The Effects of Air Pollution on Children

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Air pollutants have been documented to be associated with a wide variety of adverse health impacts in children. These include increases in mortality in very severe episodes; an increased risk of perineonatal mortality in regions of higher pollution, and an increased general rate of mortality in children; increased acute respiratory disease morbidity; aggravation of asthma, as shown by increased hospital emergency visits or admissions as well as in longitudinal panel studies; increased prevalence of respiratory symptoms in children, and infectious episodes of longer duration; lowered lung function in children when pollutants increase; lowered lung function in more polluted regions; increased sickness rates as indicated by kindergarten and school absences; the adverse effects of inhaled lead from automobile exhaust. These impacts are especially severe when high levels of outdoor pollution (usually from uncontrolled coal burning) are combined with high levels of indoor pollution. In developed countries, where indoor pollution levels are lower, increasing traffic density and elevated NO_2 levels with secondary photochemical and fine particulate pollution appear to be the main contemporary problem. By virtue of physical activity out of doors when pollution levels may be high, children may experience higher exposures than adults. Air pollution is likely to have a greater impact on asthmatic children if they are without access to routine medical care. — Environ Health Perspect 103(Suppl 6):49–53 (1995)

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Introduction

The description of the impact of air pollutants on children's health is complicated by the fact that there are a number of types of air pollution and a variety of indicators of adverse health effects. In the discussion that follows, the classification is by observed outcome; and the probable pollutants responsible are noted in each section.

Over the past 5 years, investigators have been increasingly interested in studying the impact of air pollutants on children. There are several reasons for this, of which the lack of interference from cigarette smoking is one, and the increasing evidence that adult chronic respiratory disease may have its roots in childhood is another. In addition, schoolchildren can be studied as a particularly accessible group, with spirometric tests being conducted in the classroom.

Increased Perineonatal Mortality

Premature mortality is generally regarded as the most serious effect of air pollution, and the end point is specific.

In Acute Episodes

In the London episode in December 1952, there was an increase in observed mortality in children, as the following data from the official report shows (Table 1) (*1*).

Numerically the deaths in those over the age of 45 are, of course, dominant; but in children below the age of 14, the death rate increased from 38 in the week before the episode to 67 during it, which is a not inconsiderable increase.

Increased Mortality Risk in More Polluted Regions

Observations in Britain in 1971 (*2*) and in the United States in 1977 (*3*) both indicated that respiratory mortality in children was increased in more polluted regions. New data reported in 1992 by Bobak and Leon (*4*) from the Czech Republic for the years 1986 to 1988 showed an increasing relative risk for respiratory mortality in the postneonatal years, in relation to the severity of the air pollution. Forty-six regions of the country for which TSP, SO_2, and NO_2 data were available were included. For quintiles of progressively more severe air pollution (1 being the least and 5 the most severe), Table 2 shows the relative risks found after adjustment for socioeconomic factors.

Table 2. Relative risks of respiratory mortality caused by air pollution in postneonatal years in 46 regions of the Czech Republic.

	Quintile of pollution				
Pollutant	1	2	3	4	5
TSP	1.0	1.25	2.50	1.70	3.16
SO_2	1.0	0.56	0.84	1.96	5.41
NO_2	1.0	0.85	1.00	1.72	2.73

In London in 1952, the pollution was mostly derived from open coal burning; and the same is true of the air pollution in North Bohemia, which constitutes the fifth quintile in the table above.

Barker and Osmond (*5*) from Britain reported that there was a strong geographic relationship between death rates from chronic bronchitis and emphysema between 1959 and 1978, and between the infant mortality from bronchitis and pneumonia in the period 1921 to 1925.

Risks from High Levels of Indoor Pollution

It is well known that respiratory disease is the second most common cause of death in children in the Third World (*6*). This has often been attributed to measles; but it seems very probable that the very high levels of indoor particulate pollution derived

Table 1. Deaths registered in London administrative county classified by age.

