in children range from subtle sub-clinical non-specific symptoms, through to increased cough and wheeze, increased use of asthma medication, increased rates of asthma attacks (reversible airway obstruction), increased physician and hospital respiratory visits, permanent reduction of lung capacity, and an increase in risk for sudden infant mortality (SIDS).

**Asthma**

In examining the associations between childhood/adolescent asthma and environmental factors, two central issues need to be addressed: 1) are environmental factors causing increased asthma rates in children and adolescents? and, 2) do environmental factors precipitate and/or aggravate symptoms associated with asthma? The interrelationships between the effects of air pollutants, asthma and allergic responses make the determination of causation exceptionally difficult. Further, there is compelling evidence that viral infections may have profound effects in conditioning the airways and lung to adverse responses to air pollutant exposures in many children, with or without asthma.

The Canada Health Survey\(^{28}\) of 1978 reported asthma prevalence rates of 2.6% in populations 15 years of age and older. The 1991 General Social Survey\(^{29}\) and 1994/95 National Population Health Survey\(^{30}\) estimated the asthma prevalence for the same age group, at 6% and 6.1%, respectively. Dales et al.\(^{31}\) reported the prevalence of asthma in 5 to 8 year old children to average 4.7% (range 1.8% to 9.8%) across 30 rural communities in Canada. King and Paré\(^{32}\) report that asthma prevalence in children is approximately 10%. Dockery et al.,\(^{33}\) using data from approximately 15,000 questionnaires, reported “doctor diagnosed asthma” rates in 24 non-industrial North American communities to range from 3 to 11% in children 8 to 11 years of age. Recently, a Health Canada study entitled “The Student Lung Health Study”\(^{34}\) reported a prevalence of 13% for self-reported doctor-diagnosed asthma in a study of 5 to 19 year olds. Although the prevalence of asthma has been observed to have increased over the past two decades, the levels of most ambient air pollutants reportedly has decreased in Canada.\(^{35}\) This association is consistent with observations noted in other countries and it is generally concluded that asthma is not caused by air pollutants per se. Although there is converging evidence against a causal association between ambient air pollutant exposures and the development of asthma in children and adolescents,\(^{4}\) there is substantive evidence that asthmatic and non-asthmatic children have significant adverse health responses to many air contaminants.

**Increased respiratory morbidity**

It is not possible to review here the full scope of the literature addressing the volumes of morbidity responses associated with air pollutants. Interested individuals are referred to excellent State of the Art review articles published by the American Thoracic Society, that review the health effects for children and adults for pollutants found outdoors.\(^{35,36}\)

The evidence that asthma and asthma-like symptoms are exacerbated by air pollutants and other environmental factors is substantial and irrefutable. What is not clear is why some air pollutants elicit responses while others do not and why responses vary over time. For example, exposure to sulphur dioxide\(^{37}\) (SO\(_2\)) results in marked bronchoconstriction in asthmatics while ozone, a powerful respiratory irritant and oxidant, has modest independent effects on asthmatics.

Approximately 40% of Canadian children are exposed at home to environmental tobacco smoke\(^{35}\) which increases the risks of: SIDS, emergency visits and hospitalization for respiratory diseases, middle ear disease, and chronic respiratory symptoms and asthma exacerbations. An exhaustive and excellent international review of the effects of passive smoking on the health of children has been published recently.\(^{37}\)

Biological agents are generally recognized to cause infections. Respiratory viral infections, tuberculosis, and Legionnaire’s disease all spread in the indoor environment. Biological agents such as dust mites and furry pets are allergenic, causing conjunctivitis, rhinitis and aggravating asthmatic symptoms. Approximately 80% of children with asthma are sensitized to dust mites, and >10 µg/g of dust of *Der p1* (a major dust mite antigen) are associated with a 4.8 relative risk of asthma. Cockroaches were recently found to be a common cause of allergic illness in New York City, and by inference are probably important in other urban environments.
where cockroach infestation is common. Microbiologicals also may affect children’s health through mechanisms other than allergy and infection. Hypersensitivity pneumonitis and humidifier fever, due to microbial contamination of water sources, are acute febrile illnesses, the former with pneumonia. Bacterial cell-wall lipopolysaccharides, called endotoxins, appear to synergize with dust mites to amplify asthma symptoms. Indoor fungi, quantitatively fewer and qualitatively different than those outdoors, are sources of mycotoxins and glucans which have the potential to cause respiratory symptoms. Interactions exist whereby exposure to two agents causes a health effect larger than their sum. The presence of a respiratory viral infection may cause a child to react more severely to an allergen. Prior exposure to elevated concentrations of ozone, i.e., 0.12 ppm for 1 hour, will also enhance the asthmatic reaction to allergen.

