

Total contribution of airborne lead to blood lead

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ABSTRACT A nine year study of blood lead concentrations and isotope ratios carried out on a married couple shows that pulmonary deposition cannot account for all the airborne lead in blood; that lead from bone may comprise 70% of blood lead; and that during pregnancy blood lead may double due to mobilisation of lead from bone.

The measurement of the isotope ratios of lead in human blood or other tissue may be used to identify the various sources of the metal. In a previous study I showed that the ratios in the blood of a small group of subjects in Dallas, Texas, varied systematically and demonstrated the presence of dietary, skeletal, and airborne lead in blood.¹ Analysis of the results showed that 7-40% of the lead in blood was airborne in origin. It was clear, however, from direct measurement of the exposure of three of these subjects to airborne lead that pulmonary deposition could not account for all of the airborne lead indicated by the isotope ratio study (table 1).² To understand this discrepancy, the observations on two of the subjects of the earlier study—subjects 9 and 8 (a husband and wife)—have been continued for a further period.

Subjects and samples

When the study began in 1974 the husband was 38. His first wife was 30 and six months pregnant with her second child. He had grown up in England and South Africa and started to live in Dallas in 1964. She was American born and raised. They had lived in the suburbs of north Dallas since 1968. Neither smoked. Both worked (she returned to work three months after delivery), making a daily round trip of 36 miles (58 km) along suburban thoroughfares and freeways. Their exposure to lead was similar with the exception that he travelled alone to England on three occasions. In January 1977 both were exposed to large amounts of lead from sandpapering old paint that was later found to contain 3.8% lead.

Throughout the study the isotope ratio (only the

ratio ²⁰⁶Pb/²⁰⁷Pb is reported) of lead in the air of Dallas was monitored, using composites of high volume collections from sites in north Dallas maintained by the City of Dallas. For the first three years the husband was bled twice monthly and his first wife monthly. Thereafter he was bled in 1978 and 1980 only to identify his winter minima in isotope ratio, but from 1982 to the present has been bled regularly. The first wife was not bled again until 1980, when a few samples were obtained. She was then diagnosed as suffering from ankylosing spondylitis. Subject 9 has since remarried and, for the sake of comparison, some measurements from his present wife are reported. In addition to the air, lead enters the blood from food and from the skeleton and, to characterise the sources isotopically, samples were taken of the husband's diet and a piece of bone was procured from his hip.

Results

Since 1974, there have been pronounced variations in the isotope ratio in the air of Dallas, which doubtless reflects the several geological sources of lead used for making additives (fig 1).

Except for a small increase after exposure to paint dust, the husband's blood lead concentration varied between 16 and 18 µg/dl until January 1977, when it began to decline, reaching a minimum of 11 µg/dl in 1980 (fig 2). Currently it has stabilised between 12 and 14 µg/dl. His isotope ratio (fig 1) has shown a characteristic seasonal pattern, high in autumn and low in spring, broken by exposure to lead of extreme isotope ratio while sandpapering old paint and visiting Britain.

The pattern displayed by his first wife differs appreciably from his. The amount of lead in her blood remained constant until seven months after childbirth when it began to decline, halving its value in 11 months. Her isotope ratio declined until child-

Table 1 Respired and other inputs of airborne lead to blood for some Dallas residents in 1975

Subject No	Blood lead ($\mu\text{g}/\text{dl}$)	Total lead input ($\mu\text{g}/\text{day}$)	Percentage airborne lead in blood	Total input airborne lead ($\mu\text{g}/\text{day}$)	Airborne lead 24 h (conc $\mu\text{g}/\text{m}^3$)	α	Airborne lead		
							Respired ($\mu\text{g}/\text{day}$)	Other ($\mu\text{g}/\text{day}$)	(Respired) (Respired + other) %
3	8.4	15	31	4.5	0.22	12	0.91	3.6	20
6	12.6	30	39	11.7	1.09	4.5	4.5	7.2	38
8	5.5	9.3	>33	>3.1	0.45	>4.1	1.9	>1.2	<61
9	17.4	45	20	9.0	0.45	7.7	1.9	7.1	21

Data in first five columns for subjects 3, 6, and 9 from refs 1 and 2 recalculated using revised parameters of Chamberlain *et al.* α is defined as increment in blood lead concentration ($\mu\text{g}/\text{dl}$) per unit increment in airborne lead concentration ($\mu\text{g}/\text{m}^3$). Fraction of airborne lead in subject 8 calculated from measured isotope ratio of air, blood, and diet on two occasions in 1976 (fig 1). Figures quoted are minima because skeletal input, which would have had an isotope ratio less than that of the diet, has been ignored.

birth, when it abruptly reversed and, except for a brief perturbation after exposure to paint dust, appears to follow the ratio of the air.

