Mortality in an extended follow-up of British coal workers.

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Abstract. The Pneumoconiosis Field Research (PFR) programme was established in the 1950s, to evaluate effects of coal mining exposures on the health and mortality of British coal workers. Surveys of working miners were carried out at 5-yearly intervals, initially in 24 collieries but later concentrating on 10, collecting detailed work histories and health information for each recruit. Here we report on cause-specific mortality in a cohort of almost 18,000 men from 10 British collieries, followed up for periods up to 47 years, yielding over 516,000 life-years of follow-up. External analyses compared cause-specific death rates in the cohort to those of the population of the regions in which the collieries were situated, using Standardised Mortality Ratios (SMRs). The causes investigated included lung cancer, stomach cancer, non-malignant respiratory disorders and cardiovascular disorders. SMRs showed evidence of an initial healthy worker effect diminishing over time. Several causes, including non-malignant respiratory disease and lung cancer, showed a significant deficit of mortality at the start of the study period with an excess in the latter part of the follow-up period. In these results, effects of working conditions are likely to be confounded with smoking habits. Overall, we believe our results may be generalised to the British coal industry since nationalisation.

1. Background
The Pneumoconiosis Field Research (PFR) programme of the then National Coal Board began in the early 1950s. The aims were “to determine how much and what kinds of dust cause pneumoconiosis, and to establish what environmental conditions should be maintained if mineworkers are not to be disabled by the dust they breathe during the course of their work”. The research began in the 1950s in 24 representative British collieries, later reduced to 10. All industrial-grade workers at these collieries were invited to regular health surveys (around every five years) including chest radiographs, lung function tests and questionnaires on respiratory symptoms and smoking habits. Response rates were high, generally well above 90%. A separate programme of measurement quantified typical concentrations of respirable dust and quartz at the locations of a wide variety of colliery occupations. These were linked to data from the colliery payroll systems, on the times worked by each worker in the same occupations, to produce individual and period-specific estimates of exposure to respirable dust and quartz.

Study of coalminers’ mortality within the PFR began in 1970. Several phases of tracing led to the inclusion in a mortality database of all workers surveyed during the PFR. Several reports ([1],
have compared mortality experienced in the cohort with that expected on the basis of local reference rates, and have shown increased mortality risks in those with severe pneumoconiosis at the start of follow-up, and a general increase in risk from stomach cancer. Analyses (based on subsets of the cohort with appropriate data) have also shown exposure-response relationships for risks of various respiratory causes of death with increasing exposure to dust, but little evidence of increased cancer risks from dust or quartz exposures.

The present study is based on an extended period of follow-up in a previously analysed cohort of almost 18,000 workers from the 10 continuing PFR collieries. Exposure estimates are the same as before, since the collieries were all closed by the early 1980s.

2. Methods
The entire PFR cohort had been flagged for death notification in the National Health Service Central Registers (NHSCR) administered by the Office of National Statistics (ONS) and the General Register Office (Scotland) (GRO(S)), but death notification had been suspended some years previously. Notification was reactivated, and the database used in the most recent PFR study report [3] was updated with the new death details. These included the underlying cause of death coded according to the International Classification of Diseases and Causes of Death (ICD) published by the World Health Organisation (WHO), in the contemporary revision ([6], [7], [8], [9]).

To create regional reference rates for selected causes of death, data were obtained from the national authorities on numbers of deaths from those causes and on the mid-year male populations, by age and calendar year, for each of the relevant administrative regions of Britain. In a few cases where death data for specific cause groups were not available for a small number of early years, we extrapolated or interpolated rates from years for which data existed, using regression models.

Comparisons with regional reference data used indirect standardisation to calculate expected deaths from selected cause groups, and to compare observed numbers with these, stratified by age group and calendar year within region, calculating standardised mortality ratios (SMRs) with 95% confidence intervals assuming Poisson variation. All the calculations were carried out using the table manipulation facilities of the statistical package Genstat [10].

3. Results
Miller et al. (1997)[3] analysed mortality in 18,166 men from 10 collieries within the PFR cohort. On reactivation of the cohort tracing, vital status could not be ascertained for 346 of these men, and the final analysis cohort consisted of 17,820 men.

