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Paint as a source of recontamination of houses in urban environments and its role in maintaining elevated blood leads in children

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Abstract

A detailed lead isotopic and scanning electron microscope investigation of particulates from three houses in urban Sydney, previously decontaminated by their owners, has shown that they have been recontaminated over varying periods, as short as 6 months. The source of recontamination is lead paint from adjoining dwellings whose paint is thoroughly deteriorated, as well as from unknown sources. In one house, the external to internal lead loading was > 10:1. The pathway for the lead paint contaminants is both airborne and mechanical transport into the houses. Recontamination of houses provides an explanation for the maintenance of elevated blood lead levels in the children residing in these houses. Recontamination can be a major urban problem applicable in any community which used leaded paints on dwellings in the past. It is a matter of concern for families with young children and couples, especially women who are, or intend to become, pregnant.

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1. Introduction

Recontamination is not an unexpected problem in mining and smelting communities such as Trail (British Columbia, Canada) and Port Pirie (South Australia) although it is not always recognised as a problem. In urban environments it is rarely recognised as a problem although Clark and Bornschein [1] attributed the maintenance of elevated blood leads (PbB) and dust in houses in Cincinnati to recontamination from leaded paints in a deteriorating condition from neighbouring dwellings.

Gibson [2] was the first to draw attention to the potential hazard to children of lead-based paint in dwellings, especially through ingestion by hand-to-mouth activity. Since that time, many studies have documented a correlation between elevated PbB and lead paint, especially in soil and

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dust arising from renovations or 'deleading' [3–13].

There is a commonly held belief in Australia and many other countries that because lead in paint for domestic purposes was discontinued in the early 1970s, lead in paint is not a source of elevated PbB, especially in children. Nevertheless, the regular occurrence of reported (and unreported) cases of lead poisoning in adults and children attributable to home renovations [14–16] attests to lead in paint being a major ongoing problem in urban environments. The most common statement from home renovators when confronted with lead poisoned children and themselves is that: we never considered lead in paint as a problem because it is no longer used in domestic paints. There are >3.5 million houses in Australia built prior to 1971, representing potential sources of leaded paint [17]. In the US, HUD [18] estimated that there are 14 million housing units containing lead paint in unsound condition and there are 3.8 million units with deteriorating paint occupied by young children. Rare cases of infant death resulting from chewing through outer layers of acrylic paint to the underlying lead paint have been reported as recently as August 1993 from Christchurch, NZ, a country and area long accustomed to the lead paint problem in housing [5,15].

Unfortunately, the group most at risk is the young couple purchasing their first house in inner city areas because of price differentials. These couples either have young children or intend to have families. Another group at risk is the investment speculator. In all cases, renovation is the uppermost priority and this activity may be ongoing over several years within their own house or in the neighbourhood.

Lead poisoning of the occupants of houses during renovations is understandable, but not recognised is recontamination of previously decontaminated dwellings from deteriorating paint on neighbouring dwellings, combined with the potential short or long distance transport of fine lead paint particles from home renovations.

In a pilot study to identify the sources of lead in children's blood in an urban environment, we have found that not only are adjacent dwellings with deteriorating paint potential sources of contamination but the lead particles can be transported from not easily identified sources in a neighbourhood.

2. Methods

2.1 Sampling

Three houses, occupied by children with PbB > 10 μg/dl, for whom the principal source of lead was presumed to be petrol were selected. This presumption was based on the absence, or minimal amount, of exposed soil at their residences, no exposed lead paint, the children spending minimal time at other locations and the residences being on relatively busy or narrow streets or both. An added advantage in these residences was the presence of more than one child, which allowed some degree of control.

Venous blood samples were collected following a rigorous protocol [19]. Environmental samples included: soil (if present) from a number of locations, paint if in unsound condition (flaking, powdering), cold water after a 30-s flush, house air over an 8-h period with a low volume sampler run at 2 l/s, vacuum cleaner dust (whole bag collected from owner) and ceiling dust. Initial analyses were performed on 'bulk' samples of soil and dust, i.e. no attempt was made to separate the samples into various size fractions. Later, vacuum cleaner dust and soil samples were sieved to obtain particles of differing diameters, especially those <100 μm, as recommended by P. Mushak (written commun., 1993). These fine fractions are considered critical because: (i) they adhere more strongly to skin [20]; (ii) they are more soluble in the gastrointestinal tract than coarser particles [21]; and (iii) particles <10 μm diameter can be absorbed through the respiratory tract. Selected soil and vacuum cleaner dust samples were concentrated by centrifuging the bulk sample in a heavy liquid, methylene iodide (density 3.2).

Three-month interior dustfall accumulations were collected in 85-mm diameter polycarbonate Petri dishes. In two houses, exterior dust fall accumulation was also monitored. In one house, an ~80-cm high movable platform with sides 5

cm high was constructed of PVC to hold six Petri dishes. The platform was protected from the weather by a canopy of PVC, ~ 130 cm above the dishes. The Petri dishes were removed at monthly intervals. Advantages and disadvantages of dust fall accumulation are described in Gulson et al. [22].

All houses were > 80 years old so that occurrence of lead paints was likely. Paint flakes and some house dust samples were tested for lead using micro-staining methods prior to lead isotope and scanning electron microscopic (SEM) analysis. Interior and exterior dust and soils were randomly sampled with double-sided tape and analysed qualitatively by SEM for major elements. Specific lead phases were then hand-picked for lead isotope analysis. Analytical methods for lead isotopes and SEM are described in Gulson et al. [23].

Lead concentrations in many of the hand-picked grains are only approximate and probably a minimum because they were not able to be accurately weighed. The weights were calculated from the dimensions of the grains measured on the SEM, using a density of 10, once again probably resulting in an underestimate of the lead concentrations.

In trying to draw comparison between paint sources based on chemical composition, it is assumed that 'weathering' of paint over the relatively short period of sampling of months or even weeks will have little impact on compositional changes, i.e. differential leaching of say Ca and Fe relative to Ti and Cr.

3. Results

Lead from food, water and air are estimated to contribute a 'background' value of 6 μg/dl to PbB in 'unexposed' children and adults, even in a community with a point source of lead such as Broken Hill [23]. Other exposures from point sources such as battery factories and smelters are not applicable in this study. The major contributors to elevated PbB in the children under consideration are paint and petrol. The isotopic composition of lead in air, of which > 90% derives from petrol, is well known from measurements of high volume air filters supplied by the NSW Environment Protection Authority over a 3-year period, and is shown by the shaded band in Figs. 1, 3 and 4. The lead isotopic composition of paint is highly variable from one house to another, as will be shown in the following sections. There are, however, some generalisations that can be made at this stage. The majority of paint samples and paint flakes from internal and external house dust samples have $^{206}Pb/^{204}Pb$ ratios > 17.4 and many are > 18, quite different from petrol. These paints commonly have the gross chemical compositions of PbCaFe ± Ba ± Ti. The other main paint population has $^{206}Pb/^{204}Pb$ ratios < 16.2 and they commonly contain Ti ± Zn ± Cr. Some paints have $^{206}Pb/^{204}Pb$ ratios which are similar to those in petrol and hence interpretations of sources of lead in blood and environmental samples are complex.

3.1. House A

House A is a two-storey terrace dwelling, in a densely-populated area, joined on either side by houses with deteriorating lead paint. House A was extensively renovated when it was found that a male child at 23 months had a PbB of 20 μg/dl. Five blood tests for this child over an 18-month period oscillated between 20 and 16 μg/dl and, most recently, after a 30-month period of monitoring, his PbB was 14 μg/dl (parent, written commun., 1993). This child had, and still has, considerable hand-to-mouth activity, in contrast to his 12 month-old brother, whose PbB was 4 μg/dl. Isotope ratios in the older child's blood were considerably higher than that in the house air or petrol-derived lead (Fig. 1). The house was thoroughly investigated over a 12-month period because of the relatively small decline in PbB, in spite of extensive remedial actions by the parents to minimise absorption and exposure that have demonstrated an ability to lower PbB, and visits by remediation consultants. Concern also arose because of high lead concentrations of ~ 3000 ppm Pb in the bulk vacuum cleaner dust first sampled (Table 1), even though the house was decontaminated with the 'Elite' vacuum cleaner dust system, a system which is considered to re-

