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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1 Department of Health Committee on the Medical Effects of Air Pollutants. *Asthma and outdoor air pollution*. London: HMSO, 1995.

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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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1 Buchdahl R, Parker A, Stebbings T, Babiker A. Association between air pollution and acute childhood wheezy episodes: prospective observational study. *BMJ* 1996;312:661–5. (16 March.)

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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1 Buchdahl R, Parker A, Stebbings T, Babiker A. Association between air pollution and acute childhood wheezy episodes: prospective observational study. *BMJ* 1996;312:661–5. (16 March.)

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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