TOPICAL REVIEW

Toward eliminating children’s lead exposure: a comparison of policies and their outcomes in three lead producing and using countries

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Keywords: childhood lead exposure, BLL, comparative policy, lead, Australia, Germany, USA

Abstract
Though the problem of childhood lead poisoning has been recognized for more than a century, it remains an important threat to children’s health and development. This comparative policy assessment examines and compares environmental and public health policies to prevent children’s exposure of three major lead producing countries. Germany, the USA and Australia were used to explore a range of approaches primarily at the national level to identify best practice, how the lack of finding any ‘safe exposure’ level has been incorporated into policy, and to consider if any international harmonization has occurred. We searched the peer-reviewed literature, government websites, policy documents and grey literature to identify how and when policies were developed, implemented and revised. From this analysis, we present a chronology and discussion of national policy approaches for the main sources of lead exposure for children in each country. We found significant differences in timing and comprehensiveness of environmental policies related to lead. There are significant differences among the countries in biomonitoring and identifying pathways of exposure, with limited information available in Germany and Australia. Though there are significant gaps, the US has the most comprehensive regulations relating to old lead paint in housing, while in Australia, a regulatory framework is lacking. Though all three countries regulate lead in air, the US has the most health protective standard, while Australia lacks the ability to ensure that states meet the national standard. Though each country has developed regulatory frameworks for lead that have reduced children’s exposure, none of the three countries have a comprehensive set of policies that respond to the scientific evidence that there is no identified threshold for lead exposure. While there are differences in the relative importance of lead exposure pathways among the three countries, suggesting the need for different prevention emphases, there is also a strong argument for more international harmonization of exposure standards at the most protective levels. Some environmental policies incorporate current scientific understanding of lead toxicity, however, we identified gaps in standards and enforcement, and as a result, exposure continues to affect the health of children in all three countries.

1. Background

Though the problem of childhood lead poisoning has been recognized for more than a century, it remains an important and ongoing threat to children’s health and optimal development globally. Solving the problem of childhood lead exposure continues to be a ‘moving target’ as toxicity has been continuously redefined based on accumulating scientific evidence of harm to children’s health at increasingly lower levels of exposure [1]. Low-lead levels are scientifically reported to cause neurodevelopmental harm to children resulting in decreases in IQ [2, 3], and the World Health Organization states: ‘there is no level of
exposure to lead that is known to be without harmful effects.' [4]

Until the mid 1960s, lead industry researchers shaped, and largely controlled, research about the health effects caused by exposure to lead [5]. These vested interests advocated for a threshold model for health effects which suggested that damage to health only occurred at certain levels of lead exposure—those which caused clinically observable symptoms [6]. Beginning in the 1970s, researchers free of industry influence credibly demonstrated that harm occurred at lower levels of exposure to lead than previously thought [7]. Subsequently, policies to address environmental lead exposure among children began to be developed and implemented at the federal level in Germany and the US. As environmental policies were implemented, children's blood lead levels (BLLs) began to decline. Further research in the late 1970s and 1980s, focused on subclinical effects of lead exposure and showed that lead exposure at levels previously thought to be safe, were not. First in cross-sectional and then in more robust cohort studies, so-called safe levels were found to damage children's intellectual development and negatively affect their behavior [8–10]. Studies that followed in Germany and Australia reported similar findings [11, 12].

By the 1990s, some US researchers who investigated the relationship between lead exposure and children's health and well-being posited that lead, as a neurotoxicant, might have no threshold for harm [13]. Since the early 2000s, the toxic effects of lead at low BLLs have been further elucidated. At <5 µg dL−1, effects include loss of IQ points, reduced achievement in school, problems with attention, and increases in antisocial behavior. Levels <10 µg dL−1 have been associated with problems with hearing and delayed puberty [14–16]. IQ losses associated with ‘low-level’ lead exposure include 7.4 points when ‘lifetime average’ BLLs increased from 1 to 10 µg dL−1 and did not exceed 10 µg dL−1 in one study; [2] and 6.2 points in an international pooled analysis as BLLs increased from <1 to 10 µg dL−1 [3]. Recently, the Port Pirie cohort study reported a loss of 13.5 full-scale IQ points for a 10 microgram increase in blood lead, for example from to 1−10 µg dL−1 [17]. The relationship between IQ loss and blood lead concentration is thought to be supralinear with a greater proportion of IQ loss occurring at lower levels of blood lead [18]. The lower limits of the benchmark dose for the loss of one IQ point was estimated to be between 0.1 and 1.0 µg dL−1 [19].

Low-level childhood lead exposure is only ‘visible’ in epidemiological tallies, but has significant social and economic consequences for communities and societies. Annual costs from the decrease in ‘lost economic productivity’ resulting from the neurotoxic effects of lead have been estimated at US$50.9 billion [20], and in the European Union (EU) at US$57 billion [21]. If the BLL of each child under six years old in the US were reduced to less than 1 µg dL−1, costs savings of an estimated $50 000 per child per year potentially could be realized due to improved educational outcomes and reduced crime [22].

The purpose of this comparative study is to examine how Germany, US and Australia have addressed the problem of children's exposure to lead since recognition of children's health effects from it became widespread in the 1970s. A secondary aim is to compare how prevention policies in these countries have responded to more recent changes in scientific understanding of lead's toxicity for children.

2. Method

An academic search was completed using Medline and Google Scholar using the country name as the first keyword (Germany/US/Australia) with the addition of the following keywords: lead; childhood lead exposure, childhood lead poisoning, lead paint/coatings, lead in gasoline/petrol, lead in drinking water, lead pipes, lead in food, lead in air, lead in soil, lead in house dust, lead surveillance and lead human biomonitoring. Relevant government websites were also searched with the above terms. For Germany these included the websites for the Umwelt Bundesamt (UBA), the German Federal Environmental Agency (www.umweltbundesamt.de), the Bundesministerium für Gesundheit, the Federal Ministry of Health (www.bundesgesundheitsministerium.de) and the European Commission (https://ec.europa.eu). For the US the main websites searched were those for the Centers for Disease Control and Prevention (www.cdc.gov), the Environmental Protection Agency (www.epa.gov/) and the Department of Housing and Urban Development (www.hud.gov). For Australia the main websites included were the National Health and Medical Research Council (www.nhmrc.gov.au) and the National Environmental Protection Council (www.nepc.gov.au/). Subsequently, relevant sources for known hotspot areas were followed up for each country by location name.

We then followed references to identify government reports and grey literature for each country to provide an overview of the ways in which the problem of childhood lead poisoning has been understood and framed within each country, and the policy approaches each country has taken, primarily at the federal level, to address the major pathways of exposure to lead for children over time (air, leaded gasoline, paint/house dust/soil, food and drinking water).

The countries of Germany, the US and Australia were selected because they are all high-income countries with the regulatory infrastructure and economic capacity to largely solve the problem of childhood lead exposure. Each country has a history of extensive lead production and use, and the lead industry has had a significant role in the country’s economy. All three countries have a shared federal
and state/territory governance structure, with federal environmental and health agencies and state and/or territory level analogues. Germany is embedded within the governance structure of the EU, consequently the country may set its own regulations for lead and also must comply with EU regulations. This comparative assessment of the policy approaches (table S1 (available online at https://stacks.iop.org/ERL/15/103008/mmedia)) and prevention strategies of the three countries provides an opportunity to compare and contrast these countries’ approaches to reducing children’s exposure to lead, and to make inferences about which have been the most effective approaches to date.

3. Results

Worldwide, approximately five million tons of lead are mined annually and nearly six million tons are produced through secondary means [23]. Historically, parts of Germany with lead mining/smelting include the Harz Mountains, Lower Saxony; Eifel Mountains, in the North Rhine Westphalia area; Freiberg, Saxony; and Hettstedt, Saxony-Anhalt. Today in Germany there is still active primary and secondary lead production in Stolberg, North Rhine Westphalia; Braubach, Rhineland-Palatinate; and near Freiberg at three smelters [24]. In 2012, the country’s production was 4% of the world’s total, and 5% of global recycled lead. In the same year, Germany was the world’s fifth largest user of lead [23].

The US was a significant producer of primary lead in the twentieth century, with lead mines and smelters in Missouri, Idaho, California, among other states, and a long history of environmental and health concerns around these point sources [25]. Cleanup of extensive environmental damage continues around many of them because they operated without significant pollution controls for much of their lifetimes. The last primary lead smelter in the US (Herculaneum, MO) closed in 2013. In 2012, the US mined 6.9% of the worldwide total of primary lead. The US is also the world’s second largest producer of recycled lead and the second largest user of lead [23].

Australia is the world’s second leading producer of primary lead, behind China, and produces 11.5% of the world’s total [23] from mines at Boroloola, Northern Territory; Broken Hill, New South Wales (NSW); and Mount Isa, Queensland (Qld). Smelters at Port Pirie, South Australia (SA); Boolaroo, NSW (historic) and Mount Isa, Qld, process the lead from the country’s mines. There is a long history of large scale lead mining and smelting in Australia and widespread childhood lead exposure has been, and continues to be, documented in the towns and cities near many of Australia’s lead mining and smelting sites [26–28]. Australia is not among the top ten secondary producers of lead worldwide, though there are secondary smelters (battery recyclers) in Melbourne and Sydney [29], nor are they among the top ten users of lead in the world [23].