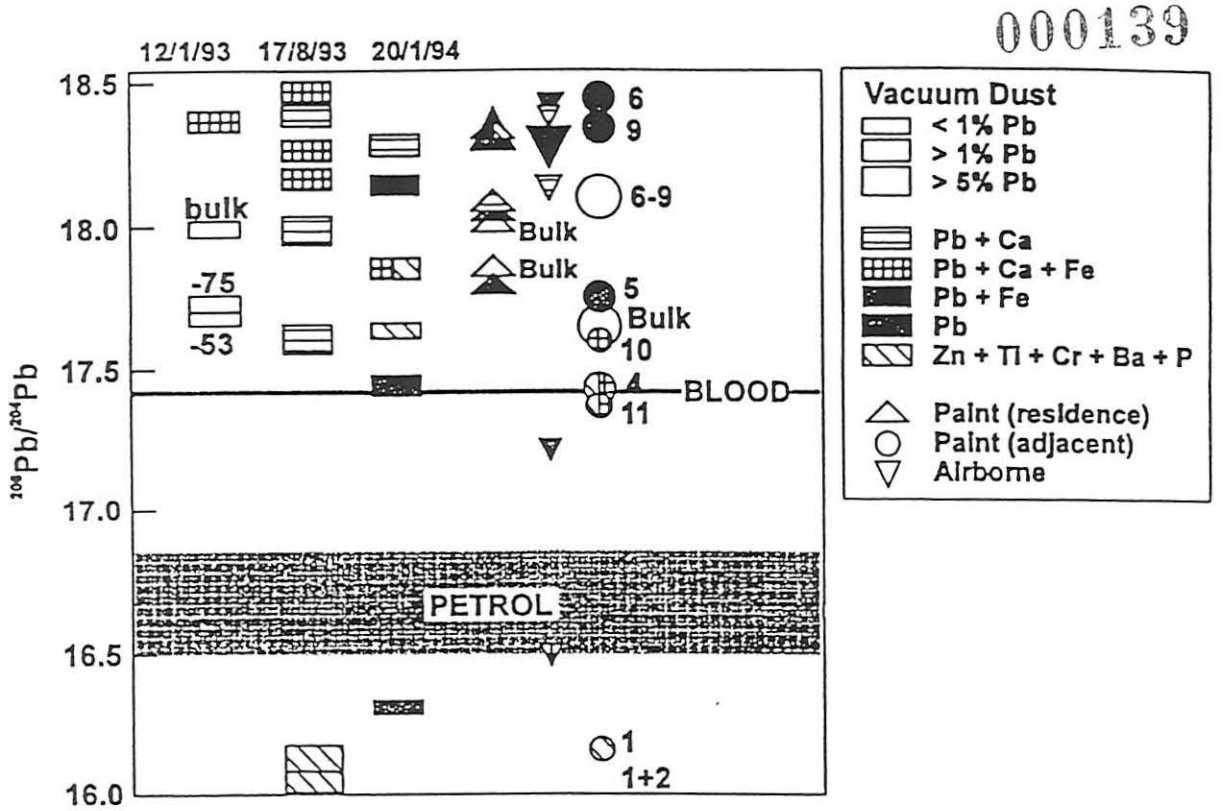

Fig. 1. Isotopic and compositional data for house A. The shaded area, PETROL, denotes data for high volume air filters over a 3-year period and represents lead from petrol; this field also includes the data for several petrol samples. Bulk and sized fractions of vacuum cleaner dust are denoted by 'bulk' and −75 (−75 μm) and −53 (−53 μm). The semi-hatched symbols denote Pb + Ca ± Zn ± Ti ± Cr ± Ba ± P. The numbers on the right side of the symbols for the paint from the adjacent residence are the individual or composited layers.

move all traces of surface lead materials from carpets and rugs (P. Body, written commun., 1993).

Vacuum cleaner dust. Carpet was present only on the stairs leading to the top section of the house with scatter rugs in the downstairs living areas. Inspection of the coarser fractions (−250 + 150 μm) of vacuum cleaner dust failed to detect any lead-bearing phases which almost resulted in a premature abandonment of the study. The main paint particles in the coarser fractions were Ti-bearing phases containing Zn, Ca, P, Fe and Mn as well as phases with CaFe, CaSi and Fe. Except for some samples of PbO, which were presumably originally lead carbonate (the SEM used is not set up for analysis of C), the majority of paints also contained Si and Al, probably as fillers.

However, lead paint was identified in finer fractions. Lead-bearing paint flakes (n = 36) from three vacuum cleaner collections over a 12-month period had compositions of: Pb oxide (?carbonate) (~25%), PbCa (~8%), PbCaFe (~25%), PbFe (~17%), PbCa(Fe) ± Ti/Zn/Ba/Cr (~25%). Data for samples analysed for isotopic composition are listed in Table 1. In spite of the random method of sampling by double-sided tape, the small sample population and potential for composite grains, there were similar amounts of paints with differing chemical compositions. Other particles observed in the finer vacuum cleaner dust had compositions of: TiZn, Fe, CaFe, CaTi, Ti, TiP, TiFeMn and CaSi.

The isotopic composition, and hence source of lead in paint, can vary widely, even in grains with

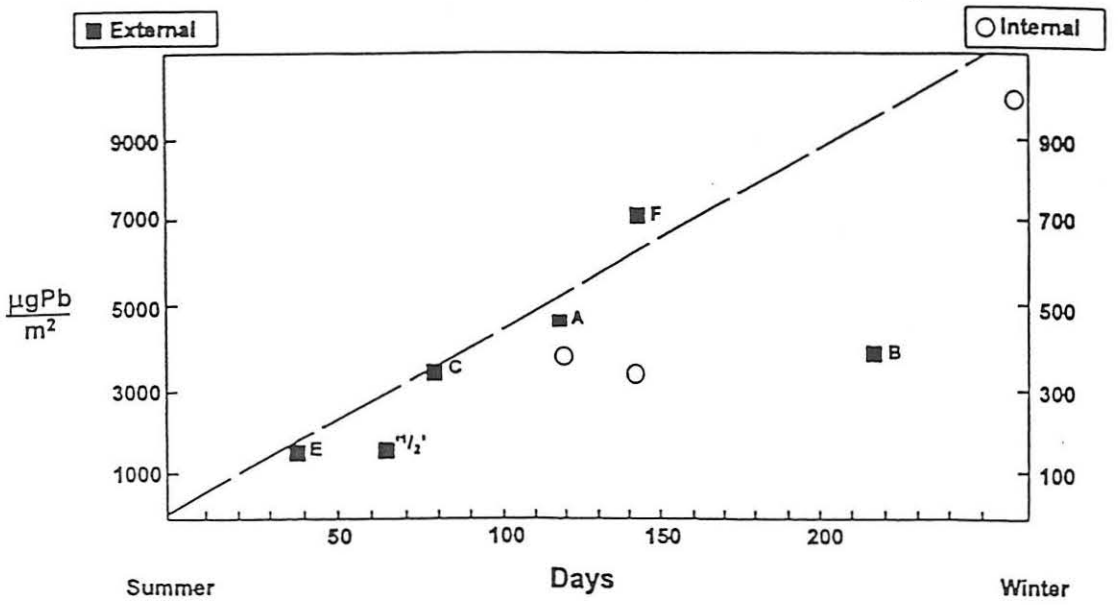

Fig. 2. Plot of lead loading in $\mu g\ Pb/m^2$ versus time for external dust fall accumulation collected in 85-mm Petri dishes in a covered platform from a courtyard enclosed on four sides. The platform was ~3 m from the deteriorating painted wall. The letters denote the different Petri dishes. & '1/2' denotes only half the contents of the dish were ana

the same overall chemical composition. For example, in the vacuum cleaner collection of August 1993, PbCaFe grains had $^{206}Pb/^{204}Pb$ ratios of 18.4 and 18.2 and PbCa grains $^{206}Pb/^{204}Pb$ ratios of 18.4 and 17.9.

External dust. Houses on either side of house A had exterior lead paint which on one side was sealed with epoxy paint. However, one of the adjoining properties had an ~10-m long wall with paint in very poor condition, peeling and in a powdery state. The main doorways and windows of house A faced this wall. During summer in particular, doors and windows of house A were kept open allowing easy access of dust. To evaluate if this wall was the main source of recontamination of house A, samples of paint from the wall and window architraves were collected and external dust fall accumulation was measured in the courtyard of house A, facing the exposed wall, using the platform system described earlier.

The chemical composition of Pb-bearing particles from the 7-month period are: Pb oxide (?carbonate) (~25%), PbCa (~15%), PbCaFe (~25%), PbFe (~15%), PbCa(Fe) ± Ti/Zn/Ba ± Cr (~25%). Even though only a small number were analysed, the types and number of particles in the external dust are similar to the internal dust detected in vacuum cleaner collections. As is the case with the vacuum cleaner dusts, particles of similar chemical composition can have quite different isotopic compositions. Other particles observed in the external dust have compositions of BaZn, FeSi, FeTi, TiFeSi, FeCa and TiCa.

The lead loading in the exterior Petri dishes exhibits a positive relationship with time except for the 5th-month collection, coming into winter, when the loading appeared to decrease (Fig. 2). Considering that the exterior dishes had a protective canopy against the weather so that the lead loading was a minimum, the exterior/interior loading was > 10:1, much higher than in earlier studies of urban environments where the ratio was 1:0.6 [24]. The isotopic composition was relatively homogeneous for the 7-month period of monitoring suggesting that the source of lead was relatively uniform. Lead from petrol may be a contributing source, in addition to paint, as a rare PbBrCl particle was found in one of the interior

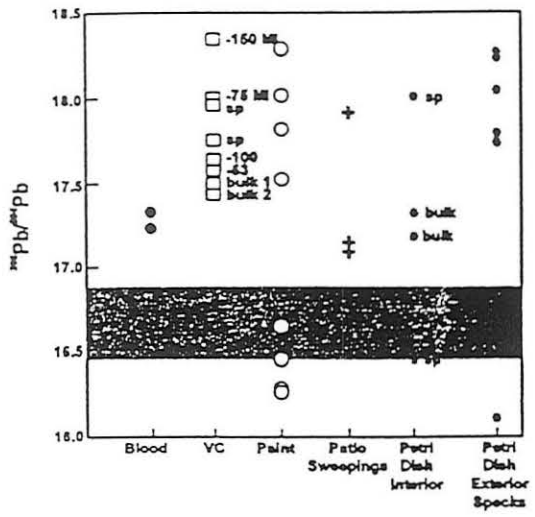

Fig. 3. Isotopic and compositional data for house B. The shaded area (HV AIR RANGE) is the same as 'PETROL' in Fig. 1. Paint denotes paint layers from the wall of the adjoining property. Patio sweepings are analyses of paint flakes of surface dust. Bulk 1 and 2 denotes bulk samples of vacuum cleaner dust collected 6 months apart. −150 MI and −75 MI denote the methylene iodide concentrates of the −150 + 75 μm and −75 μm size fractions; and −100 and −53 the −100 μm and −53 μm fractions. Bulk for the interior dust fall accumulation (Petri dish interior) refers to the analysis of the whole sample. Sp denotes hand-picked grains.

