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## Review

## Potential for childhood lead poisoning in the inner cities of Australia due to exposure to lead in soil dust

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## ABSTRACT

This article presents evidence demonstrating that the historical use of leaded gasoline and lead (Pb) in exterior paints in Australia has contaminated urban soils in the older inner suburbs of large cities such as Sydney and Melbourne. While significant attention has been focused on Pb poisoning in mining and smelting towns in Australia, relatively little research has focused on exposure to Pb originating from inner-city soil dust and its potential for childhood Pb exposures. Due to a lack of systematic blood lead (PbB) screening and geochemical soil Pb mapping in the inner cities of Australia, the risks from environmental Pb exposure remain unconstrained within urban population centres.

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## 1. Introduction

In the United States (US) and Australia there is a substantial body of evidence of widespread soil lead (Pb) contamination in large inner-city areas (Mielke et al., 2007, 2010b; Olszowy et al., 1995) and in centres for metal mining and smelting (see Taylor et al., 2010). In this paper, US and Australian research related to urban soil Pb distributions, studies of the association between soil Pb and PbB, PbB screening practices and PbB prevalence are presented. The US soil Pb exposure research is presented alongside relevant Australian research because it provides direct parallels to the Australian situation. In addition, international urban soil Pb exposure prevention methods and the current urban Pb poisoning exposure paradigm are presented. Finally, this article presents a review of the evidence of toxicity from low-level exposure (<10 µg/dL) in children typically due to chronic soil Pb dust exposure and makes recommendations to improve the long-term public health of inner-city children in Australia.

## 2. Urban soil lead distribution – United States

In the US, motor vehicles used gasoline containing tetramethyl and tetraethyl Pb additives from the 1920s to 1995 (Mielke et al., 2010a). By the 1950s, Pb additives were contained in virtually all grades of gasoline. By 1986, when leaded gasoline underwent a rapid phase-down, 5–6 million metric tons of Pb had been used as a gasoline additive, and about 75% of this Pb was released into the atmosphere (Chaney and Mielke, 1986; Mielke and Reagan, 1998). Thus, an estimated 4–5 million tons of Pb has been deposited into the US environment by way of gasoline-fueled motor vehicles (Mielke, 1994). Accumulation of soil Pb created by leaded gasoline is considered proportional to highway traffic flow (LaBelle et al., 1987; Mielke et al., 1997). About the same quantity of Pb (5–6 million metric tonnes) was also used in production of Pb based paint in the US (Mielke and Reagan, 1998), which has added to the burden of environmental Pb in urban soils.

In the 1970s, the presumed dominant source of soil Pb contamination was Pb-based house paint (Ter Haar and Aronow, 1974). A subsequent study of garden soils conducted in metropolitan Baltimore, Maryland, began to raise questions about that assumption (Mielke et al., 1983). Soil around Baltimore's inner city buildings, predominantly unpainted brick, exhibited the highest

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amounts of Pb, while soils outside of the inner city, where buildings were commonly constructed with Pb-based paint on wood siding, contained comparatively low amounts of Pb. These findings indicated that Pb based house paint could not account for the observed pattern of soil Pb (Mielke et al., 1983).

The quantity and distribution of soil Pb have been studied in numerous places in the US (e.g. Mielke et al., 2010b; Laidlaw and Filippelli, 2008; Burgoon et al., 1995; USEPA, 1996, 1998). The U.S. cities exhibited the same distance decay pattern with high soil Pb concentration values in the inner city and decreasing contamination towards the outer parts of the urban area (Mielke et al., 1983; Laidlaw, 2010). Urban soil Pb patterns are well understood. As Fig. 1 indicates, soil Pb decays exponentially away from the roadside, with the concentration proportional to historical traffic volume (LaBelle et al., 1987; Filippelli et al., 2005; Lejano and Ericsson, 2005). A large percentage of the Pb emitted from automobiles has been deposited within approximately 50 m of the roadside. Soil Pb also decays exponentially with distance away from the house-side towards the roadside (see Fig. 1). In homes that used exterior Pb-based paint, the Pb in the house garden soil is a mixture of vehicular Pb and paint Pb (Linton et al., 1980), with automotive Pb concentrated in the finer grain size (<44 µm), which is susceptible to re-suspension (Clark et al., 2006). Houses are impacted by vehicular Pb when exhaust emission particles came into contact with the house-side and are deposited in the soils adjacent to the house (see Fig. 1). This is evidenced by elevated Pb concentrations being found adjacent to brick homes facing roadways (Mielke et al., 1983; Olszowy et al., 1995).

### 3. Australian inner-city soil lead contamination

The problem of environmental lead exposure in children from paint and dust was first identified over a century ago by the Queensland doctors Gibson (1904) and Turner (1909). However, it appears that their warnings about environmental lead exposure went largely unheeded and leaded petrol was introduced in Australia in August 1932 (Cook and Gale, 2005). Unleaded petrol was introduced in 1996, and the Pb content of leaded petrol declined from 0.84 g/L in 1990 to 0.2 g/L in 1996, until it was banned in 2001 (Queensland Health, 2008). Lead levels in Australian paint were up to 50 % before the 1950s but thereafter several reductions were mandated bringing the allowable concentration to 0.1% in 1997 (Taylor et al., 2010).

