VIEWPOINT

Lessons learned on lead poisoning in children: One-hundred years on from Turner’s declaration

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Abstract: There is significant emerging evidence showing life-long negative health, intellectual and socio-behavioural impacts as a result of childhood blood lead concentrations well below the widely used intervention level of 10 \( \mu \text{g/dL} \). This issue raises serious health concerns for children in several Australian smelting and mining towns. Routine educational and home cleanliness advice to wet mop floors rather than to use a brush and pan to reduce lead exposure risks have been shown to have limited efficacy. This paper argues, as advocated 100 years ago by Queensland doctor Alfred Jefferis Turner, that childhood lead poisoning can only be mitigated via primary prevention and reduction of contaminants at source. Given that the effects of lead exposure are irreversible, there is a strong argument for the application of the precautionary principle to dealing with childhood lead exposure. There is a clear need to improve regulatory controls and emissions management to reduce environmental lead exposure risks.

Key words: adverse effect; childhood; lead poisoning; prevention and control.

Key Points

1. Educational and home cleanliness approaches such as advice to wet mop floors rather than to use a brush and pan to protect children from environmental lead emissions from mining and smelting activities have been shown to have limited efficacy. This paper argues, as was advocated 100 years ago by the Queensland doctor Alfred Jefferis Turner, that the key to successfully protecting children from lead poisoning is through primary prevention and reduction of contaminants at their source.

2. There is considerable evidence showing negative health impacts as a result of childhood blood lead concentrations at values well below the currently accepted guideline of 10 \( \mu \text{g/dL} \). These effects have been shown to delay and limit children’s intellectual development and academic achievements. They have also been linked to problematic socio-behavioural patterns including attention deficit hyperactivity disorders, learning difficulties, oppositional/conduct disorders and delinquency.

3. Given that the effects of lead exposure are irreversible, there is a strong argument for the application of the precautionary principle to dealing with the problem of childhood lead poisoning. There is a clear need to improve regulatory controls via legislation, policy and environmental management actions in order to reduce toxic emissions and improve environmental lead standards.

Introduction

One hundred years ago, Dr Alfred Jefferis Turner1 was the first medical practitioner to identify that children could only be guaranteed protection from the deleterious effects of lead by preventing exposure. Nevertheless, even today, it still remains a critical public health issue that we focus our efforts to prevent children’s exposure to lead.

The neurotoxic effect of lead has been long established in the scientific literature and children are particularly at risk if exposed.2–4 In addition with other biologic and socio-demographic factors, exposure to lead has been linked to decreased IQ and academic achievement, as well as to a range of socio-behavioural problems such as attention deficit hyperactivity disorder, learning difficulties, oppositional/conduct disorders and delinquency.5–10 These are disabling mental health issues that often persist into adolescence or adulthood.11,12 Recent research has indicated that levels well below the currently accepted blood lead guidelines of 10 \( \mu \text{g/dL} \) can result in significant impairment in terms of neurocognitive functioning.13–16 In light of compelling evidence of lead toxicity at the lowest measurable levels, a precautionary approach is critical.17 This is significant particularly in light of the fact that unlike other known risk factors such as genes and demography, we know how to prevent lead exposure.

The Effect of Legislation to Prevent Childhood Exposure to Lead

The power, value and influence of legislative tools to mitigate lead exposure in children has been demonstrated throughout history. In the early 20th century, Australian medical
Lessons learned on lead poisoning in children

MP Taylor et al.

practitioners Gibson\textsuperscript{22} and Turner\textsuperscript{1} showed unequivocally that lead paint and associated dust caused severe poisoning as a result of hand-to-mouth behaviours in young children. Lead-based paint was banned in much of Europe in the 1920s, but in Australia its removal was more gradual. Lead levels in paint were up to 50% before the 1950s but thereafter several reductions were mandated bringing the allowable concentration to 0.1% in 1997. In the United States, the Lead-Based Poisoning Prevention Act resulted in the removal of lead in paint only after 1978. Pioneering research by Herbert Needleman and Claire Patterson, among others, was instrumental in the removal of lead from gasoline in the United States from the 1970s onwards until it was totally removed in 1986 (the completion of the phase out was in 1996, although most states had banned it before then). It is clear that stricter regulatory controls were implemented only after the deleterious health effects of lead on children had been demonstrated unequivocally.

