CORRESPONDENCE

Mortality of Dutch coal miners in relation to pneumoconiosis, chronic obstructive pulmonary disease, and lung function

EDITOR,—Meijers *et al* presented a very interesting study on mortality of Dutch coal miners.[1] Most of the coal miners, 3367 out of 3790 (89%), had radiological manifestations of coal workers' pneumoconiosis (CWP). All the coal miners had an increased mortality due to ischaemic heart disease (IHD) in all four periods of follow up when compared with the total male population in the Netherlands. The increase varied between 10% and 25%. It was unlikely that smoking habits were responsible for this increase as the SMR for lung cancer was 1.02.

Fibrinogen is an established risk factor for IHD[2 3] and it has been found that coal miners with pneumoconiosis have higher plasma concentrations of fibrinogen than coal miners without pneumoconiosis.[4] Thus it seems that these findings on coalminers with pneumoconiosis could also be included in the hypothesis linking exposure to dust with increased concentrations of plasma fibrinogen and IHD.

The hypothesis of exposure through inhaled particles and the occurrence of IHD can be expressed in the following way. Long term inhalation of particles retained in the lungs will create a low grade inflammation associated with an increase in plasma fibrinogen. The high concentrations of fibrinogen will increase the likelihood of blood clotting and thereby the risk for myocardial infarction and IHD.[5 6]

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1 Meijers JMM, Swaen GMH, Slangen JJM. Mortality of Dutch coal miners in relation to pneumoconiosis, chronic obstructive pulmonary disease, and lung function. *Occup Environ Med* 1997;**54**:708–13.
2 Meade TW, Mellows S, Brozovic M, *et al*. Haemostatic function and ischaemic heart disease: principle results of the Northwick Park heart study. *Lancet* 1986;ii:533–7.
3 Yarnell JWG, Baker IA, Sweetnam PM, *et al*. Fibrinogen, viscosity, and white blood cell count are major risk factors for ischemic heart disease. *Circulation* 1991;**83**:836–44.
4 Rego GF, Achaerandio GO, Cuervo VG, *et al*. Presence of acute phase response in coal workers' pneumoconiosis. *Br J Ind Med* 1991;**48**:193–5.
5 Seaton A, MacNee W, Donaldson K, *et al*. Particulate air pollution and acute health effects. *Lancet* 1995;**345**:176–8.
6 Sjogren B. Occupational exposure to dust: inflammation and ischaemic heart disease. *Occup Environ Med* 1997;**54**:466–9.

Bronchial reactions to exposure to welding fumes

EDITOR,—We read with great interest the recent article by Contreras *et al*[1] describing non-specific bronchial hyperresponsiveness and differential pulmonary responses to welding fumes of differing constituents in a small group of current welders with respiratory symptoms suggestive of asthma. The authors concluded that in this group of symptomatic people, the reactions found were likely to represent irritant responses as there were no late asthmatic reactions, and no evidence of sensitisation.

This raises the issue of how we generalise the findings of this study to workplaces and workers with substantial exposure to welding fume.

Although Contreras *et al* noted these interesting findings in a group of symptomatic workers, no mention was made of whether asthma or wheeze had been diagnosed and whether the respiratory symptoms of cough, chest tightness, and dyspnoea were only work related, or whether these symptoms predated employment as a welder. Similarly, no asymptomatic welders were included for comparison and one person had certainly had a notable, presumably irritant, exposure in the past that would be highly likely to influence bronchial responsiveness and response to welding fume.

This interesting study now poses two further questions; what is the longer term importance of this acute response (if any), and what is the importance of acute pulmonary responses in welders with no respiratory symptoms?

We have recently studied, in the workplace, a large group of welders in New Zealand, and a similar group of non-exposed workers, welding mild steel predominantly with MIG and TIG techniques.[2] We noted that all welders as a group sustained a mean fall in FEV_1 of about 4% at 15 minutes after welding started, although within the welding group, certain individual welders sustained much larger falls (>15% in some cases). Interestingly, not all these were accompanied by current or work related respiratory symptoms. Also, we were able to show that the falls in FEV_1 were negated by the proper use of local extraction ventilation. Although we agree that the fall in FEV_1 found may represent a simple irritant response, we think that the presence of this FEV_1 response may predict longer term sequelae, and in particular may lead to the development of an asthma like state, bronchial hyperresponsivenss, and accelerated loss of lung volume. This is now the thrust of a follow up study on our original group of welders.

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1 Contrera GR, Chan-Yeung M. Bronchial reactions to exposure to welding fumes. *Occup Environ Med* 1997;**54**:836–9.
2 Fishwick D, Bradshaw LM, Slater T, *et al*. Respiratory symptoms, across shift lung function changes and lifetime exposures in New Zealand welders. *Scand J Work Environ Health* 1997;**23**:351–8.

Authors' reply—Fishwick *et al* brought up important points in their letter. Because of limited space, we were unable to put in as much clinical information as we would like. The welders studied did not have any symptoms before they started their trade as a welder. All of them were sent to us for evaluation of work relatedness of their symptoms. Unfortunately, we were unable to study a group of asymptomatic welders as controls.

The more important point brought up by Fishwick *et al* concerns the importance of this acute irritant response and whether it predicts longer term sequelae. It has been documented among workers in grain, cotton, and swine confinement industries that acute decline in lung function during a shift is an independent predictor of longitudinal decline in lung function. This subject has been reviewed in depth by Becklake.[1] Although the observations were limited to organic dust, it is likely that exposure to fumes may lead to similar changes. Fishwick *et al* are wise to follow up their welders with acute airway response to welding.

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1 Becklake MR. Relationship of acute obstructive airway change to chronic (fixed) obstruction. *Thorax* 1995;**50**(suppl 1):S16–21.

Heavy physical work and occurrence of sciatic pain: need for Poisson log linear models or for better data?

EDITOR,—In a recent issue of *Occupational and Environmental Medicine*, Nurminen presented a re-examination of data from a sample of 419 workers previously analysed by Riihimaki.[1 2] The objective given by Nurminen was to clarify the role that heavy physical work had in the production of back pain. The data on occurrence of sciatic pain according to age, earlier back accident, and occupation (concrete reinforcement workers versus maintenance house painters) presented in a 48 cell multiway contingency table were reanalysed with a Poisson log linear model. The conclusion was that earlier back accident, aging, and also heavy physical work (more precisely, belonging to the group of concrete reinforcement workers) were related to sciatic pain, whereas the first analysis had failed to show the role of heavy physical work.

The paper is interesting in that it reminds us of important methodological aspects on confounding, interaction, and the comparison between models. It also presents the Poisson log linear model. The reader may regret the lack of details in the presentation of this model, as it is less widely known than logistic or additive models. The model remains obscure, except for specialists who, presumably, are already familiar with it.

The reader may also regret the lack of discussion on several methodological points: is this model really more parsimonious than the logistic model, as it contains 24 parameters, which seems much for describing a 48 cell contingency table? The p value for occupation and sciatica is 0.048. Does it remain <0.05 if the terms in the model are slightly changed, or if age is treated differently? How far is it possible to draw statistical inference about associations between heavy work and sciatica, beyond this particular data set ?

However, the most questionable point about this interesting statistical and methodological exercise deals with its relevance for a better understanding of the occupational determinants of sciatica. The analysis presented by Riihimaki was done more than 10 years ago. In 1985 it would have been difficult, maybe impossible, to use a Poisson log linear model, as the methodological references given by Nurminen had not yet been published. Clearly, the tools for analysis have improved in the