British Journal of Industrial Medicine 1984;**41**:170–178

Long term mortality study of chromate pigment workers who suffered lead poisoning

JOAN M DAVIES

From the Division of Epidemiology, Institute of Cancer Research, Sutton, Surrey SM2 5PX, UK

ABSTRACT Long term mortality was studied in a group of 57 chromate pigment workers who suffered clinical lead poisoning, mostly between 1930 and 1945. One death was attributed to lead poisoning and there were significant excesses of deaths from nephritis (observed/expected 3/0·24) and cerebrovascular disease (9/2·20), as well as non-significant excesses for respiratory diseases (7/3·59) and accidents and violence (3/1·13). The deaths from nephritis followed long spells of service exceeding 10 years. Poisoning appeared to have more adverse long term effects on older workers: 15 men aged 40 or over at the time of acute poisoning experienced generally high mortality, and 30 years later or by the end of 1981 only two survived instead of the seven expected. The risk of cerebrovascular disease appeared to be unrelated to duration of exposure and affected even men employed for under one year. Excluding the 57 lead poisoned men, other contemporary workers at the factories showed no excess mortality from cerebrovascular disease.

Occupational lead poisoning has been a notifiable disease in the UK since 1896 and table 1 shows the number of cases notified to 1979. Fluctuations since about 1950 partly reflect changing criteria for notifications, which formerly related mainly to patients suffering acute poisoning with symptoms such as colic and some already having chronic disease (often terminal) when plumbism was first diagnosed. Since then more effective biochemical testing and medical supervision have facilitated earlier diagnosis, and notifications relate increasingly (though not exclusively) to early cases with mild symptoms or even asymptomatic cases with high lead absorption.[1,2]

The chronic ill effects of lead poisoning commonly take the form of nephropathy. In the early 1900s mortality from "Bright's disease" was four times higher than normal among lead workers, and was the main cause of death in nearly a third of fatal cases of notified lead poisoning.[3] Since the late 1950s there have been numerous reports of renal dysfunction and chronic nephritis after lead poisoning,[4–8] and epidemiological studies in the United Kingdom and United States have found excess mortality from chronic nephritis among heavily exposed smelter and battery workers.[9–11] Earlier official United Kingdom statistics had shown mortality from chronic nephritis in the lead exposed occupations of plumbing and painting to be twice as high as normal in 1921,[12] about a third higher in 1931,[13] and still a fifth higher in plumbers in 1951,[14] though normal in painters. In these occupations cerebrovascular disease (CVD) mortality was also raised by about half in 1921 and 1931,[12,13] though normal by 1951[14]; the Registrar General considered this excess to be attributable to the effects of lead,[13] and earlier, Legge and Goadby had expressed the view that cerebral haemorrhage was often a sequel to lead poisoning.[3] In 1963 a mortality study[15] of United Kingdom battery workers who had retired after at least 25 years' service showed a pronounced excess of deaths from CVD among the most heavily exposed (observed/expected (O/E) 24/9·3). In 1971 Malcolm reported a continuation of this study, analysing pensioners' deaths during 1963–7 by the proportional mortality technique.[16] Of 143 deaths, 29 were due to CVD, a figure close to Malcolm's "expected" estimate of 26, and this reported finding has often been cited[17–20] as evidence that deaths from CVD were

Table 1 *Numbers of occupational lead poisoning cases notified during 1896–1979**

Period	*All cases*	*(Fatal cases)*	*Period*	*All cases*	*(Fatal cases)*
1896–9	4780	(Unknown)	1940–9	578	(45)
1900–9	8655	(662)	1950–9	562	(1)
1910–9	5756	(594)	1960–9	814	(0)
1920–9	3601	(400)	1970–9	471	(1)
1930–9	1554	(194)			

*Including early cases among house painters and plumbers reported voluntarily.

Received 21 March 1983
Accepted 25 April 1983

no longer in excess among lead workers. In fact, however, the data appear to indicate a continuing excess, for the estimate of 26 expected deaths seems unduly high: it forms 18% of the 143 deaths, but national data[21] show that deaths from CVD did not reach such a high proportion of all deaths in any age group, and a more likely estimate might be about 13%, giving about 19 expected deaths compared with 29 observed. In 1982 Malcolm and Barnett re-examined mortality during 1925–76 among a total of 1898 pensioners using the man-years method, and found excess mortality from CVD in those with heavy lead exposure (O/E 43/33·69) which persisted into the 1960s.[10]

No other studies of lead workers' mortality have been published in Britain, nor any follow up of lead poisoned adults. A study of lung cancer in chromate pigment workers[22 23] is being extended to cover other diseases, and the opportunity has been taken to examine the mortality of a subgroup who suffered lead poisoning. Since this examination was initiated, an Australian study[24] has been published which analyses 140 long term deaths among lead poisoned smelter workers by the proportional mortality method, and finds severe excess mortality from chronic nephritis (20 deaths) and CVD (28 deaths).

