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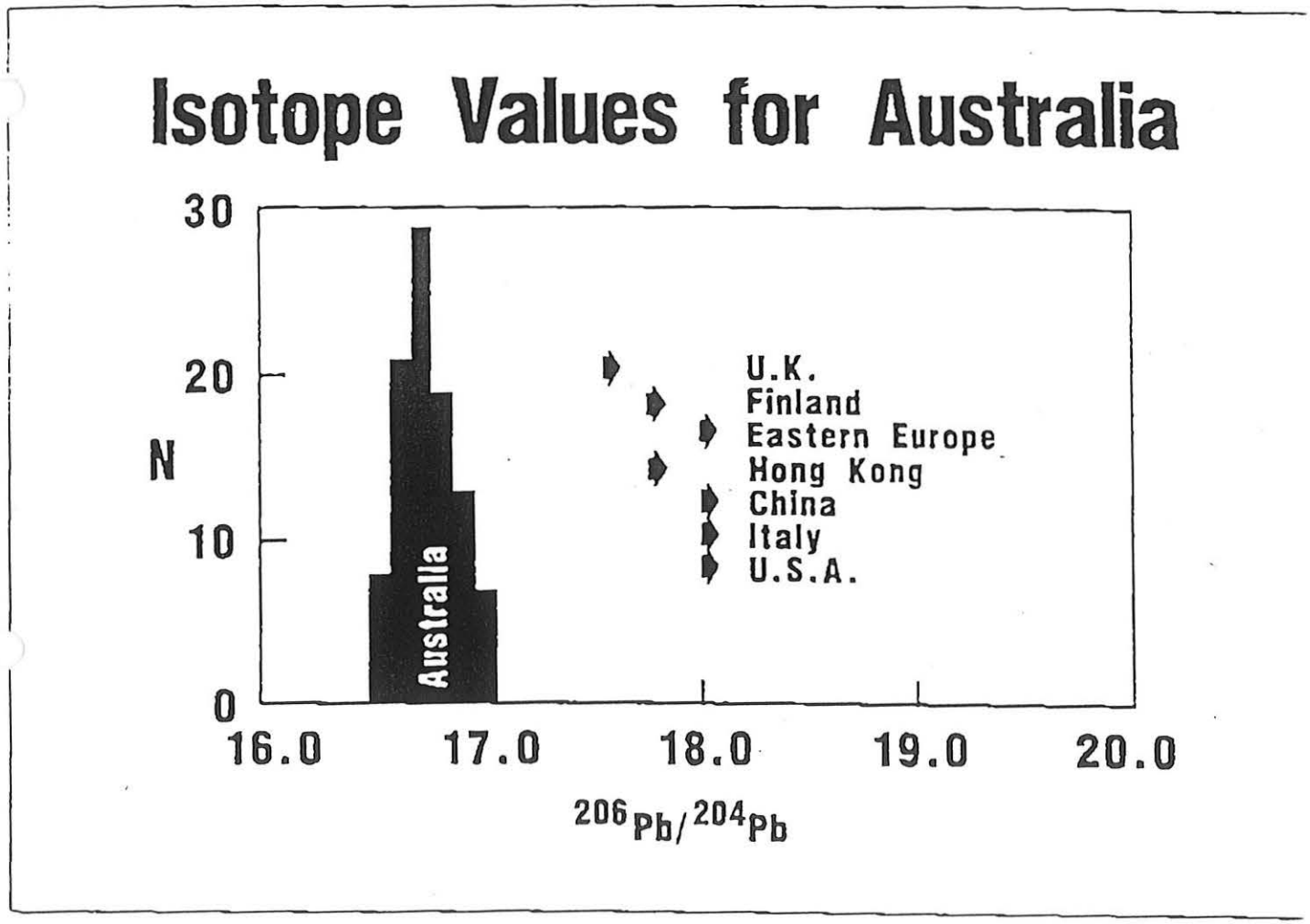
Immigrant Women. Stable Lead Isotopes

Figure 1



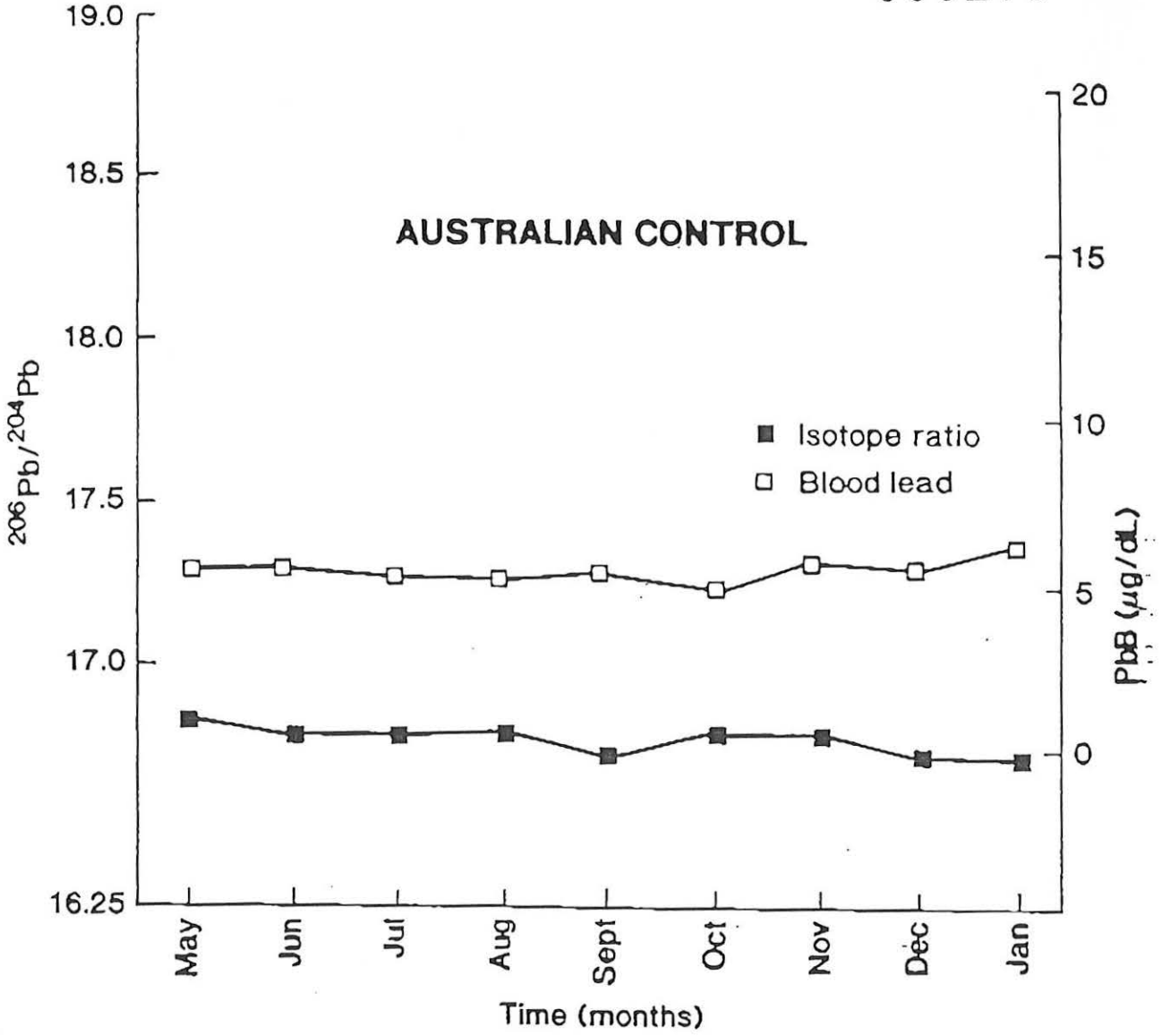

AUSTRALIAN CONTROL
Isotope ratio
Blood lead
$^{206}Pb/^{204}Pb$
PbB (μg/dL)
19.0
18.5
18.0
17.5
17.0
16.25
20
15
10
5
0
May
Jun
Jul
Aug
Sept
Oct
Nov
Dec
Jan
Time (months)