Needleman et al.\textsuperscript{5,21} research was particularly instrumental in influencing the US Centre of Disease Control in lowering its blood lead standard for children. This value was 60 \( \mu \text{g/dL} \) prior to 1970, where after it was gradually reduced to its current level of 10 \( \mu \text{g/dL} \) in 1991. The imposition of legislative instruments that removed lead from paint in the United States in 1978 coupled with the reduction of lead in gasoline from the 1970s resulted in a significant decline in the number of children with lead poisoning. In the 1970s, 88% of children younger than 6 years of age had blood lead concentration levels \( \geq 10 \mu \text{g/dL} \).\textsuperscript{24} The combined effect of banning lead paint and the reduction of lead in gasoline resulted in simultaneous reductions in children’s blood lead levels. By the 1990s, fewer than 5% of children under 6 years of age were estimated to have blood lead concentrations in excess of 10 \( \mu \text{g/dL} \),\textsuperscript{24,25} with the figure falling to 1.6% by 2002.\textsuperscript{26} It is clear that regulatory controls have been extremely powerful agents in reducing childhood lead poisoning and are likely to continue to have a mitigating effect when implemented appropriately.

The effectiveness of government legislation in Australia was clearly understood by Turner\textsuperscript{1} who argued that the problem of lead exposure in children ‘is certainly a matter which calls for legislative interference’ (p. 897).\textsuperscript{1} While regulatory authorities have been instrumental in reducing childhood lead poisoning in Australia, lead exposure remains an important public health issue for a substantial number of children. In light of more recent studies indicating that blood lead concentrations well below the currently accepted blood lead intervention levels of 10 \( \mu \text{g/dL} \) may result in permanent neurocognitive impairment in young children,\textsuperscript{18,19} concerns have been raised about the impact of chronic low blood lead levels in children across a number of Australian towns. Smelter and mining-related operations in Australia have resulted in significant environmental contamination of both airborne dust and soil in adjacent urban environments (e.g. Broken Hill,\textsuperscript{27–29} Lake Macquarie,\textsuperscript{30–33} Mount Isa,\textsuperscript{34–38} Port Pirie,\textsuperscript{39,40} Wollongong,\textsuperscript{41} Fig. 1). Unlike adults, children are at particular risk of lead poisoning following exposure to lead-contaminated dust and soil, and chronic low level exposure in children is an ongoing public health issue with significant long-term implications. The exposure to lead and its pathways are critical factors in these risks, which are examined below.

**Primary Pathways and sources of Lead**

Lead-contaminated house dust and soil in the immediate environment are primary sources of exposure for lead poisoning in children.\textsuperscript{5,42,43} It has been demonstrated that children living in housing with floor lead levels greater than 250 \( \mu \text{g/m}^2 \) are eight times more likely to have a blood lead level \( > 10 \mu \text{g/dL} \) compared with their peers.\textsuperscript{44} Importantly, children exposed to floor dust lead levels as low as 150–200 \( \mu \text{g/m}^2 \) are still 3–4 times more likely to have a blood lead level \( > 10 \mu \text{g/dL} \).\textsuperscript{44} In addition, children <5 years of age who ingest contaminated soil via mouthing behaviours are at particular risk.\textsuperscript{45} Commensurate with this data, reductions in soil lead levels have been linked to significant reductions in childhood blood lead concentrations.\textsuperscript{42}