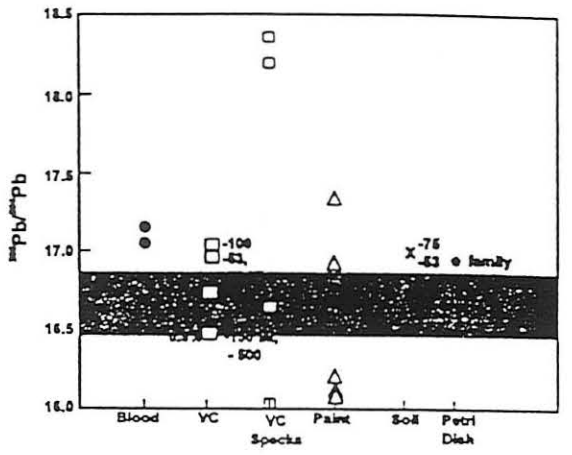

Fig. 4. Isotopic and compositional data for house C. Samples in the VC column are data for the bulk dust and various sized fractions. Samples in the VC specks column denote hand-picked paint flakes from two size fractions of dust, −100 μm and the −75 + 53 μm methylene iodide concentrate. Soil data are for the <75 μm and <53 μm sized fractions. The vacuum cleaner fraction marked −150 MI is for the −150 + 75 μm fraction concentrated in methylene iodide. The Pb oxide speck with a $^{206}Pb/^{204}Pb$ ratio of 18.90 is not plotted.

Petri dishes, and 3 of the smallest rounded grains in the exterior Petri dishes had $^{206}Pb/^{204}Pb$ ratios and chemical compositions which could be interpreted as being derived from a petrol source.

Paint. No leaded paint was present on the interior of house A but paint from an exterior wall into the courtyard had similar chemical and isotopic compositions to house dust, especially the first collection on 12/1993 (Table 1, Fig. 1). However, exterior paint had been wire brushed and sealed with epoxy paint immediately after the first blood test of the child.

12/1/1993

Paint on the wall of the adjoining residence, although only ~250μm thick, consisted of 11 identifiable lead layers (Table 2, A1). There are a similar number of layers in the architrave from around a window in this wall. Layers 4, 10 and 11 from the wall are PbCaFe ± Zn compounds, chemically similar to the PbCaFe compounds observed in dusts from the vacuum cleaner and Petri dishes, and could be interpreted to be the main contributor to the house contamination. However, the PbCaFe compounds from the vacuum cleaner dust have quite different $^{206}Pb/^{204}Pb$ values from those in wall paint from the adjoining property (Fig. 1). Furthermore, the Pb oxides in layers 6 and 9, with $^{206}Pb/^{204}Pb$ ratios of 18.3 and 18.4, have not been detected in interior dust from house A although Pb oxides of similar isotopic composition have been found in the Petri dish in the courtyard below the deteriorating wall.

3.2. *House B*

House B was a two-storey restored terrace house in an 'open' setting compared with house A. House B had no soil. Blood lead concentrations and isotopic compositions of the two girls living in this house were similar (Table 3). The $^{206}Pb/^{204}Pb$ ratio of the bulk sample of paint flakes from the paved area, collected at the same time as blood sampling, had similar isotopic ratios to those in the blood of the girls. The isotopic composition of the paint was also similar to the 3-month dust, especially that from the kitchen, which had double doors opening out onto the

Table 1
Lead isotope data and lead concentrations for samples from house A

Sample type	SEM composition	$^{206}Pb/^{204}Pb$	Pb	Comment
Child blood		17.39	17.3 μg/dl	
VC 1 −75 conc.[a]		17.81	3.1%	
VC 1 −100 + 75 conc.[a]		18.00	4.5%	
VC 1 bulk		17.87	2950	
VC/1 −53		17.69	2660	
VC/1 −75		17.73	2100	
VC/2 −75		17.44	900	
Soil −53		17.65	960	Near sand pit
Soil −75		17.01	3130	Near sand pit
Lead flashing		18.47	na	
Air filter		16.80	0.09	$\mu g/m^3$
Petri dust		17.19	72	$\mu g/m^2$/30 days lounge
Petri dust		17.14	29	$\mu g/m^2$/30 days bedroom
Sweepings bulk		17.11	na	Patio sweepings (rear)
Paint speck[b]	PbCaFe	18.43	> 0.1%	VC Jan 93 −100 + 75μm
Paint speck	PbCaFe	18.36	> 10%	VC Jan 93 −100 + 75μm
Paint bulk	na	16.23	na	Preschool wall
Paint bulk/1	na	17.83	na	Wall near back door
Paint top layer 1	PbFeZnCa	18.00	11%	Wall near back door br/or
Paint layer 2	PbCa	18.06	2.5%	Wall near back door gr/bl
Paint layer 3	PbO	17.77	2.8%	Wall near back door gr/bl gr
Paint layer 4	PbO	18.35	49%	Wall near back door gr/bl wh
Paint bulk/2	na	17.99	30%	Main part of wall
Paint bulk	PbCaFe(Mg)	18.33	0.85%	Top verandah sill
Paint speck	PbCa	18.42	> 2%	VC −150 + 75μm Aug 93
Paint speck	Pb (tr. Zn)	16.13	> 40%	VC −150 + 75μm Aug 93
Paint speck	PbTiCa	16.02	> 0.4%	VC −150 + 75μm Aug 93
Paint speck	PbCa	17.96	8.5%	VC −150 + 75μm Aug 93
Paint speck	PbCaFe	18.16	> 2%	VC −150 + 75μm Aug 93
Paint speck	PbCaFe	18.47	> 2%	VC −150 + 75μm Aug 93
Paint speck	PbCaTiP	17.60	> 5%	VC −150 + 75μm Aug 93
Paint speck	PbCaFeP	18.28	> 1%	VC −150 + 75μm Aug 93
Paint speck	PbFe	18.14	> 1%	VC −100 μm Jan 94
Speck	PbO (SiCa)	16.71	> 4%	VC −100 μm Jan 94
Paint speck	PbCa	18.29	> 3%	VC −100 μm Jan 94
Paint speck	PbCaTiFe	17.84	> 2%	VC −100 μm Jan 94
Paint speck	PbFe	17.41	> 30%	VC −100 μm Jan 94
Paint speck	PbBaZnFe	17.62	> 0.6%	VC −100 μm Jan 94
Paint speck	PbFe	16.17	> 1%	Petri dish behind TV
Paint speck	PbO (Si)	18.29	> 40%	Petri dish A external
Speck	PbCa/PbCaFe	16.50	> 0.3%	Petri dish A external
Paint speck	PbO	18.44	> 5%	Petri dish A external
Paint speck	PbO/PbFe	17.19	> 3%	Petri dish A external
Paint speck	PbCa	18.15	> 4%	Petri dish E external
Paint speck	PbCa	18.38	> 3%	Petri dish C external

[a] Heavy liquid concentrate (methylene iodide).
[b] Paints usually contain SiAI (and O), probably as fillers.
na, not analysed for Pb/composition; VC, vacuum cleaner dust; br, brown; or, orange; gr, grey; bl, blue; wh, white.

Table 2
Paint samples from adjoining properties

Sample type	SEM composition	$^{206}Pb/^{204}Pb$	Pb	Comment
House A1				
Layer 1	PbFeTiCaZnCr	16.14	0.8%	Ochre
Layer 1/2	PbCaTiCr	16.12	1.6%	Purple/white
Layer 4–7	na	17.87	22%	Orange/pink
Layer 4	PbFeCaZn	17.41	na*	Orange/brown
Layer 5	PbO	17.73	na*	Beige/white
Layer 6	PbO	18.45	na*	Beige/pink
Layer 6–9	na	18.09	10%	
Layer 9	PbO	18.35	6.8%	Orange/brown
Layer 10	PbCaFeO	17.69	3.6%	Beige
Layer 11	PbCaFeO	17.37	0.7%	White
Bulk wall	na	17.62	30%	
Bulk window sill	na	17.59	40%	
House A2				
Layer 5	PbTi/PbZnFeCaBa	17.70	> 40%	Salmon (composite)
A	na	17.61	> 50%	

na*, grain size not estimated so no weights available.

paved area; these doors were frequently left open. The windows of the girl's bedrooms face or are immediately adjacent to the peeling wall.

Vacuum cleaner dust. The carpets were replaced after renovation but, nevertheless, two analyses of bulk vacuum cleaner dust 6 months apart post-renovation gave similar lead concentrations of 500 and 460 ppm Pb and similar isotopic compositions. The coarser fractions of vacuum cleaner dust, $-1000 + 500$ μm, $-500 + 250$ μm and $-150 + 75$ μm, contained no lead phases although paint flakes containing Ti and Cr were common. The finer fractions of the vacuum cleaner dust, those containing particles in the size ranges of $-100 + 75$ and $-53 + 38$ μm, had lead concentrations higher than the bulk sample and with isotopic compositions similar to each other but higher than the bulk sample, indicating that the finer fractions contained material with a higher $^{206}Pb/^{204}Pb$ value. This was confirmed by: (i) the analysis of a heavy liquid concentrate (conc.[a], Table 3) of the finer fractions which had $^{206}Pb/^{204}Pb$ ratios >18; and (ii) the -75 μm concentrate contained several lead-bearing phases of Pb oxide composition with isotope ratios of 17.77 and 17.99 (Table 3, Fig. 3).

Interior dust fall accumulation. Dust measured in the kitchen and lounge over a 3-month period, had similar lead concentrations and slightly differing isotopic compositions to each other (Table 3), although the $^{206}Pb/^{204}Pb$ ratios were higher than for petrol-derived lead (Fig. 3). Individual grains hand-picked from this Petri dish had compositions of CaFeAlSiO ($n = 7$), FeSiAlO, FeO and TiSFeSiAlO ($n = 2$), and two of three lead-bearing grains were oxide phases (Table 3). The isotopic compositions of the lead-bearing phases were similar to paint from the neighbouring wall and in the patio sweepings (Fig. 3).

