

# Defending the data

When sick miners sued Britain's National Coal Board in the 1990s, they did so on the basis of research that linked coal dust exposure to lung disease. But that research was soon under attack in court. **Jonny Jacobsen** reports on his father's role justifying decades of work on coal miners' lung disease during landmark litigation



In January 1997 my father, Michael Jacobsen, received an email from an alarmed former colleague. The email told how decades of research, in which they had both been heavily involved, was under attack in the courts.

Welsh law firm Hugh James was representing eight miners seeking compensation from the National Coal Board (NCB).<sup>1</sup> They were arguing that exposure to coal dust had left them with a range of respiratory illnesses, and that the NCB had failed to introduce safety measures quickly enough.

For the epidemiological side of their argument, the plaintiffs based much of their case on a massive body of work that the Coal Board itself had funded: the Pneumoconiosis Field Research (PFR). The findings of the PFR had led to improved dust standards not just in Britain, but in coal mines across the world.

But the defence adopted by the NCB – which by then had become the British Coal Corporation – was to attack the very work it had commissioned. The defence experts, my father's correspondent warned, were "trashing" the PFR. The email continued: "I'd like your advice on this as well as any information you have on whether anyone is defending the PFR work."

My father, who had overseen the statistical aspects of the research for 20 years, was equally alarmed. He had already been contacted by the same law firm and, in the end, it fell to him to come to the PFR's defence.

In a trial that heard evidence for 102 days, half was devoted to considering the medical evidence – and the battle over the validity of the PFR was at the heart of the debate.

## "These people were dying"

I always knew how important this case was to my father. But as I went through his papers in the months following his death in October 2017, I got a clearer sense of just how much pressure he had been under.

The lawyers had not exaggerated the threat to the research, my father wrote in reply to his colleague. "Collectively, this represents (*inter alia*) a systematic attempt to rubbish PFR survey procedures, data, analyses and inferences."

But more was at stake than defending decades of research. Tens of thousands of miners and ex-miners across Britain were struggling with breathing problems.

"Men with very serious chest diseases were dying at relatively young ages: late 50s, early 60s," says Bleddyn Hancock, the former miner and union leader who brought the case to court. Others, some so ill they needed oxygen tanks to breathe, were being turned down for sickness benefit.

In 1989, the small mining union NACODS (the National Association of Colliery Overmen, Deputies, and Shotfirers) commenced legal proceedings on behalf of these miners, seeking compensation.

