Chapter 6. Killer lead – "let's get rid of it"

In chapter 5, we saw how scientific developments between 1890 and 1910 enabled Thomas Legge and his colleagues in the Factory Inspectorate to attack the problem of lead poisoning. The result in 1912 was the first occupational exposure limit in the English-speaking world, and a 100-page exposition in Legge and Goadby's book of what the British COSHH Regulations now call "the principles of good practice for the control of exposure".[1 2] Legge promoted the principle that the employer should provide a safe working environment, without relying on the employees to protect themselves. These things that we now take for granted were systematically explained for the first time.

But one important source of lead exposure remained outside the scope of the Factories Acts, so for that occupation there was no solid information of how many poisonings and deaths were occurring, and there was no legal right for the Inspectorate to insist on good practice. This was house-painting. G Elmhirst Duckering had developed a monitoring method, described in Chapter 5, which showed that sand-papering of paint, known to cause "severe incidence of poisoning", could give 100 mg/m^3 of lead, compared with the proposed exposure limit of 0.2 mg/m^3. Mixing of dry white lead with oil and burning off old paint were other important sources of exposure.[3] Even though reporting of poisoning in house painters was not required, 1973 of such cases with 383 deaths had been reported in the ten years 1900-1909. The number of cases every year exceeded considerably that from any other single lead industry, and according to Legge the symptoms were more severe than in other occupations, and the death rate amongst those poisoned was higher.

The battle of the ban

Why was lead in paint at all? Lead compounds had been used as a white pigment, but known to be poisonous, for centuries. It had been suggested in France in 1783 that lead carbonate could be replaced by zinc oxide.[4] Charles Thackrah, Town Surgeon of Leeds, who seems to appear in every chapter of this story, also suggested in his 1832 book that zinc salts should replace lead in paint[5]. Lead sulphide is black and zinc sulphide is white, so zinc paint survives better if there is any hydrogen sulphide in the air. In the new century, Legge, of course, was also interested in the possibility of removing lead from paint. He had written about substitution in 1902[6], and in his 1912 book with KW Goadby he gave details of the experience of six continental countries where the substitution of zinc salts had already been carried out .[7] These enquiries seem to have convinced him.

A new opportunity arose after the First World War. The Treaty of Versailles, which ended the War, recognised that universal peace required social justice, including "the protection of the worker against sickness, disease and injury arising out of his employment".[8] Under the Treaty, this was to be promoted by an International Labour Office. One of its early measures was the White Lead (Painting) Convention, 1921, which prohibited the use of white lead paints with more than 2% lead in internal painting of buildings, with certain exceptions including some railway and industrial buildings.[9] Legge was a leading member of the British delegation which negotiated the Convention. He came home expecting early ratification. A prohibition bill was introduced by the minority Labour government in 1924, but failed to pass the Commons before the government was defeated and replaced.[10] Following lobbying by the white lead manufacturers, the new government decided not to implement the Convention, but instead in 1926 brought in measures to control exposure in use. Legge resigned as Senior Medical Inspector. He had held the post with international distinction for 28 years, and had been knighted the previous year. Contrary to Civil Service conventions, he wrote to The Times and explained why he had resigned[11].

Legge's dramatic resignation, and his public announcement of the reason, led to a debate in the House of Lords[12], which revealed why the Government had preferred controlled use to a ban . The Government side argued that the annual number of deaths from lead paint in the participating countries was 400, which was "a very serious but not an enormous figure". It was claimed that the Convention discussions had been "a fight between the lead-producing and lead-using countries and the zinc-producing and zinc-using countries", and that those in favour were "influenced by the fact that their countries are the chief producers of rival pigments". But the debate highlighted the problem: there were thought to be 150,000 house-painters, often working individually or in twos and threes. As the Bishop of Southwark said in the Lord debate, without sufficient inspectors to enforce them, "the Regulations will be perfectly useless". With this usage pattern, a ban would be the only effective control.

It was also said in the debate that the white lead manufacturers had been lobbying strongly against a ban. The reasons are an illustration of some problems of substitution as a control measure. It was not possible to simply replace one substance with another without making allowance for changes in properties. Paints based on zinc oxide needed different formulation, the covering power was different, and these changes meant that a different technique was required in application. The same was true when other substitutes, such as titanium dioxide, became available. Industry sometimes resists substitution because it knows everything about its present process and the long-term properties of the product, arguing that, "Substitution looks as if it may work, but there is uncertainty and expense: let's stick with what we're sure of."