Exterior dust fall accumulation. With no exposed lead paint and completed renovations in the house, it was necessary to identify another source to explain the isotopic composition of the house dust. It was noted that the paint on the wall of the adjoining property was in very poor condition and paint flakes up to 5 mm diameter were regularly deposited on a paved area of house B, between 4–5 metres from the deteriorating neighbouring wall. To evaluate potential external sources of lead, an uncovered pyrex glass Petri dish (200 mm diameter) was placed on a hot water heater outside the house for 3 months.

Table 3
Lead isotopic and lead concentration data for house B

Sample type	SEM composition	$^{206}Pb/^{204}Pb$	Pb	Comment
Child 1 blood		17.34	10.6 μg/dl	18 months
Child 2 blood		17.23	9.5 μg/dl	36 months
VC 1 bulk		17.51	500	Jan 93
VC 1 −53 μm		17.61	870	
VC 1 −100 μm		17.65	1360	
VC 1 −75 conc.[a]		18.02	~1.5%	
VC 1 −150 + 75 conc.[a]		18.32	~1.1%	
VC 2 bulk		17.46	460	June 93
Air filter		16.68	0.94	$\mu g/m^3$
Petri dust		17.16	44	$\mu g/m^2$/30 days lounge
Petri dust		17.31	41	$\mu g/m^2$/30 days kitchen
Petri dust	PbCaBaFe[b]	17.2	0.39%	Kitchen
Petri dust	PbO	18.02	> 10%	Kitchen
Petri dust	PbO	16.48	> 4%	Kitchen
Paint bulk	PbFeCaTiZn[b]	17.22	na	Patio sweepings
Paint speck	PbCaTiFe	17.89	> 0.26%	Patio sweepings
Paint speck	PbFeTiCa	17.18	> 0.6%	Patio sweepings
Paint speck	CaFePbTi	17.83	> 0.16%	Petri dish exterior
Paint speck	na	17.79	> 0.1%	Petri dish exterior
Paint speck	PbZn(Fe)	16.05	> 10%	Petri dish exterior
Paint speck	PbFe	18.31	> 1%	Petri dish exterior
Paint speck	PbFeTiCaP	18.33	> 0.5%	Petri dish exterior
Paint speck	PbO	18.07	> 4%	Petri dish exterior
Paint speck	PbO	18.0	> 10%	Petri dish kitchen
Speck	PbO	16.5	> 4%	Petri dish kitchen
Paint speck	PbO(CaFe)	17.99	> 17%	VC 1 −75 conc.
Paint speck	PbO	17.77	> 10%	VC 1 −75 conc.
Paint samples from adjoining property				
Paint 1A	na	16.70	3.1%	Black/green bit
Paint 1B	FeZnPbO	16.31	na	Black/green bit
Paint 1C	PbCaFeTiZnCu	16.29	na	Black layer 5 bit
Paint 1A	na	16.48	3.8%	White mixed
Paint 1B	PbO	18.02	na	White bottom layer
Paint 1C	PbCa	17.84	na	Beige layer 5
Paint 2A	na	17.52	8.4%	White bottom layer
Paint 2B	PbCa (tr. Fe)	18.31	na	White bottom layer

[a]Heavy liquid concentrate (methylene iodide).
[b]Paints usually contain SiAl (and O), probably as fillers.
na, not analysed for Pb/chemical composition; VC, vacuum cleaner dust; bit, bituminous type paint.
Paint 1 and 2 refer to two different locations on the wall.
Paint A, B and C denote separate analyses of a new sample.
Paints A are mixed layers (not finely separated).

FeCaTi, FeCaZn and Pb oxide phases were the most common lead-bearing particles found in the Petri dish and patio sweepings. Other flakes had compositions of Ti and Cr ± Zn ± Ca.

Five of the six specks analysed had $^{206}Pb/^{204}Pb$ values much higher than the bulk sample of sweepings from the patio (Table 3) and two of these had the same values as the concentrates

from vacuum cleaner dust. A paint flake from the sweepings had a similar isotopic composition to the airborne flakes observed in the Petri dish. Another PbZn oxide speck had a $^{206}Pb/^{204}Pb$ ratio of 16.05, much lower than any samples of paint or house dust (Fig. 3). One half of the paint flakes from the exterior dust fall accumulation had chemical and isotopic compositions which differed from those observed in the paint from the deteriorating neighbouring wall, described in the next paragraph.

Paint. The only flaking paint on the front of the house was 'lead-free' and the children did not play in this area. Several paint flakes were brushed and scraped from the neighbouring wall in two places, one immediately outside the bedroom window of child 1. The paint samples were surprisingly simple with only two to three layers, including an unusual black/green bituminous layer in paint 1. However, this simplicity was not borne out by isotopic analyses. The first samples of bulk paint analysed (paints 1A and 2A) from the neighbouring wall had isotopic compositions quite different from the individual grains in the Petri dish, especially those with $^{206}Pb/^{204}Pb$ values < 17. The second and third analyses of the paint separated into layers showed that the bituminous type paint had consistent isotopic compositions but that there were some differences in the lighter-coloured paints. Some of these lighter-coloured paints had $^{206}Pb/^{204}Pb$ ratios similar to those found in the vacuum cleaner methylene iodide concentrates and in the dust from the interior and exterior Petri dishes but there were differences in chemical composition (Table 3), which were unlikely to be attributable to 'weathering' phenomena resulting in a loss of Fe, Ti, etc.

3.3. House C

House C is a two-storey terrace dwelling in a densely populated area which had been extensively renovated over decades by different owners. The $^{206}Pb/^{204}Pb$ ratio in the blood of the two children in this house was similar, in spite of the 100% difference in PbB concentration (Table 4).

Soil. The finer fractions of soil from the sand pit/garden contained high amounts of lead and had the same isotopic ratios, similar to those in the children's blood (Table 4, Fig. 4).

Vacuum cleaner dust. The bulk sample of vacuum cleaner dust contained 1580 ppm Pb and a $^{206}Pb/^{204}Pb$ ratio similar to that in the coarser fractions $-500 + 250$ and $-150 + 75$ μm. No lead phases were observed in the $-1000 + 500$ and $-500 + 250$ μm fractions, with the most common phases containing TiCaFe $\pm$ Zn $\pm$ Cr. The finer fractions of vacuum cleaner dust, including the $-75 + 53$ μm concentrate, contained > 2000 ppm Pb and had the same isotopic compositions, which were the same as the children's blood and soil. The most common lead phases in the -100 μm fractions and $-75 + 53$ μm concentrate were PbCaFe $\pm$ Ba $\pm$ Zn. Strangely, three PbCaFe-bearing grains from the $-75 + 53$ μm concentrate had $^{206}Pb/^{204}Pb$ ratios of 18.3–18.4, significantly higher than the bulk and fractions of vacuum cleaner dust.

Interior dust. The lead loading was higher than in the other two houses and also there were larger isotopic differences between the two locations. The lower $^{206}Pb/^{204}Pb$ ratio in the kitchen Petri dish may reflect a contribution of paint with a low $^{206}Pb/^{204}Pb$ ratio from the renovations which were being carried out around the stairwell area, which led into the kitchen.

Exterior sweepings. No lead phases were observed in the sweepings from the paved area at the rear of the house but TiCa $\pm$ Fe phases were common.

Paint. The children's sandpit was located beneath a wall exhibiting dispersed areas of peeling paint. The paint was complex with nine layers ranging from outer to inner: CaTiFe (layers 1, 2), TiO (layer 3), TiFe (layer 4), PbFeZn (layer 5), PbFeO (layer 6), PbO (layer 7) and Ca (layers 8, 9) at the base. The outer TiCa-bearing layers were chemically similar to paint flakes observed in patio sweepings and in the coarser fractions of vacuum cleaner dust. Walls near and under a stairwell in the house were undergoing renovation at the time of follow-up sampling, i.e. post blood, urine and environmental sampling. The paint consisted mainly of Ti and TiCa $\pm$ Zn $\pm$ Ba $\pm$ Cr $\pm$ Fe with a thin middle layer consisting of PbZnBaFeCa. This paint had chemical and iso-

Table 4
Lead isotopic and lead concentration data for house C

Sample type	SEM composition	$^{206}Pb/^{204}Pb$	Pb	Comment
Child 1 blood		17.07	13.6 μg/dl	
Child 2 blood		17.16	26.0 μg/dl	
VC bulk		16.73	1580	
VC −53		16.98	2150	
VC −100		17.03	2140	
VC −75 + 53 conc.[a]		16.96	1.3%	
VC 150 + 75 conc.[a]		16.45	0.8%	
VC −500 + 250		16.49	1240	
Soil −53		17.00	1490	Sand pit/garden
Soil −75		17.01	3130	Sand pit/garden
Air filter		16.65	0.16	$\mu g/m^3$
Petri dust		16.94	122	$\mu g/m^2$/30 days family room
Petri dust		16.66	91	$\mu g/m^2$/30 days kitchen
Paint bulk/1	PbFeTi/PbCaTi	16.88	na	Wall adj sand pit
Paint speck/1	PbZnCaFe	16.19	12%	Wall adj sand pit
Paint speck/2	PbFeZn(Ca)	16.10	1.7%	Wall adj sand pit layer 5 br
Paint speck/2	PbFe	16.12	> 0.9%	Wall adj sand pit layer 6 pi
Paint speck/2	PbO	17.31	> 3%	Wall adj sand pit layer 7 wh
Paint bulk	PbZnBaFeCa	16.02	na	Under stairwell brown
Paint speck	PbBa(Ca)	16.06	> 9%	VC −100μm
Paint speck	PbCaBaFeZn	16.64	> 10%	VC −100μm
Paint speck	PbCaFe	18.30	> 30%	VC −75 + 38 conc.[a]
Paint speck	PbFeCa	18.45	> 14%	VC −75 + 38 conc.[a]

[a] Heavy liquid concentrate (methylene iodide).
[b] Paints usually contain SiAI (and O), probably as fillers.
na, not analysed for Pb/composition; VC, vacuum cleaner dust; br, brown; pi, pink; wh, white.

topic similarities with some layers of paint from the wall above the sandpit. Paint flakes from the finer fractions of vacuum cleaner dust and soil from the sandpit had chemical compositions of PbCaFeAlSiO, determined by the SEM analyses, and which were similar to those in the paint from the wall. In addition, some of the paint from the wall had the high $^{206}Pb/^{204}Pb$ ratio (Table 4) measured in grains of similar chemical composition found in the vacuum cleaner dust concentrates.

