

A mortality study of lead workers 1925-76

D MALCOLM AND H A R BARNETT

From the Medical Department, Chloride Group, Eastleigh, Hants SO5 5RR, and Rodney Barnett and Co, consulting actuaries, London SW1P 3LR, UK

ABSTRACT The principal causes of mortality of 754 individuals from a population of 1898 pensioners from four lead acid battery factories during the period 1 January 1925 to 31 December 1976 were studied. In addition the causes of 553 deaths occurring before retirement was also studied. All subjects were placed into one of three groups according to their history of lead exposure. Group 1 had no occupational lead exposure, group 2 low, and group 3 the highest. Mortality was studied in 16 principal disease groups as well as all causes combined. A significant excess of deaths from cerebrovascular accidents was found among pensioners dying between 1925 and 1976 in group 3 but not among men in the same exposure group dying in employment. There was also a significant excess of deaths from renal disease among this group. After 1958, however, the causes of most of the deaths from renal disease were not those likely to be associated with exposure to lead. There was no significant excess of deaths from all causes, nor was there a significant excess of deaths in any of the exposure groups from cancer, hypertensive disease, nor any other circulatory disease. There was no excess of observed to expected deaths among any of the three groups of women in any of the cause groups examined. After the introduction of regular blood lead analysis in 1964 the blood lead distribution in men in group 3 had by 1976 come down to roughly the same level as group 2 in 1965-7. Since there was no excess of deaths in any of the 16 categories studied among group 2 subjects present conditions are probably now adequate to prevent any excess of mortality in any of the three groups.

Symptoms of occupational lead poisoning are now relatively rare in Britain, most cases occurring among men cutting old lead contaminated steel with oxyacetylene torches. It is important to ensure that control methods also prevent any long-term effects, even after a working lifetime of exposure.

Early writers found an excess of renal failure due to chronic interstitial nephritis,¹ but the evidence of an excessive incidence of hypertension is not convincing.² Dingwall-Fordyce and Lane³ showed a significant excess of cerebrovascular accidents among a large group of past lead workers with long-term exposure. The original purpose of that paper was to see if there was any excess of cancer since Zollinger⁴ and Boyland *et al*⁵ had found an excessive incidence of renal tumours in rats injected with lead compounds. It was thought worth while to bring the study of Dingwall-Fordyce and Lane³ up to date to see whether improving conditions have succeeded in eliminating long-term effects.

Study populations

The populations studied consisted of 1898 pensioners (1644 men, 254 women) from four lead acid battery companies followed up from 1 January 1925 to 31 December 1976, representing 13 865 men years experience as pensioners. The dates of first employment of the pensioners ranged from 1892 to 1971. The two largest companies also had secondary lead smelting operations. The death certificates of those who died during the study period were mostly obtained from the company pension scheme, but where certificates were not available from this source they were obtained from Somerset House. The death certificates for 23 men and one woman of this population were not traced. The size of the four companies was not constant over the years but about three-quarters of the employees were in the three factories in the north west and one quarter in the south east.

Pensioners were long-term employees who received a company pension at the age of 65 for men and 60 for women and who had, before 1961, a

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combined score of age plus service of 95 years, or 90 years for women. After 1961 the scheme became contributory, and there was no minimum service before receiving a pension, although in fact most people who became pensionable had 25 years' or more service.

Labour turnover in the factories was normally low, at about 4% a year including retirements, so that the population was unusually stable. In addition, the company tried wherever possible to find suitable work for employees whose health made them unable to continue their normal jobs. These conditions would tend to minimise the healthy worker effect.

Those with over 15 years' service and who were unable to continue employment even on light work would be kept on the employment register and reviewed after one year. Those who were unlikely to return to gainful employment would then be retired early on pension so that they would then appear in the pensioned population. No adequate records are available for employees with under 15 years' service who left the company to find other employment.

In addition, at the largest of the four factories, a list had been kept of all 553 employees who had died while still in employment. Unfortunately, the data giving the size of the working population were incomplete, so for this group the proportional mortality was determined.

