The ALLERGY ARCHIVES

Pioneers and Milestones

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A perspective on air pollution

Air pollution has a long history, and documented efforts to reduce its growing effect in Britain can be traced back to the publication by John Evelyn in London in 1661 of ''Fumifugium, or The Inconvenience of the Aer and Smoak of London Dissipated … .''

London was dependent on open coal burning for heating, and as the volume of coal consumed increased, so did the resulting air pollution. A warning of what was ahead came in December 1873 when cattle at the Cattle Show Week in London died from the effects of a severe fog.[1] This does not seem to have rung any alarm bells.

Claude Monet, the great French Impressionist painter, had brought his family to London for safety during the Franco-Prussian war and had noted the variety of colors produced by the interaction of sunlight and the mix of pollutants. He described these as ''black, brown, yellow, green, purple fogs,'' and in the winter of 1901, he came to London, renting a room with a balcony at the Savoy Hotel on the Embankment. Waterloo Bridge was to his left, and the angular Charing Cross railway bridge was to his right. Officials at St Thomas' Hospital gave him the use of a cupboard so that he could paint canvases of the House of Commons from across the river.

He went back to Paris with 96 canvases of these subjects, 36 of which were of Waterloo Bridge.[2] These provide a remarkable view of the varying mixture of pollutants, mainly SO_2 and coarse and fine particles, that made up the London winter atmosphere at that time. These canvases are now widely scattered, and I have reproduced elsewhere one from the National Gallery in Washington and another from the National Gallery in Ottawa.[3] Having the opportunity to visit the Hermitage Museum in St Petersburg (where photography is permitted), I took a picture of the Monet *Waterloo Bridge* painting there. This is shown as Fig 1 and is darker than many of the others.

In 1930, a severe temperature inversion in the heavily industrialized valley of the Meuse River in Belgium led to immediate casualties and was the subject of an enquiry that concluded that acid aerosols were probably responsible for the adverse health effect.

Devra Davis[4] has added many graphic details of the dangerous smog that enveloped the town of Donora in Pennsylvania on October 26, 1948. The factory in the valley there was plating zinc and employed 1500 men, and it continued its acidic emissions, which also had a high fluoride content, during a period of temperature inversion. Davis was present in the town as a girl and recalls how the annual Halloween Parade took place as usual. The immediate excess mortality was about 20 people, but the actual toll was in excess of 50, as Davis notes that, ''The fifty people who died in the month following the smog are nowhere counted.'' Perhaps because the town was relatively small, this episode did not lead to any widespread demand that industrial emissions should be regulated or controlled. The city of Pittsburgh, with a major steel industry needing many coke ovens, had issued an ordinance in 1946 for clean air legislation. Tarr[5] has documented the 5-year campaign that finally led to this action, the city's Director of the Department of Health taking a leading part in these events, although he lacked any epidemiologic studies that showed the harmful effects of the pollutant emissions.

The London episode of December 1952 became a landmark event.[6] It is now believed that at least 12,000 people died as a result of the pollution,[7] although at the time, the mortality was thought to be about 4000. The government of the day was defensive, arguing that steps to limit the pollution would be too expensive and that London fogs had occurred for a hundred years. Prize cattle being shown at the Smithfield Show, which was running at the same time, were also affected,[6] just as they had been in 1873. The debates in the House of Commons were not edifying, but the government was finally pushed by an aroused public opinion to establish a committee to examine the issue and provide advice on what actions might be taken. Mr Harold McMillan was the minister who answered for the government; he took a cynical view of what such a committee might achieve, telling the cabinet that it would do something to assuage public opinion but would achieve little else. In this he was wrong because the Beaver Committee correctly identified the problem, and their advice—that local authorities should have the statutory power to prohibit open coal burning—laid the foundation for effective control. Mr McMillan's autobiography covering this period does not mention the smog disaster.

The London episode triggered widespread action to reduce air pollution emissions. The relative importance of particulate and SO_2 emissions was not known, but technologic methods soon became available to reduce both of these emissions from coal burning used in major electric-generating facilities. Ambient levels of these pollutants decreased graphically in all major western centers but remained high in Eastern Europe.

FIG 1. Claude Monet, *Waterloo Bridge,* 1903. This is one of a total of 36 paintings he made of this subject, illustrating the interplay between changing light conditions and the air pollutants derived from open coal burning. This example hangs in the Hermitage Museum in St Petersburg and was photographed by the author. Claude Monet, *Waterloo Bridge, Effect of Fog,* reproduced by permission of The State Hermitage Museum, St Petersburg, Russia.

