Aspects of Air Pollution Relevant to Child Health

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Summary

Trends in air pollution over the past 50 years are considered, there having been a very considerable improvements in urban air pollution in the United Kingdom with respect to particulate matter and sulphur dioxide. In contrast, traffic-generated pollutants peaked around 1990 and have shown only a more gradual decline. The public health impacts of air pollution in the United Kingdom are reviewed based primarily on the quantification study of the Committee on the Medical Effects of Air Pollutants and the interpretation of the North American cohort studies produced for the same Committee. Evidence for the carcinogenicity of airborne particulate matter is examined and the proposition considered that this may be due to the presence of known carcinogens or could be an intrinsic property of the particulate matter itself irrespective of chemical content. The enhanced vulnerability of children to environmental agents is considered and the benefits of air pollutant abatement reviewed. Children may be at higher risk from exposure to chemical carcinogens, but current ambient air quality standards for chemical carcinogens take account of this. Finally, the role of environmental tobacco smoke as a source of exposure to air pollutants in the home is examined.

Introduction

Outdoor air pollution is an externally imposed risk factor for public health over which control is in the hands of society rather than the individual. The recognition over 50 years ago that air pollution episodes have serious detrimental consequences for human health have led to vigorous regulatory action which has brought about substantial improvements in air quality. However, the rapid growth of motor traffic has thrown up new challenges in air pollution control. This paper examines some of the trends in air pollution in the United Kingdom over the past 50 years and reviews recent evidence for the impacts of air pollution on public health. Carcinogenic risks are given particular consideration as is the vulnerability of children to the effects of both carcinogenic and non-carcinogenic air pollutants.

Air Pollution in the United Kingdom Since 1950 and Quantification of the Effects

The London smog of 1952 caused about 4,000 excess deaths as a result of exposure to smoke (black particulate matter arising largely from domestic coal combustion) and sulphur dioxide. It led to the Clean Air Acts of 1956 and 1968. Due to the introduction of legislation, and perhaps more importantly, the switch from coal to natural gas for home heating, air quality in towns improved rapidly and faster than the reduction of national smoke and sulphur dioxide emissions. Temporal trends in both black smoke and PM_{10} (approximating to particles less than 10 micrometres diameter measured by weighing) show a strong temporal decline since detailed emissions inventories were first constructed in 1970. Concurrent with reductions in emissions of smoke and sulphur dioxide was a massive increase in volumes of road traffic. Traffic-generated pollutants such as carbon monoxide, volatile organic compounds and oxides of nitrogen generally show a maximum in emissions in around 1990 just prior to the introduction of catalytic converters on petrol cars in the early 1990s. Despite a continuing increase in the volume of traffic, the improved emission controls on new vehicles have led to a substantial improvement with respect to emissions of carbon monoxide and volatile organic compounds, whilst reductions in oxides of nitrogen have been more

modest. Also, due to the complex atmospheric chemistry of oxides of nitrogen, the decrease in concentrations of nitrogen dioxide (the more toxic of the two components of NO_x) has been much slower than for NO_x concentrations. PM_{10} in urban air arises from a range of sources including both primary emissions and secondary formation within the atmosphere. Concentrations have been declining since detailed urban measurements started in 1992 and are projected to continue a steady but gentle decline.

Public health impacts of air pollution may be sub-divided into the short-term (acute) effects and the longer-term (chronic) effects. The former are now rather well quantified as a result of time series epidemiological studies linking day-to-day changes in air quality with adverse health outcomes such as mortality and hospital admissions. The UK Department of Health Committee on the Medical Effects of Air Pollutants has issued a report (Department of Health, 1998) in which estimates are made of the acute effects of exposure to PM_{10}, sulphur dioxide and nitrogen dioxide across the UK urban population and of ozone across both urban and rural populations of the UK. The effects quantified within that report of deaths brought forward and respiratory hospital admissions do not give the full picture as there are lesser effects including symptom exacerbations amongst those with respiratory disease and reduced activity days which affect far larger numbers of people.

A small number of cross-sectional cohort studies of the effects of air pollutants on mortality provide the basis for quantification of chronic effects on public health. The UK Department of Health commissioned a study which used coefficients derived from the American Cancer Society Study to estimate chronic effects of $PM_{2.5}$ exposure across the United Kingdom. It was estimated that for a birth cohort born in 2000 and followed for their lifetime, the gain of life expectancy per 1 $\mu g\ m^{-3}$ is from 0.5 to 4.5 weeks averaged across the population. The then background site $PM_{2.5}$ concentrations in London were around 18 $\mu g\ m^{-3}$ equating to between 9 weeks and 18 months life expectancy gain from a complete abolition of $PM_{2.5}$ exposure. Such effects are considerably greater than from the acute effects of PM_{10} exposure.

