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Geochemical and Man-Made Sources of Lead and Human Health

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Geochemical and man-made sources of lead and human health

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Multiple sources of Pb have been identified for human populations, but there is little agreement as to their relative significance. Marked regional geochemical anomalies in the distribution of Pb have been identified and, in one district, their impact on rural communities has been determined.

The absorption of ingested Pb is modified by its chemical and physical form, by interaction with dietary minerals and lipids and by the nutritional status of the individual. Studies on children of various ethnic groups have demonstrated differences in soft tissue Pb burdens which may in part have a nutritional basis.

Erythrocyte protoporphyrin determinations suggest that metabolic disturbances attributable to Pb may occur at soft tissue burdens previously regarded as acceptable. The problem of defining an adverse health effect in relation to environmental sources of Pb is discussed.

Pb has been known and used by mankind for at least 5000 years. Of all the potentially toxic elements it is perhaps the most widely used and has been the most extensively studied. The data now available for the toxicological sources of Pb are extensive and could provide the basis for a useful model for the study of other elements which have been more recently exploited.

Historically, Pb was known to the ancient Greeks, used by the Romans, and found numerous applications in Europe in the Middle Ages. Some of the best described episodes of Pb toxicity occurred in the 18th century and were associated with the contamination of water and alcoholic beverages by Pb in storage vessels. The first treatise on Pb poisoning as it is recognized today was written in Paris in the 19th century by Tanquerel des Planches (1829) and recorded the occurrence of Pb poisoning in that city among textile workers using lead pigments. At the beginning of this century, Pb poisoning was a well recognized industrial disease, mainly involving adults. During the last few decades the introduction of effective control measures in the United Kingdom has resulted in the disappearance of frank plumbism, and industrial deaths from Pb poisoning have not been recorded in the United Kingdom during the last 20 years.

In contrast, Pb poisoning has been increasingly recognized in childhood so that in the United States some 40 000 children per year are now found to have evidence of undue exposure to Pb. In the United Kingdom, however, only 70–100 childhood cases are recognized each year. The definition of 'undue' has aroused debate and there is no general agreement as to the degree of exposure that might be regarded as acceptable. Although considerable progress has been made in limiting the health risks associated with discrete sources, for example Pb-based paint, a consequence of this is that the contribution from atmospheric and geochemical sources for human populations has assumed a greater significance. The clinical features of childhood Pb poisoning are non-specific and substantial body burdens of Pb may be sustained before clinical, as opposed to laboratory, recognition is possible. Four major categories in childhood can be defined, and are shown in table 1.

These categories are not, however, rigidly defined and considerable overlap may be recognized between them. Thus, few children sustain Pb-induced encephalopathy at less than 100 μg/100 ml, whereas some children with values greater than this may be asymptomatic. Since Pb is not subject to homoeostatic mechanisms in the body, it follows that the values encountered in the soft tissues such as blood reflect an equilibrium between Pb absorption from the lung and gut on the one hand, and excretion and deposition in bone on the other. The values encountered in a given population are thus characteristic and cannot readily be transposed to other communities.

TABLE 1

grade	description	blood lead concentration μg/100 ml
I	normal	<30–40
II	asymptomatic	40–60
III	symptomatic	60–80
IV	neurological disturbances	>80

This problem is enhanced in the interpretation of the significance of low level exposures resulting from general environmental sources. Thus, during the last few years, the acceptable upper limit for Pb in blood has been progressively reduced. Formerly, 40 μg/100 ml would have been regarded as the upper limit of normal for children but smaller concentrations have been increasingly advocated. This process has been enhanced by the recognition of biochemical disturbances at blood Pb concentrations encountered in the non-industrially exposed population. Thus, evidence of disturbed porphyrin metabolism has been demonstrated at blood Pb concentrations values of the order of 20 μg/100 ml and inhibition of the enzyme δ-aminolaevulinate dehydratase at 15 μg/100 ml or less. The biological significances of such metabolic and biochemical disturbances at low levels of exposure is uncertain although they have been used to support arguments in favour of reduction of Pb encountered in atmospheric lead derived from automotive emissions (Federal Register 1977).