Nevertheless, despite the lack of a ban, the use of lead in paint decreased. Robin Williams explored some of the economic reasons in a 1984 University of Aston PhD thesis.[13] As early as the late 1920s, zinc oxide was cheaper per ton than white lead, and the amount produced passed white lead in the early 1930s. Titanium dioxide remained more expensive per ton, but its greater covering power meant that less was needed in the paint, so it was effectively cheaper by the 1950s. In the early 1960s, the industry felt able to agree a voluntary ban. In 1992, the ban had to be made statutory to implement a European Directive,[14] but by then lead paint had long gone out of use except for very specialised uses. Britain has never formally ratified the ILO Convention.

The pattern is familiar to those of us who remember what happened to asbestos: first arguments for controlled use, which the government accepted; then public rejection of the product and a shift in the economics; finally official bans when few users remain.

Unfortunately, Sir Thomas did not live to see these changes. He became medical adviser to the Trades Union Congress in 1930 and died two years later, at the age of 69.[15]

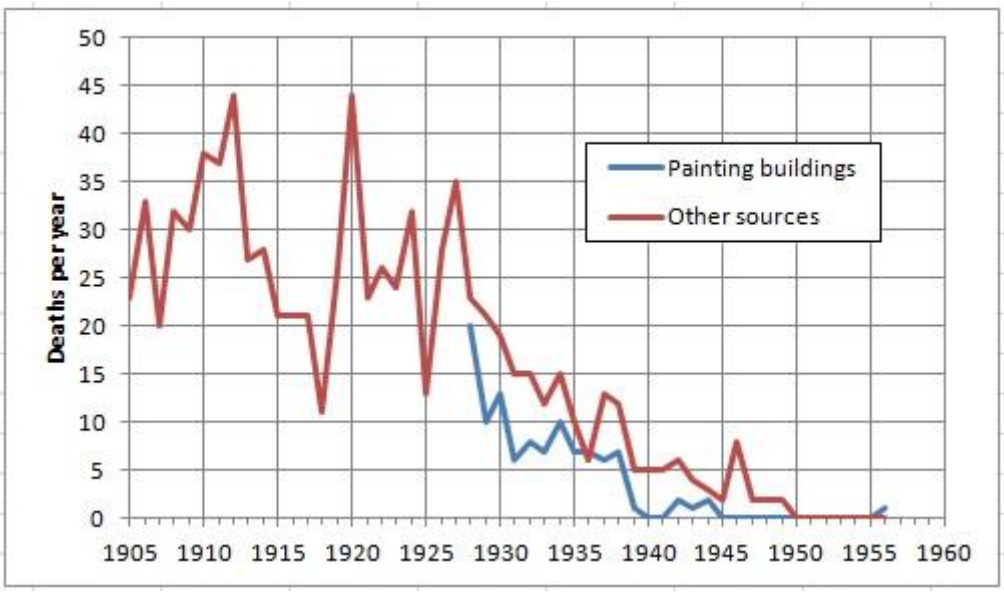

Fig 6.1. Was the government right in 1926 that a ban on lead in paint was unnecessary? The chart shows annual deaths from lead poisoning reported under the Factories Acts, and, from 1928, those in painting of buildings. From the Annual Reports of the Chief Inspector of Factories.

But would a ban have saved lives?

It is easy to feel indignant about what happened to the Convention, but we ought to ask whether the industry and government were right that a ban was unnecessary in 1926, and that other controls were enough. The controls in the 1926 Act reduced painter exposure if followed, and reinforced the economic incentive to substitute. The Chief Inspector of Factories annual reports show that deaths from lead poisoning generally and in painters in particular fell pretty steadily from 1927 onwards (Fig 6.1). Deaths in painters continued into the 1950s, totalling 108 after 1928, plus another 9 in manufacture of white lead, and another 482 painters were poisoned non-fatally. Of course there is no way of knowing how many of these casualties got their exposure from paint in place before 1926, but a ban would presumably have saved the lives of a fraction of them – perhaps some dozens of painters - and might have saved two or three hundred other painters from very unpleasant or disabling poisoning.

The decreasing death rate was perhaps due to better implementation of the control measures that Legge and Goadby had detailed in their 1912 book, and to cheaper substitutes, less public tolerance of bad industrial practice, and better enforcement tools. It is interesting that figures analysed by Robin Williams in his thesis indicate that the decrease was the same in industries subject to specific regulations, such as potteries and accumulator manufacture, as in other industries.[16]

This is not the whole story, however. Today there is concern about the health effects of lead at levels much lower than those which cause what used to be called “lead poisoning”, so old paint is still a problem, and many more people would have had their health affected than appear in the old poisoning figures. Current HSE guidance warns that “lead pigments were widely used in paints for homes, schools and offices until the 1960s”, and repeats Legge and Goadby’s 1912 warnings about sanding and burning off old paint.[17] The effects of the 35-year delay in a ban are still with us.