A review of the literature indicates that some of the soils from the inner-city suburbs of Sydney, Australia, have become contaminated with a range of metals including Pb. It is likely that these soils have been contaminated due to the use of Pb in gasoline and Pb in exterior paints. The median and mean background soil lead concentrations in the Sydney region are 15.5 mg/kg and 21.3 mg/kg, respectively (Olszowy et al., 1995). In Balmain, an Inner Sydney suburb located 2.5 km north-west of Sydney, a soil Pb survey of 41 samples found that 68 % of residential housing samples exceeded

the National Environmental Protection Council (NEPC, 1999) 300 mg/kg residential soil lead guideline (Royal Prince Alfred Hospital and Central and Southern Sydney Area Health Service, 1988). Fett et al. (1992) analysed soil Pb concentrations at 18 homes in the inner Sydney suburbs and observed a median and mean “sink” (garden) soil Pb concentrations of 1237 mg/kg and 1944 mg/kg (range = 123 to 5407 mg/kg), respectively. In addition, at 24 inner Sydney suburban homes, Fett et al. (1992) observed a median and mean “play” area soil Pb concentration of 380 mg/kg and 627 mg/kg, respectively. Fifty-four % of all soil samples exceeded the NEPC 300 mg/kg residential soil Pb guideline (NEPC, 1999).

Skinner et al. (1993) collected seven soil samples from Bradfield Park (beneath the Harbour Bridge) and three samples at distances up to 350 m from the park. Four sites were sampled farther north at distances of 50–300 m from the major arterial Warringah Freeway in North Sydney. The median values for the two areas were 708 mg/kg (range = 19 to 1451 mg/kg) and 637 mg/kg (range = 216 to 1269 mg/kg), respectively. Olszowy et al. (1995) analysed 80 surface soil samples from residential properties in Sydney and found that about 40% of soil samples exceeded the residential 300 mg/kg soil Pb guideline. Snowdon and Birch (2004) analysed Pb concentrations in 274 soil samples in Iron Cove Catchment in Sydney (located approximately 2.5 km west of Sydney). They observed that 33% of the samples exceeded the NEPC (1999) guideline for Pb. Further, the mean bioavailability of the Iron Cove Catchment soils was 70 % using an EDTA extraction (Snowdon and Birch, 2004). Markus and McBrantney (1996) analysed 219 surface soil samples for Pb and other heavy metal concentrations in Glebe, a suburb located approximately 1 km south-west of Sydney, and found that greater than 50 % of Pb concentrations exceeded the 300 mg/kg residential soil Pb guideline. Cattle et al. (2002) reported that 41% of 807 surface soil samples in Glebe and Camperdown (located immediately west of Glebe) exceeded the 300 mg/kg residential soil Pb guideline. Cattle et al. (2002) also tested four geostatistical techniques to determine which methods were best able to delineate between soil Pb concentrations above and below the 300 mg/kg guideline in Camperdown and Glebe. Markus and McBrantney (2001) compiled a brief review of Australian soil Pb studies prior to 2001. Pb in Australian urban inner city soils is highly bioavailable (Snowdon and Birch, 2004) with bioavailability likely to be positively correlated with total Pb concentration (Wu et al., 2010). Upon the NEPC (1999) 300 mg/kg residential soil guideline and a range of other studies showing a relationship between soil Pb and PbB. This suggests that soils in large areas of inner Sydney may potentially pose a toxic threat to children and adults (Table 1).

In addition to exterior soil Pb contamination in Sydney, dusts collected from vacuum bags, the interiors of some homes and ceiling cavities are also contaminated by Pb. For example, Gulson et al. (1995) collected vacuum bag bulk dust samples in five homes in Sydney (locations not indicated) which were analysed for Pb concentration. Results indicated Pb concentrations ranged between 460 and 2950 mg/kg, with a median of 1202.5 mg/kg and an average of 1344 mg/kg. Chattopadhyay et al. (2003) completed Pb dust sampling in 82 residential homes in the Sydney metropolitan area. Results showed statistically significant differences of Pb levels by region in Sydney but not for other metals. Large variations in Pb levels were found in household dust (Range = 16–16,600 mg/kg; Mean = 389 mg/kg; Median = 76 mg/kg), with the inner-west area associated with significantly higher Pb levels ( $P < 0.001$ ) compared with other regions (Table 2). Chattopadhyay et al. (2003) also observed that household dust Pb levels have remained constant over the past decade despite substantial improvements in air quality. Gulson et al. (1995) collected ceiling dusts at 38 locations in the greater Sydney area. Results indicated median ceiling dust Pb concentrations of

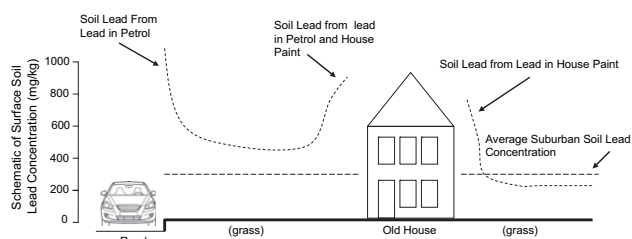


Fig. 1. A schematic cross-section through a residential suburban setting demonstrating typical urban soil Pb patterns (modified from Olszowy et al., 1995).

**Table 1**

List of Sydney and Sydney Suburbs Soil Pb Studies.