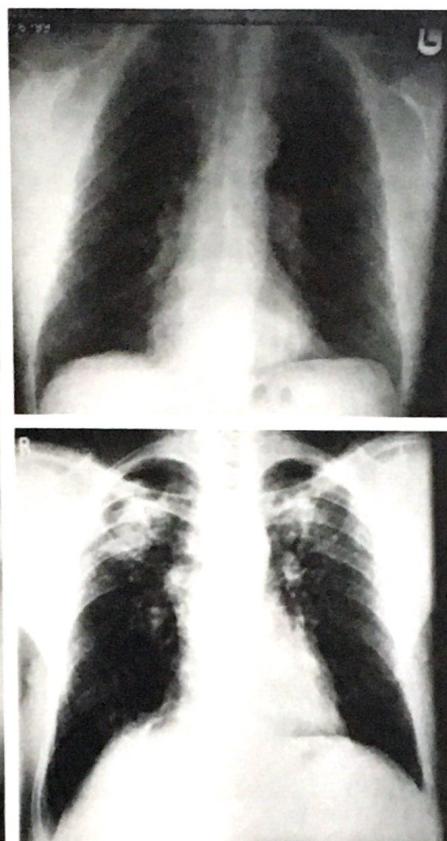
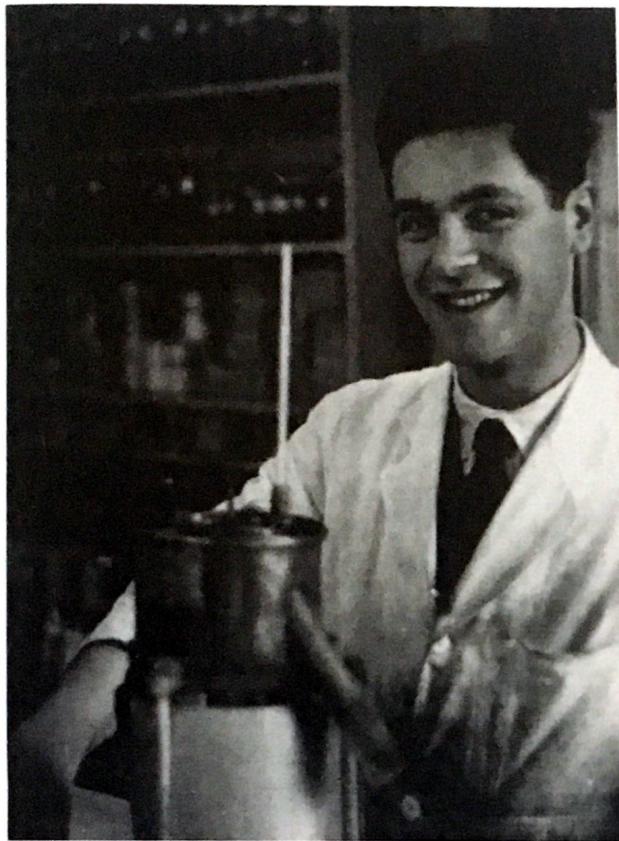
It had long been acknowledged that inhaling coal dust could provoke coal worker's pneumoconiosis, also known as "black lung". In its more serious form, progressive massive fibrosis (PMF), it could be fatal (see X-rays on page 19 and box on page 22).

But X-rays showed that these men did not have pneumoconiosis. Hancock – who was general secretary of



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Photo by Zaida Kersten



**FAR LEFT** Professor Michael Jacobsen, a statistician, epidemiologist, and former head of research and deputy director of the Institute of Occupational Medicine, who died in October 2017 aged 84. Photo courtesy of Jonny Jacobsen.

**LEFT** X-rays showing (top) a healthy pair of lungs and (bottom) a case of progressive massive fibrosis, the most serious form of pneumoconiosis. Photos courtesy of Professor Anthony Seaton.

NACODS in South Wales at the time – pressed doctors to explain what was wrong with the two dozen miners he had already tried to help. The medics suggested the breathing problems might be due to smoking.

Hancock was not convinced. "I noticed that a very high proportion of men with severe breathing difficulties had never smoked in their lives. And that made me think, 'Well what is it then?'"

The union called in a lung specialist, Dr Robin Rudd. His tests revealed that while the miners did not have pneumoconiosis, they did have respiratory diseases such as chronic bronchitis and emphysema.

So, when NACODS launched its class action it argued that coal dust was responsible for these other respiratory diseases and the attendant health issues.

Fighting the case was a major financial gamble for the union, says Hancock. "But I was convinced it was the right thing to do. I mean, these people were dying."

In fact, by the time the case opened in October 1996, two of the plaintiffs were already dead.

### A reluctant witness

The PFR, on which the plaintiffs were leaning so heavily, was a massive study, dating back to the early 1950s. Its aim was to gather data so officials could draw up new standards to protect miners from the disabling effects of coal dust. The Institute of Occupational Medicine (IOM), an independent charity set up

by the Coal Board in Edinburgh, managed the research from 1969 onwards.

My father joined the IOM from its inception. He arrived as head of statistics, completed his PhD there,<sup>2</sup> and was head of research and deputy director when he left the organisation in 1989. During that period, he oversaw the statistical analysis of data from the PFR work.

Given his key role then, it was no surprise that he was drawn into what became known as the British Coal respiratory disease litigation. Hugh James, the Welsh law firm representing some of the plaintiffs, had approached him in 1996 asking for his expertise.

At that time my father was an associate professor and deputy director of the University of Cologne's Institute of Occupational Medicine. Having escaped Nazi Germany as a child, he had finally returned to the country of his birth to see out the final years of his career.

