

Appendix D – History of Lead in NZ

The History of Lead in New Zealand

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The main sources

For much of the twentieth century, lead was widely used in New Zealand. Lead oxide was the principal paint base, lead arsenate was the main insecticide for pipfruit orchards, and various lead compounds were used for pottery glazes and pigments. Tetraethyl lead was added to petrol to prevent engine knocking, at relatively high levels of up to 0.84 g/L. Metallic lead was used for roof flashing and nails, for bird shot and bullets, and in lead-acid batteries; alloyed with tin it made solder for cans, electrical equipment and pewter utensils. Most of these uses were pervasive throughout society. It has been estimated that as many as 450,000 houses, one quarter of the present-day housing stock, were painted with high-lead paints.

Still, New Zealand did not have some sources of lead that are major contributors to lead exposure in many other countries. Lead mining and processing were rare in New Zealand; lead was only mined during the 1960s, at a single site – the Tui Mine near the small town of Te Aroha in the Waikato (Christie and Brathwaite undated) – and processed on an industrial scale only in battery factories at Onehunga in Auckland, Petone in Lower Hutt, and Woolston in Christchurch. Also, it does not seem that lead was used for potable water pipework to any significant extent in New Zealand (though joints in pre-1940s cast-iron mains pipes are known to have been sealed with lead – refer The Press, 2019). So, since New Zealand lacked either lead pipework or a lead-enriched geology, it appears that potable water rarely contained elevated lead.

Discovering the consequences

With the advent of atomic absorption spectroscopy in the 1970s, broad-scale lead screening became practical. Initial studies reported significantly elevated lead in the New Zealand diet (Dick et al. 1978), in children's blood (Shellshear et al. 1975, Kjellstrom et al. 1978) or teeth (Fergusson et al. 1988a,b,c), in the blood (Hinton et al. 1984) or hair (Fergusson et al. 1981) of adults who worked with lead, and in house dust, street dust and residential soils (Kjellstrom et al. 1978, Fergusson and Schroeder 1985).

Many of these early results were considerably above the 10 µg/dL threshold for notification to a Medical Officer of Health today:

- 1975 Christchurch surveys show that the *average* child blood lead level was greater than 14 µg/dL (Malpress et al. 1984).
- For groups of 50 1-4 year-olds from each of four Auckland suburbs, geometric mean blood lead levels in December 1977 or February 1978 ranged from 12 µg/dL (greenfield subdivision, Lynfield) to 21 µg/dL (Onehunga). Four children exceeded 40 µg/dL (Kjellstrom et al. 1978).

Adult occupational exposure could also be significant. Using burners or sandblasters to remove lead-based paint was recognised to be particularly hazardous (Cameron et al. 1984, Hinton et al. 1984); this was a common activity since the great majority of houses had been clad in painted weatherboard, and the original enamel paints were incompatible with the new acrylic paints, meaning that paint had to be removed for renovation or maintenance. There were reports of children and pets living at such houses developing acute lead poisoning (Kjellstrom et al. 1978, Cairns et al. 1979). Muffler repairers and scrap dealers exhibited even higher levels of exposure (Hinton et al. 1984).

After the peak

As time passed, some uses of lead gave way to more modern products. Levels of lead in house paints declined after the 1940s; paint lead content was generally less than 1 % by the 1970s, and less than 0.1 % by 1997 (AS/NZS4361.2:2017), except for certain metal priming paints. Lead arsenate insecticide was superseded by organophosphate pesticides such as azinphos-methyl during the 1970s. Tui Mine was found to be uneconomic, and was abandoned in 1973.

After the extent of elevated blood lead levels became clear, health authorities promptly began a range of interventions to address most of the other sources. Lead was eliminated from toys, plastics, glazes and most food cans between the mid-1970s and mid-1980s (Toxic Substances Regulations 1983, Malpress et al. 1984, Hinton et al. 1986). Sandblasting paints became a licensed activity under the Clean Air Act 1982, and occupational health guidelines were developed for other industries (OSH 1982). Reduced-lead and unleaded petrols were introduced in 1987, and lead additives in petrol were banned from 1996.

Occupational exposure to lead also appeared to fall in this period, for a variety of reasons (Hinton et al. 1984); one factor was undoubtedly that the battery factories closed, though Exide in Lower Hutt lived on as a recycling plant until 2012.

The only uses of lead that continued unabated were aviation gasoline, lead-acid batteries, and clay target shooting.

