Pollution and People

Air pollution: the "classical" pollutants

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Few disasters can have led to such great good as the four-day London "smog" of December 1952. Though its extreme concentrations of smoke and sulphur dioxide appear to have caused up to 4000 extra deaths,¹ it provided the impetus for the Clean Air Act 1956. Many lines of evidence suggest that the pollution so strikingly reduced by this Act was associated with both acute and chronic illness.² Air pollution has many components (see box) and may be of different types in different places.³ In this article I deal with smoke and sulphur dioxide pollution,⁴ mainly in Britain, and in the next I will touch on other pollutants.

Severe fogs had occurred before in Britain and elsewhere; and smoke had polluted cities for centuries—in fact, the problem was past its worst well before 1952.⁵ In the eighteenth century Benjamin Taylor lectured about "the clouds of Smoke, Vapour, and corrupted Effluvia," correctly pointing to the importance of temperature inversions, the incomplete combustion of coal, and "all the Sooty particles . . . and cloud of Smoke, which if continued, . . . its caustic quality would seize upon our lungs, restrain their vigour, and at last, render them totally incapable of their office."⁶ A century earlier the more famous John Evelyn ascribed the ill health of Londoners to the "fuliginous and filthy vapour"⁷; while a commission was set up to punish those who burnt coal in the City of London as long ago as 1307. Only in the present century, however, could air pollution become an important cause of illness and death: earlier its effects, though suspected, were overwhelmed by those of infectious diseases.⁵ Moreover, potentially lethal effects of intense fog would not be so apparent until there was a large population of susceptible people: by 1952 antibiotics enabled many people to survive with precarious respiratory and cardiovascular systems; and it was these apparently, not the fit, who were killed by the fog.¹

Action against pollution

In Britain the problem was especially serious: in 1951, for example, the death rates from bronchitis for men and women were 107.9 and 62.7 per 100 000 in England and Wales compared with 2.2 and 1.9 in Denmark, and 5.0 and 4.0 in Sweden.⁶ Certainly there were differences in nomenclature and so on, and factors such as climate and housing were seen to play a part; but the difference was attributed largely to pollution by smoke and sulphur dioxide in the Beaver Report of 1954.⁸ Apart from being a more urban country, Britain had a worse problem than others because of its profusion of open grates and low chimneys.⁵ While asking for more research the report concluded that "action to reduce pollution by smoke, grit and dust, and sulphur dioxide need not and must not be held up while

What is air pollution?

The three categories of air pollutants are solid particles, gases, and radioactivity. The fine suspended particles include smoke (black particulate matter), water-soluble material (such as ammonium sulphate), and insoluble ash (containing small amounts of iron, lead, and many other elements). Gaseous pollutants include oxides of sulphur and nitrogen, hydrocarbons, carbon monoxide, and photochemical oxidants. Sulphur dioxide ($1000 \mu\text{g}/\text{m}^3 = 0.35 \text{ ppm}$) and smoke have been the most important air pollutants and are both produced by the burning of fossil fuels. Smoke results from incomplete combustion and sulphur dioxide from the combustion of impurities present in coal and heavy oils. Estimations in cities throughout the world show a wide range of values. Typical annual means for smoke are $30\text{--}200 \mu\text{g}/\text{m}^3$, with highest daily values of $150\text{--}900 \mu\text{g}/\text{m}^3$; and for sulphur dioxide typical means are $100\text{--}200 \mu\text{g}/\text{m}^3$, with highest daily values of $300\text{--}900 \mu\text{g}/\text{m}^3$.⁴ British values tend to be relatively low.

further medical research is done. There can be no doubt that the effect of air pollution on health is wholly bad, whether measured positively in relation to growth, well being and joy of living, or more negatively in terms of death, disease and the economic loss which goes with incapacity to work."⁸

Action followed swiftly. In December 1962, when a somewhat similar episode of "smog" occurred in London, the associated deaths were estimated at only 700; several factors, including publicity about the dangers, seem to have been responsible; but the fog was much less polluted—while sulphur dioxide concentrations were similar to those of 1952 there was far less smoke.⁹ Concentrations of smoke had been falling steadily and continued to do so.¹⁰ Now we can see right across London most of the winter. Eventually sulphur dioxide diminished somewhat also. Gas and electricity were used more for domestic heating and there was better dispersion from industrial and commercial sources. In particular, there were fewer episodes of high pollution for, with less smoke, sunlight could more often warm up the air near the ground, so breaking up the temperature inversions that cause gases as well as particles to become trapped at a low level. Even the lichens—more fastidious than man about sulphur dioxide—are returning, including some not seen since 1800.¹¹