Method

Originally the data for examination closed with the year 1974, and the post-war range of 1947–74 were divided into quadrennial periods. Actuaries have found that quadrennial periods are useful for this type of investigation since “the 1949–52 Table of Mortality of Assured Lives” was compiled. The reason, given in Benjamin and Pollard,⁶ is that it has been observed to be unusual to find more than one year with heavy mortality in any four-year period, while the use of shorter periods would show results difficult to interpret due to year-to-year fluctuations caused by epidemics or climatic conditions. Nevertheless, for the data under discussion it was necessary to examine the mortality over 1925–46 in somewhat longer periods due to the comparatively small amount of data, while a two-year period, 1975 and 1976, was separately investigated in order to cover the additional years which had become available since the work started.

It is appreciated that it has been customary to use quinquennial periods in epidemiological studies, but as the Registrar General's published figures are in *annual* form the final results are not affected by how many years are combined into a period, other than by the considerations of the previous paragraph.

The use of quadrennial periods of observation should not be confused with the continued use of quinquennial age groups; the questions underlying the choice of periods of experience do not apply to age groupings, which have to follow the groupings used in the Registrar General's statistics. The “exposed to risk” since retirement—that is, the number of years of life observed at each age separately for the two sexes—were first calculated from the experience and recorded in quinary age groups to be comparable with the published national experience. For this calculation it was assumed that men retiring at 65 and women at 60 had retired on their birthdays but that retirement at any other ages were evenly spread over the year of age. In those cases where the age was not recorded it was assumed that men retired at 65 and women at 60: the numbers were not large enough for this assumption to cause errors of appreciable magnitude in those exposed to risk.

The observed deaths were counted in similar age groups and divided into 16 “cause of death” groups together with an additional group for “cause of death unknown,” comprising those deaths where particulars from death certificates had not been recorded. The causes of death were coded according to the eighth revision of the manual of the *International Statistical Classification of Diseases, Injuries and Causes of Death*.⁷

The control experience used was the national experience of England and Wales of the Registrar General's Annual Review.⁸ The eighth revision coding came into effect in 1968, but it was possible to translate earlier data to the new coding.

The expected deaths, according to national mortality rates, were calculated by multiplying the exposed to risk in each age group and quadrennial period by the appropriate national age in cause-specific rate of mortality for the same period. These represent the deaths in each subgroup that would have arisen if the pensioners and the national experience had been identical.

The only complication here arose from those cases where the death certificates had not been sighted; in the subgroups where this occurred a proportionate reduction was made in the expected deaths in each cause group, leaving the balance to be assigned to cause unknown. This is tantamount to assuming that such cases were evenly spread over the different cause groups in the same proportions as the known cases; a reasonable assumption so long as non-production of the certificate was not correlated with cause of death.

Lead exposure

Over the years, various methods of monitoring the health and absorption of lead have been used.

Before 1927 the principal method was monthly clinical screening that included the early detection of significant symptoms of lead poisoning such as anaemia, pallor, Burtonian line, colic, and wrist drop.

In 1927 punctuate basophil counting was started, supported by haemoglobin estimations. This continued as the principal control with the addition of urinary leads on a small scale in the late 1930s and on a regular basis in 1952.

In 1964 facilities for lead in blood analysis became available for all the lead workers, and this has proved much the most reliable method of controlling absorption, especially in the absence of clinical signs. Because of changes in control methods as well as improved working conditions over the years, it has not been possible to provide a clear-cut lead exposure index for individuals. It was therefore considered best to continue to allocate subjects to the three groups used by Dingwall-Fordyce and Lane.³ This has the merit of keeping the numbers high in each group.