Methods

The main study covers three factories (A, B, and C) making lead chromate pigments, which in recent decades were generally based on lead nitrate produced on site from metallic lead and nitric acid. Previously lead acetate had been more often used, and there had been some use of litharge. The most severe lead exposures usually occurred during the grinding or handling of the dried colours. No data are available on lead in air levels during the period when lead poisoning cases were occurring. Factories A and B also made zinc chromate pigments, with a resulting hazard from lung cancer.[22 23] Most jobs at the factories were semiskilled or unskilled, and the work was heavy and dirty. Labour turnover rates were high and when recruiting new operatives the managements could not be as selective as employers offering more attractive work.

The study nominal roll for factory A includes 578 men already working there in 1932 or entering employment during 1932 to mid-1974 and staying at least one year, and also 97 men entering during 1933–45 and remaining for 3–11 months only. Of these 675 men, 23 were recorded as suffering one or more episodes of lead poisoning notified to the authorities during 1922–45; they are numbered A1–A23 in the appendix table, which gives details of all 57 cases. Factory records identified a further six cases of clinical lead poisoning: A24 had suffered poisoning in 1913, and cases A25–A29 (although not notified) were recorded as having "lead intoxication" or "plumbism," or as showing a blue line on the gums as well as other signs and symptoms. In addition a series of 14 non-notified cases at the factory was described in 1954,[25] giving the men's initials and relevant dates. Six of the 14 were identified unequivocally as men on the study roll, and were added to the series; all suffered clinical poisoning, with haemoglobin concentrations dropping to 65–70% and other signs and symptoms. None of the 35 cases at factory A was fatal, but during the 1940s two other men died from certified lead poisoning in the form of chronic nephritis soon after leaving employment, without previous notified episodes of poisoning; their details are given at the end of the appendix table (cases AF1, AF2).

The study roll for factory B includes 222 men already there in 1948 or entering employment during 1948 to mid-1974 and staying at least one year. Eight men (B1–B8) suffered notified episodes of lead poisoning during 1939–59; no episode was immediately fatal, but cases B4 and B5 already had chronic nephritis when plumbism was first diagnosed, and died a few years later.

The study roll for factory C includes 180 men already there in 1946 or entering employment during 1946 to mid-1974 and staying at least one year. Seven men (C1–C7) suffered notified episodes of lead poisoning during 1939–68. Records listed 15 other notified cases among men excluded from the study roll because they left before 1946 or because they stayed less than a year; for seven (C8–C14) the names and other data recorded were sufficiently distinctive and detailed for the men to be identified and added to the present series; the durations of service of C8–C12 are not known. None of these 14 cases was fatal.

The three factories thus provided a total of 57 cases of non-fatal clinical lead poisoning. All 57 men were successfully traced as dead or as alive on 31 December 1981, and certified causes of death were ascertained. The 43 men at factories A and B were entered into observation at the earliest dates from which all records of workers had been retained (1933 at A, 1948 at B), or at the date of poisoning if later. All 14 men at factory C were entered into observation at the date of poisoning (however early) because they had been identified from contemporary records of notifications as distinct from the records used to compile the main study roll.

Man-years at risk were computed in the usual five year age groups and calendar periods, and totalled 1585 years. Each cause of death was coded by the ICD revision in force at the date of death; in the

Table 2 *Observed and expected deaths from lead related causes*

Cause	*ICD codes 9th rev*	*Observed deaths*	*Expected deaths*	*O/E ratio*
All causes	001–999	38	25·59	1·48*
Hypertensive disease	401–405	0	0·40	0·00
Cerebrovascular disease	430–438	9	2·20	4·10***
Nephritis, nephrosis	580–589	3	0·24	12·67**
Lead poisoning	E984	1	0·00	
All lead-related causes		13	2·83	4·58****

*p < 0·05; **p < 0·01; ***p < 0·001; ****p < 0·0001.

tables causes are described by 9th revision codes.[26] Expected deaths by cause at national rates were calculated using specially compiled death rates for quinquennia from 1931 to 1980; 1976–80 rates were used for 1981. One man (A30) died overseas on war service in 1944; because the rates excluded such deaths he was simply removed from observation at that date. The statistical significance of the differences between observed and expected deaths was assessed assuming a Poisson distribution. A one-tailed test was used for causes already suspect, hypertensive disease, CVD, nephritis and nephrosis, and lead poisoning (together termed "lead related causes"); a two-tailed test was used for other causes and for all causes combined.

