



Review

Key airborne pollutants—the impact on health

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Abstract

Current appreciations of the effect on health of classical air pollutants including nitrogen dioxide, sulphur dioxide, particulate matter, carbon monoxide and ozone, depend upon modern techniques of epidemiological research. These techniques, which are powerful and able to detect small effects, depend upon the power of modern computers and, though the theory was understood, their application was not possible for most research workers before about 1980. The methods involve handling large data sets and the application of complex methods of regression analysis. Dealing statistically with the many confounding factors that obscure and interact with the effects on health of air pollutants is possible using these methods. Early concerns about the adequacy of handling confounding factors have recently been allayed. This review considers short-term impacts, long-term impacts and areas where further research is required.

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1. Introduction

Air pollution and its effects on health are not a problem of the past (Holgate et al., 1999). Though the concentrations of many air pollutants continue to fall in the UK, they still have a significant impact on health. How large this impact is has only been realised during the past 10 or so years. That levels of pollutants, which are low in comparison to levels recorded in the 1950s and earlier, continue to damage health has come as an unwelcome surprise to many, including policy makers. Explaining how low concentrations of pollutants affect health is a challenge to the research community: mechanisms of effect are beginning to appear but much remains to be discovered.

2. Results*2.1. Short-term impacts*

Studies by Lawther and Waller showed, in the 1950s (Lawther et al., 1970; Waller, 1971), that concentrations of pollutants measured as daily averages were related to respiratory ill-health assessed daily amongst a panel of patients suffering from chronic bronchitis. As levels of pollutants fell the associations weakened and seemed to disappear in the early 1970s. The more recent application of the methods of time-series analysis has shown that these associations still exist and has extended the early work to link daily levels of air pollutants with daily death counts and with the number of people admitted to hospital each day for treatment of cardio-respiratory diseases (Schwartz and Morris, 1995; Morris et al.;

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Atkinson et al., 1999). The associations are clear in the case of particles, less so with regard to nitrogen dioxide and variable in terms of effects on admissions to hospital in the case of ozone. The effects of current levels of pollutants are not, in a statistical sense, strong but the implied impact on public health is large.

Calculations of impacts have shown that some thousands of deaths and hospital admissions are precipitated each year (Dept. of Health, 1998). These calculations have been important in underpinning the Air Quality Strategy for England, Scotland, Wales and Northern Ireland (Dept. for Environment, 2000). Debate about what the calculations of precipitated deaths actually mean continues: it is not possible to determine to what extent death is advanced. Some argue that though the calculated impact is numerically large the real impact is small: very ill individuals dying perhaps just a few days earlier than expected as a result of exposure to air pollution (Gamble and Lewis, 1996). That the effect is as small as this may be doubted and recent work has shown that deaths may be advanced by periods of weeks to months (Schwartz, 2001). Some hundreds of time-series studies have been completed around the world and with increasing sophistication of techniques, interesting variations in the strength of effects from location to location are beginning to appear. This heterogeneity may hold clues to the mechanisms of effect of pollutants and large international collaborative studies are focussed upon this (Samet et al., 2000; Zanobetti et al., 2002).

2.2. Long-term impacts

In parallel with the development of time-series studies a number of prospective cohort studies have been reported (Dockery et al., 1993; Pope et al., 1995). These have produced important and worrying results. The studies show that inhabiting a relatively polluted city for a prolonged period leads to a shortening of life expectancy. This effect can be represented in several ways depending on the method of analysis employed. Broadly, two approaches have been taken:

- (i) estimating the extra deaths occurring by a given date in a population (i.e. deaths not expected to occur);
- (ii) estimating the total, and thence the average, loss of life expectancy by a population that lives out its life, i.e. all members of the population eventually die.

The latter approach has been adopted in the UK to predict the benefit that would accrue as a result of policies that produce a small decrement in particle concentrations. Details of the method may be found in the report of the Committee on the Medical Effects of Air Pollutants (COMEAP) (Dept. of Health, 2001). When undertaking the calculation COMEAP cautiously examined a range of estimates of the size of the effect. These were based on the original prospective cohort studies and on an important reanalysis of the studies undertaken by an independent group of Canadian experts (Krewski et al., 2000). The “bottom line” estimate based on the population alive in 2000 and “followed” for 105 years is:

0.2–0.5 million life years gained per $1 \mu\text{g}/\text{m}^3$ fall in $\text{PM}_{2.5}$ level.

$\text{PM}_{2.5}$ was chosen as the metric because this was used in the original studies. The range (0.2–0.5 million life years) would have been wider if a larger span of the possible coefficients had been considered plausible. It is not easy to grasp this expression of impact but it equates to 1.5–3.5 days per person per $\mu\text{g}/\text{m}^3$ $\text{PM}_{2.5}$. Of course, it is not known how the impact is distributed across the population and for the purposes of cost–benefit analysis—the basis of policy development—this is not too important. But, for visualisation of the impact it should be appreciated that if only 10% of the population are actually affected, then amongst that group—and this is not an implausibly small group—the average loss of life expectancy could be a month per $\mu\text{g}/\text{m}^3$ $\text{PM}_{2.5}$. How large then is the impact of the current level of $\text{PM}_{2.5}$, about $12 \mu\text{g}/\text{m}^3$ in UK cities? If we assume linearity of effect the impact could be a year of life expectancy: this is a significant effect. That such an impact could be occurring today has struck some as implausible but in comparison with known impacts of poverty and deprivation, smoking and poor nutrition, it is not implausibly large.