The groups were: (1) no occupational lead exposure, (2) light occupational lead exposure, and (3) high occupational lead exposure. People were placed in the various groups according to their principal occupation at work. The allocation to one of the three groups was carried out before listing mortality experience. When an individual had worked in groups 2 and 3 they were allocated to the group in which they had spent the longest period. In cases of roughly equal exposure the higher exposure group was selected. There was no change from group 1 to other groups. Group 1 included all social classes and group 2 principally social class IV with about 25% of social class IIIM; those in group 3 are all social class IV (table 1). While previous methods of lead estimation lacked the precision to make comparisons of absorption possible the use of blood lead data makes such a comparison feasible. The blood lead distributions of categories 2 and 3 for 1965-6 and 1975-6 are compared in figures 1 and 2. This clearly shows a pronounced improvement that has resulted from better information, and the consequent improvement in environmental control. Nevertheless, the relative ranking of exposure related to the various departments has remained much the same, although the reduction in exposure has been greatest in those departments that were formerly the highest.

Results

DEATHS FROM ALL CAUSES

There was no significant excess mortality from all causes combined in any of the three groups of pensioners; observed deaths among men 754, expected

Table 1 *Grades of exposure to lead*

<i>Group 1</i> <i>No occupational lead exposure</i>	<i>Group 2</i> <i>Light occupational lead exposure</i>	<i>Group 3</i> <i>High occupational lead exposure</i>
General office	Casting	Oxide mills
Chemist	General stores	Battery assembly
Power house	Charging	Plate cutting
Stokers	Works offices	Forming
Sales	Internal transport	Pasting and mixing
Joiners	Mechanics shop	Plumbers
		Lead recovery
		Packers
		Tube filling (All other lead operations not included in 2)

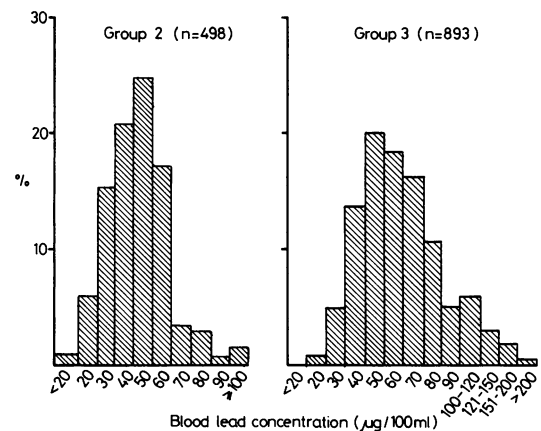


Fig 1 *Blood lead distribution 1965-6.*

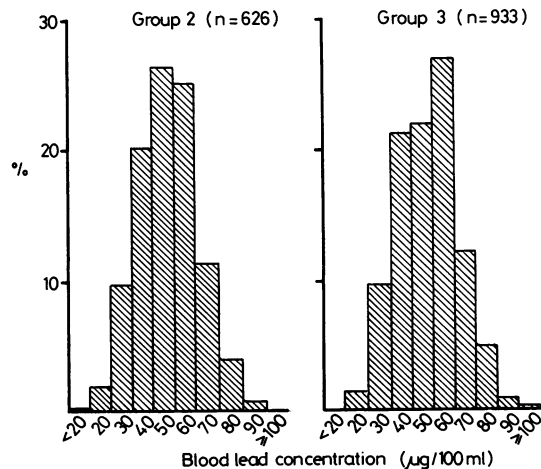


Fig 2 *Blood lead distribution 1975-6.*

Table 2 *Deaths from all causes*

Male pensioners group	Observed	Expected
1	212	244.01
2	258	254.60
3	284	265.86*

*p = 0.134.

Table 3 *Deaths due to cerebrovascular accidents 1925-76 (430-438) (compared with Dingwall-Fordyce and Lane³ (1925-61) in parentheses)*

Pensioners group	Observed	Expected
1	30 (6)	30.84 (7.9)
2	30 (6)	31.54 (3.4)
3	43 (24)	33.69* (9.3)†

*p = 0.055.

†p < 0.001.

deaths 764.47. Among male pensioners in group 3 there were 284 deaths observed compared with 265.86 expected, which is not significant (table 2).