Some biochemical changes, for example the accumulation of erythrocyte protoporphyrin, have formed the basis of systematic screening programmes for the detection of undue absorption by children in the United States. The objective of such programmes is the detection of children that are asymptomatic before serious clinical problems ensue. Such programmes do not at present exist in the United Kingdom where a recent survey of paediatricians (Barltrop *et al.* 1976) showed that only some 70 cases of undue absorption of Pb were recognized during the course of one year. Analysis of the blood Pb data for these children showed that in most cases there were marked increases in blood Pb concentrations above 40 μg/100 ml. A striking feature of the survey, however, was that in almost one-third of the children, no source of Pb was identified. The most frequently recognized source was Pb-based paint accessible to the child on surfaces within the home. It is perhaps useful to relate the Pb content of paint flakes that may be encountered in dwellings to the amount of Pb required to poison a child. Thus, a single paint flake weighing only 10 mg may contain as much as 1000 μg Pb, i.e. 10% (figure 1) and in some cases up to five times this amount may be encountered (Barltrop & Killala 1969). Although the cumulative dose of Pb required to produce severe symptomatic poisoning in a child has not been established with certainty, faecal Pb analyses have suggested that this must be of the order of 1000 μg/day (Barltrop & Killala 1967; Chisolm & Harrison 1956).

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It is unlikely that such intakes are ever encountered from general environmental sources. Thus, for adults a typical oral intake would be of the order of 300 μg/day and an atmospheric exposure of 30 μg/day. Nevertheless, the demonstration that a greater proportion of atmospheric Pb (30–40 %) is absorbed than dietary Pb (10 %) and the recognition that some atmospheric Pb must ultimately be deposited on the Earth's surface has stimulated studies to isolate the contribution from soils and dusts. This is particularly important with regard to the childhood age groups in whom the play and behavioural characteristics increase exposure to Pb from such sources and in whom pica has been shown to be prevalent. Such a study was undertaken in central England in the County of Derbyshire in an area which had formerly been the site of extensive mining and smelting during the last two millenia. Geochemical studies in

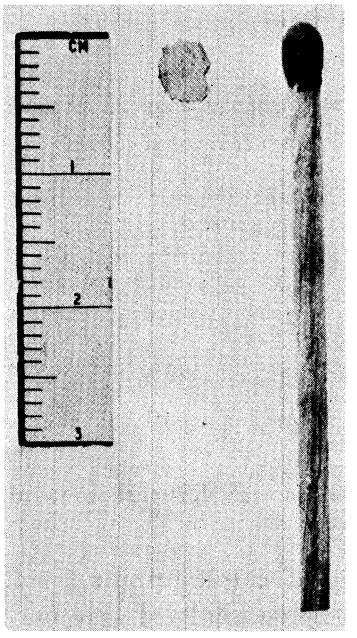

FIGURE 1. Paint flake (10 mg) from an internal surface accessible to children. Pb content approximately 10 % (1000 μg). Match and centimetre scale for comparison.

association with the Applied Geochemistry Research Group at Imperial College, London, showed that extensive areas of soil contamination exist in that county. The contamination is mainly situated on the east side of the Pennine range, however, and is minimal on the west. Apart from one small secondary smelter, the Pb industry is now virtually defunct in what is an essentially rural area.

Initial studies were related to mothers and children living in two towns situated in the contaminated and uncontaminated areas respectively. Soil analyses showed that surface Pb concentrations of 900 μg/g were encountered in the contaminated town and 400 μg/g in the control area. The control areas contrast with the average concentration in the Earth's crust which is usually given as 16 μg/g. Nevertheless, a search for control areas of similar geochemical nature (limestone) elsewhere in England failed to reveal smaller values. The study revealed no significant difference in the soft tissue burdens of Pb in children or the corresponding mothers,

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between the two districts. However, children had consistently greater mean blood concentrations than their mothers and a repeat survey showed that levels attained in the summer months were greater than those attained in the spring. These findings emphasized the need for the recognition of age and season as variables that must be controlled in any community studies associated with low level exposures to Pb (Barltrop *et al.* 1974). During the course of this study, a number of isolated villages were found in which much greater concentrations in garden soils and dusts were encountered with values of up to 30000 μg/g (3%). A similar study was repeated among the village communities and the results classified according to the soil Pb concentration surrounding the home. It was found that a significant correlation existed between both soil Pb concentration and the corresponding levels in blood and hair. Displacement in the geometric mean concentrations of Pb in blood associated with soil Pb levels in excess of 10000 μg/g (geometric mean 13969) compared with controls was of the order of 8 μg/100 ml blood (Barltrop *et al.* 1975) (table 2).

TABLE 2. MEAN CONTENT OF Pb IN BLOOD AND HAIR
(From Barltrop *et al.* (1975).)

	blood lead μg/100 ml		hair Pb	soil Pb	house dust Pb
soil lead	child	mother	μg/g	μg/g	μg/g
less than 1000 μg/g $n = 29$	20.7[a, b]	14.1[c]	7.7[f]	420	531
1000–10000 μg/g $n = 43$	23.8[a]	18.7[c, d]	10.5[e]	3390	1564
over 10000 μg/g $n = 10$	29.0[b]	14.8[d]	20.2[e, f]	13969	2582

a, a; b, b; etc.: significantly different, two-tailed t test, $p < 0.05$.