Author(s)	Suburb	Outcome of soil lead assessment
Olszowy et al. (1995)	Sydney Region	Soil Pb Background: median = 15.5 mg/kg, mean = 21.3 mg/kg
Royal Prince Alfred Hospital and Central and Southern Sydney Area Health Service (1988)	Balmain (2 km north-west of Sydney)	68 % of 41 samples exceeded the 300 mg/kg guideline
Fett et al. (1992)	Sydney Suburbs	Pb median in garden soil = 1,237 mg/kg. 54 % of soil samples (all types) exceeded the 300 mg/kg residential soil Pb guideline. (n = 22 homes)
Skinner et al. (1993)	North Sydney (3 km north of Sydney)	Median value of two groups of samples were 708 mg/kg (n = 10; range = 19 to 1451 mg/kg) and 637 mg/kg (n = 4; range = 216 to 1269 mg/kg)
Olszowy et al. (1995)	Sydney Suburbs	40 % of 80 samples exceed 300 mg/kg guideline
Gulson et al. (1995)	Inner Sydney Suburbs	Five houses were sampled with 11 total samples collected (average = 1217 mg/kg, median = 1135 mg/kg and range = 37–3130 mg/kg).
Markus and McBrantney (1996)	Glebe (1 km southwest of Sydney)	50 % of 219 samples exceed 300 mg/kg residential soil Pb guideline
Snowdon and Birch (2004)	Iron Cove Catchment (2.5 km west of Sydney)	33 % of 274 samples exceed 300 mg/kg guideline
Gulson et al. (2006)	Sydney Suburbs (located at varying distances from major traffic thoroughfares in Sydney)	In a large study in Sydney, Gulson et al. (2006) observed that the Pb concentration in soil was a significant predictor for Pb in the house dustfall, and dustfall was a significant predictor of PbB concentrations.

1960 mg/kg in the industrial area ( $n = 10$ ), 1022 mg/kg in the semi-industrial area ( $n = 17$ ), 621 mg/kg in the non-industrial area ( $n = 10$ ) and 145 mg/kg in the rural area ( $n = 1$ ).

In Brisbane, Olszowy et al. (1995) observed that about 40% of soil samples from residential properties in old areas near busy roads exceeded the 300 mg/kg residential soil Pb guideline. In Adelaide, Tiller et al. (1987) analysed about 600 surface samples for Pb from a  $90 \times 20$  km study area extending from the metropolitan area of Adelaide, South Australia, to rural areas. The Pb content of surface soils showed petrol-Pb emitted within Adelaide from automotive exhausts has measurably contaminated the rural landscape to about 50 km downwind of the city. Soil Pb concentrations in Adelaide (Tiller et al., 1987) were digested using EDTA and are not comparable to soil Pb concentrations in other Australian cities which are based on total extractable Pb concentrations. Gulson et al. (1981) measured lead isotopes in surface soil near Adelaide and concluded that Pb from gasoline was the main source of Pb in surface soil.

In New South Wales, Queensland, Victoria and South Australian cities, Olszowy et al. (1995) observed the following relative soil Pb gradient concentrations for various city areas: Old High Traffic > Old Low Traffic > New High Traffic > New Low Traffic > Rural. These findings are, in effect, similar to those of Mielke et al.'s (1997) work in the USA. Even in the old areas with low traffic flow in Brisbane, Sydney and Melbourne approximately 20% of samples were found to exceed the investigation threshold for Pb (Olszowy et al., 2005).

Soils in some areas of regional cities such as Newcastle are also contaminated with Pb. Devey and Jingda (1995) analysed Pb in 108

surface soil samples from public parks and playgrounds in Newcastle, New South Wales (NSW). This study found that soil Pb concentrations ranged from 25 to 2400 mg/kg and that 21% of samples had concentrations higher than the 300 mg/kg residential soil Pb guideline. This assessment excluded the areas of Boolaroo and Argenton, which have been severely impacted by the former Pasmenco Pb smelter (now closed) (Willmore et al., 2006; NSW Environmental Protection Authority, 2003).

#### 4. Emerging soil Pb exposure paradigm

The emerging PbB poisoning paradigm is that children in cities unaffected by Pb mining and smelting are also exposed to soil Pb dust, which can be traced to the use of Pb in gasoline and exterior Pb paint (Filippelli and Laidlaw, 2010). Contaminated dust is tracked into homes by shoes (Hunt et al., 2006), family pets, and also via resuspension and deposition of Pb dust, which penetrates interiors of homes and settles onto contact surfaces (Layton and Beamer, 2009; Laidlaw and Filippelli, 2008). Analysis of interior house dusts indicates that a large percentage of interior house dust most probably originates from outdoor soils (see Table 3). This illustrates the significance of the soil reservoir as significant potential exposure pathway for childhood Pb poisoning.

Fig. 2 shows a conceptual diagram depicting the movement of contaminated soil and airborne particulates into a residence, subsequent mixing by the organic matter in floor dust, redistribution indoors via resuspension, and removal by cleaning and exhalation with building vented air (Layton and Beamer, 2009). Once Pb has been tracked into homes, exposure to interior house dust then

**Table 2**

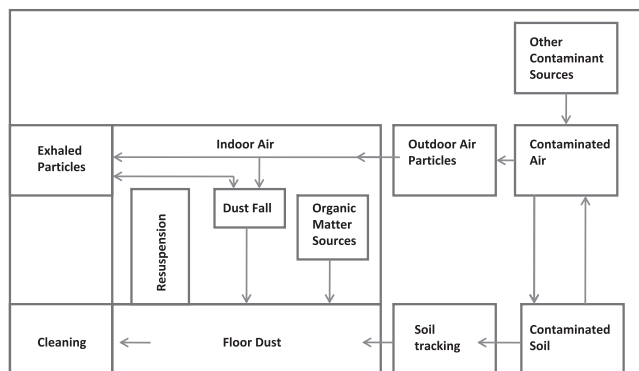
Geometric mean interior house dust Pb concentrations in Sydney by region (after Chattopadhyay et al., 2003). Note that the Sydney mean Pb dust concentration is 389 mg/kg and the median is 76 mg/kg ( $n = 82$ ).