The observed deaths occurring among women pensioners and those dying in service do not show a significant excess either overall nor in any of the 16 categories examined. Because women were prohibited from several high exposure occupations the number in category 3 is small. The data in the tables are therefore all based on analysis of the male population except for neoplastic diseases.

CEREBROVASCULAR ACCIDENTS

Table 3 shows no excess mortality from cerebrovascular accidents in groups 1 or 2. There is an excess in group 3, but this is not statistically significant (p = 0.055). More detailed examination of group 3 shows that most of this excess occurs in the 65-69 group where 18 deaths were observed compared with 7.5 expected, which is statistically significant (p < 0.001). In the 70 and over age group 25 deaths were observed compared with 26.19 expected. By comparison with Dingwall-Fordyce and Lane³ the overall excess of observed to expected deaths among group 3 pensioners is less for the whole period than in the 1925-61 period (table 3). The excess of deaths from cerebrovascular accidents (430-438) appears to have persisted into the 1960s, the last death of a pensioner in group 3 from a cerebrovascular accident occurring in 1969. Five men in the group showing excess mortality were first exposed to lead at work during 1940-43, while the other 13 men were first exposed between 1915 and 1932. The average duration of exposure was 36 years for 16 of these men and unknown for the other two.

Among the men dying in service, groups 1 and 2 showed no excess of observed against expected deaths. In group 3 there were 15 deaths observed compared with 13.48 expected, but this is not statistically significant.

HYPERTENSIVE DISEASE

Table 4 shows no excess of deaths from hypertension among men whether pensioners or deaths in service. None of the three categories showed any excess mortality due to hypertension. Only one death from hypertension was recorded among the women.

RENAL DISEASE

There is an excess of deaths due to renal diseases (p = 0.007) in groups 2 and 3 (table 5). Because most of the deaths in the years after 1958 were of an infective nature, three due to pyelonephritis and one to renal abscess, it was considered worth while to separate out nephritis and nephrosis (580-584) as being most likely to be associated with long-term lead exposure.

Table 6 shows that seven deaths occurred compared with 3.41 expected, which is of borderline significance at the 5% confidence level. The excess in 1945-6 was significant at the 5% level and in 1947-58 was just short of significant. The last two periods were not significant.

NEOPLASTIC DISEASE

There was no overall excess of deaths from malignant disease (tables 7 and 8). There was no significant gradient with lead exposure among pen-

Table 4 *Deaths from hypertensive disease (400-404)*

	Observed	Expected
Pensioners for all 3 groups	12	15.42
Deaths in service (all 3 groups)	20	23.21

Table 5 *Deaths due to renal disease (580-593)*

Pensioners group	Observed	Expected
1	1	2.41
2	6	2.60
3	5	2.71

Table 6 *Deaths due to nephritis and nephrosis (580-584) in groups 2 and 3*

	1925-34	1935-46	1947-58	1959-70	1971-76
Observed	0	2	2	1	2
Expected	0.02	0.49	0.73	0.98	1.19

Table 7 *Deaths after retirement due to neoplastic diseases (140-239)*

Men			Women		
Group	Observed	Expected	Group	Observed	Expected
1	42	50.60	1	4	7.71
2	62	53.88	2	2	2.18
3	53	54.98	3	1	1.53
Total	157	159.46		7	10.94

Table 8 *Deaths in service due to neoplastic diseases (140-239)*

Men			Women		
Group	Observed	Expected	Group	Observed	Expected
1	26	25.58	1	10	7.25
2	47	44.17	2	3	2.04
3	*63	48.58	3	5	4.79
Total	136	118.33		18	14.08

* $p = 0.020$.

sioners and the incidence of malignant disease. There is a small excess of observed over expected deaths in men dying in service. A more detailed analysis found an excess of malignant neoplasms of the digestive tract among men in group 3 dying in service, with 21 observed against 12.56 expected deaths ($p = 0.009$). This excess mortality, however, was almost entirely confined to the period 1963-6. The women pensioners show less cancer than expected, but the numbers are small, especially for those exposed to lead. The small excess of cancer among women dying in service is due to the excess in those not exposed to lead.