The carcinogenicity of airborne particulate matter

The extended American Cancer Society Cohort Study (Pope et al., 2002) calculated mortality relative risk associated with a 10 $\mu g\ m^{-3}$ change in $PM_{2.5}$ concentrations after controlling for a wide variety of potential confounding influences such as age, sex, race, smoking, education, martial status, body mass index, alcohol consumption, occupational exposure and diet. This study was the first to show a clear and significant association between exposure to fine particulate matter ($PM_{2.5}$) and increased mortality from lung cancer. Adjusted relative risks, depending on which air quality data are used, range from 8-14% per 10 $\mu g\ m^{-3}$ change in fine particles. The results of this study lead naturally to the question as to whether the observed carcinogenic effect of $PM_{2.5}$ is due to the presence of known chemical carcinogens or whether it is an intrinsic property of the particles irrespective of their chemical content.

In order to examine the proposition that the carcinogenic effect could be explained on the basis solely of known chemical carcinogens in urban air, calculations were conducted using unit risk factors published by the World Health Organisation which represent upper bound estimates of cancer risk from lifetime exposure to unit concentrations of chemical carcinogens. The results of such calculations are highly sensitive to the assumptions made about cancer latency period since there was a very sharp decline in airborne concentrations of

chemical carcinogens in the US atmosphere after 1960 when fairly comprehensive measurements were initiated. If a cancer latency period of 20-30 years is assumed, then the exposure to chemical carcinogens may be able to explain the excess lung cancer risk observed in the American Cancer Society Study, although the possibility that $PM_{2.5}$ is itself carcinogenic independent of chemical carcinogen content cannot be discounted (Harrison et al., 2004). Calculations of the estimated cancer risk from other specific air pollutants in the UK atmosphere are also presented but suggest a smaller risk than from $PM_{2.5}$.

The Enhanced Vulnerability of Children

According to Landrigan et al. (2004) children have an enhanced vulnerability to environmental toxic agents for a number of reasons:

- children have disproportionately heavy exposures to many environmental agents;
- children's metabolic pathways, especially in foetal life and in the first months after birth are immature;
- development processes are easily disrupted during rapid growth and development before and after birth;
- children have more years of future life and thus more time to develop diseases initiated by early exposures.

Specific environmental agents for which children are at enhanced risk include heavy metals such as lead and organic mercury, environmental tobacco smoke, aero-allergens, endocrine disruptors and air pollutants. Wong et al. (2004) have published an assessment of the benefits to the US of pollution reductions due to the Clean Air Act over the period 1990-2010. For children aged 1-16 in the US, annual reductions by 2010 in health outcomes are as follows:

Asthma hospitalisation – 10,000
Emergency department visits – 40,0000
School absences – 28×10^6
Low birth weight – 10,000

These estimates are derived from studies of acute affects of air pollutant exposure. Of at least equivalent concern is a study by Avol et al. (2001) who studied children between 10-15 years of age who moved home between areas of differing PM_{10} concentration. Subjects who had moved to areas of lower PM_{10} showed increased growth in lung function and those who moved to areas of higher PM_{10} showed decreased growth in lung function thus demonstrating that PM_{10} exposure of children may lead to a long-term reduction in lung function.

Lead exposures of children continue to be a matter of concern as recent research shows effects on IQ at very low blood lead concentrations and therefore environmental exposures. Reductions in blood leads in the United States have been widely attributed to the reduced use of lead in gasoline as a result of a parallel reduction in lead use and blood lead over the period 1976-1980. However, experience in the United Kingdom shows no clear coupling between lead in gasoline use and blood leads and it is important to recognise that lead is a multi-media pollutant and that exposures through diet, drinking water and soil/dust ingestion, in addition to inhalation exposures, can make an important contribution to childhood lead intakes.

Based upon studies of the carcinogenic potency of chemicals in laboratory animals, the USEPA has proposed for agents causing cancer through a mutagenic mechanism to use higher carcinogenic slope factors as follows:

- for exposures before two years of age, a ten-fold adjustment;
- for exposures between 2-15 years of age, a three-fold adjustment; and
- for exposures after 15 years of age, no adjustment.

When the UK ambient air quality standards recommended by the Expert Panel on Air Quality Standards are examined, those relating to chemical carcinogens (benzene, 1,3-butadiene and polycyclic aromatic hydrocarbons) are found to include safety factors designed to protect vulnerable groups such as children. The air quality standards for other pollutants in some cases are derived from studies on children, but in other cases, most notably sulphur dioxide, PM_{10} and carbon monoxide do not take account of possibly increased susceptibility in children, although especially for carbon monoxide this may not be of high relevance.

Most people spend in the order of 90% of their time indoors and therefore indoor air is a major source of exposure to air pollutants. Data are presented highlighting the importance of environment tobacco smoke in influencing indoor concentrations of chemical carcinogens.

Conclusions

Whilst air quality is broadly improving, much still remains to be achieved. There is a substantial public health impact of exposures to current levels of air pollutants which affects all age groups. Air pollutants contain a number of chemical carcinogens; particulate matter may be the most important, although questions remain over the mechanisms of effect and its overall public health impact. Children are especially vulnerable to the effects of environmental toxic agents and it is now suggested that they are at higher risk from the effects of chemical carcinogen exposures. The role of environmental tobacco smoke in children's exposure to air pollutants requires greater attention.

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