

Correspondence

Reply

To the Editor:

We thank Fan and coauthors¹ for their comments, in which they focus on the measurement of IL-5 protein in different nasal tissues. They claim to find IL-5 protein in nearly all nasal tissues including normal controls, subjects with allergy, and patients with chronic rhinosinusitis without and with nasal polyps, and also describe significant differences between those groups. They confirm our data in showing that IL-5 protein is higher in patients with asthma versus those without in the nasal polyp group, as we described earlier,² and conclude that IL-5 would act as a predisposing factor. We have taken a more careful position in talking about an association rather than a predisposition, and we based this statement on cluster analysis rather than on a pure comparison of protein data. Fan and coauthors¹ furthermore claim that increased IL-5 protein concentrations would predispose to asthma also in chronic rhinosinusitis without nasal polyps (CRSsNP) patients (n = 5); however, in Fig 1, B, IL-5 concentrations in nasal polyps in the absence of asthma are clearly higher than those in CRSsNP with asthma, causing doubt about this claim.

Apart from providing data on IL-5, we also gave data on eosinophil cationic protein and eotaxin concentrations, on specific IgE and total IgE, and on GATA-3 mRNA expression, which all support the T_H2 bias observed in specific subpopulations of nasal polyps,^{3,4} but not in all polyps and certainly not in CRSsNP. We also provided data on representative cytokine patterns for IL-5-negative polyp tissues, clearly confirming the bias to T_H1-cell or T_H17-cell predominance and neutrophilic inflammation. Thus, we are sure that we correctly phenotype different patterns of inflammation, and that there are plenty of patients with nasal polyps—and certainly patients with chronic sinusitis and controls—who do not demonstrate a T_H2 bias in the nasal mucosal tissue.

The discrepancies described between our findings and those by Fan et al¹ may in fact be a result of regional differences, which opens an exciting new field of research. We have observed significant differences in the current Global Allergy and Asthma European Network sinusitis cohort study,⁵ sampling patients with chronic rhinosinusitis with and without nasal polyps from all over Europe (Bachert, unpublished data, January 2011). IL-5 was detected in about a third of the CRSsNP patients versus 3% in control patients, in low concentrations, but in about 95% of the chronic rhinosinusitis with nasal polyps patients, with significantly higher concentrations. However, we also observed regional differences in the number of IL-5-positive CRSsNP samples, ranging from 7% to 50%. Similarly, we have observed

that the percentage of T_H2-biased polyps is significantly higher in Guandong (near Hong Kong, industrialized and subject to globalization) versus Sichuan province (mainland China, less globalization) and that this difference in inflammatory patterns may be linked to differences in the bacterial colonization of the airways (Luo et al, unpublished data, 2011). We assume that the 15 patients with polyps whom Fan et al¹ report on here were from the Guangdong province. Thus, the distribution of specific and distinguishable chronic rhinosinusitis phenotypes (endotypes) is likely to be an important issue for a better understanding of upper airway disease, which finally may have great impact on treatment options. Appropriate multicenter studies with adequate numbers of participants will be necessary to answer this question.

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