Lawyer Gareth Morgan of Hugh James, who worked on the case from the beginning, recalls my father's initial reservations about testifying. "He was a very principled guy. He didn't want to give evidence for the miners but wanted to be an independent witness. He wouldn't be paid ... I think in the end we had to subpoena him."

My father agreed to write up his thoughts on the defence documents attacking the PFR study and submit them to the court. He submitted a first set of notes – more than 40 pages, fully annotated and referenced – in the first week of December 1996. ▶

By the following month, the subpoena had landed: the Lord High Chancellor, Lord Mackay of Clashfern, commanded him to attend court.

As the summary judgment put it a year later: "Dr Jacobsen, reluctant at first to give evidence because he felt his impartiality could be impugned by so doing, was nevertheless prevailed upon to assist the court and gave evidence when called on behalf of the plaintiffs".<sup>1</sup>

### The battle lines

The plaintiffs' argument was that the NCB's own PFR work showed that coal dust was killing miners, not just the effects of smoking. The NCB's opening position, however, was that while coal dust might cause chronic bronchitis and emphysema, neither led to any serious loss of lung function. Where miners with these diseases were suffering lung deficiencies, it argued, this was due not to the coal dust but to the fact that they smoked. Most of the plaintiffs in this test case were smokers.

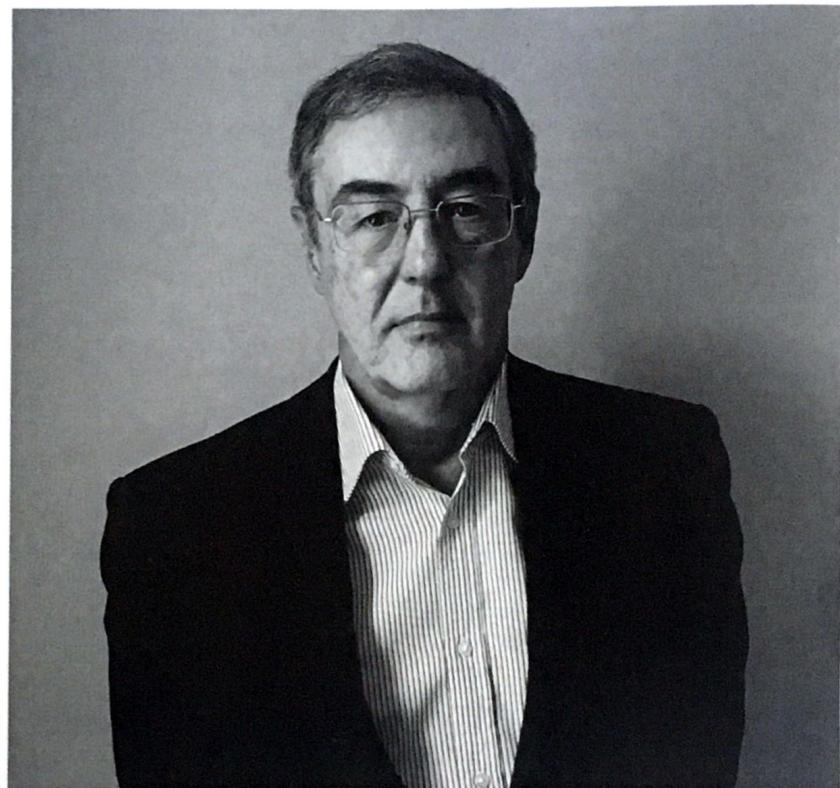
But the PFR data were telling a different story. The research, conducted over decades, looked at the effect on coal miners of their exposure to coal mine dust. Its original aim was to find out how much and what kind of dust caused pneumoconiosis, and what working conditions were required to prevent miners being disabled by the dust they breathed in.

As far back as the early 1970s, the PFR data had been suggesting there was something more to miners' ill health than pneumoconiosis, says Fintan Hurley, who recently retired as the IOM's science director. "What was crucial to the PFR was the quality of its assessments of coal miners' exposures to dust," he says.