Peaks of sulphur dioxide can still occur in cities given appropriate weather conditions, but they are much less serious. A two-day fog in London in December 1975 was the worst since 1962. Even so, although there were hourly readings of over $500 \mu\text{g}/\text{m}^3$ and the daily mean sulphur dioxide concentrations were over $1000 \mu\text{g}/\text{m}^3$ (five or six times the winter average) at many sites, these were under one-sixth of the 1962 values¹² (and

in 1952 48-hour averages of $3700 \mu\text{g}/\text{m}^3$ were recorded⁴). Daily smoke averages over $500 \mu\text{g}/\text{m}^3$ occurred at only a few sites in the 1975 fog (with some hourly readings of nearly $1000 \mu\text{g}/\text{m}^3$)—up to 10 times the winter average but under one-sixth of the 1962 figures (while in 1952 a 48-hour estimate of $4500 \mu\text{g}/\text{m}^3$ is thought to be conservative⁴). The WHO guidelines recommend limits of $100\text{--}150 \mu\text{g}/\text{m}^3$ (24-hour means) for both smoke and sulphur dioxide.⁴

Epidemiological studies

Since 1952 various types of epidemiological studies have clarified acute and chronic effects of air pollution. No later fog had such severe effects; but statistical studies showed that increases in pollution were often accompanied by increased mortality⁹—both the total number of deaths and deaths from bronchitis increased. In the London studies concentrations of smoke and sulphur dioxide both exceeding about $750 \mu\text{g}/\text{m}^3$ appeared to be the threshold for detectable effects on the death rate.² The WHO review concluded that $500 \mu\text{g}/\text{m}^3$ of both smoke and sulphur dioxide was the level at which short-term exposure produced excess mortality among the elderly and chronic sick.⁴ The effect seemed to be immediate for, in London at least, the number of deaths rose the first day of a fog; but an apparent “compensatory” decrease sometimes occurred immediately afterwards⁹: did it then merely accelerate by a few days the deaths of some whose lives were precarious? In 1952 in particular, cardiovascular but not bronchitis mortality fell almost as soon as the weather cleared.¹ By 1970 mortality changes associated with increased pollution were scarcely detectable.³ Now the death rate for bronchitis is lower everywhere than it used to be, and in London is much closer to rural rates than formerly; though other interpretations are possible, this may be the effect of reduced pollution.¹³

In the 1952 fog illness appeared to be less dramatically influenced than mortality; again, younger people were not seen to be affected. But figures based on sickness certificates and GP consultations are bound to be incomplete.¹ A series of studies based on “diaries” kept by patients with chronic bronchitis was illuminating.¹⁴ Subjective day-by-day records showed that exacerbations were related to increased air pollution—namely, to 24-hour averages of at least $500 \mu\text{g}/\text{m}^3$ of sulphur dioxide, and $250 \mu\text{g}/\text{m}^3$ of smoke (though possibly the harm was done by very brief peaks of much higher pollution). Some patients appeared to be very sensitive, others less so. Such pollution became rarer during the 1960s and by 1969–70 pollution effects were no longer noticeable.² The WHO report puts the threshold of deterioration in patients with respiratory disease at $250 \mu\text{g}/\text{m}^3$ for both smoke and sulphur dioxide.⁴

Respiratory function studies on a few healthy volunteers complemented the diary studies.^{15, 16} The men lived in the suburbs but walked through the streets to work. There were some reductions in ventilatory function associated with high concentrations of smoke and sulphur dioxide, and for a short time specific airways resistance was significantly related to pollution. In time, however, pollution effects were difficult to detect and many other factors—notably minor infections—were clearly more important. In experiments on volunteers with a range of sulphur dioxide concentrations, effects on respiratory function appeared only with deep breathing at $2860\text{--}8580 \mu\text{g}/\text{m}^3$; the highest hourly averages recorded in London have been about $5700 \mu\text{g}/\text{m}^3$ but these may conceal briefer local high peaks.¹⁷ In American work 15 minutes’ exposure to $2900 \mu\text{g}/\text{m}^3$ in the laboratory produced a slight effect on total respiratory resistance.¹⁸ Laboratory simulations of particulate pollution affected lung function only at concentrations much higher than occur in London air—though people with damaged lungs may, of course, be more sensitive.²

Many studies have shown long-term effects of air pollution on respiratory symptoms and function by comparing groups living or brought up in areas of high and low pollution.³ The

WHO review concluded that effects were detectable with annual means of about $100 \mu\text{g}/\text{m}^3$ for both smoke and sulphur dioxide.⁴ Below that level any effects would have been masked by other factors. Some interesting surveys have been carried out in children and young adults. A national survey of health and development looked at respiratory tract infections in a cohort of children born in 1946; allowing for social class, it found that they increased with increasing pollution at least up to the age of 15.²⁰ Among children living in urban and rural areas of Kent the worst polluted area gave the lowest peak expiratory flow rates, but there was no gradient from the most to the least polluted place.²¹ A national study, which started in 1973, based on St Thomas’s Hospital² did show a gradient, for respiratory illness, from places with smoke averages of $150 \mu\text{g}/\text{m}^3$ down to those with $10 \mu\text{g}/\text{m}^3$; but this preliminary analysis used pollution levels for only one month and results from later years are awaited. A follow-up study was done in Sheffield schoolchildren. In 5–6-year-olds upper and lower respiratory tract infections were related to the level of local pollution independently of socioeconomic factors.^{22, 23} Worse results in respiratory function tests were associated with a history of respiratory illness, even in these young children, and to living in the worst polluted area. Four years later pollution had diminished all over the city and there was no correlation between respiratory illness and area of residence. A comparison of 18-year-olds brought up in London and Crawley New Town also suggests some influence of air pollution: slightly higher incidences of respiratory symptoms occurred in the former group. But smoking and a history of respiratory illness appeared to be more important, and these were associated with lower ventilatory capacity.²⁴