Location	Geometric mean interior house dust Pb Concentration (mg/kg)
CBD and Eastern Suburbs	106
North Shore	66
Inner west	260
South West	110
North West	46
South	92

**Table 3**

Estimates of the relative contribution of exterior soil to house dust (Paustenbach et al., 1997).

Environmental soil and dust Pb study	% House dust from soil
Hawley (1985)	>80
Thornton et al. (1985)	20
Camann and Harding (1989)	50
Fergusson and Kim (1991)	30–50
Calabrese and Stanek (1992)	20–78



**Fig. 2.** Conceptual diagram depicting the movement of contaminated soil and airborne particulates into a residence, subsequent mixing with organic matter in floor dust, redistribution indoors via resuspension, and removal by cleaning and exhalation with building vented air (Source – adapted from Layton and Beamer, 2009).

occurs via hand-to-mouth behaviour. Acute exposure occurs typically via ingestion of paint chips from indoor and outdoor Pb paint, which is most prevalent when children are aged approximately 18–24 months. At this age children are at an exploratory phase of their development and the ingestion of non-nutrient substances may result in accidental ingestion of poisons (e.g. Pb rich soil, paint chips, or paint from toys), leading to clinical or sub-clinical toxicities (Ko et al., 2007). In addition, another potentially important exposure pathway for Pb into humans may be via ingestion of contaminated vegetables (Finster et al., 2004; Kachenko and Singh, 2004, 2006).

### 5. Association between soil lead contamination and children's blood lead levels

Howard Mielke of Tulane University and colleagues have analysed the relationship between PbB and soil Pb in Louisiana (Mielke et al., 1997, 2007). The PbB response of children to soil Pb is curvilinear in New Orleans, Louisiana (Mielke et al., 1997, 2007). Johnson and Bretsch (2002) also observed a similar curvilinear relationship between soil Pb and children's PbB in Syracuse, New York. The most recent New Orleans urban soil Pb and PbB study shows the following results: below 100 mg/kg soil Pb children's PbB response is steep at

1.4 µg/dL per 100 mg/kg, while above 300 mg/kg soil Pb children's PbB response is a more gradual 0.32 µg/dL per 100 mg/kg (see Fig. 3; Mielke et al., 2007). It may be hypothesised that similar soil Pb and PbB responses of children are expected in all urbanized areas because the physiological response to exposure is broadly uniform. However, in the USA and Australia, data have shown that African Americans (Lanphear et al., 1996) and Aboriginals (Queensland Health, 2008) tend to have higher PbB than Caucasian children.

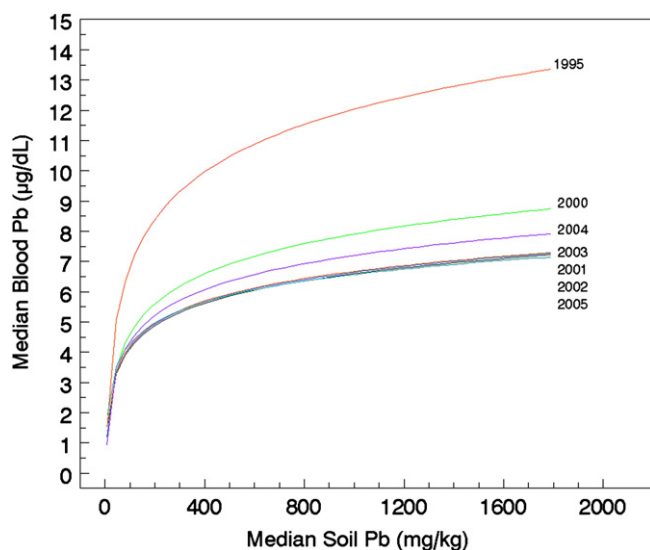
The relationship between soil Pb concentrations and blood Pb concentrations have also been modelled using the United States Environmental Protection Agency Mechanistic Exposure Uptake Biokinetic Model for Pb in Children (IEUBK) model (USEPA, 2010). Gulson used the IEUBK model to predict PbB concentrations for a range of children's ages and soil Pb concentrations (Fig. 4; Davis and Gulson, 2005).

The IEUBK soil – PbB slope (Fig. 4) predicts a child PbB level of between 4 and 5 µg/dL following exposure to soil with a Pb concentration exceeding the 300 mg/kg NEPC guideline. Mielke et al.'s (2007) empirical soil and PbB relationship slopes (Fig. 3) predict a PbB level of between 5 and 9 µg/dL for an exposure to soil with a concentration exceeding the 300 mg/kg NEPC (1999) guideline. It is noted that the slope for Mielke et al.'s (2007) empirical model is steeper than the IEUBK model for the first 300 mg/kg.

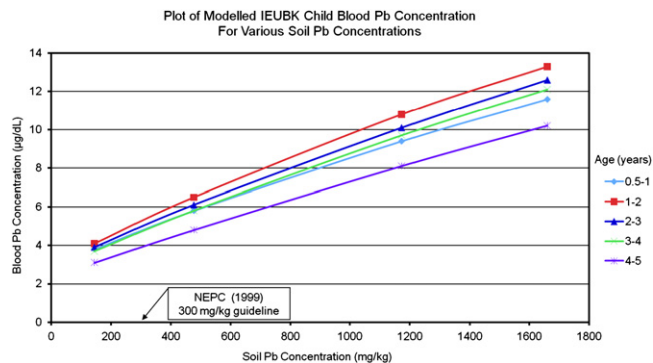
Soil Pb can also be associated with PbB concentrations greater than 10 µg/dL at soil Pb concentrations lower than suggested by the IEUBK model or Mielke et al.'s (2007) soil Pb–PbB curves. For example, Malcoe et al. (2002) found that logistic regression of yard soil Pb >165.3 mg/kg (OR, 4.1; CI, 1.3–12.4) were associated independently with PbB's greater than or equal to 10 µg/dL. Similarly, the Texas Department of Health (2004), using a large database from El Paso, Texas Area, found an odds ratio 4.5 (1.4, 14.2) for the relationship between a 500 mg/kg increase in soil Pb above background level and childhood blood lead levels > 10 µg/dL.