BRONCHITIS

Among the pensioners groups 1 and 2 showed no excess of deaths from bronchitis. In group 3 men, 31 deaths were observed compared with 22.94 expected, an excess of eight deaths ($p = 0.047$). Men dying in service do not show any excess.

OTHER CATEGORIES

None of the five groups of diseases examined—namely, other respiratory diseases, digestive diseases, infective or parasitic diseases, other circulatory diseases—nor all other causes showed any significant excess in any of the three lead exposure groups, whether among pensioners or those dying in service, neither men nor women.

Discussion

Because methods of estimating lead absorption have varied over the years, it has not been possible to give a consistent index of exposure for the years

reviewed. During this period, control of lead dust has been considerably improved, and this is well illustrated by comparing the distribution of blood lead concentrations for 1965-6 and 1975-6. The latest results show continued improvement. Evidence of earlier improvements include virtual disappearance of clinical lead poisoning after 1930 and improvements in the control data from 1927 onwards.¹⁰

For the above reasons it was decided to continue to use the three groups used by Dingwall-Fordyce and Lane³ as described above. Although the degree of exposure has clearly changed in groups 2 and 3 over the years, the use of the original groups has the advantage of not fragmenting the groups.

DEATHS FROM ALL CAUSES

The study by Dingwall-Fordyce and Lane³ found a significant excess of deaths from all causes among pensioners dying between 1925 and 1961 in group 3 exposed men—expected 75 deaths, observed 101. Deaths from all causes in this study (1925-76) do not show a significant excess of observed against expected mortality in any group, nor for all three groups combined (table 2). It would seem reasonable to conclude that any loss of expectation of life found in the previous study has now disappeared. The small excess of observed deaths in group 3 could be expected as all individuals in this group were in social class IV. The disease group most affected by social class as well as by geographical location is respiratory diseases.

CEREBROVASCULAR ACCIDENTS

This group comprised diseases in the international classification of disease group 430-438. Table 3 compares the results of this study with those of Dingwall-Fordyce and Lane.³ The excess of cerebrovascular accidents is still found in this study in pensioners with the history of highest lead exposure, and the last death recorded in the 18 observed age 65-69 was in 1969. The small excess (not statistically significant) that Dingwall-Fordyce and Lane found among men dying in service has now disappeared.

Neither this study, nor that of Dingwall-Fordyce and Lane, has found any excess of cerebrovascular accidents in group 2. Since current blood lead concentrations in group 3 are now down to, or below, earlier group 2 concentrations, it would seem reasonable to assume that present levels of control should effectively prevent any excess of deaths among all the lead exposed workers first exposed after 1975 and probably earlier.

It is not clear why the excess of cerebrovascular deaths in this study is confined to the 65-69 age

group. Social class does not make any difference to these findings since the PMR for social class IV from cerebrovascular disease aged 65-74 is 100.

CARDIOVASCULAR AND HYPERTENSIVE DISEASE

Cardiovascular disease tends to be more prevalent in soft water areas and lead has been suggested as a possible factor.¹⁰ Some early writers^{11,12} associated lead work with an excess of hypertension but if lead were a major factor in the incidence of cardiovascular disease one would expect to find an excess among this group of lead workers, who have had blood leads at a much higher concentration than the general population over many years (usually in excess of 25 years).

There is no excess of hypertensive disease nor of all other circulatory disease in either the pensioners or those dying in service. Since a large proportion of all other circulatory disease would be coronary heart disease, there is unlikely to be any excess of coronary heart disease among this group of lead workers.

One of us (DM) in an unpublished study of 1000 lead workers was unable to find any correlation between lead dosage and the incidence of hypertension. Lane⁹ reports a small study of pasters where there was no excess of hypertension; and nor did Cramer and Dahlberg¹³ find an excess of hypertension.