It thus appeared that soils and dusts can contribute to soft tissue burdens in a local population of children but that the values encountered were not in themselves hazardous. Nevertheless, proposed legislation in the United States concerning atmospheric Pb has been based on the precept that the maximal permissible contribution from lead in air to blood is 3 μg/100 ml, that is about one-third of the value encountered in the contaminated soil studies in the United Kingdom. The proposed atmospheric standards have been based on the concept that it is undesirable to permit a significant increase in the proportion of the population in which disturbances of porphyrin metabolism due to Pb may be detected. By contrast to the stringent measures which have been promulgated for atmospheric Pb, there are no comparable standards for Pb in soils and dusts even though their potential contribution to body Pb burdens may be of equal significance (Federal Register 1977).

Although the relation has now been demonstrated between Pb content in soils and dusts, and body burdens, the data might not be generally applicable to other geochemical areas, or indeed to urban districts. The Derbyshire soils were quite high in Ca content and this may have diminished the absorption of ingested Pb (Barltrop & Khoo 1975). The chemical form of Pb encountered in an old mining and smelting district may differ from that in an urban area in which the source of Pb is perhaps derived from automotive exhausts. Moreover, the association demonstrated did not identify the route of transmission which could have been the result of

oral ingestion either directly from pica, or indirectly from contamination of locally grown produce. An alternative route could have been re-entrainment of Pb from soil to atmosphere with subsequent inhalation and ingestion.

In order to determine the relation between chemical species of Pb available for absorption by the gut, a series of feeding studies were undertaken in a carefully standardized rat model. The data showed that a 12-fold variation in Pb absorption could be encountered for various compounds of Pb and that these were not necessarily related to the solubility of the compound in water or dilute mineral acids. Moreover, a further variable was noted in studies with metallic Pb preparations in which a fivefold variation in absorption could be demonstrated in relation to particle size (Barltrop & Meek 1975).

It follows that the interpretation of data for acquired Pb burdens in human populations requires knowledge of the many variables which may modify Pb absorption and that comparison of Pb burdens for even homogeneous populations in different geochemical districts should be interpreted with caution.

One important variable which has hitherto attracted little attention is that of the interaction of ingested Pb with other dietary components. One of the first systematic attempts to identify the relative importance of various dietary constituents for the absorption of Pb was that of Khoo (1976). She investigated the absorption of standard doses of Pb incorporated into diets of varying composition by immature rats and found that diets of low mineral and high lipid content markedly enhanced absorption. Thus a 50-fold increase in Pb absorption occurred in animals fed an appropriately modified diet compared with controls. Conversely, a high Ca diet inhibited Pb absorption (Barltrop & Khoo 1975). As yet it is unknown whether these findings are applicable to human populations. Nevertheless, diet and nutritional status often differ markedly between individuals in a given community and, in the United Kingdom, a rich variety of cultural and ethnic backgrounds coexist. Clearly, it would be advantageous to be able to identify groups of individuals that may be at special risk of undue absorption to Pb from given environmental sources. There is some evidence that particular ethnic groups may be relatively disadvantaged in this respect. Thus, Guinee (1972) showed that negro children tended to have greater blood Pb concentrations than Puerto Rican children living in similar environmental circumstances in New York. More recently, it has been shown that an undue proportion of Asian immigrant children in the United Kingdom are diagnosed as cases of Pb poisoning compared with their indigenous counterparts. Hitherto, such differences have been attributed to cultural or behavioural characteristics but the possibility of nutritional determinants for Pb absorption by various groups has not been fully explored. Recently, however, the preliminary findings of a study involving some 400 children aged 2–3 years in England have been explored (Strehlow & Barltrop 1978) This survey involved the random selection of children from two defined multiracial communities. It was found that the geometric mean blood Pb concentration for 155 Asian children (18.1 μg/100 ml) was significantly greater than that for 158 non-Asian controls (14.9 μg/100 ml) ($p < 0.001$). Moreover, ten of the Asian but only one of the non-Asian had blood Pb concentrations greater than 40 μg/100 ml (figure 2).