All	< 4 weeks	4 weeks–1 year	1–14 years	15–44 years	45–64 years	65–74 years	75+ years
Week before the episode							
945	16	12	10	61	237	254	335
Week of the episode							
2484	28	26	13	99	652	717	949

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from biomass fuel use without adequate ventilation (*7*) play a major part in this mortality.

Increased Acute Respiratory Illness

Hospital Admission and Emergency Visit Data

In Provo, Utah, Pope (*8*) found that hospital admissions of children for acute respiratory disease were closely associated with levels of fine particulate pollution (particles less than 10 microns in size or PM10). In southern Ontario, hospital admissions for children in the summer are associated with ambient ozone and sulfate levels (*9*). Data recently published by Burnett et al. (*10*) reported on hospital admissions for 168 hospitals in Ontario over a 6-year period. They noted that in the summer respiratory admissions were closely associated with ozone levels, and they observed that among infants 15% of summer admissions were pollutant associated; whereas among the elderly only 4% were. Recent studies of hospital admissions in Toronto have indicated that, in addition to ozone and sulfates, the aerosol hydrogen ion level and the PM10 are associated with increased admissions (*11*). In five cities in Germany, Schwartz et al. (*12*) found an association between croup and levels of NO_2.

Other Indices

In an important longitudinal study in the United Kingdom published in 1966, Douglas and Waller (*13*) followed 3866 infants adopted at birth into other families, which randomized any genetic factor. The infants were being brought up in widely different parts of the country, and the general air pollution levels of the different regions were divided into four categories. Over a 3-year period of observation, the occurrence of lower chest infections was three times higher in the most polluted category than in the cleanest. It is now realized that such data would have to be corrected for passive smoking effects and for other sources of indoor pollution such as the use of gas stoves; these possible influences on the study results are not known.

Lunn et al. (*14,15*) studied schoolchildren in Sheffield in 1967 and 1970 and concluded that air pollution levels were partly responsible for respiratory morbidity. More recently, Braun-Fahrlander et al. (*16*) in Switzerland reported on a random sample of 625 children aged 0 to 5 years followed in rural and urban areas. They showed that higher NO_2 exposures (confirmed by personal samplers) were associated with a prolongation of respiratory episodes and that particulate pollution (TSP) was a significant predictor of symptoms. The asthma prevalence was low in this population. A similar study of schoolchildren recently reported, found a significant association between respiratory symptoms and outdoor NO_2 exposure (*17*). In the Gardanne coal basin in France, Charpin et al. (*18*) showed that there were more respiratory symptoms in children in the more polluted regions. Environmental tobacco smoke exposure has been shown to be associated with exacerbations of asthma in children (*19*). It is not clear whether this effect is to be attributed to particulate exposure or to increased levels of NO_2.

Aggravation of Asthma

Increased Hospital Admissions

Studying hospital admissions in the summer in southern Ontario over a 6-year period from 1974 to 1980 (*20*), we found that asthma admissions for children up to the age of 14 were invariably higher after days when the ambient ozone level had exceeded 80 ppb, compared to all days. More recent data from Toronto have fully confirmed this relationship (*11*).

Increased Hospital Emergency Visits

The pioneer study of hospital emergency visits of children in relation to air pollution was that of Girsch et al. (*21*) in Philadelphia in 1967. Visits of 1346 patients were analyzed over 676 study days between July 1963 and May 1965. The hospital was located in a polluted area of the city. Although aerometric data were very incomplete by modern standards, the authors concluded, "There was a threefold greater incidence of bronchial asthma during days of noteworthy higher pollution." Pollen counts were available in the ragweed season, but the authors noted, "Very few asthma attacks occurred during the ragweed season."

White et al. (*22*) studied emergency visits to the Grady Children's Hospital in Atlanta in the summer of 1990, and reported a 37% increase in visits for asthma after 6 days when the ozone level exceeded 0.11 ppm. Cody et al. (*23*) in New Jersey, in a study of seven hospitals over a 5-year period, found a consistent increase in emergency visits in relation to ozone levels during the summer. In Puerto Rico, environmental factors have been shown to be related to exacerbations of asthma (*24*); and Puerto Rican children in the United States have about double the prevalence of asthma compared to Mexican–American children (*25*).

