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Distinct clinical phenotypes of airways disease: a primary-care clinician perspective

To the Editors:

As a clinician involved in the diagnosis, treatment and aetiological research of obstructive airways disease in primary care practice-based networks, I read with interest the description by WEATHERALL *et al.* [1] of four or five distinct clinical types of airways disease. Their results appear to confirm and extend observations from an earlier population-based study that distinguished asthma and chronic airway obstruction, which was termed AS-CAO (analogous to their Cluster 1), from smoking-associated emphysematous chronic obstructive pulmonary disease (COPD) (Cluster 2) [2].

I would like to offer a personal perspective on the potential value to airway disease diagnosis, treatment and research, of this new taxonomy in the primary-care setting. In my experience AS-CAO and smoking-associated COPD represent two distinct clinical patterns of airways disease that can be easily recognised by an experienced clinician armed only with spirometry and home peak flow monitoring. The distinction is potentially important since AS-CAO and smoking-associated COPD may have different aetiologies and response to treatment [3]. Distinguishing Cluster 3 (classic atopic, eosinophilic asthma) from Cluster 4 (mild, nondescript disease) required further laboratory testing (serum immunoglobulin E and exhaled nitric oxide) not currently routinely used in primary care. Pending the results of further research, the importance of making this distinction in the primary-care setting is unclear to me. An important topic for future investigation is to describe the natural history of Cluster 4. Can WEATHERALL *et al.* [1] comment on the possibility that Cluster 4 subjects had had episodes of self-limited ''acute asthmatic bronchitis'' [4] that did not evolve into more severe forms of airways disease?

I agree with WEATHERALL *et al.* [1] that AS-CAO (Cluster 1) patients are a very ill group yet are excluded from both asthma and COPD studies. An ongoing clinical trial [5] was rejected for funding by the National Institute of Health because it aimed to enrol all eligible primary-care patients with reversible airways disease regardless of smoking status or other lung comorbidities (especially COPD). Until this dogma is overcome it is unlikely that progress will be made towards conducting long-term ''real world'' effectiveness trials in reversible airways obstructive disease(s) [6]. For these reasons I support adoption of this or a similar new taxonomy that: 1) is more congruent with the realities of airways disease than the current taxonomy; and 2) will enable successful implementation of effectiveness trials.

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From the authors:

We thank D. Hahn for his interest in our paper [1]. We agree that following the natural history of individuals defined by these clusters may help define whether they represent distinct phenotypes, and we plan to do this in a follow-up cohort study of the Wellington Respiratory Survey. We agree that the fourth cluster identified by the ''Diana'' method may represent a phenotype of airway inflammation characterised by sputum production without marked elevation of exhaled nitric oxide and without marked airflow variability, as has been previously described [2, 3]. However, we are cautious in extrapolating the results of the cluster analysis too far. The analysis was of a