Atmospheric concentrations of lead are also considered to be major sources of lead exposure. In 2008, the United States Environmental Protection Agency (US EPA) revised the airborne lead standard for the first time in 30 years by reducing the maximum permissible concentration from 1.5 to 0.15 \( \mu \text{g/m}^3 \), a ten-fold decrease. In their final determination, considerations were influenced by the significant body of evidence of the effects of lead on human health (p. 66970):\textsuperscript{46}

In developing this rationale, EPA has drawn upon an integrative synthesis in the Criteria Document of the entire body of evidence published through late 2006 on human health effects associated with \( \text{Pb} \) exposure. Some 6000 studies were considered in this review. This body of evidence addresses a broad range of health endpoints associated with exposure to \( \text{Pb} \) (EPA, 2006a, chapter 8), and includes hundreds of epidemiologic studies conducted in the U.S., Canada, and many countries around the world since the time of the last review (EPA, 2006a, chapter 6).
The equivalent goal in the UK is 0.25 μg/m³, which is 50% lower than that proscribed for Australia’s Federal government lead in air goal of 0.5 μg/m³, averaged over a year. Using the example of Mount Isa and Queensland, it is noteworthy that the Mount Isa Limited Agreement Act 1985 (Queensland) permitted atmospheric Pb concentrations up to a maximum permissible rate of 1.5 μg/m³ averaged over 90 days (cf. Environmental Protection (Air) Policy, 1997, Queensland). These emission standards were replaced on 1 January 2009 by the Environmental Protection (Air) Policy 2008 (Queensland). Under these new regulations, the lead in air standard will be reduced to 0.5 μg/m³ averaged over a year, in line with the Australian Federal government guidelines. These new standards will be transitioned in over a 3-year period for Mount Isa Mine. These recent changes reflect the increased awareness and acceptance that lead in the ambient environment needs to be reduced as part of a broader regulatory and legislative framework strategy to address primary prevention and exposure.

A significant proportion of children living in Australian smelter and/or mining towns have elevated blood lead levels (Fig. 1, Table 1). For example, in Mount Isa, results of a recent blood screening programme conducted by Queensland Health indicated that almost 40% of children aged 1–4 years had blood lead levels above 6 μg/dL, and 11.25% had blood lead levels above 10 μg/dL. Similar patterns of elevated environmental contaminants have been identified elsewhere in numerous other international studies in urban areas adjacent to smelter or mining operations.10–12

The collective body of research indicates that smelter and mining towns present a significant and increased risk of elevated blood lead concentrations in young children compared with the general population. A comparison with Sweden is useful because leaded gasoline was no longer available after 1995 and it therefore provides an indication of blood lead levels unaffected by this potential contaminant source. The average blood lead level for Swedish children aged 7–11 years for the period 1995–2001 was 2.1 μg/dL for children not living near industrial sources.13 Similarly, in the United States, the average childhood blood lead concentration for children aged 1–5 years between 1999 and 2002 was 1.9 μg/dL, with only 1.6% of children having a level >10 μg/dL.14

Despite the reductions in blood lead concentrations in the broader public, it is apparent that chronic low lead exposure in children continues to be a substantial public health issue particularly at the Australian locations shown in Figure 1 and Table 1. These locations have well-established histories of environmental lead and are likely to cause adverse neurocognitive functioning in affected children, resulting in life-long social and economic impacts. For example, Jusko et al.’s study15 showed that six year olds with a mean childhood blood lead level <5 μg/dL had 4.9 more IQ points than children with mean blood lead concentrations between 5 and 9.9 μg/dL. Such data, amongst others, should precipitate a precautionary approach to mitigating, reducing and legislating against preventable primary exposure sources and pathways at levels below the currently accepted blood lead concentration guideline value of 10 μg/dL.