In a study of emergency visits in Vancouver in a population of 984,900, containing 170,300 children below the age of 14 (*26*), we found that in a calendar year, there were 2936 visits for all acute respiratory disease in this age group, of which asthma comprised 1357. In the children, acute respiratory visits were associated with SO_2 levels in the winter; but in the summer there was no such association, although it was present for both asthma and acute respiratory visits in the 15- to 60-year-old age group. No age group showed an association with ozone levels, which were generally low. Data for fine particulate pollution were not available.

Schwartz et al. (*27*) showed that emergency visits for asthma in Seattle were associated year-round with fine particulate pollution (PM10). Levels of PM10 were always lower than the current U.S. standard of 150 $\mu g/m^3$ for 1 hr. The association of increased asthma symptoms with increased particulate air pollution has also been documented by Forsberg et al. (*28*).

Increased Medication Use

Following a cohort of schoolchildren with asthma in Provo, Utah, Pope et al. (*29*) showed that their medication use cycled with the levels of PM10. They found the same thing in a panel of young adult asthmatics and observed that the peak flow rate of normal children was affected by the PM10 level.

Increase in Respiratory Symptoms

Survey Data

In the cross-sectional comparison of six cities in the United States, children's symptoms of cough and phlegm were associated with increased levels of pollutants (*30*). Dodge et al. (*31*) found evidence that respiratory symptoms in children were related to SO_2 pollution levels; the same was true in Charpin's study (*18*). Dockery and Pope (*32*) have recently provided a valuable review of the evidence linking increased fine particulate pollution (PM10) with a range of outcomes in adults and children.

There are so many possible confounding factors in cross-sectional comparisons that this method may be relatively insensi-

tive to differences. For this reason, the study by Neas et al. (*33*) of a cohort of 1567 white children aged 7 to 11 years in the U. S. Six-City Study, is particularly valuable. With measurements of NO_2 in each home, they were able to show that there was a 40% increase of risk in lower respiratory symptoms for a 15 ppb increase in NO_2 in the home. In contrast, in a prospective study of 1205 healthy infants in Albuquerque, New Mexico, Samet et al. (*34*) showed that there was no association between NO_2 levels in the home and the occurrence of respiratory infections. There was no passive tobacco smoke exposure.

Decreases in Lung Function

Summer Camp Data

There is no doubt that ambient ozone, and possibly aerosol acidity, in northeast North America causes a decrease in lung function in normal children at summer camps (*35*). Ozone, sulphate aerosols, and H^+ levels are usually all raised in these locations.

Pollution Episode Data

Brunekreef et al. (*36*) followed a cohort of 1000 children 6 to 12 years of age in rural Holland. For 9 days in January 1987 there was a pollution episode with increased levels of SO_2 and particulates. Significant decrements in pulmonary function were documented, and the depression of function continued for some weeks after the episode. This study followed an earlier one in the same country reported by Dassen et al. (*37*), who had also shown an effect on lung function.

More recently, Koenig et al. (*38*) have shown that woodsmoke air pollution in the Pacific Northwest has a significant effect on lung function in children. These authors studied 326 elementary schoolchildren and 24 asthmatic children during and after the winters of 1988 to 1989 and 1989 to 1990. Increases in the fine particulate fraction (PM2.5) were shown to be related to decreases in the function of the asthmatic children, but the normal children were not affected. Kinney et al. (*39*) in the United States documented changes in lung function in children in relation to episodes during which the ambient ozone was increased. Halfon and Newacheck (*40*), using Health Interview Survey data from the United States, have shown that the impact of asthma is more severe (in terms of increased school absences, increased hospital inpatient days, etc.) in children of lower economic status, presumably because they lack access to routine medication and advice. They also found that children in the lower socioeconomic category depended more on hospital emergency departments for routine care and less on private physicians. Montealegre et al. (*24*) showed that environmental factors, probably particulate pollution, were responsible for aggravating asthma in Puerto Rico. It is of interest that Puerto Rican children resident in the United States show a high prevalence of asthma (*25*). The fact that exposure to environmental tobacco smoke aggravates asthma has been established beyond doubt (*19*). It is not clear whether this is due to particulates in the air or to NO_2.