Hurley, who collaborated with my father on this work during the 1970s and 1980s, says the research showed a clear relationship between dust exposure and both lung function and respiratory symptoms – even after adjusting for smoking habits. "So it was known for a long time that dust exposure was related to lung disease much wider than pneumoconiosis in coal miners." That was why the PFR work was so important to the miners' case.

My father, in his court testimony, went to great lengths to explain how the research was conducted, to counter defence claims that its methodology and data were unsound.

Starting in 1953, two specially equipped lorries took chest X-rays of more than 30 000 miners at 25 mines scattered across England, Scotland and Wales. The collieries were



**ABOVE** Bleddyn Hancock, the former miner and union leader, was a key figure in bringing the British Coal respiratory disease litigation to court.  
Photo by Jonny Jacobsen

chosen to represent a range of environmental conditions, including different kinds of coal in the various UK coalfields. From 1953 until 1977, more than 53 000 miners were examined in at least one of the roughly five-yearly rounds of medical checks at these sites (though health checks continued at one mine until 1991). Researchers also recorded the miners' occupational histories.

Five years in, the research was expanded to take account of other kinds of lung disease. Researchers introduced a questionnaire on respiratory symptoms and the smoking history of the subjects.

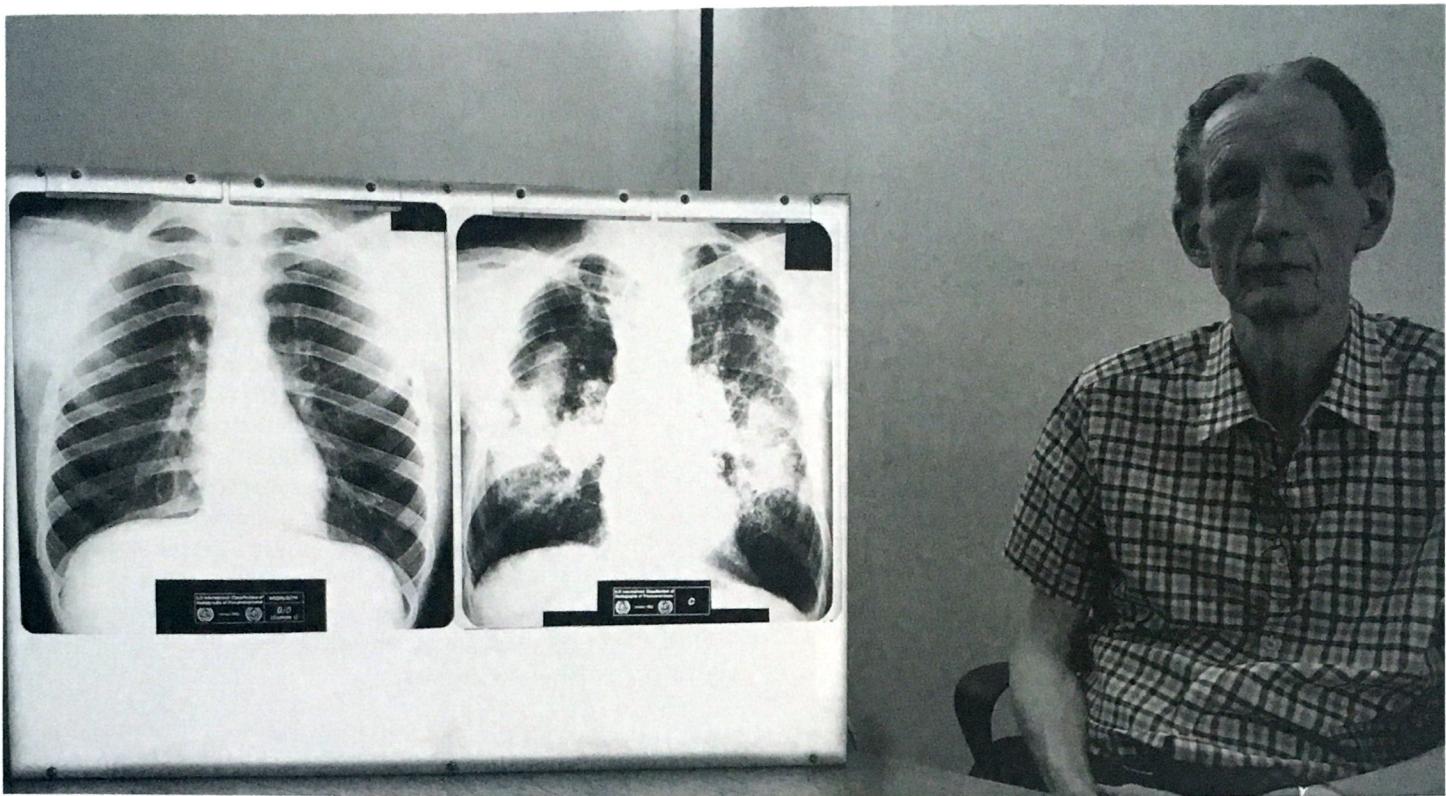
They used spirometric equipment to measure lung function and, at specially equipped vans stationed at the pit head, trained technicians conducted a test known as forced expiratory volume of air in one second (FEV1).