Figure 2

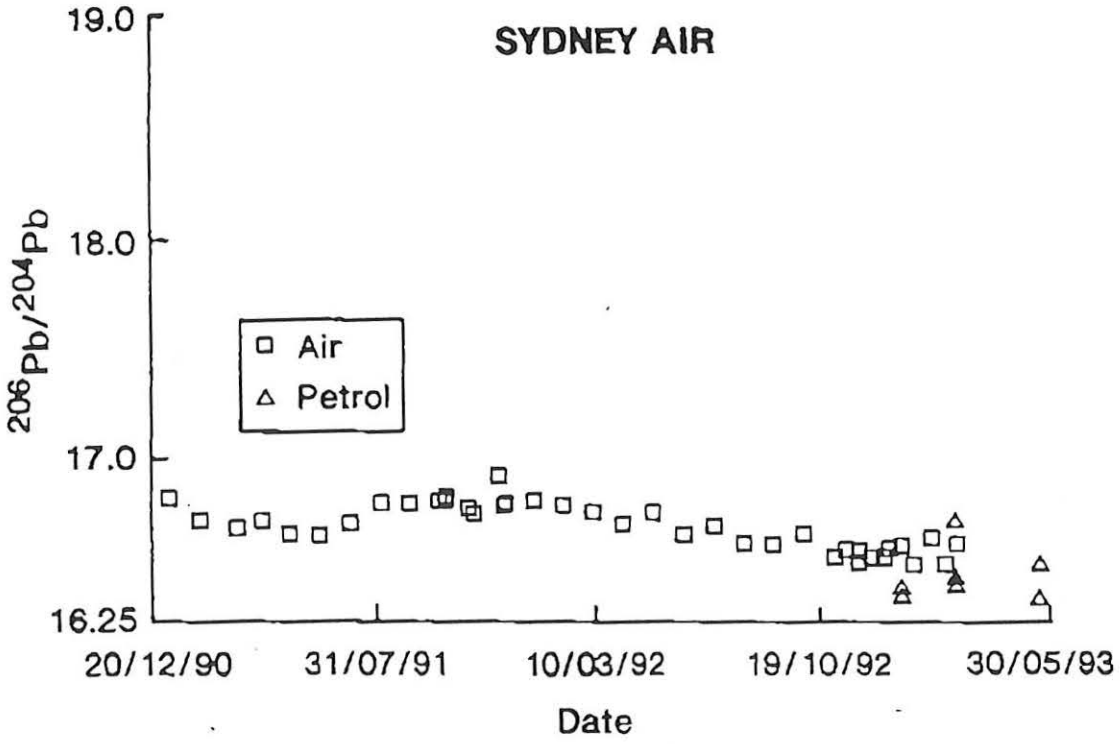

SYDNEY AIR
19.0
18.0
17.0
16.25
$^{206}Pb/^{204}Pb$
Air
Petrol
20/12/90
31/07/91
10/03/92
19/10/92
30/05/93
Date

Figure 3

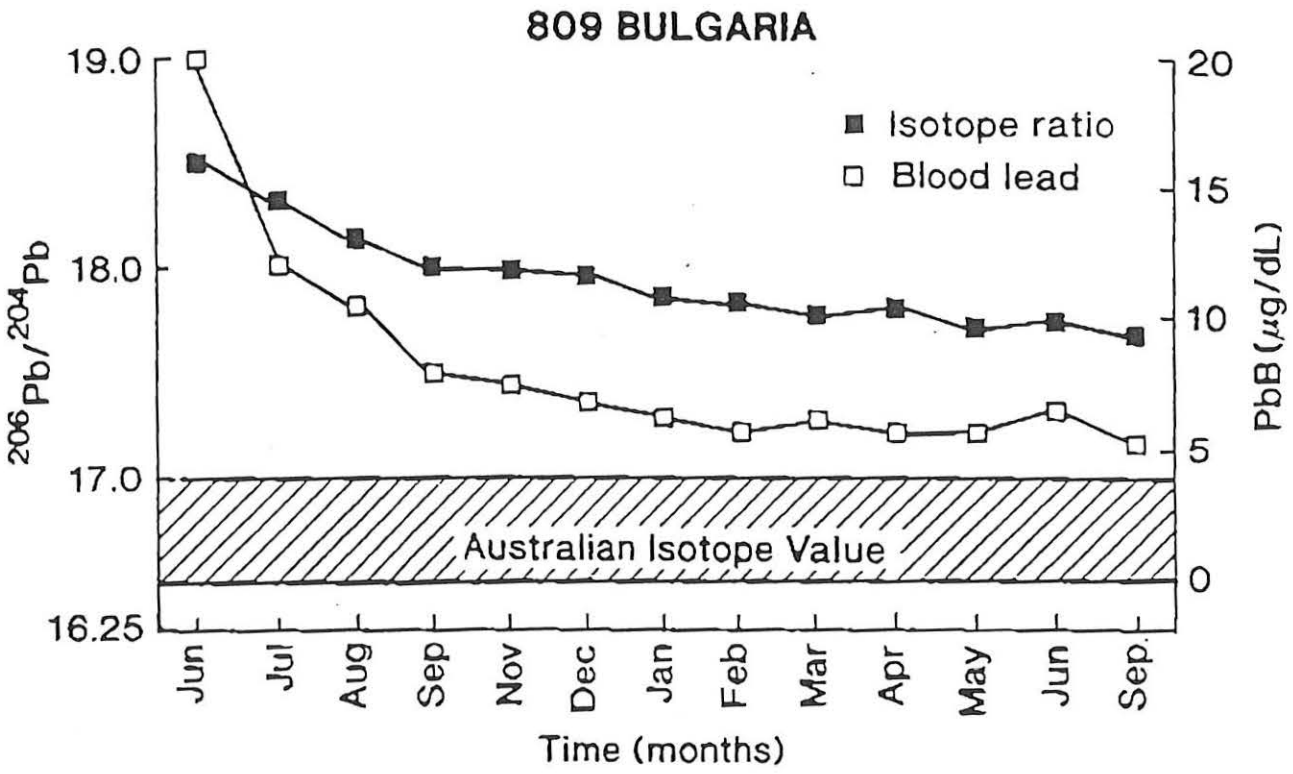

809 BULGARIA
19.0
18.0
17.0
16.25
$^{206}Pb/^{204}Pb$
20
15
10
5
0
PbB (μg/dL)
Isotope ratio
Blood lead
Australian Isotope Value
Jun
Jul
Aug
Sep
Nov
Dec
Jan
Feb
Mar
Apr
May
Jun
Sep.
Time (months)

Figure 4

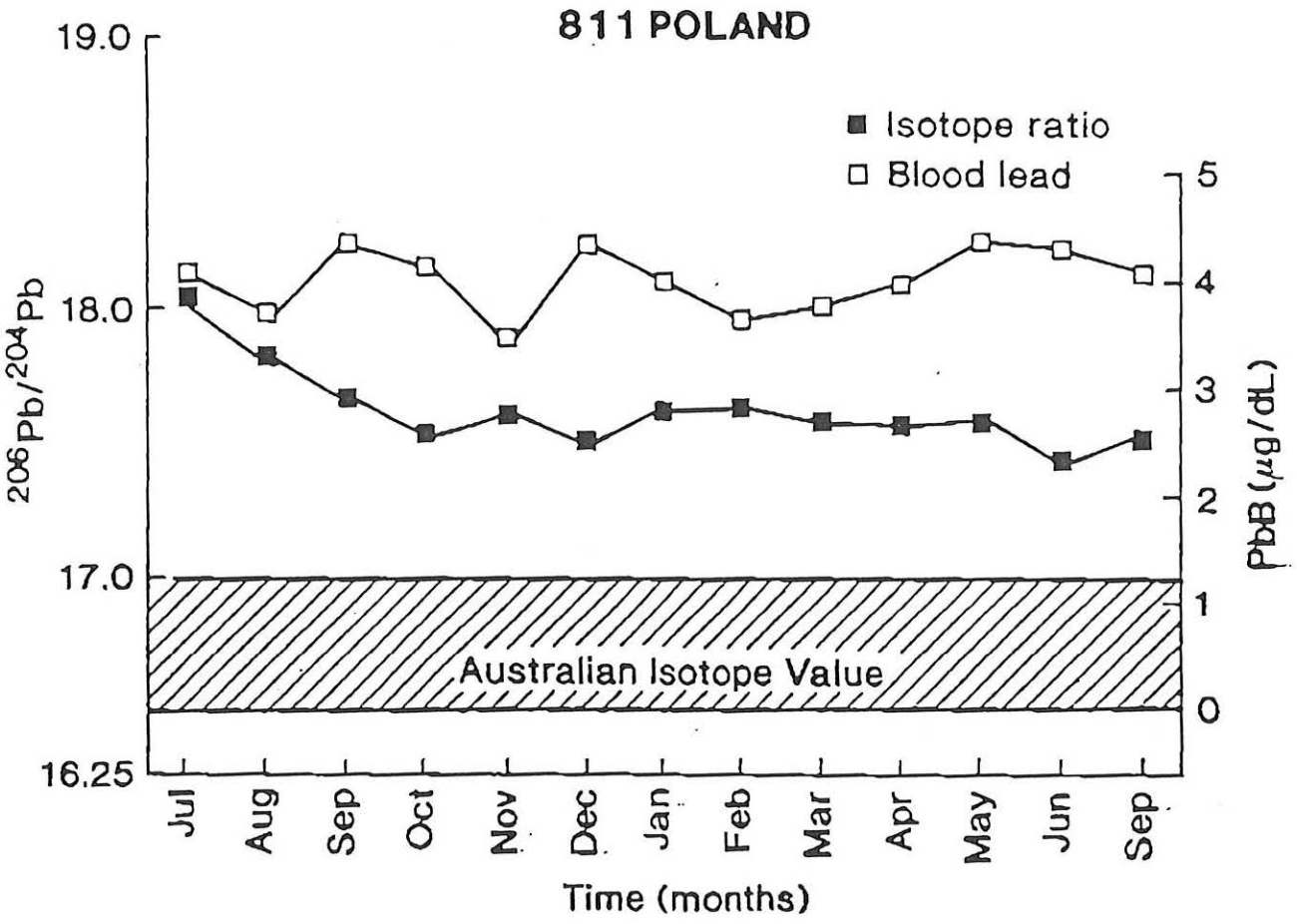

811 POLAND
Isotope ratio
Blood lead
19.0
18.0
17.0
16.25
$^{206}Pb/^{204}Pb$
PbB (μg/dL)
5
4
3
2
1
0
Australian Isotope Value
Jul
Aug
Sep
Oct
Nov
Dec
Jan
Feb
Mar
Apr
May
Jun
Sep
Time (months)