Results

Observed and expected deaths for the whole group of 57 men are shown in table 2, for all causes and for the lead related causes. There were three deaths from nephritis, significantly more than the 0·24 expected (cases A2, A28, B4). In addition B5, whose death was attributed to lead poisoning, had granular nephritis, and the two excluded fatal cases (AF1, AF2) died from chronic nephritis. No deaths were attributed to hypertensive disease, but there was a highly significant excess of deaths attributed to CVD, with about two expected and nine observed (A7, 11, 16, 26, 31; B2, 7; C8, 13). Within this category the death certificate entries were varied, as shown in the appendix table. Observed/expected ratios by interval after poisoning were 2/0·41 during the period 0–19 years, 6/1·22 during 20–39 years, and 1/0·57 from 40 years onwards.

In table 3 mortality from the lead related causes is examined in subgroups of the 57 men divided according to five different variables. Significance tests have not been applied, and because of the small numbers it would be prudent to interpret the results as indicating merely that the excess deaths are not generally confined to any particular subgroups. The results by total duration of service (before and after poisoning) are of interest; all three deaths from nephritis occurred among men with at least five years' service, but the excess mortality from CVD affected even the seven men employed for under one year, three of whom died from strokes (A7, A16, C13). Possibly the very shortness of these seven men's service may be indicative of unusual sensitivity to the toxic effects of lead or unusually severe poisoning, or both.

Table 4 shows mortality from other causes. The total of observed deaths was close to that expected, but there were non-significant excesses for respiratory disease (mainly bronchitis) and for accidents and

Table 3 *Deaths from lead related causes among subgroups of 57 men*

Division	*Subgroup*	*No of men*	*Cerebrovascular disease Obs/Exp*	*Nephritis, nephrosis Obs/Exp*	*All lead related causes**	
					Obs/Exp	*O/E ratio*
By notification	Notified	45	7/1·73	2/0·18	10/2·22	4·50
	Not notified	12	2/0·47	1/0·06	3/0·61	4·92
By factory	Factory A	35	5/1·58	2/0·18	7/2·04	3·43
	Factory B	8	2/0·12	1/0·01	4/0·17	24·10
	Factory C	14	2/0·49	0/0·05	2/0·63	3·19
By date of poisoning	Before 1950	45	7/1·95	2/0·21	9/2·51	3·59
	1950 onwards	12	2/0·25	1/0·02	4/0·33	12·27
By duration of service†	<1 year	7	3/0·15	0/0·02	3/0·20	15·31
	1–4 years	11	2/0·29	0/0·03	3/0·39	7·79
	≥5 years	34	3/1·57	3/0·15	6/1·99	3·02
	Not known	5	1/0·16	0/0·02	1/0·21	4·74
By age at poisoning	< 30	20	3/0·83	0/0·09	3/1·07	2·81
	30–39	20	3/0·86	2/0·09	5/1·09	4·59
	≥ 40	17	3/0·50	1/0·06	5/0·67	7·47

*Including lead poisoning and hypertensive disease.
†With observation starting at the date of poisoning or five years after entry into service: the later of the two dates.

Table 4 *Observed and expected deaths from other causes*

Cause	*ICD codes 9th rev*	*Deaths*		*O/E ratio*
		Obs	*Exp*	
All causes other than lead related diseases		25	22·76	1·10
Cancer of the lung, trachea, etc	162–165	4	2·77	1·45
All other neoplasms	140–161, 170–239	3	3·72	0·81
Other circulatory diseases	390–398, 401–428, 440–459	7	9·04	0·77
Respiratory diseases	460–519	7	3·59	1·95
Accidents, poisonings (except lead), and violence	E800–983, 985–999	3	1·13	2·65
All remaining causes		1	2·52	0·40

violence, which included a suicide (A9), a works acident (A27), and a street fall (A21). There was no excess mortality from neoplasms other than cancer of the lung (known to be in excess at factories A and B); the other neoplasms were cancers of the stomach (A34) and colon (A32), and lymphatic leukaemia (A23). Case C14 had multiple myeloma mentioned as a contributory cause. Mortality from other circulatory diseases appeared normal, but it may be of interest that two men (A5, A7) had arteriosclerosis and the amputation of a leg mentioned on their certificates; of some 350 certificates for deaths among the other 1027 men on the main study roll, none mentioned an amputation, although one mentioned "gangrene of leg." Expected deaths were also calculated for infectious diseases, diseases of the digestive system, and all residual causes, but the only death in these groups was that of A24 in 1945 from a ruptured aortic aneurysm, classified as syphilitic disease under prevailing coding rules.

