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Australia’s leading public health body delays action on the revision of the public health goal for blood lead exposures

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A B S T R A C T

Globally, childhood blood lead levels have fallen precipitously in developed countries since the 1970s following action by international bodies such as the WHO and Food and Agricultural Organization (FAO) of the United Nations. These reductions have been affected by the activities of national agencies such as the US EPA and US Centers for Disease Control and Prevention in the establishment of air lead and blood lead standards, the introduction of legislation to remove lead from petrol, paint and consumer products and tighter restrictions on lead emissions. The outcome of recent major international reviews of research into the effects of low-level lead exposures (e.g. by WHO, USA health and environmental agencies, German and Canadian health bodies) has resulted in recommendations to reduce and eliminate lead exposures. By contrast, Australian policy responses to the incontrovertible evidence that adverse neurocognitive and behavioural effects that occur at levels well below the current national goal of 10 μg/dL have stalled. The delayed response by Australia occurs at a time when blood lead levels in two of Australia’s three primary lead mining and smelting cities: Port Pirie, South Australia and Broken Hill, New South Wales, are rising. In the third city, Mount Isa, Queensland, there is still no systematic, annual testing of childhood blood lead values. This is despite the fact that Mount Isa has the highest lead (and other toxic metals such as cadmium and arsenic) emissions to the environment (120 tonnes of lead in 2011/12) from any single point source in Australia. It is clear that both state and national policy approaches to the ongoing risks of lead exposure need to be revised urgently and in line with contemporary international standards. Recommended changes should include a new lower blood lead intervention level of no more than 5 μg/dL, with a national goal for all children under 5 years of age to have a blood lead level of below 1 μg/dL by 2020. In order to achieve any new lower exposure goals other relevant lead standards including air, dust, soil and water must also be revised downwards.

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1. Introduction

The measurement of lead in the remains of pre-industrial humans has shown that the natural levels of lead were 0.016 μg/dL (Flegal and Smith, 1992), 625 times less than the current recommended public health goal in Australia (10 μg/dL). In the 1960s, health agencies in Europe and the USA set, and then lowered (as more evidence became available) a blood lead “level of concern” from 60 μg/dL to 40 μg/dL in 1971, to 30 μg/dL in 1985, and lastly, to 10 μg/dL in 1991 (see Fig. 1). The level of concern was used by pediatricians and other health professionals to establish priorities and manage a lead poisoned child. In 2009, Germany lowered the reference value for children’s (aged 3–14 years) blood lead level to 3.5 μg/dL (Schultz et al., 2011). In 2012, the US CDC eliminated the “level of concern” set at 10 μg/dL and established 5 μg/dL as the intervention (reference) level for individual children (CDC, 2012). Following substantive reviews of the research evidence, several other major international health bodies have also issued similar recommendations that lead exposure, particularly in children, must be reduced or eliminated (FAO/WHO, 2010; Health Canada, 2013; US NTP, 2012; WHO, 2010).

In 1993, Australia’s current blood lead goal of 10 μg/dL was established after being revised downwards from the 1983 level of 25 μg/dL. The new lower standard mirrored the blood lead level established by the US Centers for Disease Control (CDC) in 1991 (Fig. 1). The figure of 10 μg/dL was reaffirmed by the National Health and Medical Research Council (NHMRC) after a review in 2009 (NHMRC, 2009), which concluded that “The nature of the ‘dose–response’ relationship between lead exposure and children’s intellectual abilities and behaviour is also contentious.” Importantly however, the NHMRC (2009) also noted in their information paper that “No threshold

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of lead exposure below which any exposure might be considered ‘safe’ in respect of cognitive abilities has ever been identified.” The NHMRC is currently examining the existing blood lead goal for Australia and was meant to report its findings in late 2013/early 2014 (NHMRC, 2014), but the outcomes of the review have been delayed.