Figure 5

Table 1. Summary of change in blood lead over time

Subject No.	Country of Origin	"Initial" Value		Value at 6 Months or 300 Days		% E-Pb[b]	Half-life at 300 Days[c]	Subject Age (years)	Subject Children Age (years)
		PbB (μg/dL)	^{206}Pb / ^{204}Pb	PbB (μg/dL)	^{206}Pb / ^{204}Pb[a]				
802	CIS	2.6	17.67	2.4	17.31	46	-	27	3.3[d]
803	CIS	3.8	17.62	3.3,3.5	17.16,17.40	65	-	33	9.4
804	CIS	2.9	17.66	3.4	17.21	32	-	30	2
807	Bulgaria	7.2	18.32	5.1,4.7	17.62,17.54	41	43±8	26	-
808	Bulgaria	13.3	18.36	8.0,6.6	17.97,17.82	60	25±6	36	13.7
809	Bulgaria	20	18.51	6.8,6.5	17.96,17.73	48	80±13	30	7
810	Romania	8.6	18.2	4.0,4.0	17.80,17.61	51	48±7	29	-
811	Poland	4.1	18.04	4.4,4.3	17.50,17.44	42	25±6	31	8.5
812	Poland	2.7	17.87	2.7	17.32	37	-	25	5
813	Bulgaria	6.6	18.33	4.6,4.6	17.92,17.70	53	175±92	26	3
817	Bulgaria	3.7	17.79	2.89,2.7	17.72,17.58	73	-	32	1
G811	Australia	5.7	16.84	6.7,6.0	16.78,16.72		-	45	-

[a] No values for subjects 802, 804, 812 at 300 days indicate discontinued program after 6 months

[b] Approximate percentage of European lead at equilibrium (6 months or 300 days) in Australia; remainder is Australian lead

[c] Calculated for $^{206}Pb/^{204}Pb$

[d] Subject has 3-year-old twins

- Half-life calculation gives meaningless values, especially in case of constant or increasing PbB over time

Sources and Intensity of Lead Exposure in Children In Utero and Early Childhood and the Dynamic Nature of Lead in Dentine

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Submitted: Environ Health Perspectives

Key Words: Teeth, lead exposure, children, pregnancy

The sources and intensity of lead exposure *in utero* and in early childhood was determined with stable lead isotopic ratios and lead concentrations of incisal and cervical sections of deciduous teeth from 30 exposed and non-exposed children from the Broken Hill lead mining community and several other children from diverse environments. Incisal sections consisting mostly of enamel, generally have lower amounts of lead and isotopic compositions consistent with those expected in the mother during pregnancy. Cervical sections, consisting mostly of dentine with secondary dentine removed by resorption and reaming, generally have higher amounts of lead than the enamel and isotopic compositions consistent with the source of exposure. In Broken Hill the dentine results are interpreted to reflect an increased exposure to orebody lead during early childhood, probably associated with hand-to-mouth activity. Leaded paint was identified as the source of elevated tooth lead in at least two cases in Broken Hill. Increased exposure to lead from orebody and paint sources *in utero* was implicated in two cases from Broken Hill, but there is no indication of this former exposure from the mother's current blood lead suggesting an acute rather than chronic exposure for the mother. Permanent teeth from one subject have lower amounts of lead in the roots compared with the crowns and the isotopic composition of the crowns are consistent with the data for the deciduous teeth from the same subject. These results provide convincing evidence for the dynamic nature of lead in dentine.

Introduction

Exposure profiles are one of the fundamental aspects underpinning studies of lead and its impact on neuropsychological and neurobehavioural outcomes in children. Furthermore, as the skeleton is the primary storage compartment for lead in the human body, it is a potential endogenous source of lead that may be released from the bones during pregnancy (1,2), during lactation (2) and post menopause (3). Chronic exposure to lead, such as from mouthing activity in early childhood, may be camouflaged by dilution of lead in bones during periods of rapid skeletal growth in the young and adolescents, and so may not be

detected by the normal methods of blood lead analysis. Hence, knowledge of past lead exposure especially *in utero,* is fundamental to any investigations of lead toxicity.

In many human epidemiological investigations, exposure profiles may be limited to a single blood (PbB) level (4), which may provide information only of recent exposure. The use of lead in whole deciduous teeth, enamel or dentine as an indicator of past exposure of children to lead, and as a proxy for skeletal lead, has been well documented in a number of studies, many of which have been referenced in Gulson and Wilson (5). Gulson and Wilson (5) and Edwards-Bert et al. (6) assessed various aspects of previous tooth lead studies. The advantage of tooth lead over blood lead (PbB) lies in the integration of lead exposure in the tooth over several years from *in utero* to exfoliation compared with blood which has an approximately 30 day mean life (7). However, because of differences in the type of tooth analysed, the part of the tooth analysed and the analytical techniques employed for the lead measurement, comparisons between investigations have constrained the widespread acceptance of tooth lead as an indicator of lead exposure. In a pilot study, Gulson and Wilson (5) showed that stable lead isotopic analyses and lead concentrations in cross-sectional slices of deciduous teeth provided evidence of *in utero* and earliest childhood exposure using enamel and the results for dentine provided evidence of exposure during early childhood, possibly up until the time of exfoliation.

The aim of this paper was to evaluate the efficacy of the isotopic analyses of slices of teeth as an indicator of past exposure in 30 exposed and non-exposed children from a major lead mining community and in several children from other areas. These data would also permit evaluation of the hypothesis that lead in dentine does not turnover (8,9) an hypothesis which has been misinterpreted to indicate that tooth lead, apart from that in the circumpulpal dentine, is a passive reservoir for lead.

Methods

Deciduous teeth, mostly upper and lower incisors, from 30 children from the Broken Hill lead mining community with differing exposure to lead have been tested. In one subject, permanent teeth were also available and these allow a comparison of exposure

through different stages of childhood. Estimates of exposure were based on information obtained from parents and include residence in areas identified as "high risk" by a blood lead survey of 899 children aged 1 to 4 years (10), early childhood mouthing frequency, learning difficulties and behavioural problems. With one exception, the mothers of the children were long-term residents of Broken Hill (>10 years).

Except for the permanent teeth, the samples consisted only of crowns, which had undergone varying amounts of resorption. Wherever possible, central and/or lateral incisors were analysed. The crowns were cut into approximately 1-2 mm thick cross-sectional slices from the incisal and cervical areas. The incisal slice consisted of enamel and varying amounts of coronal dentine. In this paper, the nomenclature of Shapiro et al. (9) is followed; i.e., enamel, dentine, secondary dentine (the dentine zone around the pulpal canal, also called circumpulpal dentine) and pulp; the locations from which the incisal, cervical and root sections were cut for the analyses in this paper are shown in Figure 1. For lower incisors, the tooth was generally sawn in half but for upper incisors, it was possible to obtain at least three sections.

As enamel and coronal dentine are formed prior to the crawling stage of most children (11), they provide an indicator for *in utero* and earliest childhood exposure. The lower central portion of the incisal section was reamed to ensure removal of any later-formed circumpulpal dentine. As much as possible of the thin veneer of enamel was burred from the cervical section, leaving mainly dentine. The pulpal canal in the cervical section was usually resorbed to varying degrees but to ensure minimal contribution from secondary (circumpulpal) dentine, approximately 2 mm of the pulpal canal and dentine was reamed out. The dentine in the cervical section provides an integrated exposure to lead from the time of eruption of the tooth until exfoliation (12,13). For permanent teeth, 1-2 mm sections of the outer crown and an area approximately 2 mm from the root tip were sliced (Figure 1). The cementum on the root was burred off.

Tooth slices were decontaminated and analysed in the manner described by Gulson and Wilson (5) except that the decontamination employed 1% HNO_3 and tooth slices weighing approximately 2-60 mg were leached with 1.5M HCl for approximately 4 hours.

Depending on the sample size, not all the sample dissolved in the 4 hours. The 1.5M HCl approach was devised for differential leaching tests in an effort to obtain more information about lead distributions in teeth (5). However, the 1.5M HCl leaching procedure was not considered satisfactory for a general purpose method and so tests were carried out on sagittal halves of incisal and cervical sections using a 1.5M HCl leach and a 6M HCl leach for approximately 4 hours. The samples dissolved completely in 6M HCl, as they do in concentrated HNO_3. Lower concentrations observed in the 1.5M HCl, compared with the 6M HCl, leaches for enamel are consistent with incomplete solution of the sample in 1.5M HCl. There was negligible difference in the isotopic compositions for the halves using the two leaching solutions. Thus dissolution of samples using 6M HCl or concentrated HNO_3 is recommended. Processing "blank" levels were < 150 picograms Pb; no corrections for this "blank" have been made to the data as it is insignificant compared with the amount of lead in the analysed sample.