There is a wide variety of aeroallergens that may precipitate severe asthma in children (*41*).

Chronically Depressed Lung Function

The French study (*42*) of men, women, and children showed that the FEV1 and FVC of children were significantly lower in regions with higher levels of SO_2. The same has recently been shown to be true of nonsmoking women in Beijing (*43*) who are exposed to severe outdoor and indoor pollution. In Wuhan in China (*44*), there were significant differences between the FVC values of children between 8 and 12 years of age as between more and less polluted regions, with girls showing larger differences than boys. The differences in lung function were not only highly significant, but of a magnitude to be considered clinically important.

Indirect Indicators

Kindergarten and School Absence Data

Ponka (*45*) in Helsinki found that absences from school and kindergarten were related to SO_2 levels in the city, but it was difficult to be sure that effects of temperature changes had been completely eliminated. Ransom and Pope (*46*) in Provo, Utah, showed convincingly that school absences in grades 1 to 6 from 1985 to 1990 were significantly associated with PM10 levels. The response was greater in those in grades 1 to 3 (6–9 years old) than in the older children. The results were robust to different model specifications, and weather variables were unlikely to have been responsible for the findings.

Inhaled Lead from Automobiles

There is controversy over how much of the blood lead in children is directly derived from lead in automobile exhaust; indirect inhalation is undoubtedly important (*47*).

However, the recent demonstration that a fall in the blood lead of the population in the United States has precisely paralleled the reduction of lead in gasoline (*48*) and has indicated beyond question that lead in gasoline is an important source. There is justifiable current concern that this problem may be of major importance in the Third World (*49*).

Conclusions

This brief summary of our present understanding of the impact of air pollution on children shows that, when all the data are taken together, there is no doubt that relatively low levels of pollution are responsible for increased morbidity and even mortality in children. This is particularly true of the pollution that follows uncontrolled coal burning. However, in the developed world, there is convincing evidence that current levels of fine particulate pollution are responsible for aggravating asthma and may well also be responsible for increased lower respiratory illness.

Photochemical air pollution at current levels, particularly when associated with sulfate aerosols and increased H^+ levels, is having an impact on acute respiratory disease as well as on asthma in children. It seems probable that when air pollution is severe, the performance of lung function tests is depressed. It also seems likely that in children growing up in such polluted environments a "normal" level of FVC and FEV1 may not be attained—but it remains unclear how much of the cross-sectional difference in these tests in adults between regions of greater or lesser air pollution is a reflection of such an effect in childhood.

It is known that schoolchildren may have relatively high ambient pollution exposures, partly because they are physically active out of doors, but also because, at the time they may be leaving school in the afternoon, ozone levels may be at their highest. Low socioeconomic status, with a resulting lack of routine medical care, may put such children at higher risk if they have asthma—and it has been shown that children from low-income families are more often dependent on hospital emergency departments rather than private physicians for their care and have much more annual morbidity, as judged by school absences and days spent in the hospital than do "nonpoor" children (*40*). In addition, if houses are not air-conditioned, the concentration of outdoor pollutants in them will be

higher. The exposure of such children to indoor pollutants in regions with cold winters may also be higher because of relative overcrowding and inexpensive (but dangerous) heating systems such as kerosene stoves.

There is little doubt that indoor and outdoor air pollution is responsible for major childhood mortality and morbidity in the Third World. Recent research, however, has shown that there are measurable adverse effects on children in the developed world; there is a lack of understanding of the precise mechanisms that underlie the epidemiological data. It is to be presumed that in all cases events in childhood are not without repercussions in adult life.

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