### Intervention Strategies and Approaches

Unfortunately, to date there have been limited efforts to manage the problem of chronic low lead exposure in children at a Federal legislative or policy level in Australia, and interventions have had to be determined predominantly by local government health and environment agencies. The recent determination by the National Health and Medical Research Council (NHMRC)16 that the guideline value of 10 μg/dL should remain unchanged is in conflict with the precautionary principle. Although the NHMRC acknowledged that there was no evidence for a threshold of lead exposure, they failed to revise it because of the ‘uncertainty concerning the magnitude and form of causal relations. . .’ (NHMRC, p. 4).

Once a child under 5 years of age has presented with a blood lead concentration of 10 μg/dL, there is a high likelihood that they will have been negatively impacted. Thus, even under current guidelines, waiting until 10 μg/dL before medical and environmental authorities respond is contrary to the precautionary approach. In regards to other pernicious diseases such as breast, cervical or bowel cancer for example, the approach in Australia is precautionary, in that action and medical assessment are primarily geared towards a preventive approach. It is difficult to see why the risks of lead exposure should not be approached in a similar way.
Underpinning our argument that the use of a blood lead level of 10 μg/dL is inadequate is a proper understanding and application of the precautionary approach. The precautionary approach was defined formally in Principle 15 of the United Nations 1992 Rio Declaration[^54] and is encompassed within Australian Commonwealth legislation at section 391 (2) of the Environment Protection and Biodiversity Conservation Act, 1999:[^54]

The precautionary principle is that lack of full scientific certainty should not be used as a reason for postponing a measure to prevent degradation of the environment where there are threats of serious or irreversible environmental damage.

We argue that this is a well-accepted and adopted principle in environmental decision-making affecting human health (e.g. Telstra Corporation Limited vs. Hornsby Shire Council [2006] New South Wales Land and Environment Court (NSWLEC), 133). Further, when the principle is coupled to evidence demonstrating the effects of lead on children even at low concentrations, there is a more than sufficient basis to argue that there should be more stringent, lower blood lead guidelines than what exist currently in Australia.

A failure of health and environmental authorities including the NHMRC[^35] to advise communities, particularly those in Table 1, that they may be at risk of serious potential harm, even at lower levels of lead exposure, is inconsistent with a proper and well-accepted precautionary approach to the prevention and minimisation of deleterious environmental health effects. We suggest that a level of 5 μg/dL, which was used in response to the recent lead-dust problem in Esperance, would be a more appropriate guideline value.[^50] This purpose of using this lower value was to provide a margin of safety, being 50% lower than the commonly accepted and applied blood lead guideline value of 10 μg/dL.

In general, responses to elevated children’s blood lead have involved some form of education-based intervention directed towards parents regarding the importance of reducing lead-rich dust in the home environment. For example, Queensland Health’s[^34] primary response to the problem of elevated blood lead levels in Mount Isa was to promote increased community education, via the development and circulation of fact sheets, posters and stories in local publications and newspapers. Education programmes have focussed on the importance of reducing dust in the home environment through the use of a mop to clean rather than a broom and on minimising child hand–mouth behaviours and practices.[^34] The onus is on families to take ‘personal protective measures’ (p. 10) to reduce environmental lead exposure. This approach is not dissimilar to interventions taken elsewhere in lead-contaminated areas, such as in Esperance in Western Australia, where community education was employed as the primary management strategy.[^54] While blood lead concentrations in children fell after such interventions were instituted in Esperance, it is difficult to untangle the individual effectiveness of different strategies, for example, wet dust mopping versus hand hygiene.[^56]

In a handful of cases where primary prevention measures were applied including cleaning up lead contamination at the source or the closing of the smelter, there were notable reductions in childhood blood levels.[^50][^51]

Educational efforts to control environmental lead exposure have long been criticised in the scientific community for their lack of efficacy, and it is difficult to support their application in the absence of other management and primary prevention measures.[^1][^2][^57][^58] A recent meta-analysis examining the effectiveness of household interventions using a large sample size (n = 2239) demonstrated that household interventions involving education or dust control measures were not effective in reducing blood lead levels in children.[^58] Thus, Turner’s conclusion that was published in 1909 remains accurate: ‘The curative treatment consists essentially in removing the child from the source of the poison. We adopt also other measures calculated to encourage the elimination of lead from the system, but to what extent these are efficacious must be a matter of doubt. By themselves they are useless’ (p. 897).[^1]

Next we describe, in brief, the case example of Mount Isa where industrial lead emissions and environmental sources are significant. Here, we argue that in addition to the educational advice being provided to residents, there is a demonstrable case to support the implementation of more effective primary prevention strategies to reduce emissions from point (e.g. stacks) and diffuse sources (e.g. spoil heaps) and consequent risks to child health.