Mortality from these other causes was examined for each subgroup shown in table 3, and a pronounced relationship with age at poisoning was found, as shown in table 5. Even before the exclusion of the lead related diseases and the chromate related lung cancers there was a slight deficit of deaths among the men aged under 30 at poisoning, and after exclusion their mortality was noticeably low. Men aged 30–39 at poisoning showed an excess for all mortality, but after excluding these causes there was a slight deficit. By contrast, the men aged 40 or over at poisoning showed a pronounced overall excess of deaths which hardly diminished after exclusion of the causes specified, and the excess deaths from respiratory disease and accidents and violence occurred mainly in this group. Starting from entry into observation, and using interpolated cohort life table P_x values (R Bell and R A M Case, unpublished data), the numbers of expected survivors 30 years after poisoning were calculated for each age group, omitting cases A30 (the overseas war casualty) and B4 and B5, who had chronic poisoning at first diagnosis; for five men poisoned after 1951 values were calculated up to 31 December 1981. Of the 19 men under age 30, 16 were still alive 30 years later compared with 16·4 expected, and after 40 years there were still 13 survivors compared with 13·3 expected. Of the 20 men aged 30–39, 13 were still alive after 30 years, one or two fewer than the 14·5 expected. The 15 men aged 40 or over at poisoning fared worst: 20 years later only seven were still alive compared with 10·6 expected, and after 30 years (or by 31 December 1981) there were only two survivors instead of the 7·2 expected.

Given the excess mortality from certain causes among the lead poisoned men, the question arises whether mortality from these causes was also raised among the other 1027 men comprising the main study population. Pending full analysis some preliminary data are shown in table 6, distinguishing

Table 5 *Mortality from other causes by age at poisoning*

Cause	*Age at poisoning*					
	< 30		*30–39*		*≥ 40*	
	Obs/Exp	*Ratio*	*Obs/Exp*	*Ratio*	*Obs/Exp*	*Ratio*
All causes	9/10·05	0·90	13/9·76	1·33	16/5·78	2·77**
Cancer of the lung, trachea, etc	2/1·08	1·85	2/1·04	1·92	0/0·64	0·00
All causes other than lung cancer and lead related diseases	4/7·90	0·51	6/7·63	0·79	11/4·47	2·46*
Respiratory diseases	2/1·35	1·49	1/1·40	0·71	4/0·84	4·75
Accidents, poisonings (except lead), and violence	0/0·52	0·00	1/0·39	2·58	2/0·22	9·09
No of men/man-years	20/749		20/547		17/289	

*p < 0·05; **p <0·01.

Table 6 *Deaths from certain causes in the main study population*

Cause	All men in the main study Obs/Exp	Men in late cohorts Obs/Exp	Men in early cohorts		
			All men Obs/Exp	Men with lead poisoning† Obs/Exp	Other men Obs/Exp
Cerebrovascular disease	31/33·05	1/6·52	30/26·53	7/1·74**	23/24·79
Nephritis, nephrosis	5/3·16	1/0·57	4/2·59	3/0·22**	1/2·37
Respiratory diseases	60/50·49	11/9·55	49/40·94	6/3·25	43/37·69
Lead poisoning	3/0·00	0/0·00	3/0·00	1/0·00	2/0·00
Suicide	4/4·46	0/1·21	4/3·25	1/0·27	3/2·98
Other accidents, poisonings, and violence	10/13·50	1/4·26	9/9·24	2/0·72	7/8·52
No of men	1077	472	605	50	555
Man-years	25,795	8,017	17,778	1,394	16,384

†Including case C7, who suffered poisoning in 1968.
**$p < 0·01$.

results for "early" cohorts of men entering employment while lead poisoning cases were occurring and "late" cohorts entering after 1945 at factory A and after 1960 at factories B and C, when notified plumbism had become rare; the seven lead poisoned men not on the main study roll are excluded. For CVD a normal result for the whole population conceals a pronounced deficit in the late cohorts and a small excess in the early cohorts, which in turn conceals a severe excess in the lead poisoned men and a normal result for the other early workers. For nephritis the numbers are small, but the overall excess can be seen to derive from the high mortality among the lead poisoned men. Cases AF1 and AF2 died from chronic nephritis, but this was recognised (and certified) as due to lead poisoning. The excess of deaths from respiratory disease in the lead poisoned men reflects a smaller excess found generally among the early workers. For both suicide and other accidental and violent deaths mortality appears high in the lead poisoned men, normal in the other early workers, and low in the late cohorts. The three suicides among the other early workers all occurred among the 207 men with at least ten years' service, giving O/E values of 3/0·01 for this subgroup.