Results and Discussion

Broken Hill subjects.

Broken Hill is a city of approximately 25,000 inhabitants, about 930 km west of Sydney, NSW, Australia, and centred about the world's largest currently mined Pb-Zn-Ag deposit. Mining activities, including underground and open-pit operations, and smelters in the latter part of the past century, have been conducted for more than 100 y. This area is desert and subject to severe windstorms, and the dust from the mining activities and potentially from weathering of the orebody over millions of years, is considered to be the main point source of lead in children from inhalation and ingestion of contaminated soil and house dust. The isotope values discussed in this paper are the abundance of the ^{206}Pb to the abundance of ^{204}Pb, expressed as the ratio $^{206}Pb/^{204}Pb$. The isotopic composition of the potential sources of lead in the Broken Hill community are summarised in Figure 2. The $^{206}Pb/^{204}Pb$ ratio generally ranges from 16.0 to 16.2 for the orebody lead and dust from ceilings, vacuum cleaners and kitchen wipes (14). The other potential sources of lead are food, water and air. Food and water contain < 10 and < 3 ppb lead, respectively, and are the

main contributors to "background" PbB concentrations of 6 ± 2 μg/dL which have been estimated from 38 minimally "exposed" female adults (14). Apart from orebody lead, another major source of lead in air is from gasoline; approximately 60% of automobiles in Broken Hill use leaded gasoline with approximately 0.8 g/L lead and with a $^{206}Pb/^{204}Pb$ of 16.56. Gasoline is considered to be a significant source of lead in adult females from Broken Hill (14). Paint has been largely ignored as a potential contributor to blood lead in children from Broken Hill but houses built prior to the 1970's and commonly containing lead paint far outnumber newer dwellings. Renovation of houses is extremely common in Broken Hill. Isotopic ratios measured in paint (Figure 2) illustrate the complexity of lead sources but nevertheless do not negate the use of isotopic analyses for elucidating sources in Broken Hill.

It is possible to estimate relative proportions of lead from some sources using the isotopic ratios, such as the orebody and gasoline. For example, given that the orebody has a $^{206}Pb/^{204}Pb$ value of approximately 16.0 and gasoline 16.56, a tooth slice with a ratio of 16.28 would have a 50% contribution from each source. Estimating proportions for paint is more complex but in some cases it is still possible to obtain approximate contributions.

Broken Hill subjects with low exposure. Results are presented in Table 1. Male and female siblings from the same family are denoted by the same same code, suffixed by 'M' and 'F' respectively; for example 568M and 568F. In general, the lead concentrations in the *incisal sections* for such subjects are low, with a range from 0.4 to 2.1 ppm Pb with a mean value of 1.0 ± 0.5 ppm (n=13; Table 1). These low lead concentrations demonstrate that the children were exposed to low levels of lead *in utero* and earliest childhood. The low lead concentrations are consistent with those found in unexposed populations (15,16) but are up to 100 times greater than natural levels (17). The $^{206}Pb/^{204}Pb$ ratios generally range from 16.44 to 16.62 (mean 16.53 ± 0.07; n=13), consistent with those found in the blood of female adults (14).

Variable lead concentrations and $^{206}Pb/^{204}Pb$ ratios are observed in the *cervical sections* but there is no simple relationship such as increasing lead concentration with decreasing $^{206}Pb/^{204}Pb$ which would reflect an increasing amount of lead from an

orebody source as a result of hand-to-mouth activity. Likewise, there is not always a straightforward relationship between lead concentration and $^{206}Pb/^{204}Pb$ ratio in-so-far as the lead concentrations in both cervical and incisal sections of some subjects (e.g., 557, Table 1) are low and yet the isotopic composition indicates that the child has a substantial component (approximately 40 %) of orebody lead in his teeth.

In general, the cervical section has higher amounts of lead and lower $^{206}Pb/^{204}Pb$ ratio than the incisal section, indicating that during early childhood, the individuals' intake of orebody lead exceeded that from other sources.

Broken Hill subjects with high exposure. Lead concentrations and $^{206}Pb/^{204}Pb$ ratios are variable in incisal and cervical sections for these subjects (Table 2). Low lead concentrations of < 2 ppm and higher $^{206}Pb/^{204}Pb$ ratios of >16.4 in the incisal tooth slices of some subjects (e.g., 519, 548) reflect a low-lead exposure *in utero* and during earliest childhood. There is no simple relationship between lead concentration and $^{206}Pb/^{204}Pb$ ratio in the cervical sections, even excluding subjects 517M and 517F (Table 3).

For most subjects, the isotopic ratios in the cervical (and often incisal) sections indicate that during early childhood, the individuals' intake of orebody lead exceeded that from other sources.

Broken Hill subjects with lead from a paint source. The major exception to a source of lead deriving mainly from the orebody is with the siblings 517, whose teeth have some of the highest lead concentrations analysed so far for Broken Hill children (Table 3). The $^{206}Pb/^{204}Pb$ ratios in both incisal and cervical sections are in the range 16.45 to 16.67. Even though the family lived within 300 meters of mining activities and the surface dust and soil from the house had over 90% of their lead derived from an orebody source, the isotopic data indicated that there was a significant contribution of lead from another source, such as paint. Paint was not, however, considered to be a source of lead by the parents because extensive renovations were purported to have been carried out several years before the children were born. However, an initial vacuum cleaner dust sample had a $^{206}Pb/^{204}Pb$ ratio of 16.98, totally different from the orebody and indicative of a source

from paint. Inspection of the vacuum dust by optical and scanning electron microscopy identified numerous lead paint flakes with evidence of burning. It is estimated from the isotopic ratios in the paint flakes (18.37, 18.31) that the lead in the children's enamel and dentine consists of approximately 30% paint lead and 70% orebody lead (assuming gasoline to be a minor contributor). After obtaining these results, and upon further discussions with the parents, it eventuated that they had burnt off many layers of paint from skirting boards and doorways when the children were very young.

The data for the incisal tooth sections indicate that the children received a lead insult *in utero* and earliest childhood. However, the mother currently exhibits no evidence of an earlier increased lead burden as her current PbB was 4.7 μg/dL and $^{206}Pb/^{204}Pb$ ratio was 16.57, consistent with other female adults from Broken Hill. Similarly, there was no evidence of this past exposure in the father whose urine lead was normal at 4.1 μg/L and $^{206}Pb/^{204}Pb$ was 16.42. These "normal" results for the parents suggest that their exposure was acute and lead from paint was not transferred in significant amounts to long-term bone compartments. In contrast, PbB in the children are relatively elevated for their age at approximately 14 μg/dL and with similar $^{206}Pb/^{204}Pb$ ratios of 16.40 perhaps reflecting leakage (mobilisation) of lead from earlier accumulated skeletal stores. The deficiencies in reading, bilateral co-ordination and balance and visual motor control of the older male sibling may be related to a chronic insult *in utero* and early childhood.

Variations during pregnancy and between deciduous and permanent teeth. The contribution to enamel lead from different sources, and impact of renovations during pregnancy, were briefly mentioned above. In another family, it was possible to obtain several teeth, including deciduous and permanent teeth, from two male siblings. Extensive contamination from renovations of this house, located within 500 meters of the mining activities, occurred during the pregnancy with the older sibling. Additional contamination may have resulted from transport of lead dust to the residence on the clothes of the male adult, who was employed underground in the mine for over 20 years. The mine work clothes were laundered at home in Broken Hill.

Incisal and cervical tooth sections from the older male sibling (subject 534 M) exhibit the highest contribution of orebody lead of any subject so far analyzed in Broken Hill (Table 4). The data are consistent with a larger insult of orebody lead than for the younger female sibling although the lead concentrations for the same tooth type (primary molar) are similar.

Data for the permanent teeth provide an interesting contrast to those for deciduous teeth (Table 4). Crowns (enamel) of the permanent teeth contain higher amounts of lead than the roots, the opposite to that observed in most deciduous teeth from children at Broken Hill and also from this same subject. This is not unexpected as the development of enamel in permanent first molars is completed at 2.5 to 3 years (11), the time of maximum mouthing activity (18). The roots of the permanent teeth of subject 534 M contain relatively high amounts of lead and the $^{206}Pb/^{204}Pb$ ratios are slightly higher than the enamel, indicating a contribution of lead from food, water, air; the $^{206}Pb/^{204}Pb$ ratios in these media are generally >16.3 in Broken Hill (14). The PbB in the children, aged 11 and 9 years at the time of blood sampling, were elevated at 19.9 and 19.3 μg/dL Pb, and the $^{206}Pb/^{204}Pb$ ratios of 16.19 and 16.24 respectively, were consistent with the data for their teeth. The isotopic data for their blood probably reflect ongoing mobilisation of lead from skeletal tissues introduced to the bone compartments during pregnancy and early childhood (19).