To date, the majority of research on the neurocognitive and behavioural effects of lead exposure has been conducted in the USA, with very few studies from Australia examining the impacts and long-term outcomes in terms of school performances or criminality (e.g. Mielke and Zahran, 2012). The majority of Australian published research was conducted in the 1990s and was based on Port Pirie children, who had higher lead levels from smelter emissions than are typically found today either there or elsewhere. However, Rachel Earl’s, 2011 University of Adelaide PhD study investigated the effects of low-level lead exposure on 106 children (whose arithmetic mean blood lead concentration was 4.97 μg/dL) from Port Pirie and Broken Hill (Earl, 2011). This study, which drew the same conclusions as previous international studies, showed that there is no apparent threshold for the toxic effects of lead (Lanphear et al., 2005). Importantly, the study’s findings re-affirmed that the lowest levels of lead exposure are associated with proportionately greater impact on children’s cognition relative to higher levels of exposure.

In short, this means that prevention of any lead exposure must be a key goal of any public policy; the de facto goal of only aiming to keep children’s blood lead below 10 μg/dL in Port Pirie (Thumbs up for Low Levels, 2013); Mount Isa (Living with Lead, 2013); and Broken Hill (Lesjak, 2012) is obsolete and will inevitably fail to protect children from the toxic effects of lead.

2. The effects and absorption of lead exposure are non-trivial and are lifelong

The United States Environmental Protection Agency (US EPA, 2013) Integrated Science Assessment for Lead concluded that the weight of evidence shows adverse cognitive function effects of lead exposure on populations of children down to 2 μg/dL. The effects of exposure may be irreversible and persist into adulthood (US EPA, 2013; US NTP, 2012). Lead exposure is associated with delayed pubertal onset and adverse reproductive and developmental effects in young adults who have mean blood levels less than or equal to 5 μg/dL (US EPA, 2013; US NTP, 2012). Adults are not immune to low-level toxicity; causal relationships having been shown to exist between low-lead exposures (below 10 μg/dL) and adverse cardiovascular effects (e.g. increased blood pressure, hypertension, coronary disease) (e.g. Menke et al., 2006; Schober et al., 2006; US EPA, 2013). In addition, the US EPA (2013) concluded that there was a likely causal relationship between lead exposure and reduced kidney function, all of which are considered to reduce lifespan and quality. The US EPA (2013) concluded that the current toxicological evidence from studies examining the effects of elevated blood lead levels (range 5–43 μg/dL) provided strong evidence that exposure reduces semen quality and that it may also affect some aspects of female reproductive function.

In 2012, the United States Centers for Disease Control (CDC) recommended that the term “level of concern” be discarded and that the blood lead level of 10 μg/dL, as a child health goal be revised. Consequently, the CDC is now using a reference value of 5 μg/dL to identify children who have been exposed to lead and require case management (CDC, 2012). If Australia adopted a reference value of 5 μg/dL, 50% or more of the children under 5 years of age in Broken Hill, Mount Isa and Port Pirie would need greater protection and intervention unless lead emissions are drastically reduced.

Although national blood lead values are falling in major cities and mining towns in Australia (Fig. 2), the benign neglect of lead exposure is evident in Medicare statistics that show 107,810 blood lead tests were conducted on all Australians in the last decade (July 2003–August 2014). Changes to the blood lead ‘level of concern’ over the last 50 years as determined by the US Centers for Disease Control and Prevention (reproduced with permission of the Canadian Environmental Health Atlas, 2014).
2013) (Medicare Australia Statistics, 2014). It is an unfortunate and a significant lost opportunity that the results from these tests are not captured, stored and analyzed by the Australian Institute of Health and Welfare (AIHW), despite that one of their ‘primary roles is to collect, analyze and report information drawn from health services, community services and housing assistance services’ (AIHW, 2014). Indeed, part of its statutory work as listed under the Australian Institute of Health and Welfare Act 1987, the functions of the Institute include (inter alia) at Section 5 (1)(f) of the Act: ‘to conduct and promote research into the health of the people of Australia and their health services’ (Australian Government ComLaw, 2014). In the absence of collation and analysis of such raw data (we understand that the AIHW does not perform this role at present, though we have suggested that they might consider it), or a regular national blood lead survey of all ages (as have been carried out regularly in the USA since 1976), we used USA population lead exposure data to estimate that approximately 100,000 Australian children under five years of age may be exposed to blood levels above 5 μg/dL, which are likely to cause adverse health effects (Taylor et al., 2012). In the most recent study of blood lead of Australian children in a large urban city (Sydney), Gulson et al. (2008) reported that 7.5% (8/107) of their cohort of children presented with a blood lead >10 μg/dL. This is the last reference value from an urban environment and according to Gulson (2014), p. 372 “the randomness of our sampling in Sydney is sufficient to obtain a reliable estimate of PbB distributions from an urban environment”. Therefore by deduction, this study’s data suggests that around 100,000 Australian children (ABS, 2013) aged 0–4 years will experience an elevated blood lead above 10 μg/dL. The point being is that the evidence suggests that the environmental lead problem has not ‘gone away’. It would be more accurate to state that its true effect on the community is largely silent because of the dearth of contemporary blood lead sampling programs.