Laidlaw and Filippelli (2008) performed a review of multiple study designs used to analyse the association between soil Pb and PbB. The study designs included cross-sectional, ecological spatial, ecological temporal, prospective soil removal, and isotopic studies. Sedman (1989) also reviewed multiple American studies published prior to 1989 that demonstrated an association between soil Pb and PbB. In both of these reviews and examples it was shown that PbB in the various studies examined was associated with soil Pb.

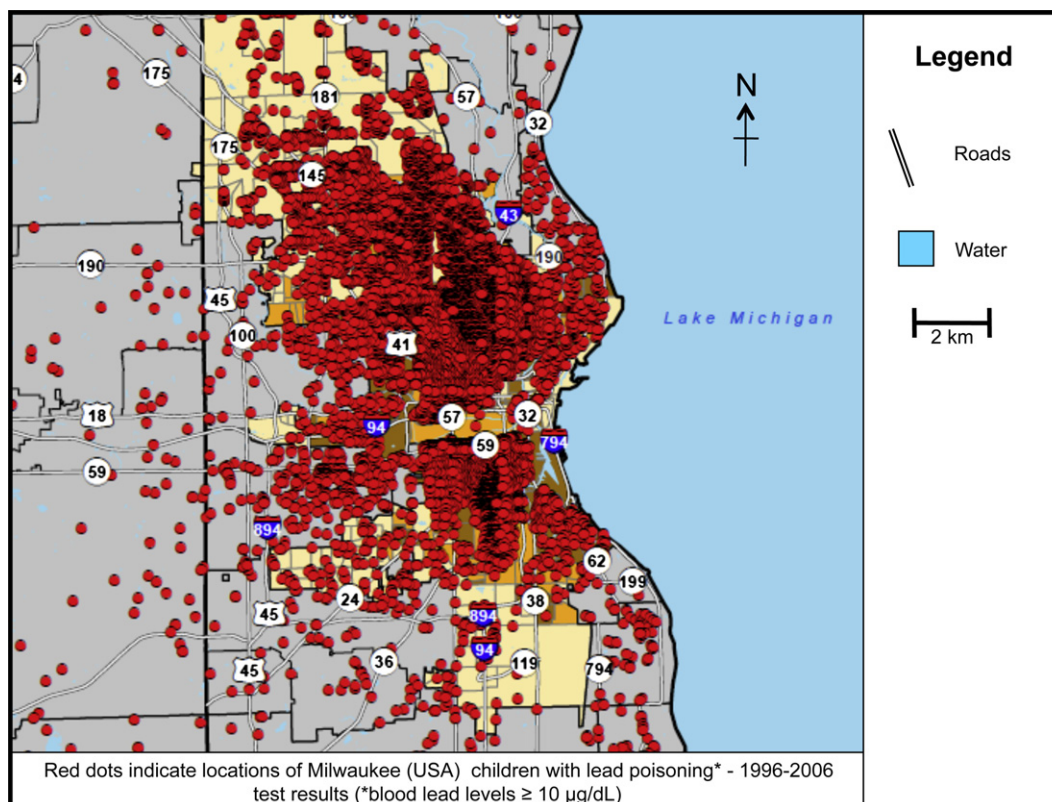
The link between soil Pb and PbB was demonstrated recently in New Orleans, where sediments in floodwater from Hurricanes Katrina and Rita (HKR) were deposited onto Pb contaminated soils (Zahran et al., 2010). High density soil surveying conducted



**Fig. 3.** Median Soil Pb and PbB Curves in New Orleans, Louisiana – 1995 and 2000–2005 (Mielke et al., 2007).



**Fig. 4.** IEUBK model of children's PbB concentrations give a full range of soil lead concentrations (modified from Davis and Gulson, 2005). This chart shows the IEUBK model of expected changes PbB concentration for a given soil Pb concentration for various age groups of children.



**Fig. 5.** Milwaukee, Wisconsin (USA) Pb Poisoning Map: 1996–2006. This map depicts the 36,856 children that exhibited PbB poisoning  $> 10 \mu\text{g/dL}$  in Milwaukee between 1996 and 2006 (source: modified from the Wisconsin Department of Health, 2010a).

in 46 census tracts before HKR was repeated after the flood. Paired  $t$  test results show that soil lead decreased from 328.54 to 203.33 mg/kg post-HKR ( $t = 3.296, p \leq 0.01$ ). Decreases in soil Pb are associated with declines in children's PbB response ( $r = 0.308, p \leq 0.05$ ). Zahran et al. (2010) found that declines in median PbB were largest in census tracts with  $\geq 50\%$  decrease in soil Pb.

Multiple studies in Australia have also shown an association between soil Pb concentrations and PbB concentrations. In Sydney, Fett et al. (1992) found that blood lead concentrations were correlated significantly with concentrations of Pb in yard soil ( $r = 0.555, p = 0.026$ ) and play area soil ( $r = 0.492, p = 0.016$ ). Young et al. (1992) also observed that soil Pb levels were significantly correlated with PbB levels near the Southern Copper smelter near Wollongong and Bellambi in NSW, Australia. In North Lake Macquarie, NSW near the former Pasmenco smelter (Boolaroo, NSW), Willmore et al. (2006) observed that geometric mean PbB was statistically significantly higher for residential soil Pb concentrations greater than 300 mg/kg. In a large study in Sydney, Gulson et al. (2006) observed that the Pb concentration in soil was a significant predictor for Pb in the house dustfall, and dustfall was a significant predictor of PbB. Dustfall accumulation was also observed to be a significant predictor for Pb concentration in handwipes.