3.1. Quantifying children’s lead burden (1970s–1980s)

Despite childhood lead poisoning attributable to the ingestion of contaminated house dust from deteriorating lead paint being first described in the medical literature at the turn of the twentieth century [30], it did not become the subject of concerted federal government prevention efforts in the US until the 1970s. It has been argued that this was in large part attributable to the lead industry influencing research and public policy, and shifting the blame for lead poisoning to ‘defective children and their ignorant families, not paint on walls and woodwork’ [31], as well as denying the widespread harm produced by burning leaded gasoline [5]. Though some US cities tried to regulate lead in paint and identify poisoned children in the 1950s, by the 1970s, the problem had reached epidemic proportions in many US urban areas where both deteriorating lead paint and leaded gasoline poisoned children [32]. Attention to the crisis in the US influenced the modern age of research in Europe and Australia.

In Germany, the prevalence of childhood lead poisoning was not well quantified in the early 1970s, but limited available data supported the notion that the very high BLLs seen in the highest exposed US children were generally not found among children in what was then the Federal Republic of Germany (West Germany). Hotspots of elevated lead exposure were identified among children living near mining and smelting sites and other industrial areas, however, in comparison to US children, and to the prevailing standard for ‘undue lead absorption’ of 40–50 µg dL⁻¹, it was not the dire crisis that was being documented in the US. For example, a 1974 study of BLLs in two to 14 year old children in a contaminated area in North Rhine/Westphalia found 15% of children with BLLs <10 µg dL⁻¹, 56% with BLLs between 10 µg dL⁻¹ and 19 µg dL⁻¹ and 1.5% with BLLs of 40 µg dL⁻¹ and over. The authors compared their findings to a study of ‘at-risk’ children in Kansas City, US (defined as low hemoglobin, anemia, pica or a sibling of child identified with elevated blood level) in which 27.3% had BLLs over 50 µg dL⁻¹. From that perspective the BLLs found in Westphalia children seemed unremarkable [33]. One small study from the town of Bautzen in East Germany reported mean BLLs ranging from 19 µg dL⁻¹ among 1–3 year olds to 37 µg dL⁻¹ in 7–12 year olds which the authors attributed to lead in air [34].

Leaded gasoline was used in Germany beginning in the late 1930s [35] and caused widespread elevations in children’s BLLs. The main sources of children’s exposure identified there in the 1970s and early 1980s were industrial pollution and motor
vehicle exhaust. Some studies also identified take home exposures from parents exposed to lead at work [36–38].

The first representative survey of BLLs was conducted in 1979 by the European Community, and included children ages six to 14 years of age. Over 2000 children in West Germany were tested. The arithmetic mean BLL in 1979 was 11.8 µg dL\(^{-1}\) and the 98th percentile of exposure was 24 µg dL\(^{-1}\) [37], quite similar to the mean of 11.7 µg dL\(^{-1}\) and 95th percentile 22 µg dL\(^{-1}\) found in US six to 19 year olds between 1976 and 1980 [39].

In West Germany, however, childhood lead exposure was not seen as the crisis it was in the US. There was a view that lead paint was the cause of the very high BLLs in US children—and that lead paint exposure was essentially a US problem [40, 41]. There was little data on very young children in West Germany, those most at risk of elevated levels. With the exception of a study that measured lead in cord blood in an industrial area of the country conducted in the 1970s [42], most studies included children six years of age and older. Even today, national biomonitoring includes only children ages three years of age and older. However, though the very high BLLs being found in inner-city children in the US during this time were not documented in West Germany, some German regulators and researchers viewed environmental lead contamination as a problem necessitating regulatory action in the 1970s and 1980s. [43]

In the US, systematic attempts to understand the extent of childhood lead poisoning from paint were just beginning in the early 1970s. A 1972 study estimated 600 000 to 708 000 children could have BLLs ≥40 µg dL\(^{-1}\) and of those, over 66 000 may have BLLs in excess of 70 µg dL\(^{-1}\) [44]. Stark racial and economic inequality compounded the problem, with the most at risk children living in low cities in dilapidated housing [7]. The first nationally representative data would be published a decade later (NHANES II, 1976–1980) and showed that only a minority, 12.2%, of US children had BLLs less than 10 µg dL\(^{-1}\). Among children ages six months to five years the mean BLL was 16.0 µg dL\(^{-1}\). African American children had a mean BLL much higher than white children—20.9 µg dL\(^{-1}\) compared to 14.9 µg dL\(^{-1}\) [45].

In Australia, in the early 1970s, despite developing some of the earliest literature on childhood lead poisoning, little was documented about the extent of the childhood lead poisoning problem. Studies conducted in the 1970s and 1980s pointed to the possibility of a significant problem, although they were geographically limited and tended not to focus on the youngest children (BLLs are highest around age two) [46], those most at risk, thereby likely underestimating average BLLs in these populations of children. For example, a 1974 study in Tasmania which included children ages four to 16 years old reported a mean BLL >17.4 µg dL\(^{-1}\) (urban) and >17.8 µg dL\(^{-1}\) (suburban). The authors reported, ‘this is of concern as 25 µg dL\(^{-1}\) is the currently suggested ‘danger’ blood lead level for children.’ [47] Garnys et al (1979) studied school aged (5–11 year old) children in Sydney and reported a mean BLL for the spring of 1974 of 20.8 µg dL\(^{-1}\) (5 to 60 µg dL\(^{-1}\)) and in the autumn of 1975 a mean of 17.0 µg dL\(^{-1}\) (1–90 µg dL\(^{-1}\)) [48]. Both studies pointed to lead in petrol as the most likely source of exposure. These findings touched off other studies, some clearly funded by industry, that criticized their methods and downplayed the problem of children’s exposure to lead [49, 50].

Over 160 000 tons of lead had been cumulatively emitted from the smelter at Port Pirie by the early 1980s. BLLs were tested in more than 1000 one to 14 year old children in 1982, though less than 10% of the sample was ages four years old or less. Predictably, the younger children (1–4 year olds) had the highest mean BLL of 22.1 µg dL\(^{-1}\). However, every age group had average BLLs >15 µg dL\(^{-1}\), and the overall average was 18.2 µg dL\(^{-1}\). Seven per cent of the children sampled had BLLs >30 µg dL\(^{-1}\) [51]. Only after the 1986 publication of McMichael et al’s prospective study of pregnant women in Port Pirie, which found that pre-term birth was positively associated with mothers’ BLLs, did the issue get national level attention [52].

3.2. Present day lead exposure

Control of environmental lead which began in the early 1970s has dramatically lowered average BLLs among children in all three countries. In Germany, the most recent available data are from the 2003/2006 German Environmental Survey (GerES), a representative biomonitoring study that measures exposure to chemicals in the population and assesses some sources and pathways of exposure. The GerES focuses on either children or adults, depending on the survey wave. Starting with the GerES in 1991/1992, which occurred after reunification, residents of the former East Germany are included in the sample. Though the most recent wave of the GerES (2014–2017) includes children, these data were not available at the time of publication. In 2003/2006, among children ages three to 14 years old, the geometric mean (GM) BLL was 1.6 µg dL\(^{-1}\) (children less than three are not tested in the GerES). Among three to five year olds the GM BLL was 1.9 µg dL\(^{-1}\) and among 12 to 14 year olds it was 1.4 µg dL\(^{-1}\). The 98th percentile of exposure for three to 14 year olds was 4.2 µg dL\(^{-1}\). Higher average BLLs were reported among those living in the former East Germany and among those of lower socioeconomic status (SES) [53].

Of the three countries, the most comprehensive representative data are from the US NHANES where, since the late 1970s/early 1980s, the median BLL in one to five year old children has fallen by 95% from 15.0 to 0.7 µg dL\(^{-1}\) [54]. The 97.5 percentile
of the 2011–2014 NHANES was 3.48 μg dL⁻¹ [55]. While the overall GM BLL decreased again among 1–5 year olds between 2013–14 and 2015–16 from 0.782 μg dL⁻¹ to 0.758 μg dL⁻¹, at the higher percentiles of exposure, GM BLLs increased in 2015–16. For example, the 95th percentile of exposure was 2.24 μg dL⁻¹ in 2013–2014 and 2.76 μg dL⁻¹ in 2015–2016 [56]. Dignam et al cite unpublished CDC data which showed that in 2015–2016, 0.2% of US 1–5 year olds had BLLs ≥10 μg dL⁻¹ and 1.3% had BLLs ≥5 μg dL⁻¹ [57].

