IL-5 in asthma

We have recently published two manuscripts on the effects of Interleukin (IL-5) administration to patients with mild asthma in the American Journal of Respiratory & Critical Care Medicine and in Thorax.1 We are addressing this letter to the Editors of both journals.

The data described in both papers have been obtained from one experiment performed in the same group of patients with mild asthma. While the paper in the American Journal of Respiratory & Critical Care Medicine reports primarily on the phenotypic changes in circulating blood eosinophils and CD34+ cells, the Thorax paper describes the changes in the airways with measurements of bronchial responsiveness and of eosinophil counts in sputum, together with etoxacin levels in the serum. Both manuscripts, however, detail the changes in blood eosinophil counts and serum IL-5 changes derived from these nine patients, which constitutes a partial duplication of the data described in the two papers. At the time of submission of the manuscripts to the two journals we did not inform the respective journals of the existence and submission of the other manuscript.

We would also like to take the opportunity of pointing out one mistake and two differences in the two manuscripts:

(1) Table 1 in Thorax contains mistakes regarding the FEV1 values; table 1 in the Am J Respir Crit Care Med paper shows the correct values.

(2) The Am J Respir Crit Care Med paper quotes the median values in the text while mean values are plotted in fig 2, and the Thorax paper the geometric mean.

(3) The blood eosinophil counts were analysed in the Am J Respir Crit Care Med paper as paired t tests while in the Thorax paper we used repeated measures analysis of variance (as recommended by a reviewer) which does not provide significant differences in eosinophil counts.

We recognise that it has been an error of judgement on our part in having the experimental data from this study reported in two separate manuscripts, and also in not informing you (and the reviewers) of the existence of these two separate manuscripts at the time of submission. We have not intentionally set out to duplicate publication of our experimental data and regret that this has happened. We also would like to apologise for the concern that this may cause to the high reputation of both journals.

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References

Bronchoconstrictor effect of deep inspiration in asthma

I was interested in the article by Burns and Gibson in the February issue of Thorax,2 but feel that the authors should exclude the following possible confounding factors which should be addressed before their hypothesis can be accepted.

(1) The time course of the bronchoconstrictor response to deep breaths in mild asthma is brief (usually 60–90 seconds), so the precise timing of the sGaw measurements after the manoeuvre is critical. Have the authors taken this into consideration?

(2) Although the pre-test and test breathing pattern was standardised as far as possible, have changes in Pco2 been excluded since hypocapnia can have a larger effect on sGaw even in normal subjects than that described here.3

(3) Why do their normal subjects show a fall from baseline sGaw after deep breaths rather than the consistent small rise seen in previous studies as quoted in the discussion? If these pressures were repeated, further investigation of their inherently speculative hypothesis might indeed be worthwhile.

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References
1 Burns GP, Gibson GJ. A novel hypothesis to explain the bronchoconstrictor effect of deep inspiration in asthma. Thorax 2002; 57:116-9.

Inhaled sodium cromoglycate in children with asthma

We admire the perseverance of Dr Edwards and colleagues to dispute the conclusions of our systematic review on sodium cromoglycate in asthmatic children.3 We note that they restrict their present comments to only a single point—interpretation of the tolerance interval. In fact, our conclusions were based not only on this finding but also on the apparent publication bias and the small overall treatment effect. Hence, we stick to our conclusion that there is insufficient evidence that maintenance treatment with sodium cromoglycate is beneficial in children with asthma.

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References
Body mass index and asthma

We read with interest the report by von Mutius et al on the association between body mass index (BMI) and asthma in children. The finding that the association between asthma and obesity may be mediated by mechanical factors or by an alternative inflammatory mechanism rather than cosinophilic inflammation is important and contributes to our understanding of the causal pathway.

With regard to breastfeeding, the authors state: “Breast feeding was no longer a significant predictor of asthma once BMI was introduced into the model. However, this may be because the intermediate variable (BMI) was introduced in the model.” The authors then surmise that BMI is an independent variable, and the results of their multivariate analyses—including breastfeeding—do not support the notion that “BMI might be an intermediate step”. However, if the former is correct, then lack of breastfeeding is the probable cause, and a reduction of BMI may not reduce asthma, as claimed by the authors. The conclusions drawn appear to be contradictory and also do not reflect the limitations of cross sectional analyses.

The question is whether: (1) lack of breastfeeding results in increased BMI and increased BMI is an intermediate step in the aetiology of asthma; or (2) the association between BMI and asthma is a spurious correlation and both are caused by lack of breastfeeding; or (3) both increased BMI and shortened breastfeeding are independent risk factors.

Acknowledging the restriction of cross sectional analyses, the authors need to clarify their conclusions and provide the respective models and path (or partial) coefficients that support one or other of the models suggested above.

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Reference
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