What happened next?

Classic lead poisoning, which caused misery to so many workers a century ago, had therefore disappeared by the early 1950s. (There was apparently a new peak in the 1960s, but this was probably due to a change in change in criteria.[18]) However, we are still concerned about possible health effects of lead today. What is the difference? The early editions of the classic book "Diseases of Occupations" by Donald Hunter, written at about the time the disease disappeared, gave diagnostic criteria for the successive stages of lead poisoning. The "commonest manifestation" was several days of constipation followed by intestinal colic. "The patient…becomes cold, pale and drenched with perspiration, and may bend over or writhe in bed in intense pain." If exposure continued, neurological damage followed.[19] These are the more severe symptoms which were no longer seen after the 1950s, but more subtle effects but still serious effects continued. From the 1970s, various biological markers, especially blood in lead, were used as triggers for control measures and the ones applied in Britain at the time of writing (2020) are shown in Table 1.

Table 1. Blood lead triggers for action under the Control of Lead at Work Regulations[20] (dl = decilitre, ie 100 ml). There are corresponding triggers in terms of lead in urine.

	Medical surveillance triggered	*Action Level*	*Suspension level*
Woman of reproductive capacity	20 µg/dl	25 µg/dl	30 µg/dl
Other person under 18	35 µg/dl	40 µg/dl	50 µg/dl
Other employee	35 µg/dl	50 µg/dl	60 µg/dl

There is a lot of individual variability in the relation between clinical symptoms and blood lead concentrations, and I have not traced any reliable blood-lead figures from the era of classic lead poisoning. However, Fig 3 shows the range of blood leads found in a works shortly after the disappearance of reported lead poisonings.[21] These figures can be compared with those in Table 1, and seem to show that many workers in the 1961 study were above the modern suspension level. However, the Fig 6.2 measurements date from before the modern interlaboratory quality control checks, so they are subject to uncertainty. As this company clearly had an interested chief medical officer, we can expect that there were many workplaces worse than this. "No cases of clinical plumbism" were observed in this study, but of the workers with blood lead >80 µg/dl, 32 showed some clinical signs of exposure, and 59 did not.

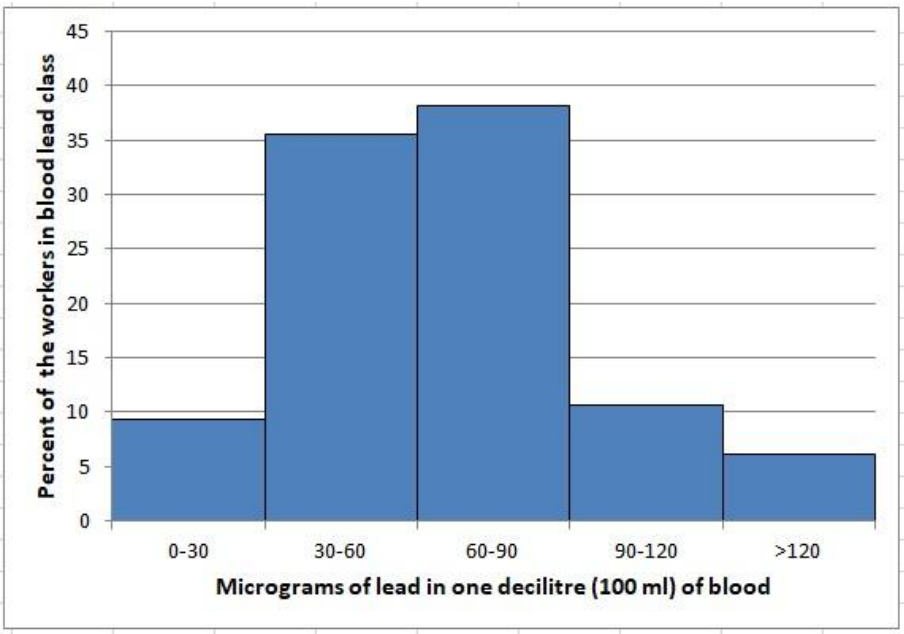

Fig 6.2. Distribution of blood leads in 540 workers exposed to metallic lead in a motor vehicle works, reported in 1961 (King and Thompson, Ann Occ Hyg 3:247-263). Compare the modern suspension levels in Table 1

There continue to be reports of reprotoxic and neurological effects at blood lead levels of a few tens of µg/dl, or lower in children, and Germany and Australia are reported to have suspension values of 10 µg/dl for women.[22] The levels in Table 1 continue to be exceeded sometimes in construction activities that disturb old lead paint.[23]

In these last two chapters, I have taken lead poisoning as an example of how what we now call principles of occupational hygiene, spelt out in 1912, came to be applied in the 20th century, to the benefit of many thousands of workers. In the next chapter, we will look at another example with many unexpected twists and turns and a less complete outcome: the example of silicosis and related lung diseases.