It is surprising in the present study with a small excess of nephritis and nephrosis in the early years, and an excess of cerebrovascular accidents up to 1969 not to find some evidence of the most likely link between lead exposure, renal disease, and cerebrovascular accidents—namely, hypertension.

The published evidence relating lead exposure to hypertension is to some extent, contradictory, but the positive evidence comes from early studies when exposure must have been very severe. Beevers's study¹⁰—which suggests a possible association between coronary artery disease and lead in soft water areas—refers to much lower concentrations of lead absorption than those experienced in this study group.

RENAL DISEASE

Historically, excessive exposure to lead has been associated with an excess of deaths from chronic interstitial nephritis. In a review of publications Malcolm¹⁴ came to the conclusion that renal failure was likely to occur in groups where exposure was likely to cause episodes of clinical poisoning. In this study the excess of deaths from nephritis and nephrosis (580-584) occurred in the 1935-58 period, which would indicate first exposure before 1933. The overall excess that persists in this study (table 5) in groups 2 and 3 is unlikely to be associated with

lead, as four cases of death occurring between 1951 and 1964 would appear to have been associated with chronic infections of the renal tract. The PMR for men aged 65-74 from nephritis and nephrosis in social class IV is 99.

NEOPLASTIC DISEASES

Neoplastic diseases were examined under four categories—namely, the digestive system, the respiratory system, leukaemias, and all other neoplasms. There was an overall excess of malignant neoplasms of the digestive system among those in group 3 dying during employment: 21 compared with 12.56 expected ($p = 0.009$). This excess was chiefly confined to the years 1963-6. Such a distribution is unlikely to be related to continued exposure to lead. No explanation has been found for this peak excess. There was no overall excess of neoplastic disease in any of the three groups of pensioners. Neither the studies of Dingwall-Fordyce and Lane³ nor Copper and Gaffey¹⁵ found any excess of cancer.

It would seem reasonable to conclude that prolonged exposure to lead at work, even under conditions regarded as unacceptable today, has not given rise to a significant excess of neoplasms. There is no association in this study between lead exposure and renal neoplasms such as those described by Zollinger⁴ and Boyland *et al*⁵ in animal experiments. Only one renal neoplasm occurred in a group 1 subject dying in service.

Cooper and Gaffey¹⁵ found a small deficit of neoplasms among lead smelters, along with a slight excess among battery workers. Their group, however, consisted of men who had worked more than one year in lead and not long-term employees, as in this study.

Conclusions

Lead exposure at work to blood lead concentrations often considerably over 80 $\mu\text{g}/100\text{ ml}$ has been associated with an excess of cerebrovascular accidents. This excess is not seen in group 2 pensioners, whose blood lead distribution in 1965-6 showed a mean of 57 $\mu\text{g}/100\text{ ml}$ (2.7 $\mu\text{mol/l}$) with 5% in excess of 80 $\mu\text{g}/100\text{ ml}$ (4 $\mu\text{mol/l}$). Since the blood lead concentration of group 3 men in 1975-6 is similar to group 2 men in 1965-6, it would seem reasonable to assume that we shall not find any excess of cerebrovascular accidents in any group under present-day conditions. There may be among present pensioners, however, those whose first exposure between 1930 to about 1970 was higher than the group 2 levels. It should be recognised, however, that exposure in group 2 before 1965, when blood lead

estimations were started, may have been higher than those recorded since.

The small excess of chronic nephritis seems to have disappeared from group 3 pensioners by 1958, although there was still some excess mortality from all renal diseases.

Exposure to lead resulting in mean blood lead concentrations of 57 $\mu\text{g}/100\text{ ml}$ (2.75 $\mu\text{mol/l}$) and 5.2% exceeding 80 $\mu\text{g}/100\text{ ml}$ (4 $\mu\text{mol/l}$) has not resulted in any excess of cancer, renal cancer, cardiovascular disease, or hypertensive disease.

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