

Correspondence

Normal nonspecific bronchial reactivity excludes occupational asthma?

To the Editor:

We read with interest the recent study by Pralong et al¹ examining the predictive value of methacholine challenge for exposed and unexposed workers in the diagnosis of occupational asthma. The authors conclude that normal methacholine challenge excludes occupational asthma in those currently exposed to a causative agent because of its high sensitivity and negative predictive value, 98.1% and 97.7% respectively; when patients who are currently unexposed are taken into account, overall sensitivity and negative predictive value are reduced (67% and 82%, respectively). These results depend critically on the criteria for a confirmed diagnosis of occupational asthma, the definition of nonspecific bronchial reactivity (NSBR), and any preselection of patients for specific inhalation challenge testing.

Canadian Thoracic Society guidelines for occupational asthma² have stated that for practical purposes the absence of NSBR excludes the diagnosis of occupational asthma in a symptomatic exposed worker; this is likely to have significantly biased referrals for specific inhalation challenges toward those with significant NSBR. We find many workers with occupational asthma to have normal NSBR while exposed, and after specific inhalation challenge, perhaps because we perform challenges to a much wider range of agents that appear to fulfill the criteria for occupational asthma with sensitization (latent interval, positive specific inhalation challenge to nonirritating levels of exposure, and improvement away from exposure). This incorporates agents such as diesel exhaust and aluminum foundry fumes, which some believe are acting primarily as irritants, but in whom the physiological responses to workplace exposures, including the presence of late asthmatic reactions, appears inseparable from those exposed to generally accepted sensitizing agents.³ In the current study, the molecular weight of the agent causing occupational asthma, either low or high, was not a predictor of NSBR. We would argue that this is unexpected: in a series of 38 patients

from our occupational lung disease unit in Birmingham, United Kingdom,⁴ occupational asthma due to low molecular weight agents was predominantly associated with noneosinophilic inflammation, with only 14 of 38 (37%) cases demonstrating sputum eosinophilia (>2.2%) while exposed at work; moreover, noneosinophilic phenotypes showed significantly less NSBR than did eosinophilic phenotypes. This observation is supported by the meta-analysis of the value of NSBR in occupational asthma,⁵ which showed that pooled estimates of sensitivity are lower for low molecular weight agents than for high molecular weight agents. We believe that NSBR should not be used to exclude patients with work-related asthma symptoms from referral to specialized units and further investigation. Many of these patients have poor health outcomes if left exposed at work.

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