### Case Example – Mount Isa

Mount Isa city is immediately adjacent to Xstrata Mount Isa Mines Pty Ltd’s mine, which is Australia’s largest emitter of a variety of elements including antimony, cadmium, copper, lead and zinc compounds.[^59] Emissions of lead for 2007–2008 alone amounted to 260 000 kg. Atmospheric dust loading concentrations range from 2420 μg/m²/day within 200 m of the lead smelter at Mount Isa to 92 μg/m²/day at ~4.7 km from the smelter.[^44] These values can be compared with the internationally recognised German TA Luft value guideline value[^61] for dust lead concentrations of 100 μg/m²/day and to the evidence that lead-rich dust is a significant pathway for elevating childhood blood levels.[^44] EPA Queensland[^40] recorded dust-wipe lead concentrations in children’s homes up to a maximum of 23 000 μg/m² (mean–1830 μg/m²). Values of 18 000 μg/m² have also been recorded in children homes who had blood lead concentrations significantly above 10 μg/dL (M.P. Taylor, unpublished data). These dust wipe values are dramatically higher than the US EPA and the US Housing and Urban Development Department (the HUD criterion) surface dust lead guideline value of 40 μg/ft² (~430 μg/m²).[^62] These values also exceed the lesser standard provided in the Australian Standards guideline value AS4874-2000 of 1000 μg/m².

There are a number of regulatory and prevention strategies that could be implemented in Mount Isa to reduce more effectively levels of airborne lead in dust and dirt. Reductions in emissions through the use of improved smelter technology (including the smelter itself as well as the filtration system) would be a first step in reducing further and ongoing lead contamination in Mount Isa. At Port Pirie and Lake Macquarie, such actions were coincident with marked reductions in children’s blood lead concentrations.[^10][^66] The use of Geotex fabric or capping of the spoil heaps at Mount Isa would also help to reduce airborne dust loading significantly. In addition,
assessment of household dust lead wipes and household gardens for soil lead would identify specific residences most at risk. Soil assessment needs to include a range of particle sizes, particularly those <63 μm, which encompass fractions more easily ingested and absorbed, rather than rely only on <2 mm fraction as specified in the National Environment Protection Council guidelines.63 Such assessment would help identify problem locations, sources and pathways more clearly. These approaches would allow for preventive strategies to be implemented before children present with elevated blood lead levels, which is often too late as the detrimental effects may well have been set in process. Environmental assessment processes as well as blood lead testing would need to be repeated over frequent time periods to (i) measure the efficacy of primary prevention and clean up and (ii) to ensure the sites are not re-contaminated from ongoing mining processing. In addition, there also needs to be a concomitant lowering of the allowable maximum permissible concentrations in environmental media (e.g. air, dust, soil, water) to further mitigate chronic and potential future lead exposures.

**Reversibility and the Implications of Blood Lead Levels Less Than 10 μg/dL**

Given the emerging research indicating the effect of low blood lead levels on cognitive ability,6,18,21,64 it is concerning that children’s blood lead levels are ≥10 μg/dL.65 It is also significant that low levels of blood lead have also been linked with a range of other negative outcomes in terms of learning difficulties, social–behavioural conduct, and physical growth and development,66 further supporting the need for intervention at blood lead levels below 10 μg/dL. Indeed, the data suggest that there may be no threshold for the adverse consequences of lead exposure.67 As a result of these concerns, Western Australia’s Department of Health instituted repeated blood lead testing as health surveillance for Esperance children aged 5 and under whose initial lead concentration was 5 μg/dL or more.69 We contend that this would be a more appropriate precautionary approach to identifying children at risk from low lead exposure because it targets those elements of the population most likely to be affected.