His exposure to lead in food (table 2) is somewhat less than the range of 100–200 $\mu\text{gPb}/\text{day}$ reported in other studies.^{3,4} The lead content of his skeleton (table 3) is similar to that encountered in the two Los Angeles residents biopsied by Rabinowitz *et al.*,⁴ but the isotope ratio reflects the Australian type of

lead to which he was exposed during childhood and adolescence.

Discussion

My previous estimates of the fraction of airborne lead in blood were made on the assumption that the isotope ratio of the dietary component of lead in blood had remained constant over a 14 month

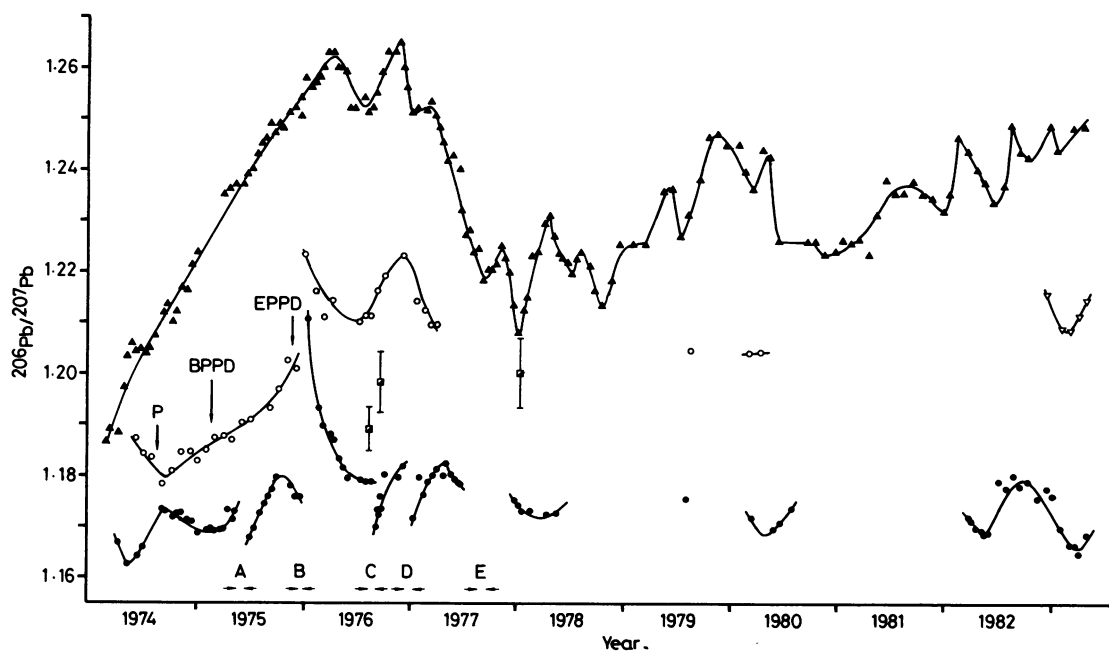


Fig 1 Isotope ratio of lead in air, blood, and diet from 1974 to 1983. Solid triangles: airborne particulate, solid circles: husband's blood, open circles: first wife's blood, open triangles: second wife's blood, half squares: diet (bars indicate 95% confidence limits). Points corresponding to parturition, P, and beginning and end of postparturition decline in blood lead concentration, BPPD and EPPD, are shown on the first wife's curve. Times of exposure to lead of unusual composition are shown between arrows. A, C, D, E correspond to visits made to Britain by husband where environmental lead is likely to be dominated by Australian lead having an isotope ratio of 1.02. B corresponds to exposure to lead in paint dust having a ratio of 1.288.

Table 2 Lead contents and isotope ratio of the husband's diet

Date	No	Lead (μg)		$^{206}\text{Pb}/^{207}\text{Pb}$	
		Range	$\bar{x} \pm \sigma$	Range	$\bar{x} \pm \sigma$
Aug 1976	12	24-122	66 ± 42	1.184-1.204	1.189 ± 0.008
Sept 1976	13	20-106	54 ± 26	1.173-1.207	1.199 ± 0.011
Jan 1978	13	19-67	45 ± 17	1.189-1.237	1.200 ± 0.013

Exact duplicates of husband's meals were taken on consecutive days over each period. One sample from August 1976 series containing $18 \mu\text{g}$ Pb with a ratio of 1.171 was excluded because it contained a meal of exotic seafood not representative of husband's diet. One sample from January 1978 series was also omitted because it contained 3.5 mg Pb with a ratio of 1.300. This ratio is found in some solders used in the United States.