There are no current nationally representative data on the mean BLLs among children in Australia since there is no national biomonitoring program or blood lead surveillance program, nor are there representative data on the proportion of children with BLLs greater than 5 μg dL⁻¹, the current intervention level. The only national study of children’s BLLs was published more than twenty years ago in 1996. At that time, the arithmetic mean BLLs in a sample of 1575 one to four year old children nationwide was 5.72 μg dL⁻¹. Indigenous children had a higher mean BLL, 7.45 μg dL⁻¹, and 18.5% had BLLs greater than 10 μg dL⁻¹. The researchers estimated that 75 000 Australian children one to four years old would have had BLLs greater than or equal to 10 μg dL⁻¹. Lead paint was one of the more important exposures identified [58].

Annual surveillance occurs only around some mining and smelting sites where children’s BLLs greatly exceed levels found in nationally representative samples in the US and Germany. For example, the GM BLL among children under five years old in Port Pirie was 6.8 μg dL⁻¹ in the first quarter of 2019. Among children aged 24 months, it was 7.5 μg dL⁻¹ and 9 children were found to have BLLs >20 μg dL⁻¹ [59]. In Broken Hill, a lead mining city where BLLs are measured annually, the GM BLL among children under five years old in 2017 was 5.7 μg dL⁻¹, and among Indigenous children it was 8.7 μg dL⁻¹ [60]. Data reported in 2018 showed that a quarter of young children in Mount Isa had a BLL >5 μg dL⁻¹ [61]. There are limited data for Indigenous children in Mount Isa, even though disparities have been documented. In Port Pirie, where annual blood lead surveillance among children is carried out, average BLLs levels have increased in the most recent monitoring results [59] and similarly in Broken Hill, the average BLL among Indigenous children showed an increase [60].

Differences in children’s average BLL, and our knowledge of them, among these three countries suggest a closer examination of their approaches to preventing childhood lead exposure primarily at the federal level is warranted. Germany has focused largely on primary prevention in the form of regulatory and policy initiatives to eliminate or control environmental sources of exposure. The US has used both primary and secondary prevention combining regulation and policies to control environmental sources of exposure with screening, surveillance and health education. Australia has implemented some primary prevention through regulation to control some environmental sources of lead, but relies heavily on secondary prevention in the form of health education and behavior change recommendations. The following sections detail these approaches so that best practices can be identified and discussed for their potential for re-application via international policy harmonization.

### 3.3. Overarching approach to prevention—Germany

Since the 1970s, Germany has focused mainly on primary prevention of lead exposure and has put less emphasis on other public health approaches such as testing BLLs and health education, though some biomonitoring has occurred near industrial sites [62] and some health education has been carried out with respect to lead exposure from water pipes.

At the federal level, most government responsibility for environmental lead research and policy is housed in the UBA, the German Federal Environmental Agency, which is responsible for many aspects of environmental policy. The Bundesministerium für Gesundheit, the Federal Ministry of Health, collaborates with the UBA regarding lead in drinking water. Many of the laws that regulate levels of lead in the environment are federal, such as those relating to air quality, soil, water quality, and waste. However, the day-to-day implementation of environmental and public health laws and policies occurs at the state level through environmental agencies and health departments and thus is subject to variability [63–65].

Since 2010, federal government publications generally endorse the scientific finding that there is no identified ‘safe’ level of lead exposure, and that exposure should be reduced as much as possible. For example, UBA’s Human Biomonitoring Commission, in 2010 ‘suspended’ previous human biomonitoring levels which were based on health effects, due to their view that deriving a health protective value for lead is not possible, given the large body of literature on lead’s health effects at low levels, and the determination of the German MAK Commission that lead is ‘to be regarded as a human carcinogen.’ Instead, a blood lead reference value, reflecting the 95th percentile of the GerES IV of 3.5 μg dL⁻¹ was set as a guideline above which investigation of a child’s sources of exposure should occur [66], though there is no national program to systematically identify these children.

Germany does not have a federal strategy for lowering children’s exposure to lead. After the removal of lead from gasoline and control of some other environmental sources, children’s BLLs declined across the population, and lead exposure has been seen as a problem that has largely been
solved [67]. However, given the scientific evidence that there is no identified safe level of lead, there is currently a reassessment of risk for the population, though this is not specific to children [68]. There are current research projects focused on assessing risk from specific sources such as food and game meat as food is thought to be the most important pathway of exposure in the general population.

3.3.1. Lead in paint
Since the 1970s when Germany began concerted efforts to control environmental lead, lead in paint has not been considered an important exposure pathway for children, likely because actions were taken early in the twentieth century to limit the use of white lead in the interiors of buildings for the protection of workers’ health. Beginning in 1905, responding to pressure from painters’ unions which sometimes saw their members fatally poisoned, Germany set limits and specifications for the handling and use of white lead to protect the health of workers. These included prohibitions on women and males younger than 18 years old from working with white lead, and prohibitions on dry sanding and mixing of dry pigments. An educational brochure, hygiene facilities and periodic medical supervision was also required [69].

Though many European countries did, Germany did not sign the International Labour Organization’s (ILO) Convention on White Lead adopted in 1921, which limited the lead content of paint used indoors to 2% [70]. However, a 1930 law aimed to bring Germany ‘into accordance with the Convention.’ In particular, the law reiterated many of the 1905 restrictions and limited the lead content of interior paint to 2% [71] but permitted exceptions for paint containing up to 5% lead until 1938. Later amendments in 1938 and 1956 allowed the interior use of leaded zinc oxides containing up to 5.5% lead [72]. In West Germany in 1980, the lead content of paint for interior residential use was limited to 1% [73]. A 1989 EU law prohibited most lead in paint for interior use, except in art restoration and historic buildings [74]. Since 2006, under REACH [75], the EU restricts the use of lead carbonates and sulfates in paint. The Global Alliance to End Lead Paint recommends that countries adopt a limit on total lead content of 90 ppm, rather than chemical specific regulations [76].

More recently, in the German construction safety literature a discussion has arisen about the presence of lead coatings on interior and exterior wooden components such as windows, wooden doors and even base paints indoors. A mid 1990s study reported on workers with who stripped lead paint from wood and then were found to have BLLs between 28.1 and 81.2 µg Dl⁻¹ [77]. Hazardous lead concentrations have been found in older layers of paint, particularly on wooden components such as windows and doors where white lead, lead-zinc and titanium blends, lacquers, and lead drying agents were used [78]. In 2015, BG BAU (provider of statutory accident insurance to the German construction industry) published guidelines for dealing with lead in coatings from an occupational safety perspective [79]. Lead present inside or outside of buildings that presents a risk to workers can also present a risk to children, particularly during demolition or renovation work. This potential risk to children has not been systematically evaluated.

Some studies of children’s BLLs in Germany have found a relationship between higher BLLs and older homes—but paint has not been regarded as an important factor [73]. Consequently, there is no federal regulatory framework to protect children in housing that may contain lead-based paint or lead contaminated dust. Children’s exposure to lead in paint or house dust contaminated by this source cannot be ruled out, particularly if renovation disturbs old lead paint that may be underneath lead-free top coats. The abandonment of a threshold for lead toxicity in children may make this pathway relevant in some cases.

3.3.2. Lead in gasoline
Ledged gasoline was introduced in Germany in the late 1930s. By the late 1960s, there were concerns about its health effects. In 1972, the maximum allowable concentration of lead in gasoline was lowered from 0.6 to 0.4 g Pb l⁻¹, and then to 0.15 g Pb l⁻¹ in 1976. As a member of the European Economic Community, Germany could not ban leaded gasoline in the early 1980s, as member states were prohibited from reducing concentrations below 0.15 g Pb l⁻¹ [80].

In 1984, unleaded gasoline was introduced to the German market, to accommodate the catalytic converter, needed in part because of damage to forests from nitrogen oxides in motor vehicle exhaust, and by 1998, leaded gasoline had been completely removed. Children’s health was not the motivation behind the ban on leaded gasoline [81]. BLLs in German children decreased substantially during the period in which lead content was reduced—identified in several studies in different regions of the country [82, 83].

3.3.3. Lead in air
In the early 1970s, significant lead exposure in children living near industrial emitters of lead was identified. The former West Germany introduced a standard for lead in air in 1974 (TA Luft 1974) of 2 µg m⁻³ in suspended particulate and concentrations of lead in air began to decline [84]. The current limit for lead in air is 0.5 µg m⁻³ (EU regulation), averaged over one year [85]. Between 1990 and 2010 there has been a reduction in lead emissions in Germany of approximately 91% [86]. The highest air lead reading in 2017 of 0.175 µg m⁻³ (annual average), was in Duisberg, a city with metal processing and a coal
port. The second highest lead in air reading in 2017 of 0.115 µg m⁻³ was in Braubach, a city with a secondary lead smelter [87].