Marked differences in nutritional status were also detected. Thus the mean haemoglobin concentration for Asian children (11.0 g/100 ml) was significantly less than that for non-Asian children (12.7 g/100 ml; $p < 0.001$) (figure 3). It is likely that this reflected a relative degree of Fe deficiency in the Asian group which in turn contributed to the increased erythrocyte protoporphyrin values for Asian compared with non-Asian groups: geometric mean

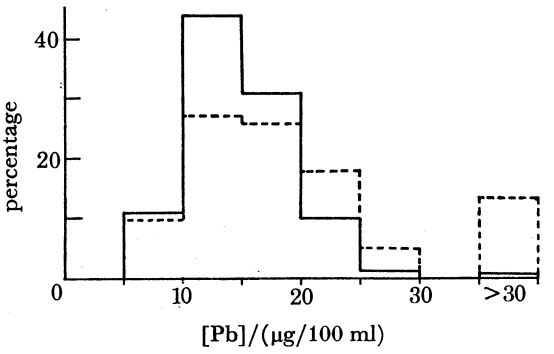

FIGURE 2

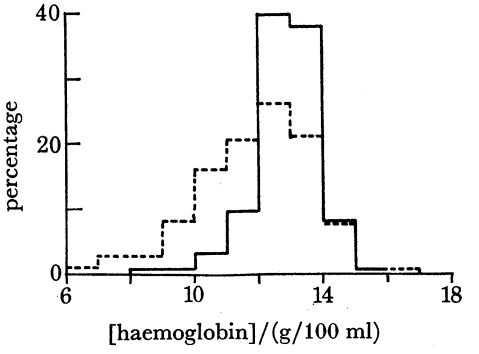

FIGURE 3

FIGURE 2. Distribution of blood Pb concentrations for 155 Asian (– – –) and 158 non-Asian (——) children.
FIGURE 3. Distribution of haemoglobin concentrations for 176 Asian (– – –) and 179 non-Asian (——) children.

34.3 μg/100 ml compared with 17.2 μg/100 ml ($p < 0.001$) (figure 4). However, even excluding children with haemoglobin levels of less than 10 g/100 ml, there was still a residue with excess erythrocyte protoporphyrin values in the Asian group compared with non-Asian (figure 4). The magnitude of the porphyrin excess was related to the concentration of Pb in blood within each group but the slope of the response differed markedly between them. Thus,

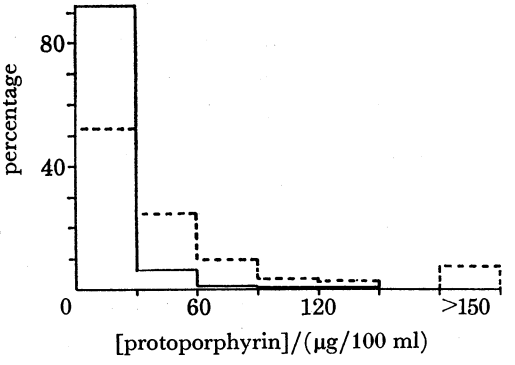

FIGURE 4

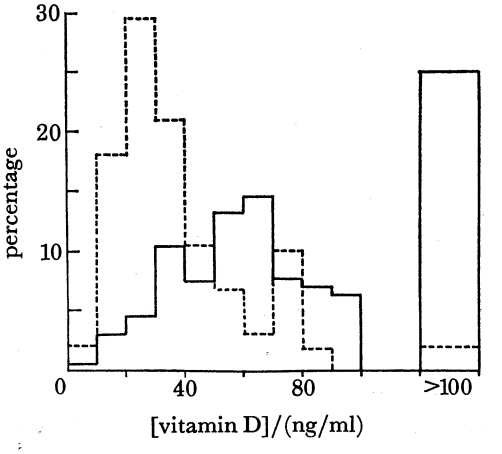

FIGURE 5

FIGURE 4. Distribution of erythrocyte protoporphyrin concentrations for 174 Asian (– – –) and 178 non-Asian (——) children.
FIGURE 5. Distribution of 25-OH vitamin D concentrations for 174 Asian (– – –) and 177 non-Asian (——) children.

for a given blood Pb concentration, Asian children appeared to have a greater erythrocyte protoporphyrin response than non-Asian children.

It is known that nutritional rickets and disturbances of Ca metabolism are more prevalent in the Asian as opposed to the non-Asian communities in the United Kingdom and in view of the previously reported relations between Pb absorption and Ca vitamin D status, plasma 25-OH vitamin D concentrations were determined in each of the children studied. A marked and significant difference was demonstrated such that the mean of 25-OH vitamin D levels

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for Asian children (36.9 ng/ml) were significantly less than the mean levels for non-Asian children (65.1 ng/ml) (figure 5).

