Smoke and dust get in your eyes: what does it mean in the workplace?

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Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality in the world. UK statistics show that it is the third biggest cause of respiratory deaths, accounting for 23% of all respiratory deaths.[1] In the USA COPD is the fourth leading cause of chronic morbidity and mortality.[2] A study by the World Bank/World Health Organization predicted COPD to be the fifth leading cause of worldwide burden of disease in 2020.[3] The role of tobacco smoking in the decline in lung function was shown in the classic study by Fletcher and Peto[4] in the 1970s, and the US Surgeon General's Report on smoking and COPD in 1984 established smoking as a causal factor for COPD.[5] Epidemiological studies also reported a link between occupational exposures and chronic bronchitis as early as the 1940s and 1950s,[6 7] but the role of occupation received less attention until Becklake[8] highlighted the evidence for a causal association between occupational exposures and COPD in a review article in 1989.

Clinicians frequently encounter patients who themselves relate their respiratory symptoms to dusts or fumes in the workplace. Although almost two decades have passed since Becklake concluded that dust exposure at work is a causal factor for COPD,[8] clinicians have still been left with considerable uncertainty concerning what advice to give to their patients, apart from the very general opinion that it is always good to reduce dust levels.

The early studies addressed the relationship between specific occupational exposures and COPD in workforce-based studies.[8 9] To reduce potential healthy worker bias (ie, selection bias due to the possibility that people entering dusty and fumy jobs or remaining in them may be healthier than the general population) in cross-sectional studies of specific workforces, epidemiological studies started to apply a population-based approach. These large studies asked rather simple questions on whether the person had exposure at work to dusts, alone or in combination with fumes or gases. They consistently found that such exposure was a risk for COPD.[8 10–14] However, the scientific community called for more precise exposure assessment, which resulted in a series of studies looking at the risk of COPD according to job title, a battery of specific exposures or applying a job exposure matrix (JEM) approach. The JEM is composed to represent the usual exposure to relevant causal agents in different jobs based on existing knowledge on occurrence and levels of exposures in each job. It can then be applied to assess a worker's exposure when only his/her job title is known. The results confirmed the impact of occupational exposures on an increased risk of COPD and a faster decline in forced expiratory volume in 1 s.[7 15–19] Exposure to silica, coal dust, other mineral dust, cadmium, solvents and second hand smoke and working in construction, foundries, transportation and trucking, agriculture, rubber, plastics and leather manufacturing, textile industry, paper industry, wood industry, professional cleaning and food processing have been implicated as specific exposures and jobs of relevance. An assessment by the American Thoracic Society from 2003 estimated that 15–20% of COPD is attributable to occupational exposures.[20]

In this issue of *Thorax* Blanc and coworkers[21] report a case-control study of 1202 cases with clinically diagnosed COPD and 302 controls recruited from a large non-profit managed care organisation in northern California, USA (***see page 6***). The strength of this study is that their definition of COPD was based on a physician diagnosis of COPD, spirometric evidence of airway obstruction and use of medication for COPD. Thus, the study population was chosen to have clinical relevance. Self-reported exposure to vapours, gases, dusts or fumes and exposure based on JEM were assessed. The risk of COPD in relation to both self-reported exposure and to exposure based on JEM was approximately twofold compared with that in subjects with no exposure or a low likelihood of such occupational exposure (OR 2.11, 95% CI 1.59 to 2.82 and OR 2.27, 95% 1.46 to 3.52, respectively). This means an excess risk of COPD of about 100% in those with these occupational exposures. The population attributable fraction (ie, the fraction of COPD in the population that can be attributed to occupational exposures and potentially prevented by reducing such exposures) was 31% when estimated based on self-reported exposure and 13% when based on JEM. The prevalence of relevant occupational exposures was smaller based on JEM assessment. Thus, one in three cases of COPD may be attributable to occupational exposures. This compares with 35% of COPD being attributable to current smoking in the same population.

The study by Blanc and colleagues[21] also tackled the important but difficult question concerning the joint effect of occupational exposure and smoking on COPD. This question has been in the air for a long time as smoking used to be almost universal in workforces exposed to dusts and fumes. The study found a significant interaction between ever smoking (current or past) and occupational exposure to vapours, gases, dusts or fumes, showing a synergistic effect. Thus, these two exposures seemed to intensify the adverse effects of each other, so that having both of them simultaneously led to a risk that was higher than multiplying the independent effects of each exposure. The joint exposure was related to a strongly increased OR of 14.1 (95% CI 9.33 to 21.2).

The limitations of the study are the relatively low participation rate (51% in the interview) and potential exposure and outcome misclassifications. Although some selection seemed to have occurred in participation in the study as the study subjects were slightly older and had a higher proportion of women and white individuals than those not interviewed, the fact that the study was nested within a cohort focusing on disability due to COPD makes selection according to specific exposures less likely. This means that the selection was probably not biased in relation to occupational exposures or smoking. Exposure assessment based on questionnaire reporting and categorisation from job titles is inevitably influenced by some misclassification, but this is likely to be random in nature, especially in the JEM-based approach. The study

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asked about the job title and exposures in the respondent's longest held job only, which adds to potential misclassification as some participants may have experienced significant exposures in other jobs but may be classified as unexposed. Both types of exposure misclassification would lead to underestimating the true effects of occupational exposures to dusts, vapours and fumes on COPD. The benefit of asking about exposure in the longest held job is that those who have already left the industry with exposures—perhaps because of respiratory symptoms—are correctly classified as having had relevant exposure, thus diminishing the healthy worker bias. Another potential limitation of the study is misclassification of the outcome, as some subjects with asthma who smoke may be labelled as having COPD, although recent studies have shown that smoking also increases the risk of asthma.[22] If a considerable proportion of COPD cases actually had asthma related to occupational exposures, this could lead to overestimation of risk. However, as the JEM classification was modified to be COPD-specific, this is not likely to be a major bias in the study.

The findings by Blanc and colleagues underline the fact that both occupational exposures and smoking should be addressed in the primary prevention of COPD at the population level. This means measures to reduce levels of dusts, vapours, gases and fumes in workplaces and enhancement of tobacco control measures directed at populations of working age. Workers with such occupational exposures should get information and education about adverse effects related to their workplace exposures and on the intensifying effect of smoking. The same strategies could be applied in secondary prevention when advising and treating individual patients with chronic bronchitis or more advanced COPD. Longitudinal and intervention studies using these strategies in clinical settings should be conducted in the future to provide information on what methods are most effective in practice and how these approaches influence the prognosis of COPD.

A question remaining open is whether this study,[21] along with other recent studies on occupational exposures and smoking,[16 18] should influence our practice of diagnosing occupational COPD. This needs open-minded discussion that should perhaps also touch on such sensitive issues as compensation for disability from occupational COPD in smokers. It seems clear that being a smoker can no more mean that the individual does not have occupational COPD, as smokers appear to be at an even higher risk of developing work-related COPD than non-smokers.

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Beyond airflow limitation: another look at COPD

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More than 40 years ago Benjamin Burrows and his colleagues[1] described the distinctive clinical, functional, radiological and pathological characteristics of the chronic obstructive pulmonary disease (COPD) phenotypes that they called *emphysematous* and *bronchial* types of chronic airways obstruction. They identified a subgroup of patients who were *"thin"* and had evidence of emphysema on chest *x* ray, while another subgroup was found to be of *"stocky build"* and had chest *x* ray changes suggestive of previous pulmonary inflammatory disease. Postmortem anatomical emphysema severity was positively