3.3.4. Lead in drinking water
As lead concentrations in air were falling due to the reduction of lead in gasoline and industrial emissions, lead exposure in drinking water became more of a research and intervention focus. Though lead pipes were outlawed in some areas of the country in the late 1800s for health reasons and apparently little used in the south of Germany, in northern Germany lead pipes as service lines and in residences were installed until the mid-1970s. [88]

Lead poisoning attributable to contaminated water from lead pipes has occurred in Germany (e.g. Leipzig, 1930) [89] and numerous studies have documented elevated concentrations of lead in drinking water from locations around Germany in more recent decades. For example, a 1985 study in Schleswig Holstein found 21.2% of drinking water samples had lead levels between 40 µg L⁻¹ and 300 µg L⁻¹, and a maximum concentration of 2470 µg L⁻¹ [90]. In the 1990/92 German Environmental Survey, the 98th percentile for lead in drinking water was 50.7 µg L⁻¹ and the maximum concentration found in first draw sampling was 5580 µg L⁻¹ [91]. A study in southern Saxonia found elevated water lead and blood lead in children ages nine to 12 years old in the early 1990s, with mean and maximum water concentration of 24 µg L⁻¹ and 2600 µg L⁻¹ and mean and maximum BLLs of 8.3 µg dL⁻¹ and 34 µg dL⁻¹. Half of the houses surveyed had lead pipes and ineffective corrosion control had occurred. The authors urged replacement of lead pipes [92]. A national study which assessed data from 2007 found 4.7% of water samples exceeded the lead standard [93].

The EU set new regulations for lead in drinking water in 1998, first lowering allowable lead content in drinking water to 25 µg L⁻¹, with a final standard of 10 µg L⁻¹ in 2013 [94]. This has provided impetus for replacing lead service lines and interior lead pipes during residential renovation, the preferred approach to the problem. In 2013 when the 10 µg L⁻¹ standard came into force, the UBA said, 'The new limit makes lead pipes in the water supply system practically useless. If the lead limit in a pipe is exceeded the public health office may oblige the water utility or the landlord to eliminate the cause of the exceedance. What it in fact usually means is that lead pipes must be entirely replaced.' [95] Water suppliers have been replacing lead service lines over the last several decades. The decline in GM BLL that occurred between the 1980s and 2009 in young adults have been partially attributed to lead pipe replacement [96].

Currently, landlords must be notified by their water company if lead pipes remain in the water distribution system [97]. Lead pipes may still be present in older buildings that have not been renovated, and, if so, landlords are required to inform tenants even if there are no exceedances of the lead standard [95]. Fixtures may contain lead and thus may be another source of exposure [98].

The European Commission is currently recommending a further lowering of the lead in drinking water standard from 10 µg L⁻¹ to 5 µg L⁻¹ to be phased in over a ten year period [99].

Rainwater harvesting is common in Germany but collected rainwater is not used for drinking water, only for other household use and for gardens. Therefore, this is not an important pathway of exposure [100].

3.3.5. Lead in food
In Germany food and drink are thought to be the primary source of exposure to lead in the general population. A 2011 WHO report estimated that children in Europe have higher average lead intake from food than children in the US and Australia [101]. Endorsing a no threshold model for lead neurotoxicity, the European Food Safety Agency (EFSA) established a benchmark dose in 2010 for exposure to lead in food of 0.5 µg kg⁻¹ bw⁻¹ day⁻¹, tied to neurotoxicity outcomes in children. Mean exposure to lead in food was estimated to exceed the benchmark dose in toddlers, and two other child age groups in a 2012 study of various European populations [102]. The EU sets maximum limits for lead in food and recently lowered these limits in some foodstuffs such as infant formula [103]. A recent study in Germany found that very young children consuming ‘baby formula in powder form’ and certain infant foods have exposure below EFSA’s benchmark dose but exposures should be reduced to levels ‘As Low As Reasonably Achievable.’ [104] Studies are on-going to gain a fuller picture of children’s exposure to lead in food across various age groups [105, 106]. A recent study in Germany of game meat shot with lead-based ammunition found that regular consumers of game meat are at risk of increased lead exposure [107]. The European Chemicals Agency (ECHA) is currently evaluating evidence and considering restrictions on the use of lead based ammunition to reduce environmental and human health harms from this source [108].

3.3.6. Lead in soil and contaminated sites
Lead contaminated soil can be an important source of exposure for children from soil ingestion outdoors or indoors as a component of household dust. Apart from contaminated sites, soil has not been a significant focus of prevention efforts. A recent survey of soils across the EU found that the highest concentrations of lead in soils were in mining and industrial areas, including in Germany [109]. Urban areas may also be contaminated from leaded gasoline or deteriorated lead paint. A study in Berlin sampled over 5000 locations including allotment gardens, playgrounds,
yards of homes, and found maximum lead concentrations at allotment gardens of 10 459 mg kg$^{-1}$, in playgrounds 769 mg kg$^{-1}$, and in yards, 391 mg kg$^{-1}$ [110].

In the late 1990s, Germany adopted two laws focused on soil contamination, one a precautionary measure to prevent contamination and the other to clean up existing contamination [111]. Under the Soil Protection and Contaminated Sites Ordinance, standards for lead in soil are 200 mg kg$^{-1}$ for playgrounds, 400 mg kg$^{-1}$ for residential yards, 1000 mg kg$^{-1}$ for parks and 2000 mg kg$^{-1}$ for industrial sites [112]. States take the lead in clean-up of most contaminated sites while the federal government is responsible for federal sites [113]. As a result of German reunification there were a large number of contaminated sites inherited from the former East Germany, some of which were former lead mining/smelting sites. About 1000 contaminated sites of various types are remediated each year in Germany, but according to the Federal Ministry of the Environment, ‘complete remediation of all contaminated sites is a task for the entire century and can only be carried out step by step.’ [114] Some remediation has also occurred in the Harz Mountain areas, where mining and smelting has been practiced for hundreds of years and soils, vegetation, water, wildlife and people have been burdened by heavy metals. Soil and sediment contamination in the Harz is still an important problem [115–117].

3.3.7. Occupational/take-home exposures

Preventing occupational exposure to lead is also important for protecting children’s health. So-called take-home exposures including lead dust on a worker’s clothing, shoes, hair, personal possessions, or in vehicles can be a source of exposure for children. Additionally maternal BLLs of less than 5 µg dL$^{-1}$ are associated with lower birth weight [15] and those as low as 5 µg dL$^{-1}$ with cognitive deficits in offspring [118]. Take-home exposure was identified as a risk factor for elevated BLLs in children in Germany in several studies in the 1980s [36–38], however we did not identify any recent studies in the literature discussing this as an on-going risk factor. The German Committee on Hazardous Substances (AGS) in 2017 proposed to lower the Biological Limit Value (BLV) for lead in blood to 15.0 µg dL$^{-1}$ [119] from 40 µg dL$^{-1}$ for men and 30 µg dL$^{-1}$ for women under 45 years of age. The ECHA recently recommended the same BLV for adoption by the EU. Additionally, in their evaluation of the science, they recognized the difficulty of setting a level applicable to women of childbearing age that would protect the fetus from cognitive effects, and that such a BLV would have to be <5 µg dL$^{-1}$ [120].

3.4. Overarching approach to prevention—US

Since the 1970s, to address childhood lead exposure, the US has used a combination of primary prevention approaches, including controlling/eliminating environmental lead; and secondary prevention, including screening and health education approaches. Responsibility for childhood lead exposure prevention at the federal level is shared among a number of agencies. Key agencies include the federal Environmental Protection Agency for regulatory efforts across environmental media, Centers for Disease Control and Prevention (CDC) for health policy and surveillance, and the Department of Housing and Urban Development (HUD) for housing policy and remediation.

US federal agencies appear to accept the scientific consensus that there is no identified safe level of lead exposure for children [54]. To identify the BLL at which intervention should begin, in 2012, the US began using a reference level of 5 µg dL$^{-1}$ of lead in blood based on the 97.5 percentile of NHANES 2007–2010 BLL distribution in 1–5 year olds [121]. In 2017, the Lead Poisoning Prevention Subcommittee recommended lowering the reference level to 3.5 µg dL$^{-1}$ to reflect the lower BLLs in more recent NHANES surveys, however, the CDC has yet to act on this recommendation [122].

The US has developed several federal strategies to address childhood lead poisoning by coordinating and focusing the efforts of federal agencies. In 1991 both the CDC and EPA developed strategies, CDC’s was primarily focused on abating lead paint to eliminate children’s exposure from this source, while EPA’s was multimedia in focus and included addressing soil contamination at Superfund sites [123, 124]. In 2000, the President’s Task Force on Environmental and Safety Risks to Children, which grew out of children’s health initiatives started under the Clinton Administration, developed the first strategy in a collaborative process across key federal agencies. These activities were mostly focused on lead exposure from paint and laid out a path to eliminate ‘childhood lead poisoning in the United States’ through a ten year plan focused mainly on remediation of housing where low-income children live [125]. The most recent federal lead strategy was released in 2018 and focuses on paint, water and soil as major sources but does not set as a goal elimination of lead exposure, only its reduction [34].