As with subjects 517 M and 517 F, described in the previous section, there is no indication from the mother's current PbB of 6.7 μg/dL ($^{206}Pb/^{204}Pb$ of 16.48) of this earlier high exposure to lead.

Children outside of Broken Hill.

Using the experience gained from working with the children from Broken Hill, where the sources of exposure are well characterised, the isotopic approach was tested with several children from other areas. Parents of these children were concerned that the children had been exposed to high amounts of lead at an earlier stage in their childhood.

The first case (A, Table 5) involved a female subject who attended, for approximately 9 months, a day-care center under the Sydney Harbour Bridge. Potential sources of lead in

this area are gasoline lead, and lead paint from bridge renovations and in the day-care center itself. No detrimental health and behavioural characteristics were noted by the parents but they were concerned that she was shorter than her younger sister and also experienced lack of concentration. The incisal section of an upper incisor had low lead concentrations with the cervical section showing low to moderate amounts of lead (Table 5). The $^{206}Pb/^{204}Pb$ ratios were slightly higher than we have measured in environmental parameters such as air, gasoline, and in blood for Sydney and is different to the data for the tooth from the male Sydney subject shown in Table 5. However, they are entirely consistent with isotopic compositions measured for lead in paint in Australia (20).

The second case involved female subjects who were raised within 400 meters of a large zinc/lead smelter. The younger sibling (B) had a PbB consistently above >20 μg/dL for 18 months and experienced learning difficulties, diminished motor functions and behavioural problems. Ceiling dust from the house contained up to 1.9% Pb and soil up to 0.5% Pb in the -75 μm particle size range. Incisal and cervical sections of her upper left incisor ('62') had lead concentrations as high as the most exposed children in Broken Hill (see Table 1). The results for the cervical section reflect the minimum lead insult to this child as there can be 2 to 3 times more lead in the roots (5). The isotopic ratios for both tooth sections indicate that the source of lead changed little during early childhood. The tooth from the older sibling (Table 5) had lower amounts of lead, especially in the incisal section, than for the younger sibling. These results suggest that the mother experienced an increased exposure to lead after the birth of the first child and in utero with the younger child. Furthermore, the younger sibling would appear to have ingested "dirt" of similar source during early childhood.

The third case (D) involved an adopted 14 year-old male who was diagnosed with porphyria, severe behavioural problems and ate leaded-paint. His current PbB was 38 μg/dL. He was born in a lead mining town of approximately 200,000 people in Brazil and came to Australia at age 13 months. The parents were anxious to determine when he obtained the lead burden. Lead concentrations of both incisal and cervical sections were very low (Table 5). The results indicate that his current high PbB was not a result of

leakage from a high skeletal burden sourced in Brazil. Furthermore, it is highly unlikely that his lead insult occurred during his early childhood (the first few years) in Australia or else some evidence for this should have been manifested in the dentine from the cervical section. No published data are available from the lead mining community in which he was born. However, the isotopic composition of his incisal crown is similar to that measured in lead ores from a neighbouring community (21).

Other data in Table 5 are for a 6 year old male Sydney resident (E) whose mother was Australian-born of Greek parents. Lead contents were low and the isotopic ratios consistent with other environmental data we have for Sydney. The similarity in isotopic ratios for incisal and cervical sections indicate no significant change in the source of lead throughout early childhood, in contrast to cases discussed above from Broken Hill.

Enamel versus dentine lead.

In more than 90% of the analyses of >150 deciduous and permanent teeth, we have found the concentrations of lead to be higher, sometimes up to an order of magnitude, in the coronal dentine compared with the enamel. In contrast, Malik and Fremlin (22) using charged particle activation analyses, found that in molars of subjects < 25 years old, lead in enamel was higher than in dentine. Incisors and canines are the most common teeth used in studies of children so that investigations of molars may not be relevant to the argument about different concentrations of lead in incisors.

Furthermore, our data have shown that analyses of cervical sections, representative of coronal dentine, substantially underestimate the exposure of a child given the high amounts of lead observed in the roots, when the roots are available for analysis. This underestimation is exacerbated if a whole tooth is analysed, because of dilution from the lower lead concentrations in the enamel, and also by the fact that considerably more weight (as against volume) of a tooth is in the highly mineralised apatite in the enamel compared with that in dentine (23). In addition, incorporation of the secondary dentine in the tooth analysis may provide an overestimate of tooth lead as the secondary dentine may dominate the tooth lead (9,24).

Dynamic Nature of Dentine Lead.

The suggestion that dentine lead does not turnover (8,9) is not consistent with the results of this study, that of Gulson and Wilson (5), nor that of Rabinowitz et al. (25). Calcification of dentine occurs first within the crown and progressively advances towards the root and inwards along a conical zone from the pulpal canal (23). Dentinal tubules with their blood supply extend from the pulpal canal through the dentine and provide channellways for introduction of mineral salts, including lead. Rabinowitz et al. (12) suggested that the permeable nature of dentine allows for loss of lead for as long as a year after calcification is complete, only later becoming a sealed repository. The higher concentrations of lead in roots and coronal dentine would appear to suggest that even if some lead is lost, there is a positive balance towards retention of lead in the dentine. Furthermore, as shown in this study and that of Gulson and Wilson (5), the significant isotopic differences between the lead in enamel and coronal dentine (and roots, when available), demonstrate an ongoing addition and possible exchange of lead from endogeneous and exogenous sources. It was argued above that analysis of whole teeth underestimated the exposure of a child to lead and the same applies to source information that is camouflaged or lost by a similar approach.

Rabinowitz et al. (13) suggest that calcification of the upper central incisors begins near the third fetal month and coronal dentine is largely completed by the third post natal month. Given that a child is unlikely to be experiencing significant hand-to-mouth activity at such a young age, the results of this study and that of Rabinowitz et al. (25) suggest that lead is not permanently fixed in the deciduous dentine. This hypothesis is supported by isotopic data on teeth from subjects who have migrated from Europe and have resided in Australia for varying lengths of time (unpublished data).

Conclusions

Results from this study illustrate that the approach of slicing teeth into incisal and cervical sections, combined with lead isotope measurements, provide an excellent history of lead exposure for children. If there are *no changes* to lead exposure over the lifetime of the deciduous tooth (e.g., approximately 7 years), then it is feasible to use a whole tooth for analysis.

Teeth from children with low lead exposure generally contain < 2 ppm in the incisal section (dominantly enamel and primary dentine) and the isotopic compositions are consistent with the lead exposure expected for the mother *in utero*. The cervical sections (dominantly dentine) contain up to 9 ppm Pb and the isotopic compositions vary depending on the source of the lead. The low lead concentrations in coronal dentine are consistent with those observed in other studies of unexposed children.

In high to moderately exposed children, the amounts of lead and isotopic compositions in incisal and cervical sections can vary widely; the cervical sections generally contain more lead and the isotopic compositions reflect the dominant source(s). Two siblings from Broken Hill, with the highest amounts of tooth lead, appear to have derived this lead *in utero* and early childhood from lead paint released during renovations, even though the dwelling is within 300 meters of mining activities. In another case from Broken Hill, increased exposure *in utero* and early childhood showed the lead in teeth was derived from an orebody source, probably also during renovations. In both these cases, there is no indication from the mother's current blood lead of any previous high exposure to lead. In the case of a subject living in close proximity to a lead smelter, the high amounts of lead and isotopic compositions of the teeth are also consistent with exposure during pregnancy.

Crowns and roots of permanent teeth from one subject have data which are consistent with those from his deciduous teeth.

Dentine appears to be a dynamic system and there is a positive balance for introduction of lead.

(Acknowledgments)

We offer our thanks to the tooth fairies from Broken Hill who kindly procured the teeth; Graham Vimpani for the opportunity to investigate the teeth from subjects B, C and D (Table 6); Garry Lenard for identification of the teeth; Michael Korsch for keeping the mass spectrometers in top condition, software development, and for ongoing fruitful discussions; Michael Korsch, Michael Moore and Michael Rabinowitz kindly reviewed an early version of this manuscript.

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Tables

Table 1. Lead isotopic ratios and concentrations for teeth from subjects who have experienced a low lead exposure.

Table 2. Lead isotopic ratios and concentrations for teeth from subjects who have experienced a high lead exposure.

Table 3. Lead isotopic ratios and concentrations for teeth from subjects whose source of lead is probably from paint.

Table 4. Lead isotopic ratios and concentrations for teeth from subjects 534.

Table 5. Lead isotopic ratios and concentrations for teeth from subjects from diverse environments.

Figure 1

Schematic representation of the sagittal section of a human tooth showing the locations of the cross-sectional slices taken for analysis in this study (modified from Shapiro et al. (9)).

Figure 2

Isotopic compositions expressed as the $^{206}Pb/^{204}Pb$ for diverse sources of lead in the Broken Hill mining community (14).