Discussion

Although based on small numbers, this study covers a substantial proportion of the cases of lead poisoning that have occurred in the manufacture of lead chromate pigments in England, and the cases were selected objectively on the basis of records from the time of poisoning; for 45 men the records were of official notifications. The other 12 men had less severe clinical symptoms and did not have sickness absence certified as due to lead poisoning, but their results were similar and in more recent years such cases would probably have been notified; doubtless some other borderline cases at all three factories have remained unidentified. Information was not generally available on the men's smoking habits, nor on other jobs they held.

As in other studies[9–11 24] there were excess deaths from nephritis. The results support the view[27] that severe chronic nephropathy does not usually occur unless there has been prolonged heavy exposure, and it is not clear to what extent episodes of acute clinical poisoning may exacerbate the long term risk. Cases A2 and A28 had suffered acute episodes during long periods of service, but B4, AF1, and AF2 already had advanced or terminal chronic nephritis at first diagnosis, after long periods of exposure free from any notified episodes of plumbism. Lane reported deaths from chronic renal disease among long service men who had not necessarily suffered toxic episodes[9]; the study of smelter and battery workers in the United States found excess deaths from nephritis (O/E 7/2·80 and 14/8·58) but contained no analyses by degree of duration of exposure or past occurrence of episodes of poisoning. The Australian smelter workers studied[24] had mostly had severe exposure in the 1920s and early 1930s, and during 1930–49 chronic nephritis accounted for 37% (12/32) of subsequent deaths among those men who had suffered notified episodes of poisoning and 8% (13/165) of deaths among the other workers; even in 1965–77 the proportion of deaths from chonic nephritis was still about three times higher than normal in both groups.

In view of findings elsewhere[10 12 13 15 16 24] the excess of deaths from CVD among the 57 men is not unexpected; but the results throw some fresh light on this risk, and suggest that an episode of acute clinical lead poisoning may render a man more likely to die from this cause even in the absence of long exposure indeed, the data in table 3 suggest that the risk was especially severe for the seven men with less than one year's service. Mortality was "normal"

among the other 555 early workers (O/E 23/24·79), and was not associated with duration of service. It might be argued that mortality from this cause should be lower than "normal" on account of the healthy worker effect, but this effect depends on the selectiveness of the workforce, and as explained the unattractiveness of this work would reduce selectiveness. Moreover a few of the 23 deaths concerned may possibly have occurred among unidentified cases of plumbism. This result for the other early workers suggests that prolonged exposure without any episode of clinical poisoning did not give rise to a risk of CVD in the English factories, or that any such risk was slight. In the Australian lead poisoned workers the excess of CVD related mainly to deaths certified as due to cerebral haemorrhage, and the proportion of these was raised at least sevenfold in 1930–64, and was still doubled in 1965–77. By contrast with the English findings, mortality from cerebral haemorrhage was also raised among the other smelter workers up to 1964. No excess mortality from other circulatory diseases were found in the other studies[10 11 24] or the present one.

Three deaths from accidents and violence occurred among the 57 men instead of about one expected, including the suicide of case A9 after 17 years' service; there were more suicides than expected among other early workers with ten or more years' service (O/E 3/1·01). The United States study[11] found significantly raised mortality from accidental and violent causes among smelter workers (O/E 59/40·52), and a table giving crude (all ages) proportional mortality values showed that 4% of their deaths were attributed to suicide instead of about 1·5% expected. These proportions represent some 13 observed deaths instead of about five, but there is no textual comment on this finding. Death rates from accidents and suicide in heavily exposed lead workers may merit further research.

The analyses by age suggest that although clinical lead poisoning at any age is associated with raised mortality from nephritis and CVD, it does not otherwise shorten life in men aged under 40. In older men there may be a more general association with increased or accelerated mortality. The men aged 40 and over suffered premature deaths from CVD, suicide, bronchitis, and also neoplasms, for cases A23 and A32 were aged only 48 and 55 when they died from leukaemia and cancer of the colon seven years after poisoning. Although there was no overall excess of cancer deaths in the 57 men (O/E 7/6·49), the mortality ratios were 6/1·57 during the first 20 years after poisoning, and only 1/4·92 thereafter; the possibility that lead poisoning accelerated these deaths cannot be excluded.

If lead poisoning is associated with generally high mortality in older men the association need not necessarily be entirely causal. Findings by Brown *et al* suggest that lead workers who smoke (especially if they smoke heavily) absorb more lead than non-smokers, either because of contamination of their cigarettes or because of impaired lung clearance mechanisms.[28] Older men who smoke heavily are therefore possibly more likely than others to suffer lead poisoning, and a high proportion of heavy smokers among the affected men might account for the excess of deaths from bronchitis. No indication was found that older men were generally more likely to suffer lead poisoning than younger men.

