traffic.⁵ 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.⁶

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, so that an effective trapping 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 require the use of particulate traps; nor will the new standards scheduled for 1997. In its 18th report the Royal Commission on Environmental Pollution recommended the use of fuels such as liquid petroleum gas or natural compressed gas instead of diesel for urban based vehicles.⁷ 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 strong correlation between levels of total suspended particulates in the atmosphere and infant mortality, and clear evidence of particles aggravating disease among bronchitics, asthmatics, cardiovascular 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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Doctors should support parliamentary bill

EDITOR,—David V Bates concludes that it may be public pressure and not the medical profession that leads to further clean air legislation in Britain.¹ Public pressure to reduce road traffic has already begun, and medical professionals should now add their weight to the campaign.

Last March saw the introduction to parliament of the Road Traffic Reduction Bill, which would require the government to set targets for reducing road traffic miles by a tenth by 2010. The bill was introduced by Mr Cynog Dafis. He has introduced similar bills in previous sessions of parliament, but they have always been opposed by the government. This bill will undoubtedly also be opposed. However, a campaign is now building behind the bill; this is being led by Friends of the Earth and the Green

party, which are trying to build enough public support to push it through.

Community based campaigns have succeeded before. Last year an alliance of local authorities, pensioners' groups, parish councils, and environmentalists managed to win government support for the Home Energy Conservation Bill, which it had previously opposed two years running. Now an act, this measure is leading to better insulated homes for a large number of people.

Doctors should be getting involved. Bates outlines our knowledge of the dangers of air pollution to health. Add to these the reduction in traffic accidents that would result from a reduction in traffic and the fitness benefits of cycling and walking, and we have a policy that doctors should back.

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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 (reversible wheeze, reversible dyspnoea, or tachypnoea 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 —a similar distribution to that in the previous year. More boys were admitted than girls (55 v 31; relative risk 1.77 (95% confidence interval 1.16 to 2.86)), 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 gave 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 largest single fall in 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 rain and no relevant changes in monitored pollution levels.

This epidemic contrasts with that reported in London^{2,3} and an outbreak in Birmingham⁴ 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,⁵

and the velocity of changes in temperature can predict hospital attendance for wheezing.⁶ Autumnal increases in acute attacks of asthma in general practice coincide with the seasonal rise in acute bronchitis.⁷ Taken with this epidemic, this supports the hypothesis that a combination of a causal agent (a virus) and a meteorological variable (fall in temperature) is more likely to lead to an attack of asthma than either in isolation.

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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,¹ 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 in the winter of 1991 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 (423 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.²

Fig 1—Daily admissions for asthma and nitrogen dioxide concentrations in London, November 1991 to January 1992

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.