Lead exposure from soils and dusts in Australian communities is dominated by three sources: (i) mining and smelting emissions, (ii) lead paint and (iii) leaded petrol. In mining and smelting affected areas, the original contaminant load is sourced primarily from smelter fallout, dust from spoil heaps or tailings that have been transported deliberately or inadvertently into and dispersed across human and natural environments. The release of such contaminants can pose a significant potential environmental and human health threat to people living and working in or near such environments. Lead levels in domestic paint were up to 50% by weight before the 1950s but thereafter several reductions were mandated bringing the allowable concentration to 0.1% in 1997 (Australian Government Department of the Environment, 2013a). Nevertheless, the legacy of paint continues to pose a significant source of lead exposure in Australia (Gulson et al., 2006), though exposures are often a complex mix of sources (Gulson et al., 2013; Laidlaw et al., 2014a).

Unleaded petrol for road vehicles was introduced in Australia in 1985, with the lead content of petrol declining from 0.84 g/L in 1990 to 0.2 g/L in 1996, until it was finally banned in 2002 (Australian Government Department of the Environment, 2013b). The consequences of the use of lead in petrol on environmental quality have been significant. In two national assessments of petrol lead emissions 3842 tonnes of lead were emitted in Australian capital cities in 1976 (Farrington, 1981), and 2388 tonnes of lead were emitted in 1985 (Farrington, 1988), despite the mandated reductions of allowable lead in petrol. As a result, soil in gardens and parks in many of the older, inner city areas of Australia became heavily contaminated with lead (Davis and Birch, 2011; Davis et al., 2011; Laidlaw and Taylor, 2011; Laidlaw et al., 2014a) and it is these sources that continue to expose children to potentially adverse levels of environmental lead. In contrast, smaller rural towns with lower vehicle use and population densities, and no mining and smelting industries are likely to have a less significant legacy of environmental lead exposure compared to major lead mining and smelting cities (Taylor et al., 2010). Exceptions in these locations include older housing where lead paint was used or where the source of drinking water is rainwater, river water or groundwater which has not been tested for lead (Green, 2013).

Although Australia continues to be a world leader in lead metal mining, smelting and processing, there has been a significant lack of research funding directed towards understanding the hazards of these operations. For example, there has never been any major longitudinal study investigating environmental lead plus other metal exposures in Mount Isa or Broken Hill, despite their importance in the Australian landscape and the current and historic childhood (and occupational) lead exposures (Taylor et al., 2011). Despite the fact that lead-impacted mining and smelting communities, councils, mining companies and government have often worked together to reduce exposures, any negative human health impacts arising from lead extraction and refining has been down-played consistently so as not to challenge the status quo and disrupt economic opportunities (Gulson, 2008; Laidlaw et al., 2014b; Lanphear et al., 2008; Taylor, 2012; Taylor and Schniering, 2010). Taylor et al. (2014) detail significant evidence of government agency obfuscation of the magnitude and human health risks associated with atmospheric emissions of lead as well as arsenic, cadmium and sulfur dioxide from the operations at Mount Isa and Port Pirie.