Epidemiologic studies

The acute health effects of ambient air pollutants on children have been examined by relating daily pollution measurements with changes in symptoms and lung function. Summer camp studies have demonstrated that ambient levels of pollutants, specifically acidic aerosols and ozone, result in transient reversible declines in lung function of children actively engaged in outdoor activities. Similar observations have been reported in other summer camp studies in the U.S. and in Europe. Asthmatic children have also been studied during periods of elevated air pollution and increases in symptoms and decreases in lung function have been reported. Several Canadian and U.S. studies have found associations between increased respiratory symptoms and/or reduced lung function resulting from acute and chronic exposures to air pollutants including particulate matter, acidic aerosols and ozone. Controlled exposure studies, also called “chamber studies” (these have exclusively been done in the U.S.), have examined the responses of children to controlled concentrations of air pollutants, excluding or controlling other environmental variables. Several ozone studies with children have revealed quantitatively similar results (transient reversible loss of lung function) to those observed in the “field” studies. Chamber studies with asthmatic children exposed to specific pollutants and combinations of pollutants, have demonstrated similar, though somewhat lower, levels of responsiveness as observed in epidemiologic and field studies to pollutants such as ozone and nitrogen dioxide. Exposures to SO₂ singly and in combination with ozone have shown an enhanced or potentiated response in adolescent asthmatics. Exercise, moderate or vigorous, combined with air pollution, elicits adverse health responses even in healthy children. Chamber studies with children have not as a whole elicited increased symptoms in the asthmatics as expected. Increased risk for hospitalizations for respiratory illnesses in children have been associated with daily levels of air contaminants in Canadian cities. Although there is no Canadian data on increased risk of mortality in children related to ambient air pollution, one recent study associated particulate air pollution with sudden infant death syndrome in the U.S. Epidemic studies on the effects of indoor air pollution have generally focused on single source or specific types of air pollutant exposures or indoor contamination. Samet et al. examined the effects of indoor nitrogen dioxide exposures in children and determined that children were at increased risk of respiratory infections and having respiratory symptoms with increasing concentrations of NO₂. Dockery et al. examined the effects of ambient indoor wood smoke exposures in children and noted transient decreases in lung function and marginal increases in respiratory symptomatology. Dekker et al. reported increased prevalence of respiratory illness in homes with indicators of water damage and visible mold or fungi contamination. Spengler et al., using data from 15,523 parent-completed questionnaires from 24 North American communities, reported increased risks of lower respiratory tract symptoms in children 9-11 years of age to be associated with: the age of the home (built before 1970) having a 12% increased risk; homes with smokers having 24% increased risk of symptoms; homes with air conditioners, cleaners and humidifiers having increased risks of 14%, 37% and 47% respectively. This study also revealed a 48% increased risk for lower respiratory tract symptoms in children who resided in homes with positive responses to indicators of “home dampness”. The risks were similarly increased in children with bronchitis and with asthma. Maier et al., using a standardized parent-completed questionnaire for 925 Seattle children aged 5 to 9 years, reported increased risk of having a doctor-confirmed diagnosis of asthma and wheezing associated with indicators of water damage in the home and exposures to environmental tobacco smoke. In contrast to other reports, these investigators found no associations with indicators of home dampness, household mould, and gas, wood or kerosene stove use. House dust mite studies and studies addressing environmental tobacco smoke were noted earlier in the text. There is considerable epidemiologic evidence of significant adverse effects, and in many cases clear dose-response relationships, between exposures to specific indoor air pollutants and health effects in children with and without any pre-existing disease or illness.

CONCLUSION

There is little doubt that air pollution exposures, outdoor and indoor, at concentrations commonly encountered in daily activities, can adversely affect the respiratory health and function of children and adolescents. There is ongoing concern by medical and environmental health researchers that exposures to air contaminants in the very early years of life, including in utero, may have strong influences in determining the level of responsiveness to air pollutants in susceptible individuals. Although some experts believe that environmental factors such as air pollution, diet and natural allergen exposures contribute significantly to an enhanced development of allergic disorders in genetically susceptible individuals, including asthmatics, the general consensus is that childhood and adolescent asthma is more causally related to genetic predisposition and that environmental pollution elicits and exacerbates the manifestation of symptoms and the diseases (allergy and asthma). As noted, there is very provocative evidence that sev-
eral air pollutants singly and, more importantly, in combination with other agents, exacerbate and aggravate many signs and symptoms of allergy and asthma. Recently, the problem of causality has been further complicated by recognizing the strong potentiating effects that viral infections may have in conditioning the responses to allergens and to air pollutants. Keeping the home free of cigarette smoke, furry and feathered pets, and dust mites will have a major impact on children’s health.

In summary, it appears unlikely that air pollutants actually cause asthma. In contrast, the evidence that air pollutants singly and in combination with other environmental factors elicit significant and adverse health responses in asthmatic and non-asthmatic children and adolescents, appears irrefutable.

REFERENCES