

Correspondence

Reply

To the Editor:

We thank Lipworth¹ for his comments. Our study included 46 individuals with asthma (30 [65%] of whom had eosinophilic asthma with sputum eosinophil counts of $\geq 2\%$) and 40 healthy control subjects. The study was not restricted to patients with only eosinophilic asthma. Importantly, this was not determined *a priori*. The objective of the study was to determine whether a panel of inflammatory biomarkers might accurately predict steroid responsiveness in asthma. As Lipworth indicates, we selected AMP over methacholine because responsiveness to AMP is a more sensitive marker of steroid effect.^{2,3} Lipworth asked what proportion of patients ended up with a “well-controlled” absolute Asthma Control Questionnaire (ACQ) score of less than 0.75 rather than a change of more than 0.5 in the ACQ score after treatment with inhaled corticosteroid. After inhaled corticosteroid treatment, 23 subjects (53.5%) were well controlled with an ACQ score of less than 0.75 while 20 (46.5%) had an ACQ score of more than 0.75. We agree that the chosen dose (ex-valve) of fluticasone propionate was high at 1000 $\mu\text{g}/\text{d}$ and in real life this would be an unlikely starting dose for such patients. However, although 1000 $\mu\text{g}/\text{d}$ is not the recommended starting dose, if we had used a lower dose in this study, the reasons for nonresponse would have been open to doubt. Was it due to too low a dose having been used? It was important to identify true nonresponders by using the highest recommended dose.

Finally, Lipworth is concerned that the high CIs for the odds ratios might negate the usefulness of these markers for predicting steroid responsiveness. We accept that the 95% CIs were wide for each of the individual biomarkers but suggest that this may be a reflection of the small size of the study population. The significance of the results, where reported, remains.

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