3.4.1. Lead in paint

One of the key drivers of elevated BLLs in US children has been, and continues to be, deteriorating lead paint that contaminates house dust or soil [14]. The history of how this occurred is recounted in several detailed accounts [5, 31]. The US was not a signatory to the ILO Convention on White Lead, and not until the early 1970s did the federal government act, first prohibiting the use of lead paint in federal housing and defining lead based paint as containing more than one per cent lead [126]. In 1977, the Consumer Product Safety Commission banned lead in paint and coatings intended for consumer use, including residential
paints, effective in 1978. Lead paints were defined as those containing more than 600 ppm (0.06%) lead content. In 2009 the definition of lead paint was revised to include paints with lead content greater than 90 ppm (0.009%) [127].

A mid-2000s HUD prevalence study estimated that 37.1 million US homes have lead-based paint, and of those 23.2 million have ‘lead-based paint hazards.’ There are still an estimated 3.6 million residences in the US in which children under six years of age may be being exposed to lead-based paint hazards [128].

Legislative efforts to address the toxic legacy of lead in paint in US housing at the federal level began with the Lead Based Paint Poisoning Prevention Act of 1971 [129] which called for identifying and treating children who were lead poisoned, and provided grants for some remediation. However, the law only applied to federally owned or subsidized housing and it did not employ a primary prevention approach, that is, eliminating risks before children are poisoned. Amendments in 1987 sought to address some of the problems by requiring inspection of federal housing and for HUD to come up with a strategy for addressing lead paint health risks in private housing [130].

The 1992 Residential Lead-Based Paint Hazard Reduction Act (Title X) addressed private housing by requiring disclosure to tenants and buyers of the possible existence of lead paint hazards in most housing built before 1978 and education on how to reduce risks [131]. It also tightened requirements for federally owned and assisted housing including assessment of lead hazards and implementation of controls if hazards are found. HUD’s Lead Safe Housing Rule specifies the agency’s rules for federally assisted housing by property type [132]. HUD has provided $2.1 billion in grants from 1993 to 2016 to reduce or lead paint hazards, with a particular focus on the 1.1 million housing units in which low-income children less than six years old reside. Between 1993 and 2016, 195,000 homes have had lead hazards reduced through HUD grants, however appropriations were significantly below what a 2000 Presidential Task Force recommended [133].

EPA’s 2008 Lead Renovation Repair and Painting rule requires lead safe work by trained contractors or workers when disturbing lead-based paint during home or child care renovation, repair or painting. The rule applies to preschools, kindergarten classrooms, childcare facilities and homes built before 1978 [134].

Though the rule has the potential to reduce children’s lead exposure, a recent report by EPA’s Inspector General finds problems with implementation and enforcement [135].

Despite this federal regulatory framework, there are still significant gaps in protecting children. For example, HUD has lower inspection standards for tenant based rental assistance programs than for federally owned public housing and there is no federal requirement to test and remediate private homes that contain lead based paint hazards [136].

In addition to federal regulations states and municipalities may have their own laws pertaining to lead risks in rental housing or other private housing. However, only a handful of states/municipalities require proactively identifying and remediating lead hazards that may harm children [137]. Massachusetts [138] is one of the states that requires a proactive approach, and a study that evaluated the state’s law found that it may help to protect children from recurrent lead poisoning [139].

The ban on lead in paint for residential use and other laws and policies to reduce children’s exposure to lead in paint have been credited as partly responsible for the significant decline in children’s BLLs that has occurred since the late 1970s [140]. However, a recent EPA modeling study of sources of exposure for children found that for infants (0-6 months) and young children (1 to <2 year olds) at the highest exposure percentiles, the soil/dust ingestion pathway, which includes lead from paint and legacy contamination from leaded gasoline, contributed the most to blood lead [141]. Due to the significant dimensions of problem of lead poisoning from paint in the US, there has been a tendency on the part of public health advocates to focus on this source to the exclusion of others.

Both deteriorating paint and outdoor lead contamination in soil from leaded gasoline and exterior paint contribute to lead in house dust and soil [142]. Several regulatory initiatives to address lead paint discussed above help to limit lead in house dust, and there are federal standards for lead in house dust under the Toxic Substances Control Act (TSCA) for concentrations on floors and windowsills. The lead dust hazard standard is used to identify hazardous concentrations of lead dust and the clearance standard is used to assess remaining hazards after abatement or other work is done that falls under HUD’s Lead Safe Housing Rule [143]. It may also be used as a standard or guideline by other government agencies or entities. The lead dust hazard standard was recently revised; new standards for assessing the risk of lead in dust are 10 µg ft$^{-2}$ (down from 40 µg ft$^{-2}$) for floors, and 100 µg ft$^{-2}$ (down from 250 µg ft$^{-2}$) for windowsills. However, EPA kept the old standards in place for post-abatement clearance, leading to a confusing situation for building occupants and contractors. Previously the pre and post abatement dust standards were the same [144, 145]. The EPA has recently proposed that the clearance standards match the hazard standards [146]. At a lead dust floor concentration of 40 µg ft$^{-2}$, a child has a 50% chance of having BLL $\geq$ 5 µg dL$^{-1}$, whereas at 5 µg ft$^{-2}$ there is only slightly more than a 10% probability [14].
3.4.2. Leaded gasoline

Concerns about leaded gasoline in the US date to when it was first added as an anti-knock agent in the early 1920s. Occupational poisoning and deaths initiated the concerns, with scientists such as Alice Hamilton, foretelling that damage to human health would follow [147]. Leaded gasoline consumption was at its highest in the US in the early 1970s, when an estimated 270,000 metric tons were used annually. More than 80% of all leaded gasoline used in western countries was attributable to US use prior to 1970 [148].

The initial impetus for removing lead from gasoline was to allow for the use of catalytic converters to meet new emissions standards for motor vehicles ushered in by the 1970 Clean Air Act. Initial steps included requiring one grade of unleaded gasoline for sale in the US by mid 1975. Children’s health concerns were a significant consideration in the gradual reductions of lead levels in gasoline through the 1980s, and by 1996 leaded gasoline was banned in the US [149]. However, getting lead out of all grades of gasoline was not straightforward. The lead industry downplayed its dangers for decades in order to keep leaded fuel in the market [5]. The removal of lead from gasoline is credited with making a large contribution to the reduction in children’s BLLs.

3.4.3. Lead in air

The contribution of industrial air emissions to children’s BLLs was recognized in the early 1970s when children living near lead smelting facilities were found to be lead poisoned. After a successful lawsuit by the Natural Resources Defense Council (NRDC), lead became a criteria air pollutant in 1976, with a national ambient air quality standard (NAAQS) of 1.5 μg m⁻³ set in 1978 [150]. Lead is also regulated as a hazardous air pollutant with additional emissions requirements. Restrictions on leaded gasoline and industrial emissions of lead have led to a reduction of 99% in ambient lead concentrations since 1980 [151]. The NAAQS for lead was revised in 2008, again after a lawsuit concerning a lead smelter in Herculeanum, MO. The new standard of 0.15 μg m⁻³, averaged quarterly, was set to prevent IQ loss in children, among other negative health outcomes [152]. EPA lists 21 nonattainment areas in 15 states [153].

Leaded aviation gas, used by piston-engine powered aircraft is the largest source for lead in air emissions currently. The EPA has been evaluating this source for regulatory action since 2010, and was expected to decide on an endangerment finding by 2018 but missed that deadline [154].

3.4.4. Lead in drinking water

Since the promulgation of the Safe Drinking Water Act in 1974, and the 1975 National Interim Drinking Water Regulations, lead in water has been a regulated contaminant at the federal level, however, deficiencies in regulatory efforts to date have precluded eliminating this exposure source [155]. Lead in drinking water is a long-standing problem in the US because lead service lines and lead pipes were widely used, and could have been installed, as late as the mid-1980s. In addition to lead pipes, lead solder and lead in fixtures also play a role in drinking water contamination, up to 8% lead was allowed in plumbing products and fixtures until a revised law passed in 2011 came into effect in 2014 [156]. The 2011 law ‘establishes the definition for ‘lead free’ as a weighted average of 0.25% lead calculated across the wetted surfaces of a pipe, pipe fitting, plumbing fitting, and fixture and 0.2% lead for solder and flux.’ [157]

The 1991 federal Lead and Copper Rule (LCR) set a ‘non-health-based action level’ for lead in water of 15 μg L⁻¹. Under the current rule, if the 15 μg L⁻¹ action level is exceeded in 10% of residences in a service area, corrosion control must be implemented along with public notifications. Corrosion control is also required of large water suppliers [158]. Corrosion control, however, is not always effective in reducing lead levels in water [155]. Lead service lines only need to be replaced if the action level continues to be exceeded, and then only 7% of them each year in the particular service area [158]. A recent study estimated that there are still 6.1 million lead service lines remaining in the US, serving between 15 and 22 million people on community water systems. This is a reduction of four million from EPA’s 1991 estimate, likely attributable both to replacement of lead service lines and better estimates [159]. EPA’s National Drinking Water Advisory Council recommends that lead service line replacement be an important part of the revision of the LCR [160].

In October 2019 EPA announced revisions to the 1991 LCR. Significantly, EPA has not proposed lowering the 15 μg L⁻¹ action level, nor are they proposing to setting a maximum contaminant level as public health advocates had recommended [161].