Researchers kept track of how many shifts each miner worked over the years, as well as what that work was – important because different jobs entailed different levels of dust exposure.

The PFR team also had to factor in the exposure of miners to coal dust before their study had even begun. That involved getting a detailed history from each miner of how long they had worked in the industry and what kind of work they had performed. That work was then classified according to the level of exposure to coal dust it entailed.

The total time worked and in what conditions – the concentration of respirable dust – was then added up, factoring in the data gathered from systematic dust sampling at the relevant collieries.

"A very high proportion of men with severe breathing difficulties had never smoked in their lives. And that made me think, 'Well what is it then?'"



The first IOM paper on the PFR work appeared in 1970 in the journal *Nature*.<sup>3</sup> It showed a clear relationship between dust exposure and the risk of pneumoconiosis (see Figure 1, page 22, for a related example). That led to tighter airborne dust standards in British coal mines and, in the years that followed, the PFR was used as the basis for national dust standards in other countries.

But of all the research published based on the PFR data, the most important, so far as the NACODS case was concerned, was a paper published in 1988: "Clinically important respiratory effects of dust exposure and smoking in British coal miners".<sup>5</sup> This was known as the Marine paper, after its main author, Professor William M. Marine, of the University of Colorado's Health Sciences Center.

The authors – my father among them – reanalysed a data set of 3380 miners studied in the PFR work. That included a subset of 451 lifetime non-smokers who "were used to predict levels of FEV1 that might be expected for men of the kind being considered here if they had not been exposed to dust". They found that non-smoking miners exposed to high levels of coal dust were at least as likely to develop obstructive airway disease as those miners who smoked but were exposed to low levels of dust (see Figure 2, page 22).

According to the Marine paper: "There is no evidence that the effect of dust exposure is more severe in miners who smoke than in those who do not smoke. Both insults are deleterious to health. Their effects on lung function and on bronchitic symptoms appear to be additive, and it follows that preventive strategies should continue to be aimed at reducing both pollutants."

ABOVE Dr Robin Rudd, a lung specialist, was an expert witness for the miners during the British Coal respiratory disease litigation.  
Photo by Jonny Jacobsen

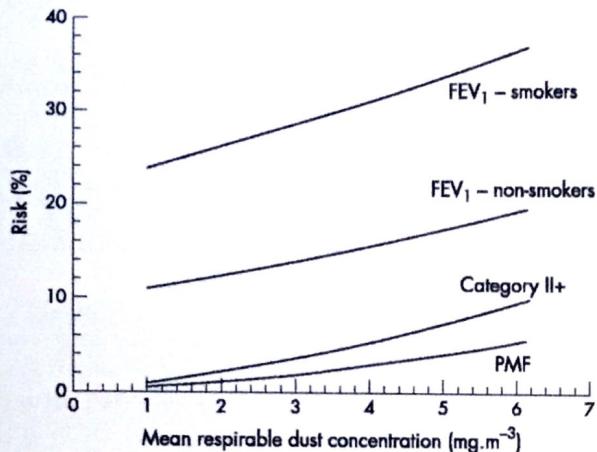
### The signal, not the static

The conclusions of the Marine paper presented strong evidence in support of the miners' claim for compensation, but three reports – drawn up by experts for the defence – advanced a series of objections to the PFR work, to which my father responded point by point. However, one major criticism of both the PFR study and the Marine paper went to the heart of the debate: the reliability of the data on non-smokers.

