

to patients and colleagues responsible for acutely ill patients with COPD. In keeping with the NICE guidelines, COPD severity should be described in terms of functional status using at least the modified MRC score, as well as previous severity of lung function impairment. It should be made clear that the severity of lung function impairment in COPD does not necessarily correlate with clinical severity or outcome of inpatient care, which may include ITU and IMV management.

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Importance of past occupational exposures in the rising incidence of idiopathic pulmonary fibrosis in the UK

We read with interest the recent article by Navaratnam *et al* highlighting the unexplained rising incidence of idiopathic pulmonary fibrosis (IPF) in the UK.¹ While we agree that this area is of great clinical interest, we feel that the rapidly rising incidence, linked with the gender, age, geographical and socioeconomic risk factors for this disease, is strongly suggestive that the cause is not wholly idiopathic. Previous work by the same group found that 20% of IPF could be explained epidemiologically by occupational exposures to metals or wood dust,² yet there is no discussion relating to how these or other exposures may have changed over the time period studied.

Mortality due to asbestosis is also likely to be highly relevant here, and in a separate paper published recently, the same research group has reported a 10-fold rise in asbestosis mortality from death certificate data (13 in 1968 to 129 in 2006).³ The authors went on to note that the rising asbestosis mortality mirrors the rising trend in mesothelioma mortality, where over a similar period deaths rose from 135 to 2058.

Given that the mortality from IPF clinical syndrome seems to be rising in parallel to that of mesothelioma, and that these diseases have similar demographic risk factors, the obvious question that arises is how much IPF is actually due to asbestos exposure that has not been recognised in life, or not recorded on the death certificate? Asbestos usage in UK industry was widespread up to the 1980s, as demonstrated by a recent mesothelioma study where two-thirds of the randomly selected male controls born in the 1940s were found to have worked in at least one high or medium risk job for asbestos exposure.⁴

These data suggest that it is likely that a large proportion of UK males presenting with pulmonary fibrosis aged 60–70 years will have previously been occupationally exposed to asbestos in the 1950s–1980s, whether or not they report it when questioned 40–50 years later. This is compounded by the similar radiological features shared by asbestosis and usual interstitial pneumonitis, as well as the problems of interpreting asbestos fibre counts if available. These diagnostic difficulties, linked with the known inaccuracies of death certificate data, and no understanding of individual susceptibility are likely to make establishing a clear epidemiological link between IPF and asbestos challenging.

While we agree with the authors that more research is required in this fascinating area, we believe the term 'idiopathic' may be misleading, and that a significant proportion of UK IPF is likely to relate to past occupa-

tional exposures. Given that UK peak asbestos usage was in the late 1960s, and with reference to a US model,⁵ mortality from asbestosis should peak around the same time as that of mesothelioma, sometime between now and 2020. The relative change in mortality from mesothelioma, asbestosis and IPF over the next decade will therefore be of great interest, and may give us a valuable insight into the true relationship between asbestos exposure and pulmonary fibrosis.

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Authors' response

We thank Drs Barber and Fishwick for their interest in our paper and would like to briefly respond to their comments.¹ Our paper describes a pragmatic epidemiological study with individuals who had an underlying diagnosis of idiopathic pulmonary fibrosis clinical syndrome (IPF-CS).² The aim was to investigate recent temporal trends in incidence and survival and not to consider environmental exposures that may have an aetiological link. Hence, although this increase may be linked to occupational or other environmental exposures, our data do not permit firm conclusions to be drawn. It is also possible that the rapid increase in incidence reported is due to an increasing tendency to investigate patients in the UK.

We agree that there is a possibility that a proportion of our cases may have been exposed to asbestos. However, 39% of registered deaths from IPF-CS (from the Office of National Statistics dataset) and 37% of cases in primary care (from The Health Improvement Network dataset) were women, who are unlikely to have had a substantial direct exposure to asbestos. We believe that factors other than occupational exposure to asbestos are important in explaining our data and until firm aetiological links are established, similar cases of pulmonary fibrosis should be considered idiopathic.

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