SMR calculations were based on follow-up to the end of 2005, yielding 516,431 person years and 10,698 deaths from all causes. The resulting SMRs and 95% confidence intervals are displayed in Table 1. (Table 1 includes the numbers of deaths described as from pneumoconiosis and silicosis, but no SMRs are calculated for these causes. Since these are diseases associated entirely with occupational exposure, there are no meaningful background rates with which to compare the observed incidences.)
Table 1 Summary results of comparisons of mortality in cohort with external reference rates. The table shows, for chosen cause groups, numbers of deaths, age-year- and region-standardised mortality ratios (SMR) and 95% confidence interval.

| Cause of death                          | Observed deaths | SMR % | Confidence bounds |
|----------------------------------------|-----------------|-------|-------------------|
| All causes                             | 10,698          | 100.9 | 99                |
| All external causes                    | 278             | 87.5  | 77.8              |
| All internal causes                    | 10,421          | 103.7 | 101.7             |
| Tuberculosis                           | 16              | 77.8  | 47.6              |
| All cancer                             | 2,732           | 98.0  | 94.4              |
| Stomach Cancer                         | 318             | 129.0 | 115.6             |
| Lung Cancer                            | 958             | 98.7  | 92.6              |
| Cardiovascular Disease:                |                 |       |                   |
| Ischaemic Heart Disease                |                 |       |                   |
| Acute Pulmonary Heart Disease          |                 |       |                   |
| Non-Malignant Respiratory Disease      |                 |       |                   |
| COPD                                   | 1,966           | 138.2 | 132.3             |
| Chronic Bronchitis                     | 849             | 115.5 | 108.0             |
| Emphysema                              | 500             | 138.9 | 127.3             |
| Pneumoconiosis                         | 70              | 164.4 | 130.1             |
| CWP                                    | 28              | 71.1  | 49.1              |
| Silicosis                              | 222             | NA    | NA                |
|                                      | 10              | NA    | NA                |

The all-cause SMR was 100.9% (95% confidence interval – CI – 99.0, 102.9), while that for all internal causes was 103.7% (101.7, 105.7). SMRs for tuberculosis, lung cancer, external causes and cardiovascular disease showed no overall excess, while those for stomach cancer and non-malignant respiratory diseases showed significant excesses.

The SMRs for chronic obstructive pulmonary disease (COPD) overall, 116%, and subcategories chronic bronchitis, 139%, and emphysema, 164% were all significantly different from 100%. Miller et al. (1997)[3] also found a significant excess of mortality from chronic bronchitis, but did not calculate SMRs for any other NMRDs. As the fashions of cause attribution change over time it is more sensible to consider the broad category of NMRD when considering changes in the SMRs over time.

The above statements on uncertainty in the overall SMRs should be interpreted with caution, since in this case average results conceal important temporal patterns. For example, the current lung cancer SMR of 99% is considerably higher than the previously reported 87% [4] and 86% [3] and a similar rise is apparent in our all-cause SMR of 101%.

All the previous reports on this cohort identified the low all-cause mortality as indicating a ‘healthy worker’ effect. The cohort was defined initially by employment, so a healthy worker effect was to be expected. However, we should also expect that as the cohort aged (and the coalmining industry disappeared) this effect would diminish and perhaps disappear. Figure 1 shows the time course of all-cause SMRs calculated for successive 5-year periods. It is clear that the initial deficit had been diminishing steadily throughout the follow-up, and that the SMR had
in fact been above 100% since the mid-1970s. The size of the apparent dip in the most recent period could be consistent with normal fluctuations, but the evidence seems clear that not only has the healthy worker effect long dissipated, the recent experience of this group is of higher all-cause mortality than in the general populations in their regions.

Figure 2 shows the time-course of lung cancer SMRs calculated for successive 5-year periods, and again shows a clear rising trend over the follow-up, more than doubling from around 60% at the beginning to around 130% recently. This pattern is seen again, although with an earlier rise, for NMRD (Figure 3), where the SMR levels out at around 140%. This was also repeated for the majority of the other causes of death considered here (Miller et al, 2007 [11]).