period at the beginning of the study. Had there been a progressive increase in this ratio, my figures would have been too large. I had hoped to test my results for consistency by observing the isotope ratio in the two subjects' blood when the ratio in airborne lead was declining, but this was precluded by their exposure to lead of extreme isotope ratio over that critical period. I note, however, that the husband's annual minima in blood lead isotope ratios for 1975 and 1978 occurred at times when ratios for airborne lead were identical. The respective values were: 20 February 1975: blood 1.168; air 1.226; 20 March 1978: blood 1.172; air 1.226. The closeness of these observations argues strongly for the absence of any significant change in the isotope ratios of non-airborne sources of lead in blood over these three years. In fact, even large changes in the isotope ratio of dietary lead would have had minimal effect on his blood isotope ratio, because his dietary intake of lead was about $55 \mu\text{g}/\text{day}$, which, using the figure of 15% for gastrointestinal absorption of Chamberlain *et al.*,¹⁴ translates to an input to blood of $4.6 \mu\text{g}/\text{day}$ or only 10% of the total input necessary to sustain his blood lead concentration at $17 \mu\text{g}/\text{dl}$.

If 20% of the input of lead comes from the air and 10% from food the remaining 70% comes from his skeleton. This figure is consistent with the isotopic

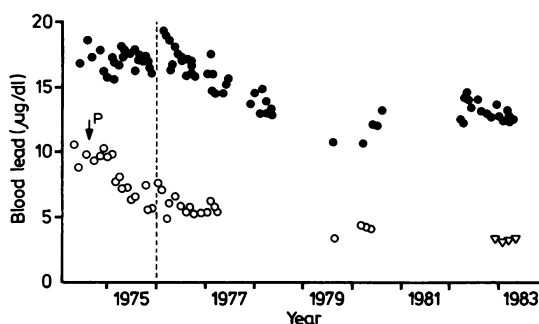


Fig 2 Change in blood lead concentrations measured from 1974 to 1983. Solid circle: husband, open circle: first wife, triangle: second wife, P designates parturition. Dashed vertical line indicates time of exposure to lead in paint dust.

Table 3 Lead concentration and isotope ratio in iliac crest biopsy

Region	Lead ($\mu\text{g}/\text{g}$)	$^{206}\text{Pb}/^{207}\text{Pb}$
Cortex	15	1.133
Trabeculae	8.6	1.163

Analyses are of two cortical tables and of delicate trabecular bone. Transitional bone present in this portion of skeleton was excluded from analysis.

data, for if the dietary and airborne contributions are subtracted from his blood the ratio of the remainder cycles between that of the cortical and trabecular bone of his ilium (fig 3). Since the biopsy showed no sign of bone disease, 70% must in his case be normal and implies that during the process

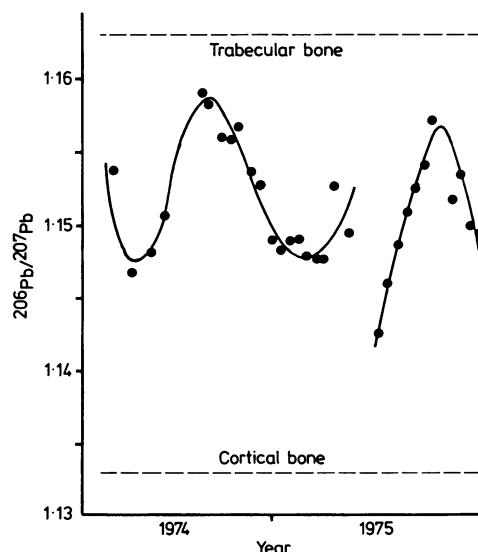


Fig 3 Isotope ratios of skeletal component of lead in husband's blood during 1974 and 1975 were obtained by subtracting from observed ratios (1) a 20% airborne component steadily increasing in isotope ratio (fig 1) and (2) a 10% dietary component with isotope ratio 1.194. Also shown are isotope ratios obtained from iliac crest biopsy in 1977. Break in curve results from exposure to lead with low isotope ratio while visiting Britain.

of remodelling of the skeleton the old bone being resorbed had a higher Pb/Ca ratio than the new bone being laid down. A large net flux of lead from his skeleton readily explains why his blood isotope ratio and concentration are so different from those of the two women with whom he has shared a common environmental exposure to lead.

One important fact should be noted. If his dietary intake of lead had been reduced to zero his blood lead concentration would have fallen by 1.8 $\mu\text{g}/\text{dl}$, so that most of the decrease of 4 $\mu\text{g}/\text{dl}$ observed in his blood lead since 1977 must be attributed to reduction in sources other than dietary. In fact, market basket surveys of lead in food show no reduction in lead content from 1974 to 1980⁶ and, in a man then entering his fifth decade net bone resorption is probably increasing with age,⁷ with the implication that this decrease in blood lead should be attributed only to a decrease in airborne lead concentration. This conclusion is supported by his blood isotope ratio which has progressively declined since 1977, whereas that in the air has slowly risen, by contrast with the situation before 1977, when his ratio grew sympathetically with that of the air.

