Correspondence

Reply

To the Editor:

We thank Huisstede et al[1] for their interest in our publication and their comments. Like the authors we agree wholeheartedly that weight reduction is key to improving asthma control and quality of life in obese patients, and that bariatric surgery is one treatment option to help achieve this. However, access to bariatric surgery varies between countries. In the United Kingdom, this option is available only for those patients who have a body mass index (BMI) range of 35 to 40 together with additional medical problems such as type 2 diabetes or high blood pressure proven to be improved by marked weight loss, or a BMI of more than 40. This does not include the obese patients with asthma who took part in our study (mean BMI of 28). We note that although Huisstede et al found no difference in the eosinophil count between obese patients with asthma and obese controls in their analysis,[2] their patients were a very different cohort (with a BMI of >45) compared with our group. Therefore, we believe it is difficult to draw comparisons between the 2 studies.

As Huisstede et al point out, we did not undertake a direct correlation between sputum eosinophil counts and the extent of lung eosinophil clearance; this analysis was not possible because the bronchial biopsies were taken from a separate cohort of patients from those who participated in the single photon emission computed tomography/computed tomography study. Our finding was unexpected, and hence bronchial biopsy was not incorporated into an already intense research protocol. We agree entirely that future single photon emission computed tomography imaging should also include healthy obese controls and indeed we plan to investigate this group at the next opportunity. Guided by our current findings, future studies will require both careful consideration of the control group and detailed analysis of the bronchial wall cellular profile. This approach may go some way to understand the causal relationship between asthma and obesity. Regardless of the initial driver of the tissue eosinophilia, the purpose of our study was to noninvasively track eosinophil uptake into the lungs. Given that obese asthma is a phenotype associated with a low eosinophil count in the sputum and blood but high submucosal eosinophil count,[3] this phenotype provided us with an ideal scenario to image lung eosinophils. Our results not only confirm previous findings that obese patients with asthma have increased lung eosinophils compared with nonobese patients with asthma but also suggest that obese patients with asthma may benefit from anti-eosinophil therapies.

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