Results of interventions

As health authorities had intended, there was a pronounced reduction in New Zealand population lead exposure from the late 1970s onward:

- In Christchurch, for both pre-school and school-age children, an average reduction of 6 µg/dL blood lead was reported between 1978 and 1985 (approximately 45 % decrease: Hinton et al. 1986).
- Similarly in Auckland, blood lead levels in 3-5 year-olds appeared to have fallen around 2-4 µg/dL between 1977 and 1984, to a baseline of 7-8 µg/dL in areas without exposure to lead-based paints or motorway traffic (approximately 25-30 % decrease: Kennedy et al. 1988).
- In the Dunedin Longitudinal Study, blood lead levels of 579 participants were assessed in 1985, when the children were 11, and the geometric mean was just over 10 µg/dL (Silva et al. 1986).
- By 1994, 12-24 month-olds in "old housing areas" of Wellington and Upper Hutt had an average blood lead of 5 µg/dL (Bates et al. 1994).

Researchers stressed that this blood lead reduction was not consistent with a predominantly petrol source, since it began before the introduction of unleaded petrol, and while regional petrol consumption and petrol lead levels were constant (Hinton et al. 1986). Rather, the drop was consistent with contemporary estimates of lead in the New Zealand diet (including the reduction in use of lead solder in canned food), which fell steadily from 412 µg/day for a young man in 1974 (Dick et al. 1978: no estimate of child intake) to 260 in 1982, 94 in 1987/88, 33 in 1990/91, and 12 in 1997/98 (Vannoort et al. 2000). The 2016 New Zealand Total Diet Survey estimated just over 10 µg/day for a young male 19-24 (MPI, 2016).

As expected from earlier case reports, some studies pointed specifically at lead-based paint removal as the key mechanism for significant lead exposure (e.g. Kjellstrom et al. 1978). In the 1994 Wellington study, three of 143 children had more than 30 µg/dL blood lead and a further 13 had more than 10 µg/dL; children living in a house more than 50 years old where paint removal had taken place in the last two years were 14 times more likely to be in these high-lead groups. Similarly in the Dunedin longitudinal study, 10 of the 579 eleven-year-olds had blood lead exceeding 24 µg/dL, and nine of them lived in houses where there was "current or very recent" paint stripping. These children's blood levels were all significantly lower on re-testing several weeks after the first measurement.

We comment that the proportion of children living in older lead-painted weatherboard houses in inner-city suburbs would have declined over the period, as cities expanded into surrounding agricultural land.

The residual

However, banning uses of lead did not remove lead from the environment. Tens of thousands of tonnes of lead had already been painted onto houses and emitted from exhausts all around the country. Consequently, lead remained in soil and dust in, on and around older houses and major roads (Cameron et al. 1984, Fergusson and Schroeder 1985, Kennedy et al. 1988, Fergusson and Kim 1991, Kim and Fergusson 1993). A study of sandblasting found elevated lead up to 6 m from the house, reaching up to 11 % in soil and 15 % in indoor dust (Cameron et al. 1984). Lead had been sprayed over approximately 6,000 ha of pipfruit orchards, principally in the Nelson and Hawkes Bay regions.

One early case report (Shellshear 1973) identified a two-year-old who had developed lead poisoning by eating contaminated soil from around the house. Larger studies indicated that this may have been a general trend. By comparing study groups from selected neighbourhoods, Kennedy et al. (1988) suggested that, for Auckland children aged 3-5 years in 1984, typical baseline levels were around 7-8 µg/dL blood lead. Average levels were up to 3-4 µg/dL higher in suburbs with many old lead-painted houses, and also up to 3-4 µg/dL higher in suburbs around major motorways. Paint and petrol lead intake appeared to be principally indirect, via soil and household dust.

Similarly, Fergusson et al. (1988a) reported that 'social disadvantage', living in pre-1950s houses, living in streets with high traffic density, and a reported history of soil / paint eating were all associated with higher dentine lead.

The respective contributions of outdoor and indoor exposure is not clear, and may be irrelevant if indoor dust derives from outdoor soil. Though the principal risk factor in

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1994 Wellington appeared to be lead paint removal, children who usually or always played outside were 3.5 times more likely to have high blood lead than children who sometimes or never played outside (Bates et al. 1994). A complicating factor is that soil lead concentrations can vary by orders of magnitude within a property, so even considering outdoor exposure on its own, it most likely matters where on the property a child plays.

Delayed effects of low-level lead exposure

In the Dunedin Longitudinal Study, after adjusting for maternal IQ, childhood IQ, and childhood socioeconomic status, each 5 µg/dL higher blood lead at age 11 was associated with a 1.6-point lower adult IQ, and statistically significant reductions in scores for perceptual reasoning, working memory and socioeconomic status (Silva et al. 1988, Reuben et al. 2017).

The Christchurch longitudinal study found small but statistically significant correlations between dentine lead and measures of school performance at the age of 9, including reported inattention / hyperactivity. However, there was no significant relationship between dentine lead and IQ (Fergusson et al. 1988a-c).

Today

It appears that the trends of decreasing blood lead and decreasing dietary lead have continued. Today, the mean blood level for children aged 5-18 is just 0.9 µg/dL, apparently with little variation by age (t Mannoetje et al. 2018).