4. Discussion

4.1. Sources and pathways

Instead of establishing that the elevated PbB in the children from the three houses was from exposure to petrol, it appears from this study that paint was the major source. Even though the lead in air from all three houses had isotopic ratios which are consistent with a derivation from petrol (Figs. 1,3,4), the differences in the isotopic composition of blood and petrol suggest that petrol is not the major source of lead in the children's blood.

It is highly unlikely that paint from house A was a contributor to vacuum cleaner dust as the exterior paint from house A had been sealed with epoxy paint and interior paint was non-leaded. As there was no obvious source of lead paint within the house and upstairs carpets were cleaned with the 'Elite' system, recontamination of the house within a few months must be from lead paint flakes from other sources. The lead-bearing paint flakes in the exterior dust observed in the courtyard of this house came from diverse sources. Based on differences in chemical composition and isotopic ratios, recontamination was not only

from the adjacent dwelling(s) but there was also a large amount of extraneous paint. With the ongoing recontamination of this house and continuing hand-to-mouth activity of the 4 year-old sibling, it is not surprising that his PbB was declining slowly, in spite of the intensive efforts to minimise exposure by his parents. After several visits and consideration of the isotopic data, the early lead insult to the older sibling was attributed to the crawling stage when, daily, he slid on his buttocks along a passageway below the deteriorating painted wall of his own residence (prior to its sealing with epoxy paint), as well as exposure from the neighbouring deteriorating wall (house A1). At this stage, it would appear that paint from other sources, as well as from the neighbouring wall of house A1, were resulting in the contamination of house A.

The distinctive chemical composition of PbFeCaTiZnCr and low $^{206}Pb/^{204}Pb$ ratio observed in the outer layers of the wall paint from the neighbouring house A1 would suggest that it could be a diagnostic indicator of source. No paint flakes have been observed so far in house A with these diagnostic characteristics although some from the 18/9/1993 1993 vacuum cleaner dust contained Ti and others with traces of Zn and had the same isotopic composition as the outer layers from the neighbouring wall. The outer layers of the neighbouring wall paint tend to disintegrate as large flakes and so probably the majority of flakes deposit immediately below the wall, as the gravitational settling speed of particles with radii > 100 μm and density 5 g/cm^3 is > 1 m/s [25].

Other pathways, besides airborne transportation, for the dust to be introduced through doors and windows to house A are mechanical transport on shoes, clothing and hands, especially when the children play outside. The soil in the garden was relatively high in lead, especially the fine fraction and the isotopic composition was consistent with the same sources of lead observed in the Petri dishes. The abundance of lead-bearing phases in the courtyard area attest to airborne transportation through doors and windows as important pathways.

A diagrammatic representation of sources and pathways for house A is shown in Fig. 5 but this is probably also valid for houses B and C.

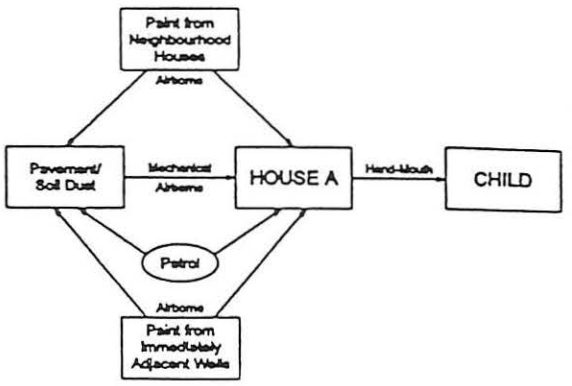

Fig. 5. Sources and pathways of lead for children with elevated PbB in an urban environment.

Interpretation of the isotopic and chemical composition data for house B are the same as for house A. That is, house B was renovated, new carpets laid and there was no obvious source of lead paint. Similarities in the isotopic and chemical composition of lead paint flakes, especially lead oxides, from external dust fall accumulation and flakes from the methylene iodide concentrates for the vacuum cleaner dust are consistent with a derivation from deteriorating paint on the adjoining building. However, as 50% the analysed paint flakes from the external dust fall accumulation are different in chemical and isotopic composition to paints from the neighbouring wall, an extraneous source is suggested.

Even though the most logical source of lead in house C was from deteriorating paint on the back wall of their property, with a pathway from the wall to the sand pit or paved area and onto children's hands, as well as mechanical transport to the house via clothing, shoes and hands, the absence of such paints in the house dust suggest that the back wall was not the dominant source of lead. For example, some grains in the vacuum cleaner dust had similar chemical compositions of PbCaFe to the paint on the wall but completely different $^{206}Pb/^{204}Pb$ values of 16.12 and 18.45, respectively; likewise, for Ba- and Zn-bearing paints. Either the PbBa-bearing paints were a residue from earlier renovations or they were a contaminant from neighbouring renovations, as suggested for houses A and B. The possibility that the Ba-containing paints were a residue of earlier

renovations is consistent with the studies of Inskip and Hutton (1988) which demonstrated that vestiges of lead paint removed by sanding could remain in a house for >3 years. Furthermore, PbZnBaFeCa (AlSi) paint was found under the stairwell in house C and its isotopic composition was similar to some paint layers on the back wall above the sand pit.

If it is conceded that the Ti and Cr-bearing paints in the patio sweepings and coarser vacuum cleaner dust in house C are indeed sourced from the back wall of the property, it might be anticipated to also observe lead-bearing phases in the sweepings and coarser vacuum cleaner fractions because of the layered nature of the paints. At this stage, no explanation for the absence of lead phases can be given apart from a sampling bias. Another concern is the smaller number of lead-bearing particles in the interior Petri dishes compared with those in the exterior Petri dish and vacuum cleaner dust, an indication that mechanical transport is more important than airborne transport.

4.2. Transportation

From where do the extraneous lead-bearing flakes come? The majority of lead-bearing particles accumulated in the Petri dishes and vacuum cleaner dust are in the range 10–70 μm diameter. Particles in this size range are those commonly generated by either mechanical or hand sanding ([14]; F. Salome, pers commun, 1993) as is the powdery paint so common in the middle to inner (older) layers of paint on buildings and is possibly removed by wind/rain action. Furthermore, the presence of such particles is not at all obvious to the home owner as particles <40 μm are invisible to the naked eye [26]. Depending on weather conditions, especially wind speed, the fine particles may be transported considerable distances of several hundred metres to kilometres [25]. For example, for gravitational settling from plumes, $\sim 50\%$ of the material of particle size 20 μm, with density 5 g/cm^3 and wind speed of 5 m/s would be deposited within 83 m of a source with a height of 1 m and 417 m for a source height of 5 m [25]. Transport distance may be enhanced by the platy nature of many of the fine particulates allowing them to remain suspended for longer periods. Another factor impacting on transport distance, redistribution or resuspension and possibly mechanical removal of deteriorated paint on walls is the building styles. Many of the terrace houses are 'connected' by an open passageway which facilitates funnelling of the wind into a vortex and often extremely strong winds according to residents.

Paint flakes up to 5 mm diameter were regularly swept from the patio of house B and similar sized flakes were collected in the external Petri dish. The ability of such coarse flakes to become airborne in the turbulent air stream that can operate in the 'alleyways' between houses was unexpected, given the particle sizes in the literature [25]. Furthermore, if it is conceded that the flakes observed in house dust, patio sweepings and external dust are from the neighbouring wall, it demonstrates the ability of wind and possibly rain to remove paint from deteriorating painted surfaces.

5. Conclusions

A combined lead isotopic and SEM investigation has shown that decontaminated houses with no obvious source of lead can be recontaminated within months by sources from adjoining buildings, with leaded paint in poor condition, as well as from unknown sources. Questionnaires with or without blood lead screening [27,28] may not have identified at risk groups such as the children investigated here.

Given the living conditions in many urban environments of 'open' air living, and enthusiastic renovators, recontamination of houses with lead paint can be a major urban problem. Unfortunately, renovation and recontamination impacts on the most sensitive population, families with young children and also on pregnant mothers. Furthermore, renovation and recontamination provide an explanation for maintenance of elevated PbB in children even though their house may have been decontaminated.

At this stage it is not possible to estimate the magnitude of the recontamination problem because of the lack of knowledge on such factors as the ability of fine particles to become resuspended. Furthermore, information on the 'bio-

availability' of paints is lacking; are the Ti and Cr bearing paints less bioavailable compared with the oxides (?carbonates) and so impact minimally on PbB?

Not only are (or were) most people ignorant of the problem of self-contamination by the homeowner, a recognised problem, but because whole blocks of houses in earlier days were apparently painted at the same time, it is not easy to attribute any particular paint species to a specific residence in a street. Hence, legal action is not a viable option to an owner whose home has been recontaminated but regulations need to be introduced to control house renovations where lead paint can be dispersed into the environment.