The excess mortality from nephritis and CVD found in this study is unlikely to be a chance finding, but because of small numbers the other results should be regarded as provisional findings that need testing in larger long term studies of lead poisoned workers. In 1979 Fox reported that a joint national study was being undertaken by the Employment Medical Advisory Service (EMAS) and the Office of Population Censuses and Surveys to examine subsequent mortality among notified cases of lead poisoning and other industrial diseases, though difficulties in identification and follow up were being experienced.[29] No detailed studies of lead workers' mortality have been published; the results reported here give no reason to suppose that current or recent workers employed in well controlled conditions have raised mortality, but further industry based studies are needed which will carefully distinguish cohorts of lead workers exposed at different dates. The long term study initiated by EMAS in 1973[30] will also provide data in due course.

The findings of this study cannot be regarded as relevant only to the past, for excess deaths are still occurring among the 57 workers, and it may be seen from table 1 that these men account for only about 2% of all those notified as suffering occupational lead poisoning during 1930–59. Fresh cases of poisoning continue to be notified from various industries, and not all such cases are mild. The official statistics of fatalities shown in table 1 understate the deaths attributable to lead poisoning because they take no account of delayed mortality.

The main study of chromate pigment workers is sponsored by the Association of European Manufacturers of Lead Chromate Pigments, and was made possible by the wholehearted cooperation of the management of the three factories. The study uses a computer program developed jointly with R Tweed; Miss Jean Miller typed the manuscript. The Institute of Cancer Research receives support from the Medical Research Council and the Cancer Research Campaign.

Appendix table Details of the 57 lead poisoning cases

Case No	Date(s) of LP Age at 1st LP	Period of employment	Alive (A) or dead (D) (age)	Certified cause of death
A1	1922 23	18 years 1919–37	A. 1981 (83)	—
A2	1922 32	18 years* 1919–38	D. 1940 (50)	Anasarca due to subacute nephritis Coroner's necropsy
A3	1929, 1931 26	41 years 1926–68	D. 1976 (73)	1 (a) Acute congestive heart failure (b) Chronic myocarditis (c) Chronic bronchitis
A4†	1931 27	20 years 1931–51	A. 1981 (77)	—
A5†	1933 25	4 years 1932–36	D. 1981 (72)	1 (a) Bronchopneumonia 2 (a) Arteriosclerosis. Amputation of leg
A6	1934, 1945 27	17 years 1928–46	A. 1981 (75)	—
A7	1934 25	5 months 1934–35	D. 1970 (61)	1 (a) Bronchopneumonia (b) Cerebrovascular accident (c) Atherosclerosis 2 Amputation leg
A8	1936, 1938 27	6 years 1933–39	A. 1981 (73)	—
A9	1936 40	17 years 1934–52	D. 1952 (57)	Shock after multiple compound fractures caused by jumping in front of a train while the balance of his mind was disturbed. Inquest
A10	1936, 1944 40	23 years* 1915–44	D. 1967 (71)	1 (a) Cardiac failure (b) Coronary thrombosis (c) Coronary atherosclerosis Coroner's necropsy
A11†	1936 26	4 years 1933–38	D. 1976 (66)	1 (a) Subarachnoid haemorrhage (b) Rupture of right internal carotid artery aneurysm Coroner's necrospsy
A12	1936 34	1 year 1936–38	D. 1944 (43)	Chronic pleurisy. Hypertrophy of heart Coroner's necropsy
A13	1937 37	2 years 1936–38	D. 1962 (62)	1 (a) Angina pectoris (b) Ischaemic heart disease
A14	1937 29	6 months 1937	D. 1974 (66)	Ischaemic heart disease
A15	1937 23	6 months 1937–38	D. 1961 (46)	Coronary occlusion due to atheroma Coroner's necropsy
A16	1937 35	7 months 1937–38	D. 1971 (69)	Cerebral thrombosis
A17	1938 30	3 years 1936–39	A. 1981 (74)	—
A18	1938 33	5 years 1934–39	A. 1981 (76)	—
A19	1939 26	3 years 1939–43	A. 1981 (68)	—
A20	1939 26	7 years 1939–47	A. 1981 (68)	—
A21	1940, 1944 41	9 years 1939–49	D. 1967 (68)	1 (a) Pulmonary embolism (b) Femoral thrombophlebitis (c) Pulmonary contusions and fractured ribs. Fall on level on street. On way from work. Inquest
A22	1943 42	3 years 1939–43	D. 1971 (70)	Myocardial infarction
A23	1945 40	6 years 1939–45	D. 1952 (48)	Lymphatic leukaemia
A24	1913 35	27 years* 1906–41	D. 1945 (66)	Ruptured aortic aneurysm Coroner's necropsy
A25	1934 24	12 years 1933–46	D. 1950 (39)	Carcinoma of bronchus
A26	1937, 1939 44	1 year* 1937–40	D. 1957 (64)	1 Right cerebral haemorrhage 2 Coronary thrombosis. Bronchopneumonia
A27	1938 34	9 years 1931–40	D. 1940 (37)	1 Cerebral contusions 2 Fractured skull caused through being accidentally caught in a machine, while at work Inquest
A28	1939 31	40 years 1932–73	D. 1976 (68)	1 (a) Uraemia (b) Chronic nephritis 2 Ischaemic heart disease