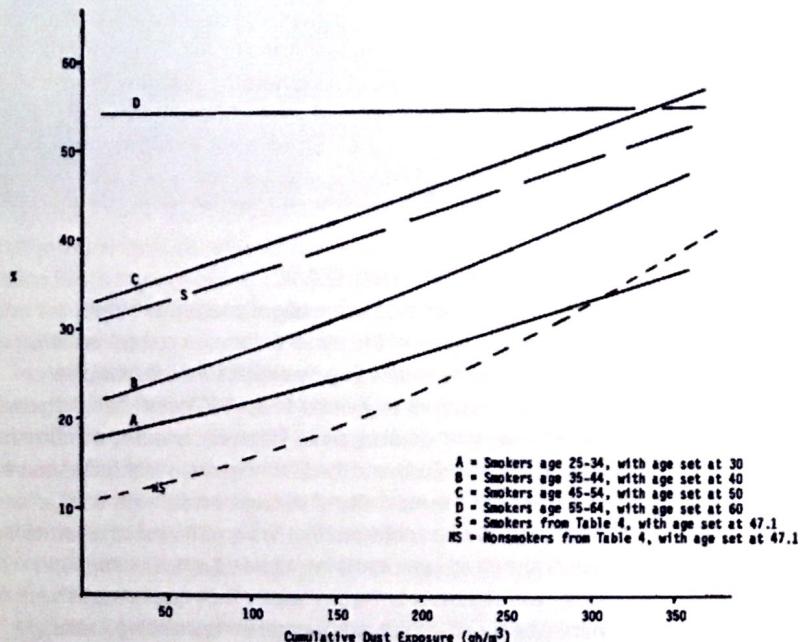
Defence experts objected that the questionnaire on smoking habits and chest symptoms introduced five years into the study was relatively limited in scope. They questioned the reliability of self-reported information on smoking habits, pointing out inconsistencies in the data to support their case. Some smokers might have described themselves as non-smokers because of the growing stigma attached to the habit, they suggested; others might have under-reported how many cigarettes they smoked a day.

In response, my father acknowledged that questionnaires, while valuable, were an imperfect way of gathering information. He conceded that doubts remained about the accuracy of self-reported smoking information. "PFR scientists were under no illusions that their data were error-free in this respect," he wrote in his court notes. That said, the smoker/non-smoker patterns the PFR study found among miners broadly matched those of the general adult population of Britain at the time.

But my father's central point was that even if some smokers had been misclassified as non-smokers, it would have made no difference to the estimated effects of dust exposure on



**FIGURE 1** This figure, from a 2004 paper by IOM researchers, shows risks at age 58–60, after 35–40 working years, of: progressive massive fibrosis (PMF); category II+ simple pneumoconiosis; 993 ml deficit of FEV<sub>1</sub> in non-smokers; 993 ml deficit of FEV<sub>1</sub> in smokers. Republished courtesy of *Occupational and Environmental Medicine/BMJ Journals*.<sup>4</sup>



**FIGURE 2** An excerpt from the Marine paper, published in 1988 in *The American Review of Respiratory Disease*.<sup>5</sup> The graph shows "Estimates of chronic bronchitis prevalence (%) from logistic regression equations, in relation to cumulative dust exposure". Reprinted with permission of the American Thoracic Society. Copyright © 2018 American Thoracic Society. *The American Review of Respiratory Disease* is an official journal of the American Thoracic Society.