Within the public health community, a focus on preventing exposure through lead in paint and soil has generally overshadowed a focus on lead in drinking water. Though drinking water may be the primary source of exposure for formula fed infants [162], until the Flint, Michigan crisis occurred, lead in drinking water did not garner the sustained attention of public health officials. In addition to drawing attention to the problem of lead service lines, the Flint crisis also brought national awareness to possible lead contamination in school drinking water due to both lead service lines and older fixtures within schools [163].

There is little published literature on lead in drinking water obtained from rainwater tanks or cisterns in the US. It is not discussed as an important source of children’s exposure.
3.4.5. Lead in food

Recent estimates of children's lead exposure from food in the US show that for the first six months of a child's life, food contributes approximately 10%–25% of a child's total lead exposure, resulting in 0.1–0.3 µg dL⁻¹ of lead in blood. For children between one and less than two years old, food contributes to 0.6 µg dL⁻¹ of blood lead on average [141]. The FDA has recently revised its Interim Reference Level (IRL) for children to 3 µg day⁻¹, FDA uses the IRL to assess whether measured levels of lead in food could raise children's BLLs to 5 µg dL⁻¹, the current CDC reference level. The FDA currently only sets legal limits for bottled water (5 µg L⁻¹) and has recommended maximum levels for lead in candy (0.1 ppm) and juice (50 ppb) [164]. The use of lead-based ammunition for hunting and subsequent exposure of people who consume game meat remains a problem in the US. The majority of ammunition used in the US for hunting contains lead. Though many states have some restrictions on its use and California has imposed a complete ban, the toxic ammunition is still widely used and can increase BLLs among those who consume game meat killed with lead-based ammunition [165, 166].

3.4.6. Lead in soil and contaminated sites

US EPA sets standards for soil lead levels which apply to federally assisted housing and are used as guidelines for remediating contaminated sites under federal programs. States may set their own more restrictive standards. The current standard of 400 ppm for ‘bare soil in children's play areas,’ and 1200 ppm on average in the rest of the yard is widely considered to be outdated as it was set in 2001, based on CDC's then BLL 'level of concern' of 10 µg dL⁻¹ [167]. Another concern is that legacy soil contamination in urban areas may be an important driver of elevated BLLs in children. For example, an EPA meeting record notes, ‘EPA's IEUBK model predicts that soil lead levels below 400 ppm are sufficient to result in childhood BLLs above the CDC’s new reference value of 5.0 µg dL⁻¹. The 400 ppm soil lead concentration likely is below typical urban lead levels in inner city soils.’ [168]

Remediation of contaminated sites under the federal government's auspices in the US occurs primarily under two laws, the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) and the Resource Conservation and Recovery Act (RCRA). A large number of Superfund sites have lead hazards. Mining, smelting and battery recycling sites have some of the highest concentrations of lead in soils. Remediation of contaminated soil from highly contaminated sites has been occurring since implementation of the Superfund law in the 1980s and in residential areas often requires removal and replacement of yards or portions of them. Near the non-operational Bunker Hill, Idaho lead smelter and in the former mining site at Butte, Montana, soil remediation has been associated with declines in children's BLLs [169, 170]. At this site, approximately half of children's lead intake was estimated to derive from house dust and the other half from soil, including the child's own yard soil as well as community soils [171]. Limited attempts have been made to clean up legacy contamination from leaded gasoline or lead paint in urban soils that are not Superfund sites, and capping/grass have shown some success [172].

3.4.7. Occupational/take-home exposures

The Occupational Safety and Health Administration set standards for occupational exposure to lead in 1978 and these standards have not been updated. Currently federal law does not require medical removal of a lead exposed worker until a worker's BLL is in the range of 50–60 µg dL⁻¹. States may set more protective standards. California and Washington state are in the process of updating their occupational lead standards. California states that a revision of occupational lead standards is needed because ‘current science strongly indicates that worker BLLs should not exceed 5 to 10 micrograms of lead per deciliter of blood (µg dL⁻¹) over a working lifetime.’ [173] Among adults in the US, most elevated BLLs are attributable to occupational exposures [174]. Due to inadequate protection for lead workers, elevated BLLs in children attributable to parental take-home exposure continue to occur in the US and are occasionally reported in the literature though we are not aware of current national prevalence estimates [175, 176].

3.5. Overarching approach to prevention—Australia

Australia has implemented some primary prevention approaches such as phasing out leaded petrol and restricting lead in paint. However, current efforts to prevent exposure primarily rely on secondary prevention strategies and put the onus on individuals to prevent lead poisoning by educating themselves about potential lead sources in their homes and environments. In mining and smelting communities this approach is augmented by suggesting a range of specific cleaning and hygiene strategies highlighted by local community groups—which are often funded by the local lead industry in partnership with the local council [177, 178]. Location specific clean-up activities are sometimes carried out by these groups, although concern has been raised by some researchers that these cleaning activities are not sufficient [179]. There remain several sources and pathways of exposure to lead in children's environments that lack robust regulatory frameworks.

The National Health and Medical Research Council (NHMRC) is the lead agency for providing advice on lead exposure prevention to the federal and six state and two territory governments. The division of
federal and state or territory governmental responsibility for policy and regulatory action is complex and sector dependent (from here on, we will use 'state' to mean both state and territory government). Although some sectors, such as public health, are a state responsibility, funding activities may involve the federal government. The federal government issues policy recommendations and health guidelines through the bodies such as the NHMRC, and the state governments may then choose to implement them through guidelines or by ratification via legislation. For the example of petrol, the federal government was able to increase excise on leaded petrol, due to powers vested in the federal government over the collection of fuel excise taxes under the Constitution, but the introduction of unleaded petrol was determined by the state governments. In this case, the federal government was able to make the price of leaded petrol higher than that of unleaded petrol to encourage the use of the latter.

Lead exposure prevention from sources other than petrol is guided by even more complicated multi-agency involvement. Relevant agencies vary over time and depending on the structure of the departmental responsibilities, with recent examples of relevance including: federal and state EPAs, state departments of health and water authorities, local government and community services departments, as well as a range of federal government departments such as health and housing.

There is no over-arching federal government strategy for childhood lead exposure prevention in Australia. In 1993, after recommending that BLLs in Australians of all ages should not exceed 10 µg dL\(^{-1}\), the NHMRC commissioned a comprehensive review of how lead exposure could be reduced. A key recommendation of this review was eliminating lead in petrol in all states and territories given that petrol was understood to be the largest source of exposure at the time. The review called for a comprehensive strategy to address other exposure pathways such lead-based paint, lead in drinking water, and contamination in hotspot mining and smelting towns. The detailed recommendations in the report to reduce BLLs were not systematically followed [180].

Although the NHMRC lowered the reference level for lead in blood to 5 µg dL\(^{-1}\) in 2015, many government agencies in Australia, such as the NHMRC, have tended to make more equivocal statements about the relationship between low lead levels children’s health than those of the current German or US governments. For example, the NHMRC summarized the evidence for low level lead exposure and health effects in 2015 the following way:

It is well established that blood lead levels greater than 10 micrograms per decilitre can have harmful effects on many organs and bodily functions... The evidence for health effects occurring as a result of blood lead levels less than 10 micrograms per decilitre is less clear. NHMRC’s comprehensive review of the health effects of lead found an association between blood lead levels less than 10 micrograms per decilitre and health effects, including reduced Intelligence Quotient and academic achievement in children, behavioural problems in children, increased blood pressure in adults and a delay in sexual maturation in adolescent boys and girls. However, there is insufficient evidence to conclude that lead at this level caused [emphasis in original] any of the health effects observed [181].

As of July 2019, the NHMRC reference level of 5 µg dL\(^{-1}\) is the notifiable level in all states and territories except for the ACT where there is only an informal notification process for ‘young children’ with no official guidance about the level for notification, or what constitutes a ‘young’ child (pers. comm. 29 July 2019) [182]. The 5 µg dL\(^{-1}\) reference level is not tied to population level biomonitoring data because Australia has no national surveillance system or biomonitoring initiative for childhood lead exposure. The last time that BLLs were systematically assessed in a nationally representative sample of Australian children was in 1996. Several NGOs, such as The Public Health Association of Australia and the LEAD Group have lobbied for better monitoring of children’s lead exposure [183, 184].

3.5.1. Lead in paint

Despite the early work of J. Lockhart Gibson in Queensland linking lead poisoning in children to paint, such exposure remains a problem in Australia [185, 186], though studies are limited. Like the US, Australia did not sign the ILO White Lead Convention and in most parts of the country allowed the use of lead paint for interior and exterior use for much of the twentieth century. Although advocacy from concerned physicians in Queensland led to the adoption of limits on the lead content of paint on surfaces children had access to in 1922, this restriction did not end the use of lead paint in the state. In 1965, federal legislation reduced the maximum allowable lead content in paint from 50% to one per cent across all states and territories. The allowable concentration was dropped to 0.25% in 1992, and in 1997 it was further reduced to 0.1% [187].