A29	1940 38	5 years 1940–45	D. 1981 (79)	1 (a) Bronchopneumonia (b) Carcinoma of lung Coroner's necropsy
A30	1936 27	3 years 1936–39	D. 1944 (35)	(Overseas war death)
A31	1936 39	46 years* 1913–63	D. 1973 (77)	1 (a) Cerebrovascular accident (b) Arteriosclerosis (c) Senility
A32	1936 48	13 years 1930–43	D. 1943 (55)	1 Bronchopneumonia after 2 Carcinoma of the pelvic colon and 3 Secondary deposits in the lungs and liver Coroner's necropsy
A33	1938 32	8 years 1929–38	D. 1973 (68)	1 (a) Bronchopneumonia (b) Congestive heart failure
A34	1939 30	5 months 1939	D. 1956 (46)	1 (a) Carcinoma of stomach
A35	1940 50	15 years 1940–55	D. 1963 (73)	1 (a) Bronchopneumonia (b) Chronic bronchitis 2 Congestive cardiac failure
B1	1939, 1944 37	27 years 1938–56	D. 1956 (54)	1 (a) Carcinoma of bronchus
B2	1944 44	45 years 1922–67	D. 1970 (69)	1 (a) Bronchopneumonia (b) Cerebral arteriosclerosis
B3	1951 35	≥30 years 1946—in employ 1976	A. 1981 65	—
B4	1952 46	11 years 1941–52	D. 1954 (48)	Hypertensive cardiac failure due to subacute nephritis Inquest
B5	1954 57	2 years 1952–54	D. 1959 (62)	Coronary occlusion due to atherosclerosis of both coronary and aortic vessels and granular nephritis. Died from industrial disease of lead poisoning. Inquest
B6	1952, 1956 34	15 years 1946–61	A. 1981 (64)	—
B7	1955 38	11 years 1954–72	D. 1981 (64)	1 (a) Cerebral haemorrhage 2 Hereditary haemorrhagic telangiectasis
B8	1959 34	2 years 1957–59	A. 1981 (56)	—
C1	1937 33	14 years 1937–52	A. 1981 (77)	—
C2	1937 26	14 years* 1930–52	D. 1957 (45)	1 (a) Carcinoma of bronchus
C3	1940 28	≥42 years 1929—in employ 1976	A. 1981 (69)	—
C4	1951 43	12 years* 1945–59	A. 1981 (73)	—
C5	1954 45	28 years* 1939–73	D. 1973 (64)	1 (a) Cardiac failure (b) Bronchitis acute on chronic
C6	1957 45	11 years 1946–57	D. 1968 (55)	1 (a) Cardiac infarction (b) Aortic valve disease
C7	1968 55	≥10 years 1966—in employ 1976	D. 1979 (66)	1 (a) Bronchopneumonia (b) Acute on chronic bronchitis Coroner's necropsy
C8	1935 19	Not known	D. 1971 (55)	1 (a) Cerebrovascular accident (b) Benign hypertension 2 Myocardial infarction
C9	1939 27	Not known	A. 1981 (70)	—
C10	1939 21	Not known	A. 1981 (63)	—
C11	1940 34	Not known	A. 1981 (76)	—
C12	1941 18	Not known	A. 1981 (59)	—
C13	1958 59	4 months 1958	D.1965 (64)	1 (a) Cerebral haemorrhage (b) Hypertension Coroner's necropsy
C14	1958 49	6 months 1958	D. 1981 (72)	1 (a) Bronchopneumonia 2 Multiple myeloma
AF1	—	12 years 1925–37	D. 1940 (33)	1 Chronic nephritis with uraemia and 2 Arteriosclerosis due to 3 Chronic lead poisoning Inquest
AF2	—	17 years 1923–40	D. 1941 (44)	Uraemia due to chronic nephritis due to lead poisoning Inquest

*Indicates broken service.
†A4, A5, and A11 were brothers.