In view of her pregnancy and lactation, the first wife's isotope ratio must be carefully interpreted. As discussed above, the husband's blood shows no evidence for a significant long term change in the isotope ratio of food, so that my previous interpretation¹ that her decreasing isotope ratio during pregnancy represented increasing amounts of dietary lead in her blood was incorrect. Instead, this decreasing ratio signifies ever increasing amounts of a third source of lead in her blood, which must be her skeleton, whose turnover rate would have increased to meet the calcium demands of late pregnancy.⁸ The increase in her ratio and the decline in her concentration after childbirth largely reflects the return of her body to normal, and it is not until 16 months later, when her blood assumed a constant concentration, that she may be considered in a steady state.

Even so, the form of her blood curve which follows closely that of the air in 1976 and 1977 has too great an amplitude to be explained by contributions from airborne lead alone. At least part of the changes in isotope ratio must be ascribed to short term fluctuations in dietary lead and, on the assumption that their exposure to lead in air and food were essentially the same, the minimum fraction of airborne lead in her blood and her α may be calculated (table 1). Too few samples were collected to attribute the 24% decline in her blood lead concentration to decreasing amounts of lead in the air, but clearly no large, long term increase in her isotope ratio occurred.

Significance

Since the evidence negates a long term change in the isotope ratio of lead in his diet, my previously published figure of 20% for the fraction of airborne lead in the husband's blood must be considered accurate. This being so, there is no reason to presume that the somewhat greater figures I measured for the other subjects are not also accurate, especially since this particular subject has so large an amount of skeletal lead in his blood. Less accurate are the estimates of the subjects' exposures to airborne lead, but the figures are within the range to be expected of suburban dwellers⁹ and the value of α in table 1 are believed to be correct to within a factor of 2. Although these α s indicate far more airborne lead in blood than do the experiments of Chamberlain *et al* with tagged aerosols,¹⁰ our respective findings are not in dispute, because two different types of experiment were involved. They designed an experiment to measure accurately the deposition of lead in the lung and its translocation to blood. An experiment such as mine is designed to measure the blood's uptake of airborne lead by all routes. Pulmonary deposition can account for only 20–40% of the blood's uptake of airborne lead (table 1). Although these figures may seem excessively low to those familiar with conventional measures of α ,¹¹ their correctness is supported by the magnitude of the declines in the husband and wife's blood during the phase out of leaded petrol. Furthermore, it is now on record that the blood lead concentrations of the population of the United States declined by 37%, or 5.4 $\mu\text{g}/\text{dl}$, from 1976 to 1980 and that this reduction correlates well with declining manufacture and sales of lead additives.¹² This decline is much greater than would be expected from an α of 1 to 2 and an average exposure of 1–2 $\mu\text{g}/\text{m}^3$.

The routes by which airborne lead may enter the body other than through the lungs are difficult to identify. Contamination of food at its source may occur but cannot be identified in this experiment, because little of the food consumed in Dallas is produced locally and different parts of the United States are characterised by different isotope ratios of airborne lead.¹³ An attempt to measure local contamination of food by airborne lead by collecting dietary samples two years apart at extremes of airborne lead isotope ratio failed because the variance was too large to detect the necessary 30% airborne contamination (fig 1). As it is with children, dustfall must be regarded as the most likely primary source of airborne lead intake, although, for an adult, the ways by which such dust might be ingested are not easily specified. None the less, ingestion does seem to occur. In this study, for example, the first wife did

no sandpapering and sandpapering does not produce particles in the respirable range, yet, by virtue of being in the same house as the paintdust, she incorporated measurable quantities into her blood.

The data obtained from the husband imply that lead stored in the skeleton is far from inert but actively contributes to blood lead and, as Chamberlain *et al* have indicated, a time integrated value of α should embody a factor taking into account lead originally of airborne origin being resorbed from the skeleton.¹⁴ The size of this factor will depend on past and present exposure. It is also implied that, even if all anthropogenic lead were removed from the environment, blood lead concentrations would not fall to zero but would be maintained at some level depending on each individual's skeletal store of lead.

At the present time the principal concern with respect to lead in the environment centres around neurological damage to young children and the fetus. In this regard I point out that the blood lead concentration of the first wife apparently doubled during pregnancy owing to increased transport of lead from her skeleton. At term, the time integrated α of a pregnant woman may greatly increase and the increment in her blood lead due to her own lifetime exposure to airborne lead may be sufficient to carry the blood lead of her fetus beyond the point where neuronal damage occurs.

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