possibility of bias but also the plausible direction in which it might operate.

We were at great pains, even in the title of our paper, to show that our findings supported a familial aetiology and that this might be genetic. On the other hand, the segregation analysis makes the genetic aetiology more plausible; we made it quite clear that this was not proof. We also pointed out that voluntary childlessness might not have been exactly the same in both groups, and therefore we performed a sensitivity analysis to see what effects this bias might have had on our conclusions—a point that Gregson and colleagues overlook.

The high incidence of subfertility would not seem to explain the striking similarity in the sperm counts across the three families in which extended sampling was permitted.

Our paper shows that we were aware of all the biases identified by Gregson and colleagues, and more besides. We believe that our findings should be corroborated and that they are an intriguing clue. If Gregson and colleagues can suggest a method to overcome bias arising from what must inevitably be verbal evidence then we would love to discuss it with them. We would, for example, like to know whether they really think that observers could be blinded in a study of genetic histories and whether this has been accomplished successfully for other topics for which genetic histories are required from cases and controls.

With regard to the letter from F J Stewart and A J Hill, absence of the vas deferens causes azoospermia, whereas we were concerned with oligozoospermia. There is no particular reason for thinking that oligozoospermia might be associated with mutations in the cystic fibrosis gene.

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Natural history of childhood asthma

Other studies have been ignored

EDITOR,—In his editorial Malcolm R Sears concludes that two thirds of children with asthma will outgrow it (especially those with mild asthma).¹ This statement is primarily based on the work of Mark A Jenkins and colleagues,² but it does not reflect several other solid research findings that show that asthma persists into adulthood in half of the children who have it.³⁻⁵

The study of Jenkins and colleagues is presented as a prospective study based on the 1968 Tasmanian asthma survey. In this survey Tasmanian parents were retrospectively asked whether their children, then at school, had ever suffered from asthma or wheezy bronchitis. Jenkins and colleagues did a follow up study 23 years later (again partly retrospectively) in which a random sample of the original population was asked whether they suffered or had previously suffered from asthma. The questionnaire seemingly did not define the term asthma.

Jenkins and colleagues concluded (a) that “in most cases, childhood asthma did not seem to persist . . . and most asthma in these adults . . . seems to have developed after the age of 7” and (b) that, “onset of parent reported asthma or wheezing before the age of 2 did not predict asthma as an adult.” To base findings on parents’ recollection, however, has limited scientific merit because the recall bias must be great and the diagnostic reliability small.

Jenkins and colleagues begin their introduction: “Few population based and truly prospective cohort studies have been published of the natural course of asthma from childhood to adult life,” but

several relevant studies spring to mind. One is Blair’s prospective study over 20 years of children from a London group practice.³ In Blair’s study asthma was clearly defined as a minimum of three attacks of paroxysmal dyspnoea with wheezing. Blair concluded that asthma persisted in half of all the cases, irrespective of time of onset, and that it disappeared or was only mild in the other half; that the age of onset of asthma was under 2 years in 57% of the 267 asthmatic children; and that there was no correlation between early age of onset and severity of asthma 20 years later.

Another important study is that of Martin *et al*, which was based on a cohort of 7 year olds in Melbourne.⁴ They found that half of the children who started wheezing before the age of 7 years still had asthmatic symptoms at the age of 21. These results were confirmed by Oswald and colleagues when the cohort was 35.⁵ Martin *et al* commented that they had not used a control group “because children who had wheezed in infancy probably were not identified because of failure in parents’ memory.”

Given the introduction to Jenkins and colleagues’ paper, I wonder why Sears does not mention or discuss the above studies in relation to the natural history of asthma. Blair’s study is a truly prospective follow up study, while Jenkins and colleagues’ study is partly retrospective but presented as a prospective study. Conclusions drawn from a study based on the methods used by Jenkins and colleagues seem dubious.

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- 3 Blair H. Natural history of asthma: 20 years’ follow up. *Arch Dis Child* 1977;52:613-9.
- 4 Martin AJ, McLennan LA, Landau LJ, Phelan PD. The natural history of childhood asthma to adult life. *BMJ* 1980;280:1397-400.
- 5 Oswald H, Phelan PD, Lanigan A, Hibbert M, Bowes G, Olinsky A. Outcome of childhood asthma in mid-adult life. *BMJ* 1994;309:95-6. (9 July.)