Today, this type of pollution is still a problem in parts of Asia.[8]

It was in the same year as the London disaster, 1952, that a Dutch plant physiologist working in Los Angeles (Arie Haagen-Smit) discovered that tropospheric ozone was being formed from oxides of nitrogen in the presence of hydrocarbons and sunlight; he thereby ushered in a new era in air pollution control. The use of automobiles was expanding rapidly in every city where they could be afforded. Dangerous levels of ozone were being generated, especially in those cities in which the summer sunlight intensity was high. The US Clean Air Act of 1970, drafted after a series of Senate hearings on every aspect of air pollution, established the infrastructure for the control of the major criteria pollutants and also delineated a large group of hazardous compounds for which emission limits had to be set. The progressive control of automobile emissions over the next 30 years constituted a remarkable tango between the industry and the regulators. The state of California had the right to set its own standards and led the battle to reduce vehicle emissions (introduced in 1966); on a broad perspective, this was a highly successful endeavor, with the emission reductions more than keeping pace with the growing automobile fleet. Policy in Europe and North America diverged in the 1990s, with Europe building much larger numbers of diesel vehicles and with agencies in North America becoming increasingly concerned with the apparent risks of human exposure to the particles in diesel exhaust.

In 1979, the US Environmental Protection Agency changed the indicator for particulate pollution from total suspended particulate to particles with a mean aerodynamic diameter less than or equal to 10 microns (PM10). Measurements were made every sixth day initially. From 1987 onward, studies began to be reported from a number of different urban locations that indicated a significant association between daily mortality (particularly from cardiac and respiratory diseases) and the PM10 levels the day before. This development transformed the medical evidence on which control of air pollution had to be based. Earlier studies had relied on comparisons between different populations living in areas of different air quality to show the effects of air pollution. Because different populations usually differ in many other respects that will affect their health, such as socioeconomic status, smoking habits, occupation, housing differences, and so on, such comparisons are a relatively insensitive method to show the effect of ambient air pollution. In studies designated as time-series studies, an association is sought between some health outcome (eg, daily mortality or hospital admissions) and the level of ambient pollution the day before. Also, 2 large-scale longitudinal studies had been launched in the United States in which the smoking history and occupational history were known, and these provided reinforcement for the effects shown by time-series methodology, although the confounders affecting the 2 types of study were very different.

A third research method that began to be exploited was the recruitment of a panel of subjects, often with a disease such as asthma, and the recording of the daily fluctuations in their medical status and lung function as their exposure to different levels of particulate and gaseous pollutants changed. The planning and execution of such panel studies have reached a remarkable degree of sophistication, as shown by a recent panel study of 19 asthmatic subjects conducted in the town

of Alpine in California.[9] These powerful methods have greatly increased the scope and credibility of the data relating adverse health outcomes to the fluctuating exposures encountered by the normal population. They have also revealed the existence of especially vulnerable populations, particularly asthmatic patients and those with cardiovascular disease.

So rapid has been the advance of sophisticated methods of detecting induced changes in physiologic and biochemical parameters that we are now in a position of being able to detect such changes that precede any alterations in simple function tests or the development of any symptoms of disease. A recent study reported from North Carolina involved placing air-monitoring equipment, including PM2.5 recorders, on the rear seats of police patrol cars during a shift of duty from 3 PM to midnight. Electrocardiographic recordings and blood sampling revealed that the low levels of urban particulate pollution encountered were inducing measurable changes, and the authors concluded that, ''The observations in these healthy young men suggest that in-vehicle exposure to PM2.5 may cause pathophysiologic changes that involve inflammation, coagulation, and cardiac rhythm.''[10] Another example is the recent demonstration in asthmatic subjects that exposure to PM2.5 can induce an inflammatory response, as indicated by changes in the expired nitric oxide concentration that precede any change in lung function or other parameters.[11]

We have therefore come a long way from the days when changes in mortality were the only indicators of the effects of air pollution on a population. Even though the interpretation of studies of air pollution effects is still the object of considerable dispute—mostly between interested parties and the regulators—we are now in a position to plan effective action to reduce air pollution to levels at which it could no longer be shown to be exerting any adverse effect. This, after all, must remain the objective. It formed the stated objective of the US Clean Air Acts, but it is easily buried beneath the papers generated by bureaucratic paper wars. It is impressive that over this space of more than 50 years, it has been public opinion and pressure that has led the fight for better control of emitted pollutants. In Britain, Lord Ashby remarked 40 years ago that it was difficult to find instances in which government initiative alone had led the way forward to reductions in air pollution, and the same conclusion could be reached today—it is the exceptions that prove the rule.

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