Though any house built prior to 1970, which would comprise millions of homes, may contain lead-based paint on interior or exterior walls or woodwork, there is limited systematic data on the prevalence of lead paint hazards in Australia’s housing [188]. The
1996 national survey of children’s BLLs sampled lead paint from the 30% of residences that were judged to have deteriorating paint. The study found an association between interior lead paint hazards and BLLs in children [38].

Australia lacks a comprehensive regulatory framework to address children’s exposure to lead paint. The federal government provides limited online information about the potential for harm to individuals via a booklet aimed at homeowners/renovators [189]. Despite bearing a publication date of 2016, this booklet provides out of date information regarding blood lead reference levels. On page three it states: ‘The national recommendation for all Australians is to have a BLL below 10 µg dL⁻¹.’ This is particularly problematic because several state governments also refer homeowners/renovators to it [190].

Standards Australia, an independent, non-profit organization, provides guidelines for management of lead paint and dust related hazards for builders and trades people, and these standards are often referred to in state legislation. The current guidelines, AS4361.2(2017), provide an update of the definition of lead paint of >0.1% lead (from >1% lead in the previous standard AS4361.2 [1998]). These guidelines have not been updated for dust.

Some state governments have laws and programs for addressing environmental and occupational health risks related to lead paint. For businesses employing workers in the building trade where lead paint removal may be a regular work hazard, there are legal obligations under worker health and safety laws at the state level. In NSW for example, the BLL for people conducting lead risk work will be lowered to 5 µg dL⁻¹ for females of reproductive capacity (from 10 µg dL⁻¹) and 20 µg dL⁻¹ for all others, (from 30 µg dL⁻¹) in July 2021 [191]. This approach is similar for workers in Queensland where workers in lead-risk positions are required to have regular health monitoring [192].

3.5.2. Lead in petrol

Leaded petrol was first used in Australia in 1932, with unleaded petrol introduced in 1985 [193]. The reduction and eventual phase out of lead in petrol occurred later than in Germany or the US, even though its use accounted for approximately 90% of airborne lead in cities (outside of major point source locations) [194]. In 1993, when the NHMRC report Reducing Lead Exposure in Australia was published, only 50% of cars in Australia used unleaded fuel. The report recommended immediately lowering the permissible petrol lead content from 0.84–0.3 g L⁻¹ to 0.15 g L⁻¹ and that the government consider a further reduction to 0.026 g L⁻¹. As a result of these recommendations, a nationwide phase-out began in 1993, with the federal government announcing a complete phase-out of leaded petrol in 2000 [195]. By 2002 this transition had been successful, however, the extended use of leaded petrol in such a highly car-dependent country resulted in an estimated 240 000 tons of lead being deposited largely in urban areas [193].

3.5.3. Lead in air

The federal government, under the non-binding Ambient Air Quality National Environmental Protection Measure (AAQ NEPM), sets standards which state and territory governments are expected to monitor and report on several common (criteria) air pollutants, including lead. The AAQ NEPM can be used for either legislation or guidance by state governments independently although performance against the AAQ NEPM is not enforced by the federal government, as it is believed that accountability for meeting standards ‘will be met via the public reporting requirement.’ [196] Despite acknowledging that, ‘It [lead] remains a potential risk at some specific industrial sites, which are addressed through jurisdictional controls such as licence conditions,’ current AAQNEPM consultations are recommending the removal of lead as a criteria pollutant and instead for it to be put into the Air Toxics NEPM [197].

The current standard for lead in air pollution is 0.5 µg m⁻³ averaged over one year, with no exceedances allowed. This standard is generally met nationwide, although until relatively recently, hot spot communities had monitoring locations where the air regularly exceeded these levels—sometimes allowable under special ‘transitional licenses’ which permit levels of lead pollution higher than state annual averages. These spatial and temporal differences in lead in air pollution standards are described in detail in Dobbie and Green (2015) [198]. Since the national lead standard is not binding on the states, a lack of enforceability ensues. Annual averaging masks emission spikes and downplays the significance of short-term acute exposure. For example, exceedances of the lead AAQ NEPM have been regularly documented in recent years in Port Pirie, where a 24 h maximum value peak of 22.57 µg m⁻³ was recorded in 2011 [26]. One monitoring location (Oliver St) in Port Pirie exceeded the national standard every year from 2002 until 2008, with readings up to 0.7 µg m⁻³ [199]. And in 2017, the annual average for lead in air (averaged across monitoring sites) was 1.13 µg m⁻³ [200]. Due to a transitional license that has allowed less stringent lead in air emission levels in comparison to the national standard for Mount Isa (up to 1.5 µg m⁻³ measured quarterly) [201], there is no directly comparable exceedance record against the national recommended level. Broken Hill reports monthly lead in air data as part of its operating license, the most recent of which indicated a maximum reading of 0.633 µg m⁻³ at one of the two monitoring sites [202].

Air emissions of lead from smelters have been tied to children’s lead exposure. For example, a recent study in Port Pirie found average lead in air
concentrations at four different monitoring sites over twice as high as the national standard of 0.5 μg m⁻³ in 2017. These authors estimated that to ensure that BLLs in children less than 5 do not exceed 5 μg dL⁻¹, air lead levels in Port Pirie would need to be substantially reduced to 0.11 μg m⁻³ [200].

Leaded avgas is in use in Australia and has either a max lead concentration of 1.12 g L⁻¹ or 0.56 g L⁻¹ (the low lead avgas is used to prevent adverse consequences from sniffing) [203]. Despite reductions in lead content in avgas, fuel sniffing, especially in the Northern Territory, has contributed to elevated BLLs. Recent testing found that out of 178 young people tested, 154 were found to have elevated levels of lead. The average BLL found in this group of young people was six times higher than the notification level [204].

3.5.4. Lead in drinking water
There are no federally enforceable general standards for lead in water in Australia. The NHMRC guideline is that lead in water should not exceed 10 μg L⁻¹ [205]. Levels of lead in reticulated water supplies in Australia are generally below this guideline, however, a study conducted in 2016 which measured lead concentrations at the tap in NSW houses found that more than half of the first draw samples had measurable amounts of lead, and exceedances of the guideline were found in eight per cent of samples [206]. It is thought that lead in drinking water found at the tap derives mostly from lead in brass fittings, which under Australian standards may contain up to 4.5% lead.

A recent review recommended lowering this level to 0.25% [207], and a government committee recently recommended that every effort should be made to reduce exposure to lead in the environment, including lead that may be dissolving into drinking water from some plumbing products. [208]

Rainwater tanks are used in many rural communities throughout Australia as potable water or for household or use on garden and lawns. Water collected and stored in these tanks may be contaminated with lead either from roofing or flashing materials, solder used in the tanks or from atmospheric deposition [209, 210]. Atmospheric deposition is a particular concern in mining and smelting towns, including Port Pirie, Mount Isa and Broken Hill. Though health authorities recommend against drinking rainwater in these towns [211], it may be used on gardens due to the need to regularly damp down dust, and due to the excessive costs of reticulated water in these locations.

3.5.5. Lead in food
Food standards are legislated and enforced at the state level. A national agency, Food Standards Australia New Zealand (FSANZ) develops and manages the standards for food which recommends maximum levels of lead in a range of food items [212]. The FSANZ works with government agencies and departments but it does not have power to enforce the code. Enforcement of the code is a state government agency, or local council, responsibility. The exception to this is imported food, which is managed by the Federal Department of Agriculture. Estimates of mean lead exposure through food for two to five year old children are thought to be below the dose determined by Joint FAO/WHO Expert Committee on Food Additives (JEFCA) in 2010 to result in 0.5 point IQ loss among 1–4 year olds (0.3 mg kg⁻¹ bw⁻¹ day⁻¹) [213].

An additional source of lead exposure from food is game meat killed with lead shot. In the Northern Territory, Indigenous people regularly hunt wildlife using lead shot [214]. In some areas, over half of the children tested had levels above 5 μg dL⁻¹ [215, 216].

3.5.6. Lead in soil and contaminated sites
Legacy pollution from leaded petrol and deteriorating lead paint are common sources of lead in urban soils. VegeSafe, a community science program begun in 2013 has tested over 1700 soil samples from schools, community and domestic gardens for lead [217]. Its findings showed that inner city gardens were the most polluted. Soil lead in concentrations greater than the national health level (300 mg kg⁻¹ for residential gardens) was found 40% of Sydney homes, while 15% of homes had soil lead in excess of 1000 mg kg⁻¹ [218].

High soil lead concentrations occurred more frequently in the inner-city areas. Lead concentrations fell as distance from the inner-city areas increased [219]. The elevated lead levels were associated with exterior paint on pre-1970 homes and traffic density, as well as legacy pollution from the use of lead paint and leaded petrol.

Similar levels of soil contamination were observed in residential areas in older inner-city areas of Brisbane [220], Adelaide [221] and Melbourne [222]. Recent studies in Melbourne and Sydney found that at the soil lead levels measured, 11.6% and 5.6% of two year old children could have BLLs >5 μg dL⁻¹ respectively [223, 224].