In contrast, the excess of stomach cancer seen here is consistent with that found by Miller et al. (1997)[3] who calculated an SMR of 124% (95% CI 110-141), suggesting that the healthy worker effect was not relevant to these stomach cancers. Figure 4 shows no convincing evidence of a healthy worker effect during the follow-up period.

**Figure 1** Standardised Mortality Ratio (SMR) for all internal causes over the length of the follow-up period where year is grouped. The solid line is the SMR while the dashed lines represent the 95% confidence interval. The dotted line shows the SMR equal to 100%.
Figure 2 Standardised Mortality Ratio (SMR) for lung cancer over the length of the follow-up period where year is grouped. The solid line is the SMR while the dashed lines represent the 95% confidence interval. The dotted line shows the SMR equal to 100%.

Figure 3 Standardised Mortality Ratio (SMR) for non-malignant respiratory disorders (NMRD) over the length of the follow-up period where year is grouped. The solid line is the SMR while the dashed lines represent the 95% confidence interval. The dotted line shows the SMR equal to 100%.
4. Discussion

Our data on coal workers’ mortality are unique in their combination of response rates at the studied collieries, detailed exposure characterisation and length of follow-up. The original collieries were chosen to represent the range of coal types and regions in the British coalfields, and the continuing 10 collieries retained this coverage. The response rates for the health surveys were high because screening for pneumoconiosis was accepted generally by workers and their representatives. Non-ascertainment of vital status was low, as is typical of British registration systems. Overall, we believe our results to be representative of the British coal industry since nationalisation.

Our extended follow-up has permitted examination of the time-trends in the comparison of mortality rates with regional reference rates. We interpret many of the early deficits as evidence of ‘healthy worker’ selection effects, because all cohort members had to be healthy enough to be employed in coalmining at study entry. However, the five-year breakdown of SMRs shows clearly that this effect was temporary, and the later SMRs for NMRD, lung cancer and all-cause mortality were in clear excess. Given such trends, interpretation of summary SMRs for the whole follow-up period is problematic and potentially misleading.

It is not surprising that there is a significant excess of mortality from NMRD in the cohort as coal mine dust has long been associated with respiratory disorders, specifically pneumoconiosis and COPD: and respiratory symptoms might well have been taken into account in the recruitment process, so a healthy-worker effect for this cause is highly plausible. However, not all of the trends found can be due to the selection of healthy men to work in coal mines. Lung cancer, with its poor cure rate and short expectation of survival from diagnosis, would not be expected to show strong healthy-worker selection effects, but the early deficits are evident in these data. These deficits in lung cancer mortality were not explainable by the prevalence of smoking as reported at the health surveys, which in this cohort was higher than typical of that era. A more likely explanation is in the strict prohibition on smoking within coalmines, limiting total
cumulative doses in those coalworkers who were regular smokers. That prohibition disappeared with the dissolution of British coalmining industry around 1980, and presumably the workers made redundant then had on average greater freedom to indulge their tobacco habit. This could partly explain the steep rise in NMRD deaths from the late 1970s onwards, and latency for lung cancer would be consistent with the later rise in those deaths. However, we have no data on actual cigarette consumption after the collieries closed.

Our results for stomach cancer add to a body of data that has demonstrated consistently elevated risks in coal workers. Other studies have shown similarly elevated risks in non-miners and in miners’ wives in coalmining communities. Possible causes have been suggested in diet, other lifestyle factors, coal-based local air pollution and infectious agents such as Helicobacter pylori. Whatever the cause of the excess, the quality of our exposure data, in analyses of the deaths contribution to the earlier excesses, has given us confidence that the cause is not occupational exposure to respirable dust or quartz ([3], [11]).

Now that death notification has been reactivated, death events will continue to be notified until the cohort is extinct, so there are opportunities for future analyses over even longer follow-up periods. Analyses relating cause-specific mortality to individual exposures to respirable dust and quartz will be reported separately.

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