In a large WHO study on children aged about 10 in several European countries, air pollution was closely associated with respiratory symptoms and lower peak expiratory flow rates, smoke having a greater effect than sulphur dioxide.¹⁹ Whether a threshold exists for pollution effects cannot be decided from the results; but no association was seen in the less polluted areas—the Danish data, for example, suggest that there are no ill effects below $69 \mu\text{g}/\text{m}^3$ for sulphur dioxide or $29 \mu\text{g}/\text{m}^3$ for smoke. The report recommends that all future work should take account of parents’ chest symptoms and smoking habits.

Should we do more about sulphur dioxide?

In the general environment ill effects of sulphur dioxide are detectable at much lower levels than the threshold limit value ($14\,300 \mu\text{g}/\text{m}^3$) based on industrial experience with this gas.² Possibly it is a marker for other air pollutants or acts syner-

Air sampling apparatus at the Medical Research Council Toxicology Unit (clinical section), St Bartholomew's Hospital Medical College. Reproduced by courtesy of the unit.

gistically with them—or the general population may be more susceptible. Though obvious effects of air pollution, both acute and chronic, have disappeared with the reduction of smoke, some individuals are likely to suffer in the urban areas still subject to high concentrations of sulphur dioxide. But how much are the vulnerable people exposed to it, out of doors and in adverse weather? Indoors sulphur dioxide concentrations are much less now that ventilation rates are lower and open coal fires uncommon⁴; and such people will benefit in that respect. Smoking is clearly a much more widespread cause of respiratory impairment and disease.

Further reductions will not be easy. Power stations emit the most sulphur dioxide,¹⁰ but in Britain at least the stacks tend to be high enough to disperse it widely. Washing the gases to remove sulphur dioxide has disadvantages: it is extravagant of energy, and by cooling the plumes it makes them disperse less readily. In Britain emissions from industrial, commercial, and other such sources are now the main problem. The City of London enforces the use of low-sulphur (1%) oil; supplies and finance would not allow this to be done universally, but it would be desirable in the worst-affected areas. The WHO guidelines⁴ for sulphur dioxide are exceeded, at least sometimes, in many places; but the scientific basis—in terms of health benefits—is admitted to be uncertain. Moreover, standards that are at present unrealistic for particular areas will be ignored; the strategy that worked so well after the Clean Air Act was to reduce pollution as vigorously as possible by the “best practicable means.” Epidemiology can rarely be conclusive about individual pollutants in a complex environment and a sounder approach, it has been said,³ might be to identify and control specific hazardous environments rather than concentrating on numerical standards.

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Does ovulation usually occur in the left and right ovaries alternately? Are non-identical twins the result of ovulation in the left and right ovaries in the same month, and are they more likely to result from the first ovulation after a delivery, when ovulation might be incoordinated, than from other ovulations? Could breast-feeding influence bilateral ovulation?

After each menstruation several new follicles begin to develop, but normally only one of them progresses to full maturity. How this selection occurs is unknown: the process appears to be random, and therefore over a woman's lifetime each ovary will contribute roughly half the ovulations. So far as we know the same ovary may ovulate in successive months, and when one ovary is removed the other will ovulate repeatedly. Since it is now possible to study ovulation by ultrasound,¹ more definite information may soon become available. Non-identical twins result from double ovulation, either in a single ovary or in two ovaries simultaneously. (As long ago as 1787 John Hunter, studying pigs, found that unilateral oophorectomy in the sow did not prevent twinning.) The epidemiology of human non-identical twins is well-documented²—for example, the rate of twinning increases with age, and twins are commoner among illegitimate pregnancies and among women who conceive in the first three months after marriage—but there is no evidence that twins are commoner in the first ovulation after delivery. Indeed, the reverse may be the case. Pituitary overproduction of follicle-stimulating hormone may contribute to twinning (just as excessive exogenous follicle-stimulating

hormone during artificial induction of ovulation produces multiple pregnancy) and if there is any truth in this suggestion twinning rates would be decreased during breast-feeding, when concentrations of follicle-stimulating hormones are lower than normal.³

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A fit 30-year-old man had a proved episode of optic neuritis when he was 25, and possibly when he was 19. Are there any statistical studies to show the likelihood of him developing any other demyelinating disease?

Unilateral optic neuritis is rarely due to any disorder other than multiple sclerosis, but further evidence may be delayed as long as 20 years or never appear.¹ The proportion of patients presenting with optic neuritis and going on to develop signs of multiple sclerosis within the next 10 years has been stated as 20-50%,²⁻⁴ but it will be a considerably smaller proportion of patients who are debilitated by the disease.

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