Studies on the reversibility of the adverse effects of lead using chelation therapy such as DMSA or DMPS (which binds and removes lead from the body) indicate a poor longitudinal prognosis for children with elevated blood lead. A number of large US trials with follow-up at 7 years, have shown no improvement on measures of cognitive and neuropsychological functioning despite dramatic declines in blood lead concentrations in children following chelation therapy.68,69 In line with the United States, Australian data have shown that decreases in children’s blood lead concentrations correlated with only small and inconsistent improvement in intellectual functioning.70 These studies suggest that with or without chelation therapy, neurocognitive impairments related to blood lead exposure in childhood are irreversible and continue into adulthood. Therefore, early exposure to lead places children at risk of a negative developmental trajectory characterised by diminished social and educational achievement and reduced long-term life potential. The longitudinal impact on society in terms of academic performance, economic costs to the community and negative social outcomes related to antisocial behaviours and criminality have been well established.6,71–73 Gould74 (2009) demonstrated that for every (US) dollar spent on controlling lead hazards, some $17–221 is returned to society. Vaccination programmes, which are often promoted as the most cost-effective public health and medical intervention strategies, return a much lower figure of $5.30 to $16.50 per dollar spent on immunizations.75 Thus, as well as the individual health impacts of lead, the broader socio-economic costs of lead further support the importance of primary prevention as the most effective strategy to prevent lead-related neurocognitive dysfunction, just as was advocated for by Turner in 1909.1

**Conclusions**

The utility of primary prevention has been demonstrated throughout history with removal of lead from gasoline and the banning of lead in household paint. Primary prevention has been effective in reducing childhood lead exposure and has been calculated to yield large social and economic benefits. For example, the economic benefit associated with IQ-related increases in income following the reduction of leaded gasoline, has been estimated at $110–$319 billion (US) for a single cohort.76 Since Turner’s declaration,1 there have been widespread collective efforts to promote primary prevention of childhood lead poisoning as the only appropriate response to the problem of lead toxicity,4,6,18,40,66 The challenge for governments is to develop more direct interventionist approaches to the problem of environmental toxicants, which can be implemented in an Australian context. The case study of Mount Isa illustrates the sorts of strategies that might be considered, particularly where there are significant and identifiable point and fugitive sources of contaminants. Lead exposure in children has been shown to contribute to developmental outcomes in children over and above other known risk factors. Unlike other factors, lead exposure from environmental sources is modifiable and reductions in childhood blood lead levels should be a primary focus for health authorities in places of significant risk.

Emerging evidence indicates that there should be a heightened cause for concern regarding the neurocognitive development of children who are exposed to lead at lower levels than were previously considered harmful. At the 100th anniversary of Turner’s1 declaration on the importance of primary prevention, we would like to see a greater emphasis on the primary prevention of lead exposure. This would amount to a precautionary approach to dealing with childhood lead poisoning, which is particularly relevant for young children because of their unique developmental vulnerabilities and the irreversibility of the effects of lead. These approaches ought to involve greater regulatory action at a legislative and policy level as well as environmental management change and closer work with public health departments across Australia. This model could be applied equally to other similarly affected countries. This strategy fits well with Australia’s National Priority around creating ‘A healthy start to life’ for children by mitigating unnecessary exposure to a well-known neurotoxin, which reduces well-being and life potential. The long-term benefits are likely to be
Lessons learned on lead poisoning in children MP Taylor et al.

a more productive and better educated society, especially in those areas that are subject to significant environmental lead exposure arising from ongoing and historical industrial and mining processes.

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Lessons learned on lead poisoning in children


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