

EDITORIAL

The case for confirming occupational asthma: why, how much, how far? (revisited)

J.-L. Malo

In a prior editorial, the point that occupational asthma (OA) should be confirmed by objective methods, which are justified from the medical and sociopsychological points of view was made [1]. To date, a combination of various functional and immunological methods, used in a stepwise fashion, has been proposed [2]. However, none of these tools addresses the real physiopathological mechanism of OA, which induces eosinophilic airway inflammation. Bronchoalveolar lavage and bronchial biopsies can only be considered as research tools, since they cannot be used for routine purposes.

Two articles published in the current issue of the Journal address this problem, the first in a situation of "normal" work exposure [3] and the second, following inhalation challenges in the laboratory [4]. In 16 patients investigated for possible OA, LEMIERE *et al.* [3] found a significant difference in sputum eosinophils and eosinophilic cationic protein (ECP) in the 10 subjects with confirmed OA by comparing periods at work and periods away from work. In all these subjects forced expiratory volume in one second (FEV₁) and provocative concentration causing a 20% fall in FEV₁ (PC₂₀) were also reduced while at work. OBATA *et al.* [4] examined sputum eosinophils and exhaled nitric oxide in 17 subjects who underwent challenges with plicatic acid. The nine responders had a significant increase in sputum eosinophils at both 6 and 24 h postchallenge. However, three nonresponders also had an increase in sputum eosinophils, and both responders and nonresponders, without distinction, had an increase in exhaled NO.

HARGREAVE *et al.* [5] recently proposed a comprehensive review of the history of the examination of induced sputum in the assessment of airway inflammation. Eosinophils were first identified in the sputum of asthmatic subjects more than 100 yrs ago and proposed in the assessment of the effect of treatment in asthmatic subjects 40 yrs ago [6]. Although the assessment of sputum cells was later proposed [7], standardized and reliable methods for inducing and examining sputum have only been made available in the past few years [8, 9]. It is possible to obtain sputum by inhalation of hypertonic saline in 60-85% of subjects who cannot produce sputum spontaneously [5].

Although the place of measurements of indices of airway inflammation in sputum in clinical practice has not been established [5], it may well be of interest in the investigation of OA. Assessment of responsiveness to histamine and methacholine, which provides indirect evidence

of airway inflammation, has been shown to be a sensitive tool for the detection of OA before changes in FEV₁ are apparent [10-12]. However, linking peak expiratory flow (PEF) measurement with airway responsiveness does not augment the validity of PEF measurement alone in the investigation of OA [13, 14]. The two articles published in the current issue of the Journal, which can be considered pilot studies, appear to indicate that induced sputum can be considered an interesting tool to: 1) investigate OA if the subject is still at work; and 2) support evidence related to the occurrence of a late asthmatic reaction following specific inhalation challenges in the laboratory. An extra useful application may be the prediction of a subsequent asthmatic reaction on the examination of sputum on the day of an apparently negative testing as indicated by functional indices (C. Lemièrè personal communication). It is interesting to realize in this context that the three nonresponders in the study by OBATA *et al.* [4] had an increase in sputum eosinophils. It would have been interesting to know whether more intense exposure to plicatic acid would have turned these nonresponders into responders. The validity of this test in the investigation of OA remains to be assessed in studies with a larger number of participants.

References

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