As home renovation is a way of life, the owner should take action to minimise exposure to occupants as well as to the neighbours. Effective measures can be taken by parents of young children to minimise their exposure to lead as shown by the low PbB in the younger sibling from house A compared with the older sibling who had elevated PbB. Some of the measures include: (i) safe work practices for renovation, documented in reports by, for example, Canada Mortgage and Housing Corporation [29], the US National Centre for Lead Free Housing [30] and the Commonwealth Environmental Protection Agency [31]; (ii) actions in the house such as washing paved areas, covering sand pits, replacing the top layer of sand at regular intervals and closing doors and windows on windy days, if possible; and (iii) personal hygiene such as regular washing of hands and toys and discouraging mouthing of objects/fingers. In addition, there is much literature available from environmental protection and health authorities describing measures to minimise lead exposure. To overcome neighbourhood contamination, especially from deleading using sanding methods, it may be necessary to enact regulations for containment in much the same manner as required for structures such as bridges.

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CONTRIBUTION OF TISSUE LEAD TO BLOOD LEAD IN ADULT FEMALE SUBJECTS BASED ON STABLE LEAD ISOTOPE METHODS

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ABSTRACT

Public health and medical recommendations on prevention of lead toxicity rely on use of blood lead concentrations to assess lead exposure and predict onset of adverse health effects. Blood lead levels have generally been thought to reflect recent environmental lead exposures. However, tissue lead stores are accumulated over a long time period (i.e., years). These tissue stores, primarily from bone, can be remobilized as part of both normal physiological, as well as pathological, processes. Although chemical analyses do not differentiate lead isotopes, mass spectrometric determinations can differentiate the quantities of stable lead isotopes present in particular samples (e.g., ^{207}Pb, ^{206}Pb, ^{204}Pb, and ^{202}Pb). Selected geographic locations may have distinct isotopic profiles. For example, on mainland Australia the $^{206}Pb/^{204}Pb$ ratios reported in both environmental lead sources and blood samples are typically less than 17.0. By contrast, lead stable isotope profiles in blood samples of adult women immigrating from Eastern Europe and former USSR usually have $^{206}Pb/^{204}Pb$ greater than greater than 17.5 and, as high as, 18.5 on entry into Australia. Longitudinal monitoring of blood samples to determine lead stable isotope profiles by mass spectrometry and chemical analyses of blood samples for total lead content were conducted over a 300-day period. These data show that between 45 and 70% of lead in blood comes from long-term tissue lead stores. Recognition that the predominant source of lead in blood was tissue stores rather than the contemporaneous environment should greatly modify recommendations on use of blood lead to monitor occupational or environmental interventions. In addition, internal biokinetics of lead, documented through presence of tissue lead in blood, underlie the long-term health risks of lead exposure. Transfer of lead to the fetus from maternal tissue stores represents a special area of concern.

INTRODUCTION

Lead toxicity is a major public health concern in the United States and other Western countries, notably Australia, and is a primary environmental health issue.[1-4] In recent

years. United States recommendations on screening programs aimed at prevention have identified target blood lead concentrations[1,2,5] associated with adverse health effects of lead. Although the neurobehavioral deficits associated with low level lead exposures are best documented for lead exposures early in childhood,[6-8] effects of lead on the developing nervous system are of concern. Lead readily crosses the placenta.[9,10] Cord blood lead concentrations are approximately 85 to 90% of maternal blood lead concentrations.[10] Changes in maternal blood lead are thought to result in alterations to fetal lead exposures. Mobilization of lead from body stores incorporated prior to pregnancy has been established in experimental animals.[11,12] Public health recommendations for pregnant women include maintaining blood lead concentrations less than the levels of concern for young children; i.e., < 10 μg/dL (0.48 μmol per liter).[1,3] However, current recommendations for acceptable occupational concentrations are substantially higher for adults of reproductive age [e.g., 30 μg/dL (1.44 μmol per liter)][5] and standards for medical removal in occupational settings are 50 μg/dL (2.40 μmol per liter) or higher.[5]

Lead enters the blood from both the contemporaneous environment (e.g., lead absorbed from air, food, water, dust, etc.), as well as lead mobilized from tissue stores. Such stores are assumed to largely reflect lead accumulated in bone because > 90% of tissue lead stores are in osseous tissues among adults.[13]

Analyses of total lead concentration based on chemical methods cannot distinguish one source of lead from another. However, additional methods can be applied to identify specific lead sources under some environmental circumstances. Lead is unusual among elements in that four stable lead isotopes occur in nature (^{208}Pb, ^{207}Pb, ^{206}Pb and ^{204}Pb). Isotopes have identical chemical properties, but have slightly different atomic weights due to the different number of neutrons in the nucleus. Stable lead isotopes result from long-lived radioactive decay of the parent uranium isotopes 238 and 235 and the parent thorium isotope 232 to ^{206}Pb, ^{207}Pb and ^{208}Pb, respectively. These decay products of uranium are formed at very different rates because of their vastly different half-lives. ^{204}Pb is the lead that

was present when the earth was formed and its abundance has remained essentially unchanged over this interval. These naturally occurring lead isotopes vary with the geological age in the ore deposit bodies. Thus, the ~1700 million year-old lead-zinc-silver deposits in Broken Hill, New South Wales, Australia have a $^{206}Pb/^{204}Pb$ ratio of 16.0, while the ~400 million year-old deposits in the same country have ratios >18.0. The ratio of these stable isotopes to one another (e.g., $^{206}Pb/^{204}Pb$ or $^{206}Pb/^{207}Pb$) provide a characteristic "fingerprint", "signature" or profile of the lead source.

Stable isotopes of lead can be determined using thermal ionization mass spectrometry (TIMS). This methodology is extremely sensitive, precise and accurate. TIMS has been used in earth sciences for decades, however, limited application of TIMS to biological samples has occurred primarily within the past 15 years.[14-18]

Ethical considerations restrict addition of lead to the environment of pregnant women. Consequently research findings on comparative importance of various environmental sources of lead must be based on naturally occurring circumstances. In many regions of the world, the stable isotope profiles are quite variable. However, some countries (e.g., Australia) have a characteristic profile of stable lead isotopes. For example, extensive environmental and biological monitoring of the stable isotope profiles in several Australian communities has revealed that the $^{206}Pb/^{204}Pb$ ratios are typically 16.5 to 17.0.[19-21] By contrast most other countries have stable isotope profiles in which the $^{206}Pb/^{204}Pb$ ratios are greater than 17.5.[15,17,18,22] (Figure 1). Because of the precision and accuracy of TIMS, the width of a 95% confidence interval for differences in $^{206}Pb/^{204}Pb$ ratio is generally less than 0.05. Thus differences between Australian isotope ratios and those from other countries, being of the order of 0.5, are easily detected.

Information on the tissue turnover and redistribution of absorbed lead (termed biokinetics) for female adults is almost non-existent. In addition, there is not only uncertainty on whether or not lead is mobilized from human skeletal stores during pregnancy and lactation, but also how much is mobilized and under what conditions[23-25], although

skeletal lead mobilization during pregnancy has been established for experimental animals.[11,12]

To address the extremely limited data on lead biokinetics in adult female subjects, we have undertaken a long-term study employing high precision measurements of stable lead isotope ratios on adult women in natural environments (i.e., nonoccupational exposures). Because of ethical considerations we have relied on natural circumstances that permit comparison of the contribution to blood lead of long-term body burden of lead and the contribution of lead absorbed from the contemporaneous environment. This has been possible because of immigration of persons into a geographic region with a distinctly different stable isotope profile from that of their country of origin. Australia has had an active immigration program for subjects from Europe for several decades and in recent years, there has been an increase in the number of immigrant families from Central Europe and the former USSR. Typically the $^{206}Pb/^{204}Pb$ profiles of these immigrant women of child-bearing age upon arrival to Australia is in excess of 17.5, and as high as 18.5.

This paper presents results from a pilot study to determine the response of total blood lead concentration and lead isotope profiles of these women after arrival in Australia. In addition, these data permitted assessment of the extent and time needed for these concentrations and isotope ratios to approach those of native-born Australian women of comparable age and city of residence. This enabled us to estimate the relative contributions of lead from the contemporaneous environment and lead mobilized/remobilized from body stores. The former was determined through monitoring of the subjects' sources of lead exposure. The latter is assessed from knowledge of the women's blood lead isotopic profile and concentration within four weeks of her entry into Australia and from tooth analyses of spontaneously shed deciduous teeth of children who accompanied their mothers from Central Europe.[a] As lead in blood is constantly exchanging with lead in the skeleton, under conditions of equilibrium such as residence in the one locality for several years,

[a]Stable lead isotope ratios of these teeth are available from one of the authors (BG).

an analysis of blood can provide an estimate of the skeletal isotopic lead. An analysis of blood from each subject as soon as possible after arrival in Australia ("initial" lead) thus provides a proxy for skeletal lead.

METHODS

We have recruited 11 recently arrived female immigrants of child-bearing age (20 to 36 years of age) from Eastern European countries (Bulgaria, former USSR (Commonwealth of Independent States), Poland, and Romania) whose blood lead isotope profiles upon arrival are significantly different from the relatively uniform profiles found in the Australian population (Gulson et al.[19]). One 45-year-old, long-term Australian female resident was enlisted as a concurrent reference for Australian stable lead isotope profiles in blood (Figure 2). Recruitment of the immigrants was as soon as possible after arrival in Sydney, always within one month and commonly within two weeks. All claimed to have come directly from their place of origin. Ten of the 11 were married and with either no children or, more commonly, one or two children.

Subjects were typically recruited by "networking" within their ethnic communities. If, following a visit by persons involved in the project, subjects agreed to participate, they were asked to sign a consent form describing the project, its risks and benefits to them. This consent form had been reviewed and approved by the Ethics Committee of St. Vincent's Hospital in Sydney, Australia, the University of Adelaide in Adelaide, Australia and the United States National Institutes of Health.