Atmospheric lead emissions from current mining and/or smelting operations continue to pose a major source of exposure at Broken Hill, Port Pirie and Mount Isa, with 28 tonnes; 46 tonnes and 120 tonnes being emitted to the respective ambient environments in 2011/12 (National Pollutant Inventory, 2014). Scrutiny of annual blood lead statistics from Port Pirie and Broken Hill show that childhood blood lead exposures in these towns are elevated and by some measures increasing by comparison to the national and global trend of falling levels. In Broken Hill the geometric mean blood lead of children aged 1–4 years rose from 4.8 μg/dL in 2011 to 5.4 μg/dL in 2012, with the proportion of children over the national goal of 10 μg/dL increasing from 2010 to 2013: 12.6% to 13% to 21% (age–sex standardized values) (Lesjak et al., 2013). In Port Pirie, a similar pattern is emerging with the percentage of children younger than 5 years of age with a blood lead >10 μg/dL remaining above 20%. In 2011, 24.2% of children younger than 5 years of age (excluding maternal blood lead as surrogate values) had blood lead values above 10 μg/dL. In 2012 the proportion of Port Pirie children above the Australian national goal was similar to 2011 at 24.9% and slightly lower in 2013 at 22.7% (Simon and Lewis, 2014). The geometric mean of children aged 24 months, continues to remain elevated: 6.1 μg/dL in 2011, 6.2 μg/dL in 2012 and 6.0 μg/dL in 2013 (Simon and Lewis, 2014). The geometric mean blood lead value for all children under 5 years of age rose in the last reporting year from 4.5 μg/dL to 5.0 μg/dL, indicating strongly that the problem of exposure at Port Pirie persists.

Similarly detailed annual analysis is not available for Mount Isa despite the fact that in 2008 a survey of 400 children aged 1–5 years revealed that 11.3% had a blood lead level above 10 μg/dL (geometric mean 5 μg/dL; Queensland Health, 2008). A smaller survey in 2010 (n = 167 children) showed that the number of children impacted was lower with 4.8% having a blood lead level above 10 μg/dL (geometric mean 4.27 μg/dL; Queensland Health, 2010). From March 2014, Queensland Health Authority has resolved to undertake routine screening for lead when other blood tests are ordered for children aged 0–4 years at Mount Isa Hospital. The objective of the new program is to obtain a more accurate assessment of blood lead, particularly in high risk groups such as Indigenous children and those from lower socio-economic groups (North Queensland Register, 2014).

3. Conclusions

The lead pandemic is not a problem of the past. Further efforts to remain vigilant from both governance and community perspectives are urgently required, particularly in light of the evidence for the effects at much lower levels than the current Australian goal for blood lead. This vigilance needs to be supported by adequate state and national...
government funding given that environmental lead exposures are a matter of public health and its damaging effects are borne ultimately by the whole community. There is now an overwhelming body of evidence showing that Australia’s lead level for children is too high. Therefore, in conclusion, we ask why would it not be the NHMRC so long (2012–2014) to undertake what appears to be a review of reviews (NHMRC, 2014), in the anticipation that they might come up with something different from other global experts? Procrastination on this issue will be the thief of an equitable and healthy start to life for Australia’s lead-exposed children.

References


Davis BS, Birch GF. Spatial distribution of bulk atmospheric deposition of heavy metals in the world. Environ Pollut 2011;159:1


Miekle HW, Zahran S. The urban rise and fall of air lead (pb) and the latent surge and re- treat of societal violence. Environ Int 2012;41:48–55.


Miekle HW, Zahran S. The urban rise and fall of air lead (pb) and the latent surge and retreat of societal violence. Environ Int 2012;41:48–55.


Miekle HW, Zahran S. The urban rise and fall of air lead (pb) and the latent surge and retreat of societal violence. Environ Int 2012;41:48–55.


Miekle HW, Zahran S. The urban rise and fall of air lead (pb) and the latent surge and retreat of societal violence. Environ Int 2012;41:48–55.


Miekle HW, Zahran S. The urban rise and fall of air lead (pb) and the latent surge and retreat of societal violence. Environ Int 2012;41:48–55.


Miekle HW, Zahran S. The urban rise and fall of air lead (pb) and the latent surge and retreat of societal violence. Environ Int 2012;41:48–55.


Miekle HW, Zahran S. The urban rise and fall of air lead (pb) and the latent surge and retreat of societal violence. Environ Int 2012;41:48–55.