References

[1] Ministry of Labour. *Annual report of the chief inspector of factories on industrial health, 1962.* London: HMSO, 1963.

[2] Bonnell JA. Lead smelting in Avonmouth. *Br J Ind Med* 1973;**30**:199–201.

[3] Legge TM, Goadby KW. *Lead poisoning and lead absorption.* London: Edward Arnold, 1912:58–60.

[4] Danilovic V. Chronic nephritis due to ingestion of lead-contaminated flour. *Br Med J* 1958;i:27–8.

[5] Radosevic Z, Saric M, Beritic T, Knezevic J. The kidney in lead poisoning. *Br J Ind Med* 1961;**18**:222–30.

[6] Lilis R, Gavrilescu N, Nestorescu B, Dumitriu C, Roventa A. Nephropathy in chronic lead poisoning. *Br J Ind Med* 1968;**25**:196–202.

[7] Emmerson BT. Chronic lead nephropathy. *Kidney Int* 1973;**4**:1–5.

[8] Baker EL Jr, Landrigan PJ, Barbour AG, *et al.* Occupational lead poisoning in the United States: clinical and biochemical findings related to blood lead levels. *Br J Ind Med* 1979;**36**:314–22.

[9] Lane RE. The care of the lead worker. *Br J Ind Med* 1949;**6**:125–43.

[10] Malcolm D, Barnett HAR. A mortality study of lead workers 1925–76. *Br J Ind Med* 1982;**39**:404–10.

[11] Cooper WC, Gaffey WR. Mortality of lead workers. *JOM* 1975;**17**:100–7.

[12] Registrar-General. *Decennial supplement. England and Wales 1921.* Part II. *Occupational mortality, fertility, and infant mortality.* London: HMSO, 1927: 26, 29, 57.

[13] Registrar-General. *Decennial supplement. England and Wales 1931.* Part IIa. *Occupational mortality.* London: HMSO, 1938: 233, 244.

[14] Registrar-General. *Decennial supplement. England and Wales 1951.* Part II. *Occupational mortality.* Vol 2: *Tables.* London: HMSO, 1958: 78, 114.

[15] Dingwall-Fordyce I, Lane RE. A follow-up study of lead workers. *Br J Ind Med* 1963;**20**:313–5.

[16] Malcolm D. Prevention of long-term sequelae following the absorption of lead. *Arch Environ Health* 1971;**23**:292–8.

[17] Waldron HA, Stofen D. *Sub-clinical lead poisoning.* London: Academic Press, 1974: 107.

[18] Baloh R. Neurological and behavioural toxicology of increased lead absorption. In: Carnow BW, ed. *Health effects of occupational lead and arsenic exposure.* (A symposium.) Washington: US Department of Health, Education, and Welfare, 1976.

[19] Bridbord K. Epidemiology of lead exposure among occupational groups. In: Carnow BW, ed. *Health effects of occupational lead and arsenic exposure.* (A symposium.) Washington: US Department of Health, Education, and Welfare, 1976.

[20] Shannon HS, Williams MK, King E. Sickness absence of lead workers and controls. *Br J Ind Med* 1976;**33**:236–42.

[21] General Register Office. *The Registrar General's Statistical Reviews of England and Wales for the years 1963–1967.* Part I. *Tables, medical.* London: HMSO, 1965–8.

[22] Davies JM. Lung-cancer mortality of workers making chromate pigments. *Lancet* 1978;i:384.

[23] Davies JM. Lung cancer mortality among workers making lead chromate and zinc chromate pigments at three English factories. *Br J Ind Med* 1984;**41**:158–69.

[24] McMichael AJ, Johnson HM. Long-term mortality profile of heavily-exposed lead smelter workers. *JOM* 1982;**24**:375–8.

[25] Goldblatt MW. Research in industrial health in the chemical industry. *Br J Ind Med* 1955;**12**:1–20.

[26] *International Classification of Diseases.* 9th rev. Geneva: WHO, 1977.

[27] Malcolm D. The effects of lead on the kidney. *Transactions of the Society of Occupational Medicine* 1970;**20**:50–3.

[28] Brown CP, Spivey GH, Valentine JL, Browdy BL. Cigarette smoking and lead levels in occupationally exposed lead workers. *J Toxicol Environ Health* 1980;**6**:877–83.

[29] Fox AJ. The role of OPCS in occupational epidemiology: some examples. *Ann Occup Hyg* 1979;**21**:393–403.

[30] Department of Employment. Employment Medical Advisory Service. *A report of the work of the service for 1973 and 1974.* London: HMSO, 1975.