► **FEV<sub>1</sub> levels.** Several analyses of PFR FEV<sub>1</sub> data had yielded highly significant estimates of the effect of dust exposure among self-declared current smokers, he said – and they were statistically indistinguishable from those applicable to miners who were self-declared non-smokers. Removing spurious "non-smokers" from the analysis might lower the level of statistical significance of the result. But even that was unlikely to make much difference to the overall findings.

Lawyer Gareth Morgan remembers one defence report in particular that listed a string of errors in the PFR data. It pointed out X-ray results showing a medically impossible improvement in a miner's pneumoconiosis, and a miner's date of birth that would have made him 126 years old.

"I remember when the study pointed out these errors I was quite concerned," he says. But then one defence expert explained that, actually, if the overall conclusions of the PFR work remained intact despite these errors, it underlined the strength of the research. And that was what my father argued, during more than a week on the stand. "It's like radio signals," says Morgan. "If there's a lot of interference, it has to be a stronger signal to get through." In this case, the signal was stronger than the static.

### The judgment and beyond

When Mr Justice Turner handed down his judgment on 23 January 1998, little remained of the lengthy debate over the PFR study.

The defence experts' critique of the PFR work had revealed some errors in the recording of lung function that had been incorporated into the Marine study. But those errors made no appreciable difference to its conclusions, wrote the judge.

On the other hand, he noted, substantial parts of the defence reports attacking the PFR work "were exposed by Dr Jacobsen to be ill-informed, inaccurate and either unjustifiable or plainly mistaken".

"The irony was that by attacking the PFR data, they ended up showing how strong it was," says Morgan. "I can remember being in court when this dawned on the defendants."

The judge also ruled that the NCB was at fault on safety measures, noting: "There was abundant evidence which emanated from a variety of sources that officials interpreted their duties as requiring the production of coal first and the taking of precautions in respect of health second."

On the key medical issue, the judge agreed with the plaintiffs that coal mine dust could indeed cause emphysema and that this could lead to breathing difficulties. And while it might not be possible in any given case to establish if that breathlessness was caused by smoking or coal dust, the claim still stood. Any smoking miner exposed to heavy dust, wrote the judge, "is more likely than not to have had a significant contribution to his breathlessness made by that exposure".

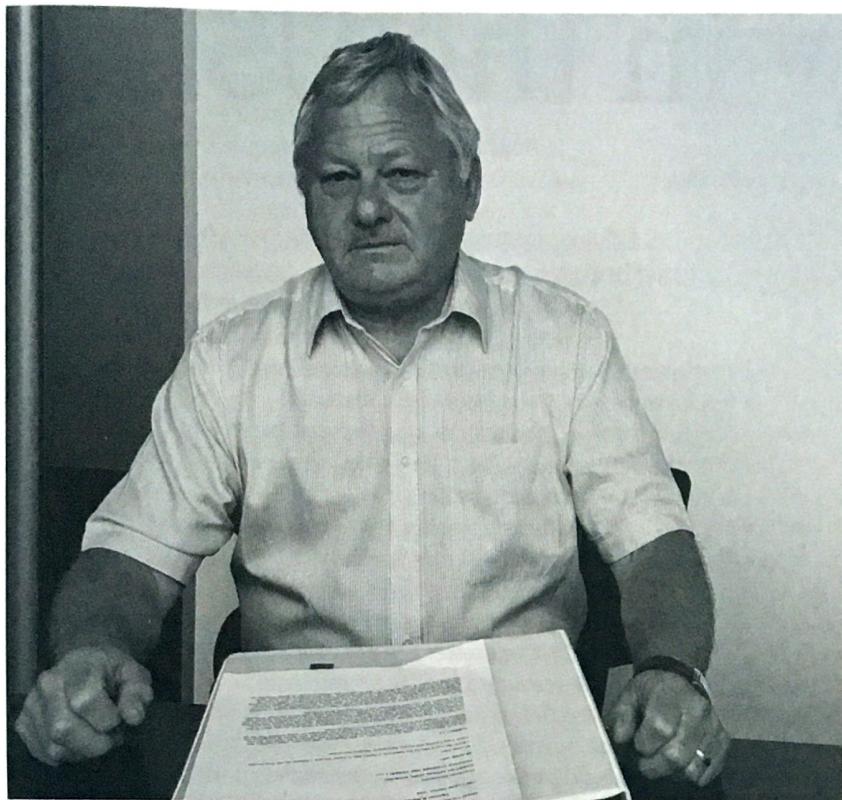
Dr Robin Rudd, the lung specialist who acted as an expert witness for the plaintiffs, puts it this way. "The epidemiology is the evidence. You can't look at a chap's lungs and see how much was caused by smoking and how much by dust. You can only reach a conclusion on the basis of the epidemiology."