## 6. Australia and United States – PbB screening

Universal blood Pb screening is not performed in Australia (NHMRC, 2009). In 1993, the National Health and Medical Research Council (NHMRC) stated that its specific goal was to achieve for all Australians a blood Pb level of below  $10 \mu\text{g/dL}$  (NHMRC, 1993). This document also recommended a graduated response to PbB levels for both individuals (children of all ages over  $15 \mu\text{g/dL}$ ) and

communities where  $>95\%$  of one-to-four-year-old children were below  $25 \mu\text{g/dL}$ , but  $>5\%$  were above  $15 \mu\text{g/dL}$ . This guideline was rescinded in 2005. In 2009, the NHMRC published an information paper titled *Blood Lead Levels for Australians* (NHMRC, 2009). The document once again supported a  $10 \mu\text{g/dL}$  PbB level guideline and suggested that representative samples of children aged 1–4 living in high and low Pb exposure areas should be screened for PbB. However, while such a blood lead study has been recommended, this has not been undertaken in the inner cities of Australia to date. Currently, due to the lack of universal screening, it is not known what the spatial distributions and incidence levels are of children with elevated PbB levels. Therefore it is difficult to establish the exact nature of the risk in urban city areas.

In the US, the current PbB screening practices were described by Cole and Windsor (2010), who stated: "...lead screening practices are created at the state level, with each state identifying and agreeing on its own lead screening guidelines. States vary widely in their approach to lead screening. Most states have a plan targeting children under the age of 6, but these plans vary greatly. Some states advocate universal screening (ex. Tennessee, Connecticut) while some advocate risk-based screening (ex. Illinois, Florida). Risk-based screening is usually accomplished through a parent questionnaire that identifies children who may be at higher risk for lead exposure and then only testing those at-risk. In addition, some states test children of certain SES designations, or who live in lower income areas or in older housing."

## 7. Australian and United States blood lead prevalence

In comparison to PbB studies in mining towns (see Taylor et al., 2010), there have been few PbB prevalence studies completed in

inner city areas of Australia. Fett et al. (1992) determined the distribution of PbB levels in 158 preschool children in inner Sydney and observed that 50.6 % of the children had PbB levels > 10 µg/dL, 17.1 % had PbB levels > 15 µg/dL, and 2.5 % had PbB's > 25 µg/dL. In a PbB survey of 718 children in central and southern Sydney, Mira et al. (1996) observed that 25 % of the children had PbB > 10 µg/dL and 7 % had PbB > 15 µg/dL. The only nationwide survey of PbB concentrations was conducted on 1575 children in 1995 (Donovan, 1996). Donovan found that the geometric mean PbB concentration in 1995 in 1–4 year-olds was 5.1 µg/dL, with 7.3 % exceeding 10 µg/dL and 1.7 % exceeding 15 µg/dL. A PbB prevalence study in Fremantle (Willis et al., 1995) of 120 children from day-care centres and 44 hospital inpatients observed that 25.6 % had PbB's > 10 µg/dL. A recent five-year longitudinal study of 113 children living in Sydney, aged six months to 31 months at recruitment, showed a mean PbB concentration of 3.1 µg/dL (range = 0.6–19.0 µg/dL) (Gulson et al., 2006). A PbB survey of 100 participants in Fremantle Western Australia in 2008 (Guttinger et al., 2008) found that none had PbB's ≥ 10 µg/dL. It is likely that PbB levels in Fremantle (Willis et al., 1995; Guttinger et al., 2008) have declined due to the elimination of Pb in petrol. However, it must also be noted that PbB prevalence studies with low sample numbers of around 100–150 subjects, as was done in Fremantle by Guttinger (2008) and in Sydney by Gulson et al. (2006), are not likely representative of the geographic distribution of PbB levels of large populations. For example, in Mt. Isa, Australia, Queensland Health determined that a sample size of 400 (approximately 25% of the Mount Isa population of children aged one to four) was required to have sufficient power to provide reliable information on PbB levels (Queensland Health, 2008). A city the size of Sydney would require a much larger sample to be statistically significant compared to Mount Isa.

The United States Center for Disease Control (CDC) indicates that the prevalence of PbB < 10 µg/dL in the US during 1999–2002 survey period for children aged 1–5 years was 1.6 % (CDC, 2005). However, the national results are arguably misleading because of the emerging evidence of the effects of low levels Pb exposure (Canfield et al., 2003; Schnaas et al., 2006; Surkan et al., 2007; Chiodo et al., 2007; Lanphear et al., 2005; Miranda et al., 2007; Chandramouli et al., 2009; Zahrn et al., 2009; Nigg et al., 2010). The National Health and Nutrition Examination Survey (NHANES) III 1999–2002 database indicates that approximately 2.4 million children aged 1–5yrs old have PbB levels between 5 and 9.9 µg/dL (Iqbal et al., 2008). Within the population sample with blood Pb levels of 5 µg/dL or higher, the prevalence was 47% for non-Hispanic black children, 28% for Mexican American children, and 19% for non-Hispanic white children (Bernard, 2003). Further, the distribution of affected children is highly spatially skewed. The prevalence of PbB poisoning > 10 µg/dL in inner cities of the US exceeds 10 to 20% in many cities. For example, the city of Milwaukee, Wisconsin, which has a population of approximately 1.7 million, has soils in the central city area contaminated with Pb (mean = 640 mg/kg, median = 280 mg/kg) (Brinkmann, 1994). Milwaukee's childhood PbB levels peak in the summer and early autumn and have been correlated to particulate matter less than 2.5 µm (Havlena et al., 2009). The seasonal variation of PbB was hypothesised by Havlena et al. (2009) to be related to the availability of dust and airborne particulates during summer months. Fig. 5 shows the distribution of Milwaukee's 36,856 children with PbB poisoning (i.e. >10 µg/dL) between 1996 and 2006. In 2008 the citywide prevalence rate for PbB > 10 µg/dL was 4.8 %, but was much higher in some neighbourhoods as indicated on a PbB incidence map on Fig. 5 (after Wisconsin Department of Health, 2010a). This demonstrates that while average PbB levels may be relatively low, the incidence of PbB poisoning exceeding 10 µg/dL (or even 5 µg/dL) may be elevated and represent large numbers of