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[1] Legge TM amd Goadby KW, *Lead Poisoning and Lead Absorption.* London, Edward Arnold, 1912, p 291. https://ia600209.us.archive.org/35/items/cu31924003449752/cu31924003449752.pdf

[2] *Control of Substances Hazardous to Health (6th Edn)*, Regulation 7(7). Health and Safety Executive, 2013 http://www.hse.gov.uk/pubns/priced/l5.pdf

[3] Legge TM amd Goadby KW, *Lead Poisoning and Lead Absorption.* London, Edward Arnold, 1912, p 291. https://ia600209.us.archive.org/35/items/cu31924003449752/cu31924003449752.pdf

[4] Thomas Legge, *Lead and its compounds*, Chapter 19 of *Dangerous Trades, The Historical,Social, and Legal Aspects of Industrial Occupations as Affecting Health* (Thomas Oliver, Ed), p293.. London, John Murray, 1902. https://archive.org/details/dangeroustrades00olivgoog/page/n5. A recent review of this book by J Tim Carter and Anne Spurgeon is at Occupational Medicine 68:75-76 (2018) https://academic.oup.com/occmed/article/68/1/75/4866343 .

[5] C. Turner Thackrah, *The Effects of Arts, Trades, and Professions, and of Civic States and Habits of Living, on Health and Longevity: with Suggestions for the Removal of many of he Agents which produce Disease, and shorten the Duration of Life.* London, Longman, Rees, Orme, Green and Longman; 2nd Edn 1832, p 105 https://books.google.co.uk/books?id=ennv5jq4FbEC . A recent review of this book by J Tim Carter and Anne Spurgeon is at Occupational Medicine 67:500–501 (2017)

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[8] Treaty of Versailles, Part XIII, https://www.firstworldwar.com/source/versailles387-399.htm

[9] CO13 – White Lead (Painting) Convention, 1921 (No.13). http://un-act.org/wp-content/uploads/2015/11/C013-White-Lead-Painting-Convention-1921-No.-132.pdf

[10] RA Williams. *The formation and impact of hazard control policy. A study of the regulation of white lead paint in Brtain.* PhD Thesis, University of Aston in Birmingham, Jan 1984. https://research.aston.ac.uk/portal/en/theses/the-formation-and-impact-of-hazard-control-policy(92990de3-2a81-43c2-be95-c54cada57e61).html

[11] *The Times* 1st Dec 1926

[12] Lead Poisoning, debate 23 February 1927, House of Lords Hansard 66:179-210. https://api.parliament.uk/historic-hansard/lords/1927/feb/23/lead-poisoning

[13] RA Williams. *The formation and impact of hazard control policy. A study of the regulation of white lead paint in Brtain.* PhD Thesis, University of Aston in Birmingham, Jan 1984. https://research.aston.ac.uk/portal/en/theses/the-formation-and-impact-of-hazard-control-policy(92990de3-2a81-43c2-be95-c54cada57e61).html

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[18] RA Williams. *The formation and impact of hazard control policy. A study of the regulation of white lead paint in Brtain.* PhD Thesis, University of Aston in Birmingham, Jan 1984. https://research.aston.ac.uk/portal/en/theses/the-formation-and-impact-of-hazard-control-policy(92990de3-2a81-43c2-be95-c54cada57e61).html

[19] Donald Hunter, *Diseases of Occupations.* London, English Universities Press, 3rd Edn 1962.

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[21] King E, Thompson AR (1961) The measurement of lead absorption in industry. *Ann Occup Hyg* 3: 247-263. The classes of blood lead concentrations in their paper appear not to be contiguous, but the total number of cases in the classes is equal to the total number of workers they give. For example, one class goes up to 80 µg/dl and the next starts at 90 µg/dl. I have assumed that this is because they have rounded each result to the nearest 10 µg/dl, and I have drawn the class boundary half-way between, in this case at 85 µg/dl.

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