

Correspondence

Remodeling and inflammation in Chinese versus white patients with chronic rhinosinusitis

To the Editor:

The authors of the article entitled “Distinct immunopathologic characteristics of various types of chronic rhinosinusitis in adult Chinese”¹ claim to confirm our data² on the deficit of forkhead box protein 3 (or T-regulatory cells) and a decreased production of TGF- β 1 in Chinese patients with chronic rhinosinusitis with nasal polyps versus controls; they also claim that, in contrast with our findings in white patients,^{3,4} mucosal samples of patients with chronic rhinosinusitis without nasal polyps (CRSsNP) in China would be characterized by a decrease in T-regulatory cells and TGF- β expression.

However, their data do not allow such conclusions, and we do have solid evidence from work in China that their conclusions concerning CRSsNP are incorrect and misleading.

Whereas we provided evidence for an upregulation of TGF- β 1 on the mRNA and protein levels, of TGF- β 2 protein concentrations, of TGF- β receptors RI and RIII mRNA expression, and of corresponding downstream events such as an increase in the number of TGF- β activated cells and total collagen amount in CRSsNP versus controls,^{3,4} the conclusions in the article by Cao et al¹ are based on TGF- β 1 mRNA data only. Moreover, those data are presented as “proportions of patients having positive mRNA expression” and do not allow any quantitative conclusions, which needs explanation. We also wonder why Cao et al¹ could not detect TGF- β 1 mRNA in approximately 50% to 60% of their samples, and how these findings fit with their own “pathological classification,” (P480) showing a low edema score for CRSsNP.

In the same way, the data for forkhead box protein 3 expression in the article by Cao et al¹ are given as “proportions of patients having positive mRNA expression,” whereas we provided quantitative mRNA and protein data (immunohistochemistry) for white patients with CRSsNP.³

Further concerns include the definition of “eosinophilic polyps” based on number of cells, because we have shown that eosinophils may be present but not activated²; the fact that the patients in the immunologic study are not identical with those from the histologic study; and the statistical evaluation of 5 groups in parallel (Fig 2) without necessary correction (eg, Bonferroni).

In contrast with their findings, we are able to show that the remodelling pattern in Chinese patients of Chengdu, a city in

West China, does match our findings in white patients with CRSsNP, with a significant upregulation of TGF- β proteins and of downstream events such as the total collagen content versus controls. Furthermore, mucosal forkhead box protein 3 mRNA in Chinese subjects with CRSsNP is quantitatively measurable and also significantly increased versus controls (Li et al, Aug 2009).

In summary, in clear contrast with the unjustified conclusions of the article by Cao et al,¹ mucosal remodelling patterns and forkhead box protein 3 expression in patients with CRSsNP and chronic rhinosinusitis with nasal polyps in China do resemble those in white patients, whereas the T-effector cell patterns and the types of inflammation do differ between Chinese and white subjects with chronic rhinosinusitis with nasal polyps, as published earlier.² These findings are extremely helpful in clarifying pathomechanisms and phenotypes of upper airway inflammation and may affect therapeutic approaches to these diseases.

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