Comparison of the impact predicted by the time-series studies with that predicted by the cohort studies is intriguing. The predictions of impact are not

directly comparable: each method looks at the effect from a different perspective and it is possible that neither reflects the whole. It seems likely that the cohort studies more closely predict the whole impact than do the time-series studies (Künzli et al., 2000).

3. Discussion

3.1. Causes and effects

That such impacts occur today when levels of air pollutants, especially particles, are probably lower in UK cities than at any time since industrialisation has seemed to some commentators to be implausible (Gamble, 1998). This viewpoint is weakly based and takes little account of the power of modern epidemiological methods to detect small effects with considerable confidence. One line of attack on the predicted effects has been to argue that though associations have been demonstrated they are merely “statistical” and do not represent causal relationships. Much has been written on this point, and the characteristics of causal associations—defined by Sir A. Bradford Hill, a leading worker in the early days of research on smoking and air pollution—have been examined with unprecedented vigour (Bates, 1992). Of such debate there is no end but opinion has swung clearly in favour of causality. One reason for this has been the development and testing of mechanistic hypotheses to explain the epidemiological observations. These have been especially helpful in explaining the observation that the impacts are mediated by effects on the cardiovascular system as well as, and perhaps more importantly than, on the respiratory system.

Several UK workers have figured prominently in this area. Seaton and his colleagues advanced the hypothesis that ultrafine particles could pass through the lung epithelium and trigger changes in factors affecting blood clotting (Seaton et al., 1995). This built upon work by Oberdörster in the US and Peters in Germany (Oberdörster et al., 1995; Peters et al., 1997). This hypothesis has been supported by both epidemiological studies and by studies in experimental animals. In the latter case, work using models of human diseases has been especially productive (Godleski et al., 1996). It is not possible to describe all this

work here but one example that shows impressive linkages will be given.

In 1989, Pope described the beneficial effects on health of a reduction of particle emissions from a steel mill in the comparatively heavily polluted Utah Valley (Pope, 1989). Particles were collected in the valley during and after the period when the operation of the steel mill was prevented by a strike. Experimental animals have been exposed to the particles: the particles collected during the strike were significantly less toxic than those collected during the period of mill operation (Dye et al., 2001). It is notable and expected that during the period of the strike particles collected in the valley contained a lower concentration of iron than those collected when the mill was active.

This study, and others, has focussed attention on particle composition. Work in the UK has shown that metallic components of particles may play an important role in mediating the toxicological properties of the particles. Iron and nickel have been identified as potentially important (Gilmour et al., 1996; Zhang et al., 1998). Running in parallel with this work have been studies of the dose expressed not in terms of mass but in terms of particle number and of total surface areas of the deposited particle load (Brown et al., 2001). These studies have led to the important conclusion that small particles may play an unexpectedly important part in determining the toxicity of the ambient aerosol; they have also led to calls for revision of air quality standards to reflect better the presence of small particles. Deciding on a metric for a standard is, however, a complex process and PM₁₀ continues to be an appropriate base for policy. It should be noted that the great majority of the epidemiological evidence that links particles and effects on health is based on measurement of PM₁₀. This evidence was considered recently in the UK and a recommendation to continue the use of PM₁₀ was made (Dept. for Environment, 2001).

Studies of other pollutants have lagged behind those of particles. Ozone, however, remains a problem and its association with effects on health is clear (World Health Organization, 2000). Levels of ozone in urban areas of the UK are predicted to rise—an unusual example of an air pollutant to which exposure is increasing in the UK (Dept. for Environment, 2000).

An area that has attracted considerable attention recently is the possible link between air pollution and asthma. Some patients suffering from asthma find their condition is adversely affected by exposure to air pollutants. This is unsurprising given that air pollutants, such as sulphur dioxide and ozone, are notable airway irritants (Dept. of Health, 1991, 1992). It is also asserted that air pollution is, in part, responsible for the dramatic increase in the prevalence of asthma that has occurred in many developed countries during the past 20 or so years. This proposition has been reviewed in the UK and the case has not, at the time of writing, been found to be persuasive (Dept. of Health, 1995). Indeed, there are several strands of evidence that argue against an association: levels of pollution have been falling in developed countries as asthma rates have increased, and asthma rates are not high in countries with high levels of air pollution. That air pollution could be playing a minor part, perhaps as an adjuvant to sensitisation to allergens is perhaps plausible but, here again, the actual evidence may be less than generally applicable (Ishizaki et al., 1987; Takano et al., 1997; Tunnicliffe et al., 1994). An association between asthma rates and proximity to busy roads has been demonstrated in some studies but not in others and it is not clear that all confounding factors have been adequately taken into account in earlier studies that do find an association (Venn et al., 2001; Edwards et al., 1994). It is fair to say that if air pollution is playing a part in the rise in asthma rates then it is playing a small part and the mechanisms by which it is doing so remain obscure.

3.2. Future research

The London Smog of 1952 seems far in the past. Air pollution continues to be a cause of ill-health and as levels fall and become less easy to reduce further, much research will be needed to find the components of the current and future air pollution mixture which if reduced will lead to significant improvements in health. If we divide pollutants, for the moment, into gases and particulates it is clear that our understanding of the effects of the gases exceeds that of the particulates. In the case of the gases, the problem is one of clearly demonstrating an effect at low concentrations. Understanding the exact mechanism of that effect is, in comparison with particles, relatively unimportant.

The ambient aerosol is, however, exceedingly complex and identification of those components which if reduced would offer the best option in cost–benefit terms is a challenge. This challenge is being taken up in developed countries and research workers in the UK are to the front of the field in epidemiological and mechanistic studies. This is encouraging. Research workers in the UK played a key role in the early years of air pollution research and do so still. The next 50 years should be interesting.

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