children in the inner-cities. It is notable that the soil Pb concentrations in some of the inner Sydney suburbs are higher than those in Milwaukee. This might suggest at least an equal or greater risk than that which has already been demonstrated to exist in Milwaukee.

## 8. Toxicity of low level Pb exposure typically caused by exposure to Pb in soil dust

The current Pb guideline in Australia is a PbB concentration of 10 µg/dL (NHMRC, 2009). However, emerging evidence (see below) suggests that the definition of Pb poisoning in Australia may need to be reduced to 5 µg/dL, or even lower at 2 µg/dL (Taylor et al., 2010). In Australia, this could result in the emergence of a large number of Pb poisoned children. Low PbB levels (<10 µg/dL) typically associated with urban soil Pb exposure are associated with a myriad of health outcomes. Low PbB levels (<10 µg/dL) are associated with Attention-Deficit Hyperactivity Disorder (ADHD) (Nigg et al., 2010), a reduction in children's tests scores for reading (odds ratio = 0.51,  $p = 0.006$ ) (Chandramouli et al., 2009), writing (odds ratio = 0.49,  $p = 0.003$ ) (Chandramouli et al., 2009; Miranda et al., 2007) and mathematics (Miranda et al., 2007). Canfield et al. (2003) observed that when lifetime average PbB concentrations in children increased from 1 to 10 µg/dL, the intelligence quotient (IQ) declined by 7.4 points. Jusko et al. (2008) observed that compared with children who had lifetime average PbB concentrations < 5 µg/dL, children with lifetime average concentrations between 5 and 9.9 µg/dL scored 4.9 points lower on Full-Scale IQ (91.3 vs. 86.4,  $p = 0.03$ ). Similarly, Surkan et al. (2007) observed that children with 5–10 µg/dL had 5.0 (S.D. 2.3) points lower IQ scores compared to children with PbB levels of 1–2 µg/dL ( $p = 0.03$ ). Interestingly, multiple studies have shown that the strongest Pb effects on IQ occurred within the first few micrograms of PbB (Schnaas et al., 2006; Canfield et al., 2003; Lanphear et al., 2005). Low PbB levels (<10 µg/dL) have also been associated with various physiological outcomes such as kidney damage (Fadrowski et al., 2010), dental caries (Moss et al., 1999), puberty delay in boys (Williams et al., 2010) and girls (Selevan et al., 2003) and cardiovascular outcomes in adults (Navas-Acien et al., 2007).

## 9. Urban soil lead exposure prevention

Although the currently acceptable soil Pb guideline for residential housing is 300 mg/kg Australian, (NEPC, 1999), in Norway the soil Pb guideline for children's play areas is 100 mg/kg, and in California the draft soil Pb guideline is 80 mg/kg (California, 2009). Currently, a full-scale national program is underway in Norway to reduce soil Pb values below 100 mg/kg at all childcare centres, elementary schools and parks in the 10 largest cities (Ottesen et al., 2008). The driver of this cleanup was that in 2005 the Norwegian government promised that every child between the age of 1 and 6 years should have access to daily care in day-care centres, if desired by their parents. As a result, many new day-care centres were established, especially in the cities, and about 75% of all children in this age group spend 6–9 hours in such centres on work-days. Studies of soil pollution in day-care centres in Norway's three largest cities, Trondheim, Bergen and Oslo between 1996 and 2007 revealed the need for soil remediation (including Pb) in up to 38 % of locations (Ottesen et al., 2008). Given the known relationship between environmental Pb and traffic and its impacts on adjacent soils and dusts (LaBelle et al., 1987; Laidlaw and Filippelli, 2008), it is quite probable that schools and day care centres on high traffic volume streets in Sydney and other larger Australian cities may also require remediation similar to that conducted in Norway.

In Australia, precedent for the remediation/isolation of urban soils has been set in Boolaroo NSW near the location of the former Pasmenco smelter. The NSW Department of Environment and Climate has approved a soil Pb abatement strategy for approximately 4000 properties surrounding the former Pasmenco smelter at Boolaroo (Lake Macquarie Council, 2009). Soils with Pb concentration ranging between 300 mg/kg and 2500 mg/kg will be covered with clean soil and areas where soil concentrations exceed 2500 mg/kg the top 5 cm of soil will be removed. In the US, Mielke et al. (2006) demonstrated that exposure to Pb contaminated urban soils can be prevented by covering contaminated soils with about 15 cm of low Pb (median ~5 mg/kg) soil. To achieve that, clean soil is simply graded over the old soil layer, hydroseeded (a slurry of seeds and moisture-retaining fill mixture sprayed onto the ground), and left to grow into a lawn. This approach “caps” the Pb-contaminated soils, and prevents children from coming into contact with soil-borne metals. Yard remediation has been demonstrated to effectively reduce PbB levels. For example, Maisonet et al. (1997) found that yard soil remediation was a protective factor for elevated PbB levels in children (odds ratio, 0.28; confidence interval, 0.08–0.92).

