

## CORRESPONDENCE

### Bronchial hyperresponsiveness and airway inflammatory markers in nonasthmatics with allergic rhinitis

To the Editor:

We thank Dr. Hargreave and colleagues for raising comments on our recent study on nonasthmatic subjects with allergic rhinitis [1].

Although bronchial hyperresponsiveness in the asthmatic range may help to identify patients with rhinitis who are at risk from asthma [2, 3], we disagree with their suggestion that our patients might have had asthma simply because they were hyperresponsive to inhaled methacholine and adenosine monophosphate (AMP). Firstly, it is well known that bronchial hyperresponsiveness is present in nonasthmatic patients with allergic rhinitis [4–6] and this may be a reflection of subclinical inflammatory changes of the lower airways [7–9]. Secondly, bronchial hyperresponsiveness *per se* is not always associated with asthma, and in fact it is also occasionally present in normal individuals [10]. Thirdly, the possibility of unrecognized asthma was excluded by further reviewing their case histories and subjects were eligible for inclusion in the study if at least two specialists in allergic diseases agreed they did not have any clinical history or symptoms suggestive of asthma. Finally, it must be noted that our rhinitic subjects were studied during the pollen season and were specifically selected from a larger pool of patients on the basis of their positive response to inhaled methacholine and AMP thus providing an explanation for their low level of nonspecific bronchial hyperresponsiveness.

We can not rule out the possibility that sputum eosinophilia was the result of contamination with post nasal drip. Pulmonary aspiration of a radiolabelled marker released into the nose was detected in a substantial number of individuals during sleep [11]. However, Bardin et al. [12] were unable to document aspiration of radionuclide in a study involving 13 patients with chronic rhinosinusitis and asthma. In view of these findings we agree that the possibility of sputum contamination with post nasal drainage must be seriously considered and investigated in controlled studies.

Our subjects with sputum eosinophilia had a history of seasonal rhinorrhoea, nasal itch, sneezing, and nasal obstruction in absence of chronic cough. Dr. Hargreave and colleagues would agree that their clinical history is more in line with a diagnosis of allergic rhinitis than eosinophilic bronchitis. In addition, sputum eosinophilia is not invariably present in nonasthmatic patients with allergic rhinitis. We have recently completed a 2-yr longitudinal study of nonasthmatic patients with seasonal allergic rhinitis to evaluate the efficacy/effect of specific immunotherapy. In the present study we were able to confirm substantial sputum eosinophilia during

the pollen season; however, when studied out of season the eosinophil count in their sputum resulted near normal (unpublished data).

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#### References

1. Polosa R, Ciamarra I, Mangano G, *et al.* Airway hyperresponsiveness in allergic rhinitis: a risk factor for asthma. *Eur Respir J* 2000; 15: 30–35.
2. Townley RG, Ryo UY, Kolotkin B, Kang B. Bronchial sensitivity to methacholine in current and former asthmatics and allergic rhinitis and control subjects. *J Allergy Clin Immunol* 1975; 56: 429–442.
3. Braman SS, Barrows AA, DeCotiis BA, Settipane GA, Corrao WM. Airway hyperresponsiveness in allergic rhinitis: a risk factor for asthma. *Chest* 1987; 91: 671–674.
4. Stevens WJ, Vermeire PA. Bronchial responsiveness to histamine and allergen in patients with asthma, rhinitis, cough. *Eur J Respir Dis* 1980; 61: 203–212.
5. Cockcroft DW, Killian DN, Mellon JJA, Hargreave FE. Bronchial reactivity to inhaled histamine: a method and clinical survey. *Clin Allergy* 1977; 7: 235–243.
6. Crimi N, Palermo F, Oliveri R, *et al.* Influence of asthmatic and rhinitic symptomatology and duration on bronchial responsiveness to histamine. *Int J Tissue React* 1987; IX: 515–520.
7. Bradley BL, Azzawi M, Jacobson M, *et al.* Eosinophils, T-lymphocytes, mast cells, neutrophils and macrophages in bronchial biopsy specimens from atopic subjects with asthma: comparisons with biopsy specimens from atopic subjects without asthma and normal control subjects and relationship to bronchial hyperresponsiveness. *J Allergy Clin Immunol* 1991; 88: 661–674.
8. Djukanovic R, Lai CKW, Wilson JW, *et al.* Bronchial mucosal manifestation of atopy: a comparison of markers of inflammation between atopic asthmatics, atopic non-asthmatics and healthy controls. *Eur Respir J* 1992; 5: 538–544.
9. Foresi A, Leone C, Pelucchi A, *et al.* Eosinophils, mast cells, and basophils in induced sputum from patients with seasonal allergic rhinitis and perennial asthma: relationship to methacholine responsiveness. *J Allergy Clin Immunol* 1997; 100: 58–64.
10. Power C, Sreenan S, Hurson B, Burke C, Poulter LW. Distribution of immunocompetent cells in the bronchial wall of clinically healthy subjects showing bronchial hyperresponsiveness. *Thorax* 1993; 48: 1125–1129.
11. Huxley EJ, Viroslav J, Gray WR, Pierce AK. Pharyngeal aspiration in normal adults and patients with depressed consciousness. *Am J Med* 1978; 64: 564–568.
12. Bardin PG, van Heerden BB, Joubert JR. Absence of pulmonary aspiration of sinus contents in patients with asthma and sinusitis. *J Allergy Clin Immunol* 1990; 86: 82–88.