from CF patients were also taken at routine visits. The study was approved by the local ethics review committee and all parents of the children gave written informed consent.

A mean (SD) number of 7.5 (2.7) (range 2–13) and 5.1 (1.8) (range 2–9) oropharyngeal cultures were taken from CF patients and healthy controls, respectively. During the study period six children with CF (30%) had at least one *P aeruginosa* positive culture compared with seven (37%) healthy controls. Cultures following a positive culture in healthy children were always negative for *P aeruginosa*, while in four of six (67%) CF children short term follow up cultures remained positive for *P aeruginosa* and anti-pseudomonal treatment was started.

This study showed that *P aeruginosa* acquisition frequently occurs in periods of ARI in both children with CF and healthy controls. While healthy individuals easily clear *P aeruginosa*, most CF patients remain positive and require anti-pseudomonal treatment. In the present study we sampled during periods of ARI, which are highly related to respiratory viruses in otherwise healthy children.[3] In line with former data in CF, these results suggest that respiratory viral infections facilitate *P aeruginosa* acquisition and colonisation.[4] The high prevalence of *P aeruginosa* in the airways of healthy children during ARI is in contrast with earlier findings which suggest that *P aeruginosa* colonisation rarely occurs in the airways of healthy individuals.[3] Our data could suggest that even healthy individuals with ARI are a potential source for *P aeruginosa* acquisition in CF patients. If confirmed, it could have major consequences for current segregation policies which simply avoid contacts between CF patients. It might imply limiting contacts between both CF and non-CF individuals in periods of ARI. Or should we conclude that prevention of *P aeruginosa* acquisition is practically unrealistic?

Our data urge for studies on the relationship between respiratory viral infections and bacteria in CF, and on the transmission of *P aeruginosa* between healthy individuals and CF patients. New insights might change current prevention rules and might open new approaches to effective prevention of *P aeruginosa* acquisition in patients with CF. Prophylactic treatment with anti-pseudomonal antibiotics in periods of ARI might be an interesting option.

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Association between sibship size and allergic diseases in the Glasgow Alumni Study

We read the interesting study by Kinra *et al*[1] which gives us important information on the relationship between sibship size, birth order, and allergic disease in British students born in the first half of the 20th century. There are, however, a few points which we would like to raise:

(1) The authors observed a stronger association between sibship size and allergy in the oldest cohort and interpreted this finding as supporting the hygiene hypothesis because of a postulated larger difference in hygiene between larger and smaller families in this cohort compared with younger cohorts. However, another possible explanation—not related to the hygiene hypothesis—is the change of determinants of family size. With modernisation and emancipation of women and the discovery of the biochemical rhythm in the female reproductive cycle[2] and the increasing popularity of condoms, all taking place in the first half of the 20th century, the determinants of family size may have shifted considerably during this period with probable consequences for the association between family size and allergy.

(2) Similarly, an interaction between socio-economic status (SES) and birth order was interpreted as—if not a chance finding—supporting the hygiene hypothesis. However, other explanations cannot be excluded if we assume a prenatal birth order effect: a stronger relationship between birth order and allergy in lower SES categories might be due to a possibly higher rate of spontaneous abortions in these groups,[3] leading to differential underestimation of birth order (or, rather, number of pregnancies). This scenario would also explain the fact that such an interaction was not observed for sibship size.

(3) In the comparison of the results with those of other studies, the authors point out that two "negative" studies[4 5] were due to lack of power. Firstly, it should be noted that these studies were not negative. In the study by Jarvis *et al*[4] a significant negative association (adjusted for birth order and relevant determinants) between allergy and sibship size was found, while in our study[5] the corresponding association with birth order was highly significant. Secondly, in our study the adjusted association with sibship size was indeed not significant (p value for trend 0.34), but the adjusted odds ratio (OR) for one extra sibling (allergy/no allergy) was *positive* (1.07) while its 95% confidence interval (95% CI) of 0.85 to 1.34 excluded any important negative trends (OR and 95% CI for trend not shown in paper).

(4) The contents of table 2 are not in agreement with the title: the results for asthma and combined allergic diseases are not shown.

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Dr Kinra was asked to comment, but no reply was received by the time this issue of *Thorax* went to press.

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CORRECTION

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In the paper entitled "COPD exacerbations – 4 · Prevention" by S Scott, P Walker and P M A Calverley which appeared in the May issue of *Thorax* (2006;**61**:440–7), the dose of tiotropium used in the studies by Casaburi and Brusasco referred to in table 1 on page 444 which currently reads "18 µg twice daily" should read "18 µg once daily". The publishers apologise for this error.