## An ongoing problem

There is growing concern about a resurgence of progressive massive fibrosis among coal miners in the United States.

In February, epidemiologists at the National Institute for Occupational Safety and Health (NIOSH) reported a surge in PMF cases among coal miners.<sup>6</sup> They counted 416 cases among 11 200 coal miners examined at three black lung clinics in southwest Virginia.

Scott Laney, one of the co-authors of the NIOSH report, told National Public Radio (n.pr/2zLg5of): "We've gone from having nearly eradicated PMF in the mid-1990s to the highest concentration of cases that anyone has ever seen."



The key point was that "we assume that what applies to groups applies to the individuals ... But as you can't measure it in individual cases, you assume the individual has the same susceptibility".

Twenty years on, Rudd has no doubt as to the importance of the PFR work to the miners' case. "Without that, they wouldn't have had any chance of success," he says.

The settlement in the British Coal respiratory disease litigation was the largest ever industrial injury group action in the United Kingdom. Approximately £2.4 billion was paid out to the miners or their families ([bit.ly/2uHy2hR](http://bit.ly/2uHy2hR)). But it took more than a decade for all the claims to be settled, and thousands of miners died while they waited. Of some 456 600 pay-outs, more than half went to the widows or the estate of the now deceased miners. The last claim was settled as late as 2012.

In the meantime, in a related case, ex-workers at the Phurnacite coke plant in Abercwmboi, South Wales, won compensation after establishing "the necessary causal link between exposure to dust and fumes ... and the development

**ABOVE** Gareth Morgan, a lawyer for the Hugh James law firm, worked on the British Coal respiratory disease litigation from the beginning.  
Photo by Jonny Jacobsen

of lung cancer, chronic bronchitis and chronic obstructive pulmonary disease" ([bit.ly/2zLT3ha](http://bit.ly/2zLT3ha)).

Here again the plaintiffs drew on the work of the IOM, including data from the PFR. "The Coal Board spent about 10 years defending that before it came to court in 2012, wasting millions defending the indefensible, in just the same ways that they had done with the coal miners," says Rudd.

Who it is that makes the decision to fight these claims to the bitter end, and why, is something that still puzzles him. "They must have spent far more to defend them than to just pay everyone off," says Rudd. "It is a strange mentality."

British Coal, like the NCB, no longer exists. Some of its functions are now managed by a public body called the Coal Authority, but liabilities for coal health compensation claims rest with the UK government and are managed by the UK Department for Business, Energy and Industrial Strategy (BEIS). *Significance* sought comment from BEIS but received no response.

### No regrets

Going through my father's papers, it is clear he had mixed feelings about getting involved in the British Coal respiratory disease litigation. In one of his letters, he told how Dr John Rogan, chief medical officer of the NCB, once advised him to avoid legal cases surrounding medico-scientific matters.

"Everything that I have experienced since then convinces me that JMR [Rogan] was right. But the attacks on the integrity of the PFR ... were so outrageous that I felt that I had to respond.

"I do not regret that decision." ■

### Acknowledgements

This article draws on my father's papers, on the judgment itself, and on interviews with Bleddyn Hancock, Dr Robin Rudd and Gareth Morgan. My thanks, too, to Fintan Hurley, recently retired science director at the IOM; Dr Michael Atfield, formerly of both the IOM and the NIOSH; and former IOM director Professor Anthony Seaton for their help.

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