Environmental lead assessments were also performed. The main sources of environmental lead are air, food, and water. As approximately 50-60% of automobiles in Australia still require leaded-gasoline, the dominant source of lead in air is gasoline additive lead (Figure 3). For this pilot study, systematic environmental monitoring was not undertaken. However, no significant changes to the environmental variables occurred compared with data reported by Gulson et al.[19,20] Verification of stable isotope data in

Sydney was from an on-going monthly air-monitoring program in south-eastern Australia[19,20] and from consistency in the isotopic data for the Australian female reference subject and a 50-year-old, Australian, male reference subject.

Monthly venipuncture blood samples were taken by a trained phlebotomist, following a strict protocol to minimize contamination, and placed in cleaned, preweighed, teflon containers without anti-coagulant. To minimize sample heterogeneity, the total weighed blood sample was pre-digested in concentrated nitric acid and an aliquot of known weight removed to a clean teflon vessel. A ^{202}Pb solution of known isotopic composition and lead concentration (a "spike") was added to the aliquot to obtain the concentration of lead and isotopic composition of the unknown sample in the one analysis; this is the isotope dilution method. [^{202}Pb is not naturally occurring and is produced in cyclotrons as a byproduct of preparation of thallium used in treatment of thyroid abnormalities.] Lead was separated from interfering elements such as Fe and Zn by anion-exchange chromatography in a hydrobromic acid medium. High precision isotope abundance ratios ($^{206}Pb/^{204}Pb$, $^{207}Pb/^{204}Pb$ and $^{208}Pb/^{206}Pb$) were measured on the purified lead sample in an Isomass 54E solid source thermal ionization mass spectrometer run in fully automatic mode. Once the ^{202}Pb solution is mixed with the sample, further quantitative recovery of lead is not necessary. The isotopic ratios are presented in this study as $^{206}Pb/^{204}Pb$, in contrast to many earlier papers that expressed the data as the $^{206}Pb/^{207}Pb$ ratio, because of the difficulty in precisely measuring the low abundance ^{204}Pb isotope identified in earlier studies. Accuracy of the isotopic ratios is controlled by measurement of about 10 nanograms (ng) of the NIST Common Lead Standard SRM 981 at the same time as the unknown samples are measured. Precision of the $^{206}Pb/^{204}Pb$ ratio is ± 0.20% (2 sigma) for a blood sample containing < 5 μg/dL (0.24 μmol per liter) and is based on replicate analyses of SRM 981 and real samples. Precision may be considered as repeatability of analyses and is controlled by: 1) repeated measurements of NIST 981 with each batch of samples; 2) replicate analyses of the one blood sample (as in validation for Phase I of this study[19]). Additional unpublished data from the CSIRO

laboratories showed a ±0.1% (1 sigma) variation in $^{206}Pb/^{204}Pb$ for sequential blood samples from the one male subject over a six-month period. This included differences arising from phlebotomy techniques.

Contamination levels (blanks) for the processing of blood samples varied from 70 to 200 picograms. No correction for this blank was made to our data because it contributed less than 1% to the amount of lead being processed and so made insignificant changes to the data. A correction of + 0.08% per mass unit due to fractionation of the isotope ratios by heating during the mass spectrometer measurements has been applied to the data.

The half-life ($t_{½}$) of lead in blood (or elimination rate of lead from blood) for each subject was calculated using a non-linear least squares fitting routine. The half-life or $t_{½}$ of $^{206}Pb/^{204}Pb$ was calculated by fitting a model of the form $^{206}Pb/^{204}Pb \approx a + c * \exp(-t/lambda)$ to describe the decay in the lead isotope ratio at time "t". "t" = 0 was taken to be the time of the initial measurement. The method of non-linear least squares was used to estimate the parameters "a", "c" and "lamba" in the above model. These parameters have the following interpretation: the half-life of the decay is given by $t_{½}$ = lambda ln2; the parameter "a" represents the ultimate (steady-state) level of the $^{206}Pb/^{204}Pb$ ratio; "c" is the difference between the initial and final values of the ratio or the total reduction in the ratio. Blood lead concentrations and $^{206}Pb/^{204}Pb$ ratio over time for each subject were tested using both a single exponential model (reflecting only the blood compartment) and a double exponential model (two compartments reflecting blood plus other compartments such as skeletal tissue). Because of the variability in blood lead concentrations, especially for some subjects with low blood lead concentrations which actually increased with time, the half-life calculations for variations of blood lead concentration are meaningless. As the data for a two-compartment model do not provide a better fit statistically than does the one-compartment model, the results are presented in terms of a one-compartment model. The half-lives would be lower (faster) if a two-compartment model were used in the calculations.

The $^{206}Pb/^{204}Pb$ ratio is the preferred ratio as differences are expressed in the first decimal place of a large number, and are simpler to view than, for example, differences in the third or fourth decimal place for the $^{208}Pb/^{206}Pb$ (or $^{206}Pb/^{208}Pb$) and $^{207}Pb/^{206}Pb$ (or $^{206}Pb/^{207}Pb$) ratios, whose values may be of the order of 2.1000 or 0.9000 respectively.

RESULTS

Lead isotope ratios (expressed as the $^{206}Pb/^{204}Pb$ ratio) and lead contents were determined for a three-hundred day period. These data were compared with the lead isotope profile present in the Australian environment as assessed by environmental monitoring (Gulson et al.[19-21]) and by inclusion of one native-born Australian woman residing in Sydney as a concurrent reference for Australian lead isotopic profile in blood. Individual variation in the rate of change in blood lead concentration and modification in the isotope profile occurred [individual data are available from one of the authors (B. Gulson)]. Data for the two subjects showing the greatest differences in rate at which they approached the Australian lead isotope profile are shown in Figures 4 and 5. Statistical treatment of these data permit calculation of the half-life of lead in blood, the half-lives ± one standard error are presented in Table 1.

The blood lead concentration of these women within one month of entry into Australia varied between 20.0 μg/dL (0.96 μmol per liter) (subject number 809 from Bulgaria) and 2.6 μg/dL (0.12 μmol per liter) (subject number 802 from Commonwealth of Independent States). Subjects with blood lead concentrations greater than 5 μg/dL (0.24 μmol per liter) exhibited significant decreases towards this concentration within six months. Subjects with blood lead concentration less than 5 μg/dL (0.24 μmol per liter) fluctuated within a small range about this value.

The $^{206}Pb/^{204}Pb$ ratios of the immigrant women soon after arrival in Australia were initially greater than 17.6 with the highest $^{206}Pb/^{204}Pb$ ratio being 18.5 for subject 809 from Bulgaria. The $^{206}Pb/^{204}Pb$ ratio decreased to a plateau after three to five months. However,

in no case did the $^{206}Pb/^{204}Pb$ ratio decrease to less than 17.2. This difference for subject 809 is at least 8 standard errors above Australian levels and is thus highly significant statistically. During this 300-day period, the blood lead concentrations for all subjects ranged between 2.4 (0.12 μmol per liter) and 8.0 μg/dL (0.39 μmol per liter). The $^{206}Pb/^{204}Pb$ varied between 16.67 and 16.84 for the Australian control subject.

The rate of attainment of the plateau or "steady state" values for stable isotope profiles of the immigrant women was independent of blood lead concentration, country of origin, age, or the subject's parity. Least squares regression analyses of the time-series data for subjects 809 and 811 are shown in Figures 4 and 5 and analyses for all subjects are listed in Table 1. The calculated half-lives for lead in blood from 5 subjects over a time interval of ~300 days using $^{206}Pb/^{204}Pb$ values were 25 to 80 days. Subject 813 from Bulgaria appears to be an outlier. Her initial $^{206}Pb/^{204}Pb$ analyses were determined on blood samples obtained within two weeks of her arrival in Sydney and her blood lead concentration was not particularly high.

DISCUSSION

Detailed information on the lead isotope ratios for non-lead-exposed women of child-bearing age living in several communities in Australia have been described in detail by Gulson et al.[19,20]. These isotopic ratios are characterized by a relative homogeneity ranging from 16.4 to 17.1 in blood. In no individual case, among native-born, mainland Australian women have we observed a $^{206}Pb/^{204}Pb$ ratio in blood greater than 17.1 (only in Tasmania, have isotopic values at this upper end of the range been observed). As an additional method of assessing temporal changes in lead isotope profiles, monitoring has included blood samples from a native-born Australian subject living in Sydney. Likewise, environmental monitoring in Sydney, contemporaneous with the period of observation of the immigrant women indicated that, apart from leaded-paint, no $^{206}Pb/^{204}Pb$ ratios exceeded 17.0 based upon monthly air monitoring, long-term interior dust-fall accumulation (three- and six-month periods), vacuum

cleaner dust. water and soil sampling. Combined. these data indicate that the contemporaneous lead exposures in Australia had a $^{206}Pb/^{204}Pb$ ratios less than 17.0. The one exception to this were an occasional 6-day duplicate diet sample that contained a $^{206}Pb/^{204}Pb$ ratio as high as 17.3. However. these samples contained extremely low amounts of lead: the lead concentrations in these samples were less than 10 μg/kg.

The decline in $^{206}Pb/^{204}Pb$ ratios from the higher values present within one month of arrival in Australia to lower values represents absorption of lead from the contemporaneous environment. This response is consistent with the traditional view that blood lead concentrations are indicative of recent lead exposures. However. the $^{206}Pb/^{204}Pb$ ratios of blood lead remain higher than the ratio typical of the contemporaneous environment for mainland Australia. Even after 300 days. the isotope ratios had a contribution from a source with a higher $^{206}Pb/^{204}Pb$ than present in the contemporaneous environment.

The immigrant women do not live in close proximity with one another in Sydney and have no discernible association. Their only common source of lead with $^{206}Pb/^{204}Pb$ ratios greater than 17.6 was lead stored in their bodies from earlier exposures in Eastern Europe. For these reasons the most probable source is lead stored in body tissues. Although there are very few data on adult. female subjects. the majority of tissue lead for these women can be considered to be bone. Over 90% of lead in tissues of adult subjects is found in bone[13]. Although the majority of Barry's subjects were males[13]. we have accepted the prevailing assumption that gender-based differences in the pattern of tissue lead distribution are minor.