Table 1. Lead isotopic ratios and concentrations for teeth from Broken Hill Subjects who have experienced a low exposure of lead.

Family	Tooth section	Tooth	type	206Pb/ 204Pb	Pb (ppm)	Age (yrs)
510M+	I	LCI	71*	16.44	2.2	7
510M	I	LLI	72/82	16.54	0.55	
510M	C			16.44	4.27	
516F	I			16.41	0.80	6
516F	C-1			16.37	0.79	
516F	C-2			16.37	0.79	
521F	I	UCI	51	16.62	1.06	9
521F	C			16.48	1.11	
521F	I	ULI	62	16.64	0.79	
521F	C			16.44	2.05	
521F	WT	UCI	61	16.58	1.27	
530M	I	LCI	71	16.49	1.88	7
530M	C			16.45	4.93	
532M	WT	LCI	81	16.47	0.95	5
538M	C	UCI	61	16.91	1.75	8
546F		MOL	64	16.49	2.18	12
557M	I-1	ULI	52	16.31	1.14	7
557M	I-2			16.33	0.97	
557M	C			16.25	4.08	
562F	I	UCI	61	16.44	3.45	7
562F	C			16.43	5.81	
566M	I	ULI	52/62	16.53	2.08	7
566M	C			16.46	7.41	
568M	I	MOL	65	16.50	0.40	7
568M	C			16.37	2.97	
568F	I	ULI	62	16.52	0.68	9
568F	C			16.36	8.27	
569M	C	UCI	61	16.60	2.53	7
569M	I			16.52	1.24	

+ M denotes male, F female
I Incisal section; C Cervical section; WT whole tooth leached with 1.5M HCI
- 1,-2 Repeat analysis of same tooth
UCI Upper central incisor; ULI Upper lateral incisor;
LCI Lower central incisor; LLI Lower lateral incisor;
MOL Molar
72/82, 52/62 indicate uncertainty in tooth identification
* International nomenclature, see reference 16

Table 2. Lead isotopic ratios and concentrations of teeth from Broken Hill Subjects who have experienced high exposure of lead.

Subject	Tooth section	Tooth type	206Pb/ 204Pb	Pb (ppm)	Age (yrs)
519M+	I	51*	16.40	0.95	11
519M	C		16.21	13.7	
519M	I	62	16.33	1.49	
519M	C		16.28	3.04	
524F	C	51	16.24	13.2	7
524F	I		16.31	1.28	
526F	m	51	16.25	4.65	6
536M2	I	61	16.25	2.46	7
536-7M2	WT	83	16.25	9.42	
536-8M1	WT	51	16.22	7.47	11
536-9M1	CR+RT	73	16.21	7.63	
536-9M1	CR		16.38	1.47	
536-9M1	RT- 1		16.14	24.9	
536-9M1	RT-2		16.16	26.4	
536-9M1	RT-3		16.17	31.5	
536-10M1	CR	53	16.33	1.74	
536-10M1	LCR		16.23	7.17	
536-10M1	RT- 1		16.19	20.6	
536-10M1	RT-2		16.20	29.5	
536-16M1	I	64	16.22	4.59	
536-16M1	I		16.19	3.52	
536-16M1	C		16.23	11.6	
536M1	C	61	16.25	6.42	
542F	I	61	16.31	8.91	5
542F	C		16.34	12.3	
548M	I	51	16.44	2.47	7
548M	C		16.35	13.4	
548M	I	71	16.56	1.51	
548M	C		16.36	12.3	
552F	I-1	65	16.29	3.14	11
552F	I-2		16.31	3.13	
552F	C		16.26	9.87	
553F	I	73	16.36	4.73	9
553F	C		16.33	8.91	
563M	I	71	16.34	3.42	6
563M	C		16.31	18.4	

I Incisal section; C Cervical section; m Middle section
WT Whole tooth acid-leached as in Table 1
CR Crown; CR+RT Crown and Root; RT Root; LCR Lower section of crown
- 1,-2 Repeat analyses of the same tooth
+ M denotes male, F female
* Tooth identification as in Table 1.

Table 3. Lead isotopic ratios and concentrations of teeth from Broken Hill subjects who ingested paint.

Subject	Tooth section	Tooth type	206Pb/ 204Pb	Pb (ppm)	Age (yrs)
517F	I	71*	16.62	5.43	6
517F	C		16.59	30.6	
517F	I-1	53	16.67	3.33	
517F	C-1		16.49	15.5	
517F	I	63	16.61	4.53	
517F	C		16.64	15.5	
517M	I-2	53	16.67	4.70	8
517M	C-2		16.49	11.9	
517M	I	61	16.61	9.16	
517M	C		16.45	10.0	
533M1	I	61	16.37	1.91	6
533M1	C		16.37	4.57	
533M2	I	53	16.61	0.99	8
533M2	C		16.41	4.38	
549M	I	54	16.54	0.94	10
549M	C		16.92	6.79	

I Incisal section; C Cervical section
- 1,-2 Repeat analyses of the same tooth
M1, M2 denote Male 1, Male 2; F denotes Female
*Tooth identification as in Table 1.

Table 4. Lead isotopic ratios and concentrations in teeth from one Broken Hill family.

Subject	Tooth section	Tooth type*	206Pb/ 204Pb	Pb (ppm)	Age (yrs)
534M	I	51	16.18	3.05	1 1
534M	C		16.14	10.5	
534M	I	54	16.22	1.56	
534M	C-1		16.16	10.5	
534M	C-2		16.21	9.06	
534M	EN	24-3	16.15	9.36	
534M	RT- 1		16.15	6.97	
534M	RT-2		16.18	6.96	
534M	EN	34-3	16.12	7.88	
534M	RT		16.22	5.09	
534M	EN	5 6	16.10	8.62	
534M	RT		16.14	7.17	
534M	I	84	16.17	3.94	
534M	C		16.15	8.12	
534M	EN	86	16.11	10.4	
534M	RT		16.20	5.14	
534F	I	M	16.29	1.67	9
534F	C		16.17	9.98	
534F	I	54	16.29	1.73	
534F	C		16.20	9.07	

I Incisal section; C Cervical section; EN Enamel from crown;
RT Section from Root; -1 ,-2 Repeat analysis of same tooth; M Molar
* Tooth identification as in Table 1.

Table 5. Lead isotopic ratios and concentrations of teeth from subjects from diverse environments.

Subject	Tooth section	Tooth type*	206Pb/ 204Pb	Pb (ppm)	Age (yrs)	Sex
Sydney A	I	51	17.16	1.49	7	F
	C		17.29	6.47		
Smelter B	I	62	17.19	7.43	7	F
	C		17.24	19.9		
Smelter C	I	63	16.96	2.18	12	F
	C		17.02	13.3		
Brazil D	I	61	17.56	0.84	14	M
	C		17.43	1.43		
Sydney E	I	71	16.77	1.77	6	M
	C		16.71	2.61		

I Incisal section; C Cervical section
* Tooth identification as in Table 1.

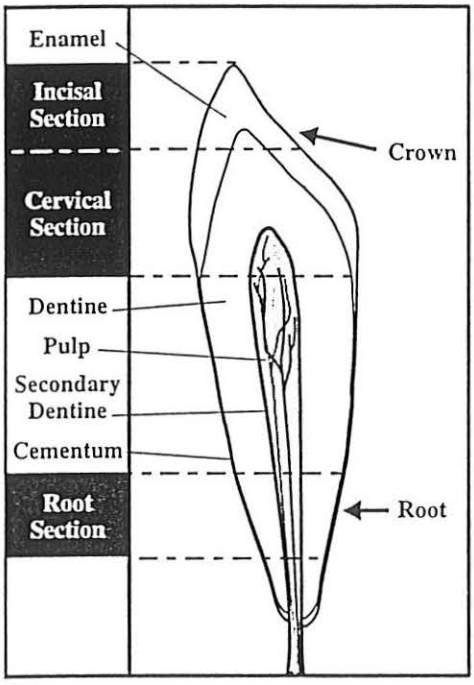

Enamel
Incisal Section
Crown
Cervical Section
Dentine
Pulp
Secondary Dentine
Cementum
Root Section
Root

