

## Correspondence

**Are rhinoviral proteinases responsible for mixed T<sub>H</sub>1 and T<sub>H</sub>2 immunity in chronic obstructive pulmonary disease?**

To the Editor:

We read with interest the article by Dr Singh et al<sup>1</sup> in the June issue of the Journal. Rhinoviruses are thought to play an important role in acute exacerbations of chronic obstructive pulmonary disease (COPD),<sup>2</sup> and in this study, the authors have highlighted the role of immunity to rhinovirus proteinases, particularly proteinase 2A.

The authors have presented data showing that rhinovirus proteinase 2A induces dendritic cell maturation and activation (Fig 3) and T-cell cytokine production (Fig 4). However, it is not clear whether these *in vitro* cellular immune responses differ between patients with COPD and healthy control subjects. Fig 3 contains data from a mixture of patients and control subjects, whereas no information is provided on the source of the cells used for the experiments shown in Fig 4. To reach a full conclusion, it is important to know whether patients with COPD and healthy control subjects have fundamentally different immune responses to rhinovirus proteinase 2A, and whether this might contribute to the immune dysregulation in acute exacerbations of COPD.

Moreover, given the conclusions reached by the authors, it would have been particularly interesting if the investigators had

provided information on whether the particular viruses detected during acute exacerbations of COPD were correlated with the differing cytokine profiles detected in bronchoalveolar lavage or produced *ex vivo* by peripheral blood T cells (Figs 1 and 2, respectively). If rhinovirus proteinase 2A is indeed skewing immunity as the authors suggest, one might expect that the data shown in Figs 1 and 2 would vary depending on whether rhinoviruses or other viruses were detectable during the acute exacerbation. There is therefore an alternative possibility: that any acute exacerbation of COPD, regardless of the microbial trigger, will exhibit a mixed T<sub>H</sub>1 and T<sub>H</sub>2 immune response that is distinct from that seen in stable COPD or healthy subjects.

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Disclosure of potential conflict of interest: J. W. Upham has received research support from the National Health and Medical Research Council (Australia). A. Pritchard has declared that she has no conflict of interest.

## REFERENCES

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doi:10.1016/j.jaci.2010.07.009