Author’s reply

EDITOR,—Distinctions must be drawn between the natural history of a disease in clinical practice and that determined in an epidemiological study. Blair reported a 20 year follow up of 267 children treated for asthma before the age of 12.¹ In these children, who were not a random sample drawn from the community, asthma persisted into adult life in 21% and went into remission and then caused a relapse in 28%. Very mild asthma was detectable in a further 24% of cases, leading Blair to conclude that most children with asthma did not grow out of their disease. Martin *et al* followed up the same Melbourne cohort as Oswald and colleagues.^{2,3} Although initially the Melbourne study used a randomly selected sample, this was “enriched” with subjects with more severe asthma to provide adequate numbers for long term follow up. The outcome was that most (but not all) asthma persisted.

In contrast, Jenkins and colleagues⁴ studied a community sample in which the severity of asthma ranged from trivial to severe and a large proportion of subjects had no symptoms. In this population sample mild asthma tended to remit while more severe asthma persisted. This finding is not contradictory to the studies mentioned by Marianne Stubbe Østergaard, but it shows the importance of clearly identifying the population under study when outcome is determined.

The Melbourne sample^{2,3} and the children studied by Blair¹ are not epidemiologically representative of their communities but are subjects

selected for having asthma. They are therefore likely to have somewhat more severe and persistent disease. Even so, some grow out of asthma, but remission is more evident among subjects with milder disease.

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- 1 Blair H. Natural history of childhood asthma. *Arch Dis Child* 1977;52:613-9.
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- 3 Oswald H, Phelan PD, Lanigan A, Hibbert M, Bowes G, Olinsky A. Outcome of childhood asthma in mid-adult life. *BMJ* 1994;309:95-6. (9 July.)
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Epidemic of asthma possibly associated with electrical storms

EDITOR,—Intense nationwide interest has arisen in an epidemic of asthma that occurred overnight on 24 and 25 June in the south of England and coincided with electrical storms. Between 11 pm on 24 June and 7 am on 25 June our accident and emergency and acute admissions departments saw 39 asthmatic patients. Over a similar period last year the two units saw only one patient with asthma. The patients tended to be young adults, with a median age of 28. Thirteen patients had not previously been diagnosed as asthmatic, and 22 were regularly affected by hay fever.

Atmospheric pollution, which has previously been implicated in epidemics of asthma¹ and is the subject of much interest in the media, was high. Zero level ozone measured in centres adjacent to Cambridge and Peterborough was at a two week high (45 ppb, compared with a daily average of 28.7 ppb over the preceding and following weeks). Pollen counts were considerably increased in the 48 hours before the rainfall. It has been shown that an allergy to pollen causes an increase in non-specific bronchial reactivity.² The high proportion of patients with regular seasonal hay fever who were affected by the epidemic might support this.

There has been interest in the fungal spore *Didymella exitialis* and the yeast *Sporobolomyces* as aetiological factors in asthma.^{3,4} *Didymella* however, showed only a moderate rise in concentration, and *Sporobolomyces* rose only after most of the patients with asthma had attended, but a massive rise occurred in the concentration of ascospores of *Phaeosphaeria nigrans* and *Diatrypaeaceae*, which have not been previously associated with asthma and may merit further investigation.

It has been suggested that electrical storm activity, through the medium of positive ions, may cause bronchospasm,⁵ but measurement of lightning strikes from cloud to cloud and from cloud to ground by the Meteorological Office showed 37 strikes in the Cambridge catchment area and two in the Peterborough catchment area. The widely varying levels of electrical activity but equivalent attendances for asthma in our two areas do not support this suggestion.

The aetiology of this epidemic was probably multifactorial, and a study relating attendances because of asthma at hospitals and general practices to factors known to precipitate asthma is required to establish the most important causative factors, as Virginia Murray and colleagues have proposed.⁶ This would also allow for a more