Figure 1

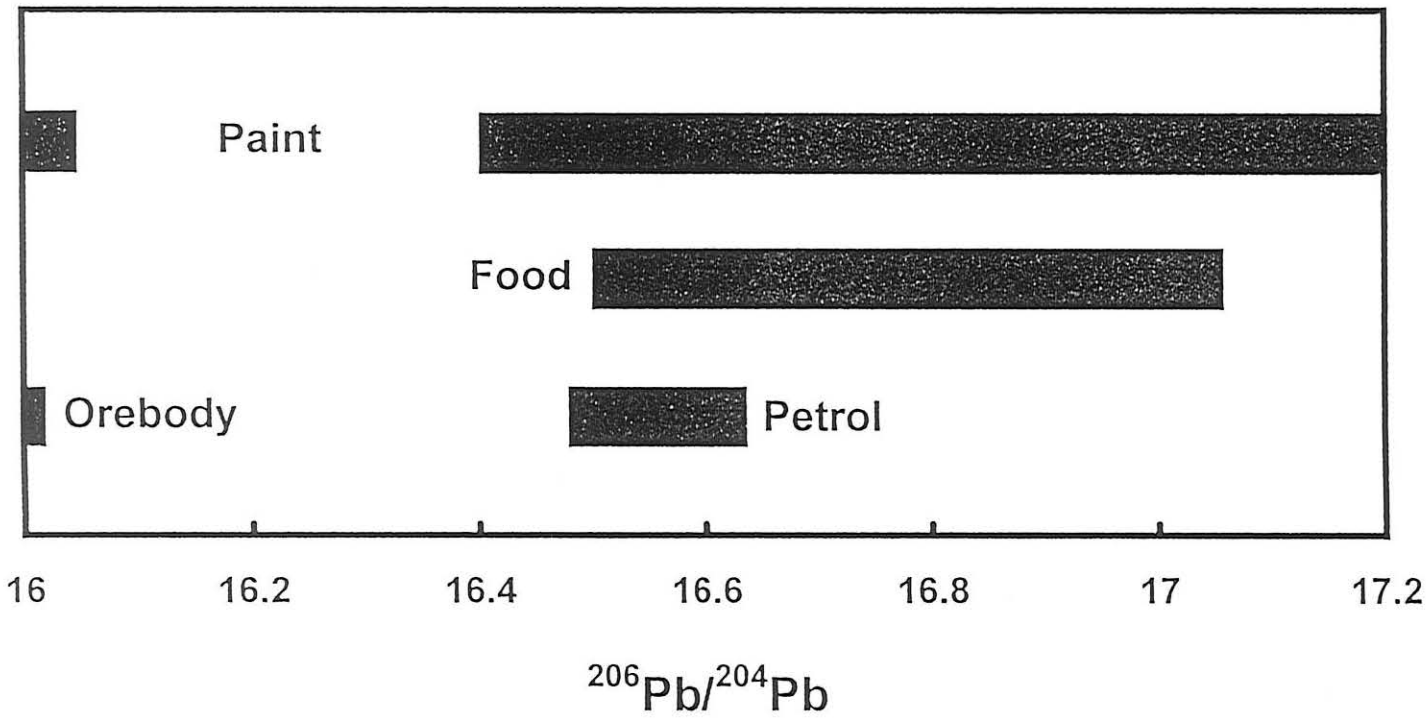


Figure 2

Chapter II. 6: Lead

000206

Introduction and Sources

1. Lead is the most widely used non-ferrous metal and has a large number of industrial applications, both in its elemental form and in alloys and compounds. The single largest use globally is in the manufacture of batteries (60-70% of the world consumption of some 4 million tonnes of lead), but other uses are as a pigment in paints and glazes, in alloys, in radiation shielding, tank lining and piping. As the compound tetraethyl lead, it has been used as a petrol additive to enhance the octane rating. With the recognition of the adverse effects of lead on human health and the growing use of catalytic converters, which are poisoned by lead, this use is declining rapidly.

2. Most of the airborne emissions of lead in the UK arise from petrol engined motor vehicles. A summary of the UK inventory of emissions of lead is given in Table II.6.1.

3. Most of the lead in the air is in the form of fine particles with an aerodynamic diameter of less than 1 micron (1 micron is one millionth of a metre). In the immediate vicinity of smelters, the particle size distribution usually shows a predominance of larger particles. However, these particles settle out of the air at distances of a few hundred metres or 1-2 kilometres, so that further away from these sources the particle size distribution is similar to that at urban sites mainly influenced by traffic emissions of lead.

4. Direct human exposure to lead occurs through food, water, dust and soil, and air. Most people receive the largest portion of their daily lead intake via food, although other sources may be important in specific populations (e.g. water in areas with lead pipes and plumbosolvent water supply; air in populations living near point sources of lead; soil, dust and paint flakes in young children living in houses with leaded paint or contaminated soil). The percentage of lead absorbed from the gastrointestinal tract is about 10% in adults, and 40-50% in children. Absorption through the respiratory tract ranges from 20% to 60%. Children are also likely to be more susceptible to lead and may be at particular risk if they have a deficient intake of calcium, iron or vitamin D.

5. Lead exhibits toxic biochemical effects in humans which are manifest in the synthesis of haemoglobin, acute or chronic damage to the nervous system, effects on the kidneys, gastrointestinal tract, joints and reproductive system. These problems are well-described in workers exposed to high concentrations. Also some have been observed as a consequence of ingestion of lead, especially by young children. In conditions of low-level and long-term lead exposure such as are found in the general population, the most critical effects are those on haem biosynthesis, erythropoiesis, the nervous system, and blood pressure.

6. Most studies of the adverse effects of lead are based on blood lead levels. On absorption, lead is rapidly distributed through the body, where it accumulates. Typically about 2% of the body burden is in the blood where it is most biologically active. Its half life in blood is about three weeks. The rest is stored in bone (>90%), teeth, skin and muscle, where it is slowly released into the blood and potentially available for excretion via the kidneys.

7. In terms of exposure to lead, the consensus is that long-term exposure (over periods of the order of a year or more) is the relevant measure and this will be discussed further below in relation to air quality standards.

8. During the 1970s and early 1980s the lead content of petrol was gradually reduced, maintaining total emissions from vehicles broadly constant. At the end of 1985, the maximum permitted lead content of petrol was reduced significantly from 0.40 g/l to 0.15 g/l, and in 1987 unleaded petrol was introduced.

9. Emissions of lead from petrol vehicles are estimated annually and the results from 1980 to 1995 are shown in Figure II.6.1. The large increase in unleaded petrol consumption after 1988 has been responsible for more than halving emissions from motor vehicles since 1987.

Table II.6.1: Emissions of lead in the UK, 1995

Source	Emission (tonnes)	Percentage of total* in 1995
Road Transport: Petrol	1067	72
Road Transport: Diesel	1	0
Non-Ferrous Metals	140	9
Iron and Steel	46	4
Waste Related Sources	105	7
Industrial Processes	23	2
Industrial Combustion	44	3
Power Stations Combustion	28	2
Domestic Combustion	9	1
Other Combustion	5	0
Total	**1492**	**100**

* Figures rounded to nearest 1 %

Health Effects: Standards and Guidelines

10. The effects of lead on human health referred to above have generally been quantified by using the concentration of lead in blood, or blood-lead, as the indicator of exposure. Contributions to blood-lead levels arise primarily from air, water and food.

11. A brief qualitative description of the adverse effects of lead on human health was given above. The World Health Organisation has recently reviewed the literature on this subject and summaries of the lowest observable adverse effect levels (LOAELs) in adults and children are given in Tables II.6.2 and II.6.3.

12. Anaemia occurs only in cases of severe lead poisoning, but effects on red cell survival and haemoglobin production are found at lower levels. The lowest observed effect level at which impaired haem synthesis is found is at a blood lead concentration of about 50 µg/dl.

13. Acute neurological effects of delirium, confusion and convulsions are very rare and occur at blood lead levels above 100 µg/dl. More chronic effects include wrist drop and impaired mental function. Peripheral nerve dysfunction has been detected at levels above about 30 µg/dl. Subtle effects on neuropsychological function may be found in children at blood lead levels below 10 µg/dl. It is worth noting in this context that the Committee on Toxicity of Chemicals in Food, Consumer Products and the

Environment (CoT) has concluded that it is not possible to identify a threshold for effects of lead on health.

14. Workers are subject to biological monitoring at frequencies dependent on their blood lead levels. Standards for airborne lead in the workplace are 100 $\mu g/m^3$ 8-hour time-weighted average for tetra-ethyl lead (the anti-knock additive used in leaded petrol) and 150 $\mu g/m^3$ 8-hour time-weighted average for other forms of lead.

15. An ambient air quality standard exists within the EU via Directive 82/884/EEC and is 2 $\mu g/m^3$ as an annual average. In their 1987 publication, the WHO set a guideline for lead at 0.5-1.0 $\mu g/m^3$ also as an annual average, but have recently revised this guideline to 0.5 $\mu g/m^3$ as an annual average. This was based on the target of ensuring that at least 98% of the exposed population should have blood lead levels below 10 µg/dl. On this basis the median blood lead level would not exceed 5.4 µg/dl. Using the relationship between blood lead and airborne lead, namely that 1 $\mu g/m^3$ of airborne lead would contribute to 5 µg/dl blood lead (to allow for uptake by other routes), and that the maximum non-anthropogenic level of blood lead is 3µ g/dl, the WHO arrived at the value of 0.5 $\mu g/m^3$ (or 500 ng/m^3) as the guideline for an annual average for airborne lead.

