

Respiratory medicine

# Occupation and COPD

D C Christiani

## Time for action

Chronic obstructive pulmonary disease (COPD) is now the fourth leading cause of death worldwide and, by all reasonable projections, its incidence will increase. The worldwide burden of COPD morbidity and mortality is already large, and will become staggering in this century. In this issue of the journal, Meldrum *et al* describe the impact of COPD on the UK.<sup>1</sup> In their poignant review, they point out that while the main cause of COPD in the UK remains cigarette smoking, there is a substantial body of epidemiological evidence linking occupational exposures to dusts, gases/vapours, and fumes to chronic airflow obstruction, with a substantial population attributable risk (15–20%) in non-smokers. There is also evidence for at least additive effects among smokers for a variety of workplace exposures. Epidemiological and pathological studies have linked exposure to coal mine dust, silica, cadmium, and asbestos.<sup>2</sup> In addition, longitudinal studies show that workers with chronic exposure to cotton and grain dusts develop chronic airflow obstruction.<sup>3–5</sup> A study from the USA, using the National Health and Nutrition Examination Survey (NHANES III) identified a number of occupations with increased odds ratios for COPD. The fraction of COPD attributable to work was estimated at 19.2% overall, and 31.1%

among never-smokers.<sup>6</sup> In June 2002, the American Thoracic Society issued an official statement on the occupational contribution to the burden of airway disease.<sup>7</sup> The ATS committee concluded that about 15% of both asthma and COPD is likely to be work related based on international population based studies. A conservative estimate of the annual costs of the occupational components of asthma and COPD is 7 billion dollars.

The scenario in the industrialising world is even more daunting, as the size of the at-risk population is growing steadily. In some areas of the world, COPD ranks at or near the top of the leading causes of death, and this shift in mortality is not explained by smoking patterns alone. The role of occupational and environmental exposures in COPD morbidity and mortality can be expected to increase in these societies.

What is to be done to prevent COPD? Firstly, regulators need to take action to reduce workplace exposures through engineering and other hygiene measures. Secondly, health practitioners, workers, and employers need to be made aware of the hazards posed by airborne exposures at work. The term “nuisance dust” should be eliminated in both the scientific and regulatory spheres. Thirdly, clinicians must consider potential occupational causes for

obstructive airway disease, as early identification offers the opportunity for preventing disability and mortality, and for epidemiological case reporting. Fourthly, efforts to reduce tobacco smoking should be accompanied (where appropriate) by initiatives to reduce or eliminate occupational exposures. Lastly, epidemiological monitoring should include occupational data and dissemination of knowledge to industrialising countries.

Meldrum and colleagues have sounded a call to action for the UK. We should heed the message globally.

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Correspondence to: Prof. D C Christiani, Harvard School of Public Health, Harvard Medical School, Boston, MA 02115, USA; dchristi@hsph.harvard.edu

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