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.

There is no evidence that this was associated with raised concentrations of ozone (or of particles (PM₁₀) or nitrogen dioxide; data not shown).

Fig 2—Attendances at accident and emergency departments for asthma and daily ozone concentrations in London, May to August 1994

These data show clearly that the episode of air pollution in 1991 was not associated with an epidemic of asthma. Conversely, the epidemic of asthma was not associated with an episode of air pollution. These findings are reassuring so far as the acute effects of air pollution are concerned. The epidemic of asthma in 1994 occurred within hours of a thunderstorm,³ and this, in conjunction with high pollen counts on previous days,⁴ is the most likely explanation for the abrupt rise in attendances for asthma at accident and emergency departments on 24–25 June.

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Ozone and childhood wheezy episodes

Needs testing as an a priori hypothesis

EDITOR,—Roger Buchdahl and colleagues conclude from their analysis of associations between air pollution and the incidence of acute childhood wheezy episodes that a critical ozone concentration exists, above or below which children are more likely to develop symptoms.¹ Their finding of a relation with very low ozone levels has not been previously described. We have some concerns about the way in which this finding is presented.

The emergence of the finding from a series of complex statistical tests bears all the hallmarks of a classical a posteriori hypothesis. In effect, the hypothesis was generated by the study rather than the study being set up specifically to test it. Nor do the authors put forward a plausible explanation for the observation. Some caution is therefore necessary in interpreting the finding, and although it is entirely apt to consider its implications in the paper's discussion section, we

believe it is inappropriate to give it such prominence in the paper's abstract and key messages. The validity of the finding will remain uncertain until confirmatory evidence is available from further work which tests it as an a priori hypothesis. In the meantime, the finding should be treated with greater circumspection.

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Meteorological confounders must be considered

EDITOR,—Roger Buchdahl and colleagues report a study of attendances by children to an accident and emergency department with acute wheezy episodes.¹ These were measured against day to day variations in gaseous pollutants using the OPSIS system situated on the roof of a hotel presumably at least 30 metres above mouth level. The authors state that they have taken into consideration meteorological variables but seem to have assessed only 24 hour temperature and wind speed. No measure was made of particulate pollution, whether as black smoke or particles less than 10 microns in size (PM₁₀).

The subsequent results showing a U shaped association between ozone and cases of acute wheezing illness is most unusual and has not been seen in any previous study. The most likely explanation of this finding is that inadequate account of confounding variables was taken, notably rates of change or absolute variation in temperature over 24 hours or, crucially, particles. Without these measures the data are uninterpretable and it is perhaps remarkable, in a field where there is so much evidence of the effects of particles on health, that they have been completely ignored in this study. The conclusion that a critical ozone concentration seems to exist in the atmosphere, above or below which children are more likely to develop symptoms, cannot be made using these results.

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Results may have been confounded by viral infections and faulty equipment

EDITOR,—Roger Buchdahl and colleagues report that hospital attendances for acute childhood wheezy episodes were significantly elevated on days when ozone concentrations were very high and very low.¹ They suggest that this U shaped relation may indicate an optimal protective concentration of atmospheric ozone, with adverse health effects being associated with higher or lower values. An alternative explanation is that the observed association is the result of confounding by exposure to viral infections that

are known to be important causes of wheezy episodes in childhood.²

Most ozone at ground level is formed photochemically by the action of sunlight on nitrogen oxides and volatile organic precursor pollutants. In the United Kingdom the highest concentrations of ozone usually occur in summer and are considerably lower in the autumn and winter. The days with very low ozone concentrations in the study by Buchdahl and colleagues are therefore likely to represent autumn and winter conditions, when viral infections are more common. This suggests that the excess incidence of wheezing at such times is more likely to be the result of increased exposure to viruses than to any causal effects of very low levels of ozone.

The lower incidence of respiratory infections during the warmer months offers less scope for these to confound any association between summer ozone peaks and the incidence of wheezy episodes. However, a distinctive feature of Buchdahl and colleagues' study was that a substantial proportion of the days with high ozone were recorded during a period of consistently high readings during October and early November 1992. Continuous periods of high ozone levels are unusual at that time of the year and none were recorded at any of the national automated network sites in the region.³ A possibility is that high values in Buchdahl and colleagues' study may have been the result of technical problems after a failure of the air pollution monitor from 26 September to 6 October.

In most years there is an early autumn peak of hospital admissions for asthma in children, which may be related to viral epidemics after the return to school.⁴ The observed excess on days with high recorded ozone may therefore not represent a causal relation but a coincidence between the period of possibly unreliable high ozone readings and the usual early autumn excess of wheezy episodes related to viral infections.

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Authors' reply

EDITOR,—The implied criticism by Merion R Evans and colleagues of not using an a priori hypothesis in our study perhaps fails to appreciate that the study was observational, undertaken in response to the controversy concerning air pollution and childhood asthma. The findings have not emerged as a result of performing "a series of complex statistical tests," as can be seen from the plots of the raw data.

The following a posteriori hypotheses may be made. Firstly, there may be a critical concentration of ozone which has a beneficial health effect. A second, and in our view a more plausible, hypothesis recognises ozone as a secondary pollutant. Under certain atmospheric conditions predisposing to very low or very high levels of ozone, other air pollutants may accumulate and these may be related more closely to incidence of wheeze. We speculate that volatile organic compounds or other hydrocarbons may be relevant. A test of the hypothesis would be to see