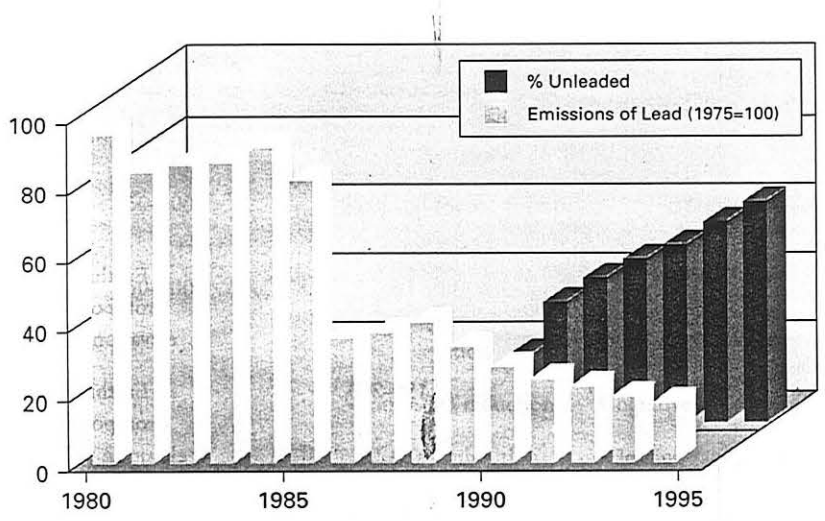
Current Air Quality

16. Lead concentrations in air have decreased significantly in the past decade. Indeed, apart from the very large decrease in black smoke and sulphur dioxide concentrations following the Clean Air Act of 1956, it is arguable that the decline in airborne lead levels has been the next most dramatic change in pollutant levels in the UK in recent years.

17. The reason for this decrease is twofold. The major reduction in the maximum permissible lead content of leaded petrol from 0.4 µg/l to 0.15 µg/l in January 1986 almost halved urban air lead levels in the space of a few months. This reduction was reinforced and sustained by the introduction of unleaded petrol in 1987 and the continued increasing market share of this fuel ever since, to the point where, since 1993, all new petrol engined cars are catalyst equipped and therefore must run on unleaded petrol.

18. In the mid-1980s, annual average urban levels of airborne lead were broadly in the range 0.15-1.0 $\mu g/m^3$, with one measurement in 1985 in

Figure II.6.1: Emissions of lead from petrol vehicles



Cardiff of 1.28 μg/m^3, and one in Manchester, also in 1985, of 2.04 μg/m^3. Kerbside levels at the Cromwell Road site in West London were of the order of 1.4 μg/m^3. Following the reductions in the lead content of petrol discussed above, urban levels have reduced to the extent that the maximum values are now of the order of 0.2-0.3 μg/m^3 even at the Cromwell Road site. Rural values, as expected, are rather smaller and currently range from about 0.005-0.050 μg/m^3. In industrial areas in the vicinity of processes which emit lead, such as secondary non-ferrous metal smelters, levels can be higher than in urban areas where motor vehicle emissions are the main source of lead. Levels at such sites in 1995 ranged from about 0.14-1.0 μg/m^3.

19. The WHO guideline figure is therefore currently unlikely to be exceeded in urban areas. This is not the case for some industrial areas however, where levels, at the Walsall sites for example, exceed the WHO guideline, although they remain within the current EC Directive limit.

The Strategy

20. Unleaded petrol currently has a market share of some 70% of petrol sales. The increasing penetration of catalyst-equipped cars into the fleet will mean a progressive increase in this proportion, to the extent that emissions of lead from petrol vehicles should decrease by over 80% by 2005 compared with 1995 levels, and to a very small amount by 2015. Levels of airborne lead will therefore continue to decrease in urban areas where traffic is the major source, and, in the great majority of the UK, annual average levels should decrease to the extent where in most urban

Table II.6.2 : Summary of Lowest Observable Adverse Effect Levels for lead-induced health effects in adults

Lowest-observed-effect levels of blood lead (μg/l)	Haem synthesis and haematological effects	Effects on nervous system
1000-1200		Encephalopathic signs and symptoms
800	Frank anaemia	
500	Reduced haemoglobin production	Overt subencephalopathic neurological symptoms, cognition impairment
400	Increased urinary Ala and elevated coproporphyrin	
300		Peripheral nerve dysfunction (slowed nerve condition velocities)
200-300	Erythrocyte protoporphyrin elevation in males	
150-200	Erythrocyte protoporphyrin elevation in females	

Source: *Update and Revision of the Air Quality Guidelines for Europe*, WHO, 1994

areas levels are of the order of 0.1-0.2 μg/m^3 even at busy roadsides. The revised WHO guideline of 0.5 μg/m^3 should therefore be achievable by 2005 in urban areas of the UK.

21. In the vicinity of individual industrial plants which may be significant emitters of airborne lead this may not be the case. Such plants will be subject generally to BATNEEC and the provisions of the Environmental Protection Act 1990 and emissions should be reduced to the lowest level possible in accordance with BATNEEC.

Conclusion

22. The Government recognises that EPAQS has not yet made a recommendation for a standard for lead. In the meantime, the Government will adopt the revised WHO guideline figure of 0.5 μg/m^3 as an annual mean, as the standard, with the objective of achieving it by 2005. Noting the advice of the Committee of Toxicology, the Government further proposes to reduce levels of airborne lead below this target level wherever practicable.

23. The Government has decided that the objective for lead should apply in the following non-occupational near-ground level outdoor locations: background locations; roadside locations; and other areas of elevated lead concentrations where a person might reasonably be expected to be exposed (e.g. in the vicinity of housing, schools or hospitals etc), over the averaging time of the objective. Consideration of potential exceedences of the lead objective at potentially high concentration locations should be carried out in conjunction with data from urban or other appropriate background locations. It should be noted that exposure to lead is from a variety of sources, and it is UK Government policy to reduce exposure to lead from all sources including food, drinking water, paint, air, soil and dust, and thus restrict as far as possible the environmental accumulation of lead.

Table II.6.3: Summary of Lowest Observable Adverse Effects Levels for lead induced health effects in children

Lowest-observed-effect levels of blood lead (μg/l)	Haem synthesis and haematological and other effects	Effects on nervous system
800-1000		Encephalopathic signs and symptoms
700	Frank anaemia	
400	Increased urinary ALA and elevated coproporphyrin	
250-300	Reduced haemoglobin synthesis	
150-200	Erythrocyte protoporphyrin elevation	
100-150	Vitamin D3 reduction	Cognitive impairment
100	ALAD - inhibition	Hearing impairment

Source: *Update and Revision of the Air Quality Guidelines for Europe*, WHO, 1994.

Table II.6.4: Annual mean airborne lead concentrations at UK sites 1980-1995 (ng/m^3)

	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995
KERBSIDE																
Cromwell Road (W. London)	-	-	-	1,370	1,410	1,450	660	-	-	-	380	360	348	255	244	199
URBAN																
Central London	640	580	630	470	520	480	270	280	300	220	-	120	99	78	85	60
Brent (London)	770	710	890	990	-	640	300	290	320	-	220	200	174	147	144	80
Leeds	650	370	450	440	260	310	180	190	140	-	120	-	-	106	80	76
Motherwell	260	230	300	240	180	260	190	180	-	-	200	160	50	86	23	50
Glasgow	460	330	240	420	190	270	120	180	130	140	95	92	93	90	39	51
Cardiff	-	-	-	-	-	1,280	630	630	620	570	460	440	384	311	233	165
Manchester	-	-	-	-	-	2,040	810	810	760	640	510	460	339	305	123	133
Newcastle	-	-	-	-	-	180	130	150	110	110	70	70	67	70	27	25
North Tyneside	-	-	-	-	-	290	150	190	140	120	81	100	90	90	26	-
RURAL																
Cottered	-	-	-	-	130	130	77	98	76	75	41	45	44	36	19	20
North Petherton	-	-	-	-	-	-	70	65	69	81	53	62	-	-		-
Eskdalemuir	-	-	-	-	-	29	8	13	14	13	10	13	10	7	6	5
Chilton	110	56	66	65	86	90	33	60	52	48	38	38	27	22	25	25
Trebanos	82	80	78	92	98	81	39	48	55	64	43	63	60	37	32	39
Styrrup	178	135	172	115	170	130	66	70	94	86	57	65	55	35	47	44
Windermere	47	39	47	45	48	35	24	20	23	21	15	20	8	8	12	13
INDUSTRIAL																
Walsall Industry 1	-	-	-	-	-	-	930	1,370	1,160	880	470	570	540	570	500	697
Walsall Metals Industry 2	-	-	-	-	-	-	2,260	2,950	3,580	2,430	1,300	1,390	1,440	1,220	1,400	1,020
Walsall Metals Industry 3	-	-	-	-	-	-	760	730	-	640	-	340	410	-	-	-
Walsall Metals Industry 5	-	-	-	-	-	-	-	1,110	1,590	1380	680	620	680	470	470	660
Brookside 1	-	-	-	-	-	-	310	330	-	260	150	150	220	160	150	177
Brookside 2	-	-	-	-	-	-	990	890	1,310	1,100	1,140	710	560	470	440	464
Elswick 1	-	-	-	-	-	-	-	1,650	1,350	530	370	660	340	190	340	481
Elswick 2	-	-	-	-	-	-	-	610	510	600	330	250	230	150	150	141
Elswick 6	-	-	-	-	-	-	-	-	-	780	450	460	300	190	220	192