Around the mining and smelting communities, elevated concentrations of lead in soils is a significant problem. In Broken Hill, children living closest to the orebody are thought to be at high risk of elevated BLLs from lead in soil [225]. Soil testing in Mount Isa has shown elevated levels near the smelter [226], and soil testing in Port Pirie has shown elevated levels of above 600 ppm for 20% of the 353 sites tested (with a range of 6–7546 ppm) [227].

There is no national law or policy to address contaminated sites in Australia. Contaminated sites are dealt with in an ad hoc manner and are a state government responsibility. As such there is no national, standardized approach to levels, timing or methods of remediation or clean-up of contaminated locations.

Some remediation work has been done at each of the three largest mining/smelting towns, in each case
being funded and managed by a committee usually including state government departments, local government representation and the relevant company at the time. In Port Pirie, 2200 houses of children with the highest BLL were decontaminated at a cost of AUD$20M during the period of the first 10 year state government funded Lead Program which began in 1994 [228]. In Broken Hill the NSW Department of Health and the NSW EPA carried out extensive remediation of public land close to the mine and smelter between 1994 and 1997 focused on covering contaminated soil and replanting vegetation [229]. For children with substantially elevated BLL, home remediation efforts were employed such as removing ceiling dust, removing or covering contaminated soil, addressing flaking lead-based paints, and cleaning or replacing carpets. In recent years, efforts to carry out remediation have been revived due to the persistence of elevated BLLs in children in the community [230].

Remediation work was carried out at Mount Isa's Leichhardt River between 1991 and 1994, 2008 and 2009 to remove contaminated sediments left over from historical mining practices of the 1940s and 1950s. [231] In total, the mining company has calculated it spent approximately AUD$2.7 M on the remediation around the River and relocated around 160 000 tonnes of soil from the river onto the mine site. The NSW Department of Environment and Climate has remediated soil around approximately 4000 houses located near the former lead smelter at Boolaroo, either by covering contaminated soil with clean soil or by removing top.

However, the type of large-scale Superfund clean-up at smelting sites in the US (e.g. Bunker Hill smelter site, Tacoma smelter site) aimed primarily at removing contaminated soil from living environments has typically not been carried out in Australia. Similarly, there has been no concerted attempts to remediate urban soil lead.

3.5.7. Occupational/take-home exposures

There is little information about the nature of general workplace exposures to lead in Australia. Regulations and data collection tend to focus on activities deemed as ‘lead risk work’ where high levels of workplace exposures may occur frequently. However, the Australian Work Exposures Study for lead and lead compounds found that approximately 6% of Australian workers are estimated to be exposed to lead [232].

Occupational exposure is managed at a state level in Australia, with federal level advisories frequently relied upon to provide frameworks on which to base state level legislation. Safe Work Australia provides model legislation to states. In the most recent model legislation, ‘lead risk work’ is defined as lead process work that could result in a BLL in a worker of more than 20 µg dL$^{-1}$ (or 5 µg dL$^{-1}$ for females of reproductive age). Medical removal of a worker would occur when their BLL is 30 µg dL$^{-1}$ and for women of childbearing age, 10 µg dL$^{-1}$. It also includes provisions for washing and showering and laundering clothes in order to reduce the risk of take home exposures [233]. Due to recognition and community/industry and state government efforts, in the hotspots of lead mining and smelting communities, take home exposure has fallen due to policies at worksites to prevent dust leaving the sites on clothes or vehicles, however we are not aware of any recent studies providing prevalence estimates [234].

4. Conclusion

The three countries have very different histories and consequent development of environmental and public health policies with respect to childhood lead exposure prevention. Germany’s primary prevention approach has been relatively effective under a threshold model of exposure in that BLLs have declined substantially over time, although more stringent environmental standards and further prevention activities will likely need to be implemented to address the evidence that there is no safe level of lead exposure. Exposures once thought to be minimal may need to be addressed, such as lead paint and house dust, soil contamination, lead in interior plumbing in unrenovated buildings, and lead in food. This may necessitate the use of further primary and secondary prevention strategies, including, for example, soil remediation and education regarding the possible existence of lead pipes in unrenovated buildings/homes.

The framework of precaution endorsed by the EU and Germany may over time help to lower exposure to lead in the population. Examples of precautionary policies include REACH, Germany’s soil protection law, and new German limits on sewage sludge used as fertilizer. Some EU environmental standards may not offer enough protection under a no threshold model of exposure and should be reviewed in light of scientific evidence. The lead in air standard is an example.

Challenges to eliminating children’s exposure in Germany include limited biomonitoring data (e.g. the 10+ year gaps between GerES II, IV and V), and no lead biomonitoring data on children younger than three years old, who are likely to be most at risk. Improving biomonitoring and data on exposure pathways would permit a greater understanding of pathways of children’s exposure and help to focus prevention priorities. Understanding the extent of disparities in lead exposure, such as by geographic location, income, race and or immigration status could also help to focus prevention. Another challenge is that there is little public awareness about the issue of low-level lead exposure. While governmental and academic experts have pointed to the need to reduce the population’s exposure to lead, this is generally not an issue that has been taken up by environmental
advocacy groups or NGOs and not something that the general public is aware of.

Though the US has relied on both primary and secondary prevention approaches which have been somewhat effective under a threshold model, more needs to be done in light of the evidence that there is no identified safe level of exposure to lead for children. The US regulatory framework at the federal level addresses most sources and pathways of lead exposure; however, standards (e.g. lead in drinking water, occupational exposure) and enforcement (e.g. EPA’s RRP rule) will need to be tightened to achieve prevention goals, and new regulations crafted, (e.g. leaded aviation gas), and further research and intervention is needed to address legacy lead in soil.

The US must focus more on primary prevention such as remediating/abating lead paint from millions of homes and schools, replacing millions of lead service lines and removing lead from the water systems in countless schools across the country. The elimination of these sources of exposure will require a significant commitment of resources from governments. The US can learn from Germany’s near elimination of lead service lines from its water distribution system over the last several decades, the scale of the removal and replacement suggest that such a strategy is possible in the US. The environmental justice dimensions of the childhood lead exposure problem have been well described in the US but need to be better integrated into policy, regulation and funding priorities, such as prioritizing funding for permanent remediation for communities most affected by childhood lead exposure [235].

Australia has the furthest to go in terms of public health and environmental policy development and implementation to prevent children’s exposure to lead. In particular there is a need to develop a more robust regulatory framework for lead, to address lead in paint and household dust, lead in air and water, and to prevent exposure in mining and smelting communities. Australia would benefit from developing and implementing a coordinated national strategy such as what was suggested in the NHMRC-funded strategy document published in 1993. There is an official reluctance to fully endorse the science that lead causes harm at low levels of exposure. The framing of the problem as an individual’s responsibility, with prevention of exposure to be achieved by the individual homeowner or resident in mining/smelting community, rather than through government action, is a challenge to making significant progress on children’s exposure. Further, the lack of population-based biomonitoring or surveillance data limits knowledge of the extent of the problem and impedes prevention efforts. For many decades, researchers and advocates have called for improved data to inform comprehensive policy and practice to reduce lead exposure [182, 183, 236].

In comparison to Germany and the US, national standards or guidelines are generally similar (with the exception of the US lead in air standard), but the authority of the national government to gain compliance differs. A good example is the Australian lead in air standard which is set at the same level as the EU/German standard. The problem lies in the fact that the Australian standard is not binding on states to enforce. Instead, state governments negotiate pollution licenses with lead emitters and there is no requirement that they meet the national standard. Even if the national standard is written into the license, there may be few penalties for violating it. Enforcement is convoluted and complex, leading to ‘no easily identifiable legal framework regarding the regulation of air pollution.’ [198]

Lead mining and smelting communities should be the focus of more proactive and evidenced-based prevention which would include reducing lead in air, soil and dust. Many people who live in these communities are of lower socio-economic status than the Australian average, and the need for additional resources to effectively and comprehensively reduce their pollution burden is increasingly recognized as an environmental injustice. With a higher percentage of Indigenous families living in rural mining/smelting areas, this issue is also one of environmental justice. With a higher percentage of Indigenous families living in rural mining/smelting areas, this issue is also one of environmental justice (or lack thereof) [226, 237] Similarly, in heavily trafficked urban areas, research is showing a disproportionate air pollution burden from traffic, and the compounded legacy lead burden from petrol with the ongoing burden from leaded paint which urgently needs formal state government acknowledgment and action [238].

With respect to lead exposure from paint, Australia can learn from regulatory framework developed in the US aimed at protecting child occupants of homes and daycare centers, including mandated disclosure of the potential for lead paint in older housing to renters and buyers, lead dust standards, and rules for renovation and repair.

All three countries should review environmental standards and reference levels in light of the evidence that there is no identified threshold for lead exposure and all should evaluate the extent to which lead exposure prevention relies on a primary prevention model.

Lastly, a limitation of this research is that it only considered three high income countries and their policies on lead exposure prevention. Future comparative policy research should examine such policies among Latin American, Asian and African countries since lead continues to be produced and consumed on these continents.

Data availability statement

No new data were created or analyzed in this study.
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