Ministry of Health data for the period 2013-June 2017 showed 38 notifications of lead absorption exceeding 10 µg/dL in under-18s. Half of those (19 cases) were in the 0-4 years age group. The principal attributed source was lead-based paint (20 cases) though "pica" and "unknown" were also significant sources (10 cases each). These cases are thought to be the 'tip of the iceberg' in that the symptoms of mild lead poisoning are not distinctive, so that lead absorption is likely to go unnoticed.

For all ages over the same period, there were 361 notifications of lead absorption exceeding 10 µg/dL. The source was often unknown (141 cases) but lead-based paint was again a key source (98 cases) with indoor rifle ranges also prominent (51 cases, plus 24 for bullet and sinker manufacture). The issue of firearms-related lead exposure was recently highlighted (Russell et al. 2019) with identified pathways including combustion of lead-containing ammunition close to the face, lead dust at indoor ranges, and consuming lead-shot meat. It was noted that "air rifles, which are often considered to be a beginner's or child's gun, are frequently used with lead pellets".

Unsurprisingly, there is no indication that soil lead has somehow 'gone away'. A recent systematic survey of Dunedin City, sampled on 1 km spacing, showed median lead concentrations were an order of magnitude higher in urban residential properties (130 mg/kg) than in rural properties (17 mg/kg); 10 of 113 samples, five from residential properties and five from schools, exceeded the current 210 mg/kg soil contaminant standard for residential use.

Data deficiencies

Nonetheless, there are things that New Zealand blood lead studies are not telling us. Firstly, there were no population-level studies before 1974 or from the mid-1990s to the mid-2010s, so we do not have a continuous record.

Secondly, few studies have investigated lead levels in the 1-2 year-old age group, and the most recent study (t Mannelje et al 2018) is not one of them. The investigation into lead exposure around the Port Pirie lead smelter, South Australia, showed a pronounced peak in blood lead for this age group (Baghurst et al. 1985, Searle et al. 2014, etc.). This could be a consequence of normal mouthing behaviour in this group (Baghurst et al. 1985, MoH 2012, Searle et al. 2014, DSM -5) and hence increased paint, soil and dust ingestion; it could also be because young children absorb a greater fraction of the lead they take in than older children or adults (MoH 2012). Whatever the mechanism, this peak is most unfortunate given this age is also a particularly important time for neural development.

Thirdly, soil lead concentrations are well known to be highly variable within a property and between properties of similar ages (e.g. Kjellstrom et al. 1978, Fergusson and Kim 1991). In our experience, lead levels can be many times higher close to a lead-painted house, than at the rear of a section. To a lesser extent, soil lead levels are well known to decrease rapidly away from high traffic roads, over scales of a few metres (e.g. Ward et al. 1979). Those New Zealand studies that do report soil lead concentrations, report them as single numbers, and provide no detail on sampling or statistical treatment of results. It is not clear whether these numbers are actually representative of potential soil lead exposure, indeed it appears unlikely that a single value *can* be representative of soil lead exposure. Certainly catchment soil sampling on the kilometre scale (e.g. Turnbull et al. 2019) cannot be related to child blood lead levels.

Fourthly, while lead's toxicity derives largely from its tendency to mimic calcium in biological environments, and calcium intake is known to be partly protective (MoH 2012), we do not know to what extent New Zealand children may be protected. The last children's nutrition survey, in 2002, found that 15 % of children aged 5 and over had inadequate calcium intake, increasing to 20 % for Maori girls and 45 % for Pasifika girls (MoH 2003), suggesting that these groups may be particularly vulnerable. Again this survey did not look at the critical 1-2 year-old age group.

Conclusions

Lead is dangerous to children and has been very common in the home environment. By today's standards, the average city child of the 1970s was suffering notifiable lead absorption, probably affecting brain development, and later loss of socioeconomic potential. In subsequent decades, great reductions in dietary lead, appear to have resulted in correspondingly great reductions in average child blood lead; removing lead from petrol may also have played a part.

However, nothing has been done about lead released into the environment. Key sources are lead-based paint removal and the combustion of leaded petrol. Paint removal is a particular concern since it results in long-term soil contamination close to houses where children live, potentially at very high levels.

A case of child poisoning due to eating lead-contaminated soil was documented in 1973* but we do not know how many cases have occurred since, or how many are occurring today. 1-2 year-olds living in pre-1950s houses appear to be at much the greatest risk from exposure via soil, especially if they have a low calcium intake. While it is probably uncommon, the adverse effects of lead absorption seem significant enough to warrant further investigation.

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* Note that this is extremely unusual in the contaminated land field. For almost all contaminants, the potential for harm is inferred by linear interpolation from studies of highly exposed groups, and simple mathematical exposure models, not from direct observation of effects.

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