traffic. As long ago as 1983, Ames tests carried out by the Swedish National Environmental Protection Board showed diesel to be 10 times more mutagenic than leaded petrol, which in turn was 10 times more mutagenic than emissions from a car running on unleaded petrol and equipped with a three-way catalytic converter.4

Even more important from a public health point of view is that four fifths of the mutagenic effect resides in the particulate fraction of diesel exhaust which is being trapped or filtering system will virtually eliminate the risk to health. Despite this, the European Union’s current emission standards for taxis, buses, and lorries do not introduce the use of particulate traps. 


The British government has done nothing to implement these proposals. Instead it has deregulated the bus services and left the choice of fuel to individual commercial operators.

Concern about diesel is not new. I quote from a memorandum submitted by Friends of the Earth to the House of Lords European Community Committee (Environment) in 1987: ‘There is a direct correlation between levels of traffic particulates in the atmosphere and infant mortality, and clear evidence of particles aggravating disease among bronchitics, asthmatics, and susceptible patients and people with influenza.’ While it is gratifying to read a BMJ editorial on the need for tighter legislation to control pollution, environmental pressure groups have been aware of similar data from the United States for over a decade.

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Asthma and the Environment

Asthma epidemics and air pollution

Upper respiratory tract infection and fall in atmospheric temperature may lead to attacks of childhood asthma

EDITOR—Adrian Bauman suggests that epidemics of asthma may yield clues to causal agents.1 We report such an epidemic in east Birmingham in 1987.

Between 14 September and 7 October, 86 children meeting our case definition for asthma (a reversible wheeze, reversible dyspnoea, or ratchy coryza with wheezing) were admitted to Birmingham Heartlands Hospital, a rate 3.58 times higher than that in the preceding four weeks and 3.44 times higher than that in the same period in 1986. There were 12 patients aged under 2, 37 aged 2-4, 26 aged 5-9, and 11 aged 10-11—a similar distribution to that in the previous year. More boys were admitted than girls (55:31; relative risk 1.77 (95% confidence interval 1.16 to 2.64)), as in the previous year. Sixty four children were white, 20 Asian, and two African-Caribbean, reflecting local birth data. Sixty eight had previously been diagnosed as having asthma (including eight aged under 2), and a further six had a clear history of nocturnal cough or exercise induced wheeze, or both. Forty eight had been admitted before.

Seventy four met our definition for coexisting clinical respiratory infection (coryza, visualised pharyngitis, or cough with fever). However, only one of 86 throat swabs collected on admission yielded a positive result on bacteriological testing (Streptococcus pyogenes) and two of 32 throat swabs collected later in the child’s illness (and only in the later part of the epidemic) yielded a positive result on viral testing (one adenovirus and one echovirus). Local meteorological data and pollution data for the period 1 August to 5 October showed that the single day in which mean air temperature between days (3.9°C) occurred on 13 September, the day before the start of the increase in admissions; it was accompanied by reduced humidity and increased barometric pressure, but no relevant changes in monitored pollution levels.

This epidemic contrasts with that reported in London and an outbreak in Birmingham2 in that it occurred in autumn, affected children (mostly known to be asthmatic), and was associated with a high prevalence of presumed viral respiratory infection and a fall in temperature without a thunderstorm. Viral respiratory infection is associated with exacerbation of asthma, and the velocity of changes in temperature can predict hospital attendance for wheezing.3 Autumnal increases in acute attacks of asthma in general practice coincide with the seasonal rise in acute bronchitis.4 Taken with this epidemic, this suggests that both asthma and chronic respiratory infections are triggered by respiratory infections.5

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1 Bauman A. Asthma associated with thunderstorms. BMJ 1996;312:590-1. (9 March.)


Epidemic of asthma was not associated with episode of air pollution

EDITOR—Are epidemics of asthma caused by outdoor air pollution or do episodes of air pollution cause epidemics of asthma? These and other questions relating to asthma and air pollution were addressed in detail by a recent report,8 but the answers emerge clearly from a simple inspection of data available for London in recent years.

Figure 1 shows daily concentrations of nitrogen dioxide in London, September and October 1993, together with daily admissions to hospital for asthma. An episode of pollution occurred, during which nitrogen dioxide concentrations in central London reached historically high levels (40 ppb; one hour average), which were five times higher than the seasonal average. Particles increased to a similar degree. No obvious effect on hospital admissions for asthma was discerned.

Figure 2 shows daily ozone concentrations in London in the summer of 1994. Together with daily attendances for asthma at the accident and emergency departments of 12 hospitals in London (unpublished data). A striking epidemic of asthma occurred, during which attendances were nearly five times the seasonal average.