## 10. General comparisons – Australia and the United States

Differences in climate, urban space and socio-economic, ethnic, and racial make-ups may affect exposure to Pb in soil dust in the US and Australia. The climates in the two countries are highly diverse geographically. Sydney has a climate similar to San Diego California, but with more precipitation, Melbourne is located in a climate similar to North Carolina, Adelaide and Perth have similar climates to Los Angeles or San Francisco and Brisbane has a climate similar to the state of Florida. It appears that the garden or lawn spaces in the US inner city areas are larger than in Australian inner cities due a higher building density in Australian cities. Some areas of Australian older inner cities have minimal garden or lawn spaces. This may reduce exposures to lead in soil dust compared to US inner cities. The socioeconomic makeup in the inner cities in the US is also different. In Australia, the inner city residents are comprised primarily of Caucasian, Asian and other ethnic groups with relatively higher socioeconomic status than their US counterparts. In the US, large portions of the inner cities are of lower socioeconomic status consisting of African American, Latino and lesser numbers of Caucasians. The behaviours of people of different socioeconomic levels and racial groups could result in different exposure patterns. Furthermore it is possible that the contribution to PbB caused by exposure to interior Pb paint may be different between the countries due to different physical properties of the paint.

## 11. Blood lead prevalence

It is not possible to quantify the number of children with blood lead poisoning in Australian inner cities because no recent statistically adequate blood lead prevalence studies have been conducted. The soil lead concentrations in Sydney, presented above, may be roughly similar to the soil lead concentrations in New Orleans or Milwaukee. The blood lead prevalence in New Orleans in 2005 was 10.7 % for PbB > 10 µg/dL and 94.1 % for PbB > 2 µg/dL (Howard Mielke, personal communication). In Milwaukee the citywide prevalence for PbB > 10 µg/dL was 4.8 % in 2008. However, PbB prevalence rates > 10 µg/dL are insufficient indicator of risk due to the clear toxicity of PbB concentrations > 5 µg/dL. The 2002 NHANES data indicated that in the US, that for every case of PbB > 10 µg/dL (CDC, 2005), there were approximately 7.7 PbB cases between 5 and 9.9 µg/dL (Iqbal et al., 2008).

## 12. Summary

A review of the scientific literature from Australia and the US indicates that some of the inner-city soils in both countries are variously contaminated with Pb and that soil Pb correlates with children's PbB levels. However, unlike the US, the spatial and temporal pattern of children's PbB levels in Australian inner-city children remains poorly characterised, with the exception of a few limited non-systematic studies. Similarly, the soil Pb distribution in large and regional Australian cities is also characterised by ad-hoc non-systematic studies. Therefore, it is argued here that the risks from low-level Pb exposure from urban soils, the likely predominant Pb reservoir, are unconstrained. Consequently, it is not possible to determine what the health risks are or what appropriate prevention strategies ought to be, although the data point to the potential for a high prevalence of PbB poisoning (>5 µg/dL) in some older inner-city areas of Australia's major cities.

Lead concentrations in some inner-city Sydney areas indicate that soils are highly contaminated and approach the soil Pb concentrations near the former Pb and zinc smelter in North Lake Macquarie (Willmore et al., 2006). We suggest that there is an urgent need for high density soil Pb mapping and universal PbB screening in older areas of large Australian cities with a history of high traffic volumes. Widespread soil Pb remediation should also be evaluated as a method of preventing children's exposure to soil and dust containing Pb in Australia's large inner cities. This is necessary if Australia is to take a precautionary approach to the risks of environmental Pb exposure (Taylor et al., 2010).

## 13. Recommendations

- 1) High density soil Pb mapping should be performed in the inner cities of Australia (see Mielke, 1991, 1994; Mielke et al., 2005);
- 2) On completion of soil Pb mapping in large Australian inner cities, we recommend that an initial PbB screening be targeted in areas where soil Pb concentrations exceed the 300 mg/kg guideline. The PbB screening should be sampled during the summertime because PbB is known to be highest during the summertime (Laidlaw et al., 2005; Laidlaw and Filippelli, 2008; Laidlaw, 2010). Following the initial PbB screening, targeted screening should be terminated in areas that exhibit a low percentage of PbB > 5 µg/dL. However in areas with high percentages of children with PbB > 5 µg/dL, PbB screening should continue until the Pb source or sources are remediated and PbB levels reduced below 5 µg/dL for at least 95% of the children. A PbB concentration of >5 µg/dL was used as the intervention PbB level in Esperance (Western Australia Government Committee of Inquiry Education and Health Standing Committee, 2007). This has recently (2007) become the default action level for children < 5 years old for Western Australia. In addition to remediating soil sources in these areas, it would also be prudent to seal indoor and outdoor flaking Pb paint to prevent further interior particle contamination and exterior soil contamination (Gulson et al., 1995); and
- 3) For transparency, we recommend that all PbB cases > 5 µg/dL be plotted on a GIS map of each city and be made available on the Internet as has already been done by the Wisconsin Department of Health (2010b). In addition, the proposed high density soil lead maps of the large Australian cities should be placed in the same location on the internet. This will allow residents to monitor evidence of progress in the elimination of children's PbB levels and will allow current and future residents to make an informed choice about any potential risks with respect to choices of homes and schools.

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