It would thus appear that it is no longer possible to ascribe absolute significance to particular low-level environmental sources of Pb. Not only must detailed information be obtained concerning the chemical and physical form of the environmental Pb source but account must also be taken of the detailed nutritional characteristics of the populations exposed. Dietary practices and nutritional status may determine not only the magnitude of the fraction of ingested Pb that is absorbed, but may also modify the metabolic response of the individual. It is suggested that these findings may explain the differences observed in both Pb burdens and clinical response in the children in communities living adjacent to certain primary Pb smelters. Thus Clark (1977) reported unusually high blood Pb levels in children in a Zambian community in which malnutrition was common and who lived near to a smelter in spite of relatively modest environmental exposures. By contrast, a well nourished group of Mexican–American children living adjacent to a lead smelter with much greater exposures to Pb had relatively modest blood Pb values and minimal, if any, clinical sequelae (McNeil & Ptasnik 1974).

Variations in the interaction between potentially harmful agents and the host are familiar in medicine and toxicology. Recognition of these variations may modify our traditional approach to the interpretation of toxic elements in the environment and encourage a selective rather than indiscriminate search for susceptible population groups.

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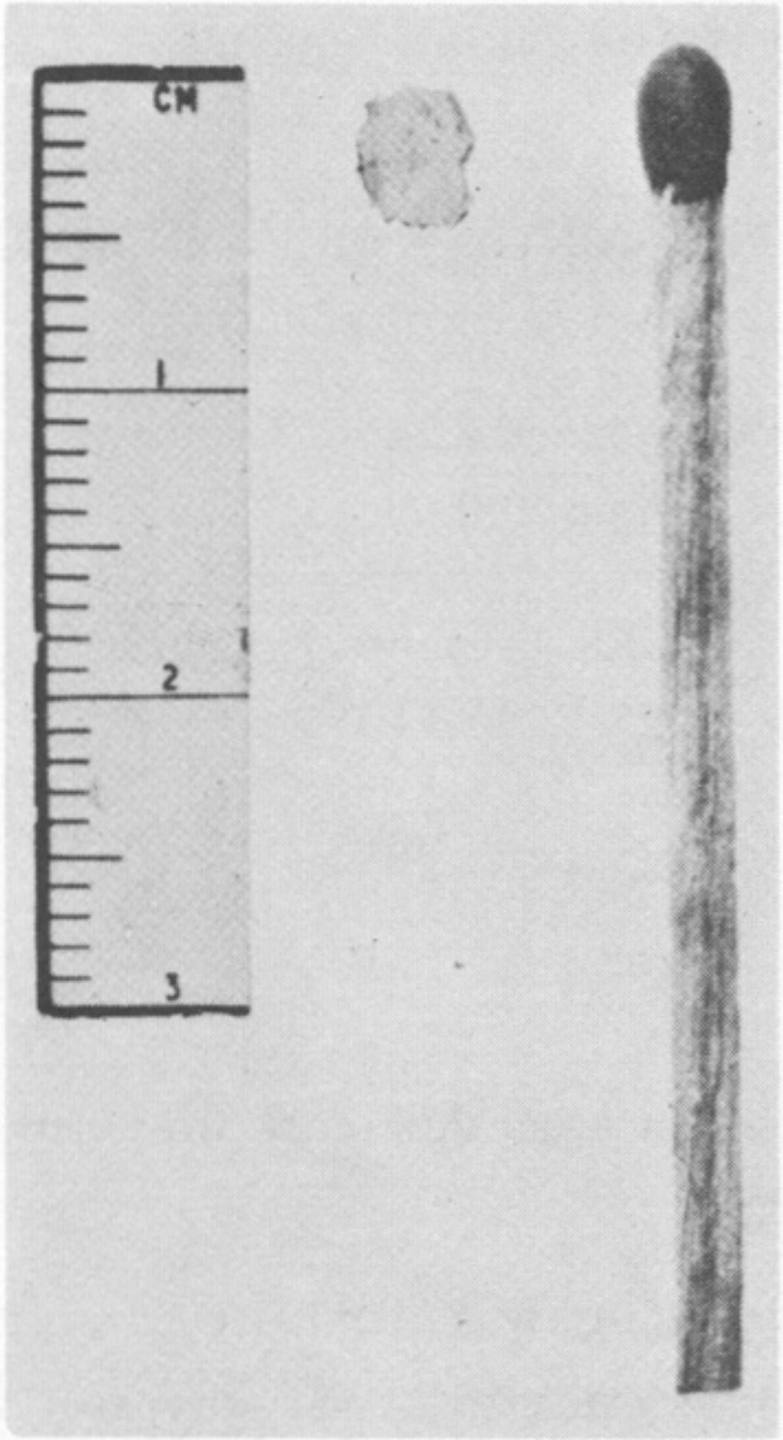

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