Skeletal lead burden. and hence its impact on contribution to the blood compartment. has changed considerably over the past two decades. Earlier estimates of skeletal lead were ~ 200 mg Pb[14]. Given that the half-life of lead in blood and in soft tissues (e.g.. liver. kidneys) is similar. these compartments are considered as one and have a volume in the average adult of 10 L. Thus. in a subject with a blood lead level of 5 μg/dL (0.24 μmol per liter). the blood compartment of 500 μg Pb represents a very small reservoir (0.25%)

compared with that in the skeleton. More recent estimates of skeletal lead for unexposed populations are considerably lower, probably because of decreasing amounts of lead in gasoline, air, food and water. Thus, Erkilla et al.[25] measured skeletal lead burdens of ~ 8 mg in a control group of adult men with a mean blood lead concentration of 3.7 μg/dL (0.18 μmol per liter). Their X-ray fluorescence spectrographic measurements of tibial lead (representing cortical bone) and calcaneal lead (representing trabecular bone) indicated a proportion of cortical/trabecular lead of 2.9, so that the amount of lead associated with trabecular bone is ~ 2000 μg; only a factor of ~5 times greater than that in the "blood" compartment. As it has been shown that lead in trabecular bone has a much shorter half-life of 7-13 years, ~3-10 times longer half-life of lead in cortical bone (e.g., ICRP[27]), then it is the trabecular lead reservoir that is most available for exchange with that in blood.

Data for estimates of skeletal contribution of blood lead are limited to stable and radiogenic isotopic studies of adult subjects. Most of the subjects previously assessed were males. Rabinowitz et al.[14] studied only male subjects and estimated that about 45% of blood lead was derived from inhaled lead and resorbed skeletal lead. He estimated that 7 μg/day came from the skeleton, which amounted to only 12% of the daily inputs into the blood pool of these men, whose blood lead concentrations averaged 20 μg/dL (0.96 μmol per liter). Furthermore, these men were maintained on positive metabolic balance with regard to calcium and phosphorus. Chamberlain et al.[28] re-interpreted these data and suggested that 25-33% was derived from endogenous sources. The pioneering studies of Manton[16,29] of two women provided further evidence of the skeletal lead contribution to blood lead. In his 1977 publication, Manton[16] attributed 4-8% of blood lead to a skeletal source during the late summer and 23-25% in winter. In a later publication, using an extended data set, Manton[29] revised his estimate up to 70% for blood lead derived from bone. Our data are consistent with these estimates.

Measurements of the isotopic ratios allow for estimations of the proportions of lead in blood derived from the skeleton and those derived from environmental inputs of the

Australian environment. Linear relations on isotope ratio plots (e.g. $^{207}Pb/^{204}Pb$ versus $^{206}Pb/^{204}Pb$) result from mixing of lead from two sources (e.g., Faure[30]). Such plots of the immigrant women's blood lead data (not shown) conform to linear trends and can thus be interpreted by a simple two-component mixing model. One component of lead is derived from the skeleton, probably the trabecular compartment. The other is lead from environmental inputs of air, food, and water which would be present in blood if there were no skeletal contribution or in blood from a person who was exposed to these lead sources over long periods of time (e.g., decades). An estimation of the isotopic ratios for the current Australian mainland environment are based on house dust, air-borne dust, food and water samples described in Pisaniello et al.[21] and blood and urine values in the mainland Australian female population (based on observations of 130 adult female subjects)[15]. Using the isotopic values of the initial blood samples of the immigrants as representative of the skeletal values and a maximum $^{206}Pb/^{204}Pb$ of 17.0 as the Australian environmental inputs obtained from direct measurements and also from blood analyses, we calculated that the skeletal lead contribution ("European lead" or "E-Pb") to blood lead of the subjects (at equilibrium or 'steady state') at 300 days varied from 41 to 73% (Table 1).

The data for the immigrant women indicate a rapid turnover of blood lead towards the Australian values in the first three or four months of their exposure to the Australian environment. Previous studies mainly based on X-ray fluorescence measurements of lead-exposed workers suggested that the trabecular bone is probably the more mobile component of the skeleton[26,31-33] with a biological half-life of lead in this bone of about seven to 13 years.

Given that the life of an erythrocyte is ~100 days and the mean life of lead in blood is ~30 days[34-35], lead can potentially exchange several times during the life of the erythrocyte. If no lead were entering the blood compartment from other tissues stores (especially the skeleton), there should be a complete exchange of lead in blood with the Australian environmental values within less than 12 months. As the lead in blood of none

of the immigrant women reached the Australian value, then lead must be coming from tissue stores, of which the skeleton is the greatest reserve. Among subjects monitored over a 15-month period, the data therefore show that considerable amounts of lead in their blood was skeletally derived.

Mobilization of lead from tissues stores among adult female subjects has several practical implications. These data indicate that a substantial portion of the lead present in blood comes from noncontemporaneous sources. Subjects who have a history of high lead exposures, with consequent body lead stores, are vulnerable to release of lead from these stores. Periods of metabolic stress in which bone mineral is mobilized could be predicted to be associated with increased release of lead from stores. Examples of such periods are physiological conditions, such as pregnancy and lactation, or pathological conditions, such as osteoporosis. It is documented that maternal blood lead concentration closely parallels fetal blood lead concentrations[10]. Lead freely crosses the placenta[9,10]. Consequently availability of tissue lead stores to the maternal blood supply could result in transfer of lead to the fetus[11,12].

A second practical implication of these data is that current blood lead reflects both current and past lead exposures. Blood lead analyses have been frequently used to assess exposure to environmental lead. For such analyses to be valuable for this purpose, however, it is critical to recognize that the time-period of lead exposure integrated by a current blood lead measurement is greater than 300 days. This estimate of time is based on the observation that even after 300 days, the stable lead isotopic profile of the immigrant women did not reach that of their contemporaneous environment. The time-period needed for the stable isotope profile in the blood of the immigrant women to be indistinguishable (based on analytical error of the TIMS lead determinations) from the native-born population remains to be determined, but exceeds 200 (or 500) days. If a multi-compartment model were used, the blood half-life would be shorter. Consequently monitoring of the rate of decline in blood lead concentration, in response to abatement of an environmental or occupational

lead source, is likely to differ markedly from the rate of increase in blood lead following a change in lead exposure during lead accumulation. Use of blood lead in evaluating response to remediation of environmental lead or removal of a worker occupationally exposed to lead will be complicated by tissue stores of lead. The slow rate of decline in blood lead levels observed in many abatement programs could be wrongly attributed to ineffective abatement if the continuing contribution to the blood pool by skeletal lead stores laid down during higher exposure periods is ignored[36]. Interpretation of blood lead data obtained for the purpose of screening to identify persons at elevated risk of lead mobilization during period of metabolic stress (e.g., pregnancy, lactation, bone remobilization) must be provisional based on understanding both the contemporaneous and historic contribution to blood lead.

CONCLUSIONS

Our data indicate that sequential blood lead measurements may be a better index of body lead burden than previously documented. Among adult female subjects ages 25 to 36 years, 41 to 73% of lead in blood was derived from tissue stores. After 300 days of residence in Australia, blood lead concentration decreased toward blood lead concentrations of ~5 μg/dL (0.24 μmol per liter) whole blood, typical of adult women residing in the same city, Sydney, or other cities from south-eastern Australia. The proportion of blood lead attributable to tissue lead stores (based on ratios in the $^{206}Pb/^{204}Pb$ in blood) was independent of the subjects' initial blood lead concentrations, age, parity, or country of origin. Tissue lead stores reduce the responsiveness of blood lead to changes in the contemporaneous environment. Use of blood lead as an indicator of changes in environmental lead must be reconsidered in view of the contribution of tissue lead to blood lead; particularly as this pattern persists for over 300 days. Whether or not the contribution of tissue lead to blood lead among pediatric subjects differs from adult values remains to be established, but is the subject of on-going research. Attenuated response of blood lead

to abatement of environmental sources of lead (e.g., worker removal from lead industries) may be due to remobilization of tissue lead stores. Because the adult females who were subjects of this research were heterogenous in terms of age, peak bone mass and state of bone remodelling could have influenced skeletal contribution of lead to blood lead.

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Captions for Figures

Figure 1. Lead isotope ratios expressed as the $^{206}Pb/^{204}Pb$ ratio illustrating the relative isotopic uniformity in the unexposed Australian population and difference in isotopic profile compared with other countries. Data sources: United States (Yaffee et al., 1983; Tera et al., 1985); United Kingdom (estimated from the $^{206}Pb/^{207}Pb$ data of Delves and Campbell, 1993); Finland (Keinonen, 1989, for bones).

Figure 2. Time-series plot of sampling month versus $^{206}Pb/^{204}Pb$ on the left-hand axis and Pb content (μg/dL) in blood on the right-hand axis for the Australian female reference subject.

Figure 3. Isotopic variation with time in Sydney air based on measurements of particulates from high-volume air-samplers and compared with gasoline lead.

Figure 4. Time-series plot of sampling month versus $^{206}Pb/^{204}Pb$ on the left-hand axis and Pb content (μg/dL) in blood on the right-hand axis for subject 809 from Bulgaria. Note scale changes for the $^{206}Pb/^{204}Pb$ and blood lead compared with Figure 2. The top segment of the "field" for Australian isotopic values is marked.

Figure 5. Time-series plot of sampling month versus $^{206}Pb/^{204}Pb$ on the left-hand axis and Pb content (μg/dL) in blood on the right-hand axis for subject 811 from Poland. Note scale changes for the $^{206}Pb/^{204}Pb$ and blood lead compared with Figures 2 and 4. The top segment of the "field" for Australian isotopic values is marked.