

# The Lead Sandwich Syndrome

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Only now is the extent of the effect of lead on people's health being recognised. In this context it is frightening when we realise just how much lead is present in our everyday foods.

THE VEIL OF NIGHT draws back its mantle revealing a fresh morning awakening with a new found crispness. Green hues mellow, framed by a golden penumbra from desiccating leaves.

The sound of the fair rings through the air again and mingles with the wafted scent of steaming toffee apples and brandy snaps.

Birds line the wires from pylon to pylon and plan their long flight south.

Autumn has come and the fruit of the year are gathered alike by animal and man. Pears and apples, plums and tomatoes; roads congested by motorists eager for the bounteous free harvest of blackberries- and lead.

Idyllic reflections blemished by the ubiquitous nature and insidious effects of the metal, element atomic number 82, lead.

Lead occurs widely in nature primarily as the sulphide in the ore galena, and to a lesser extent as the sulphate, anglesite, and carbonate, cerussite.

It has the unique combination of physical properties: low melting point (327.5 °C), low solubility in water, corrosion resistance, malleability and poor electrical conductivity, which ideally suit it to the wide variety of uses to which it is put, from glazing to water pipes, in batteries, and, more recently, as radio active screens.

In compound form it has found use in paint as pigment, in plastics as a stabiliser and in petrol as an anti-knock agent. And yet it is a poison, the toxic nature of which has been recognised for very many centuries.

Evidence for the use of lead extends back almost 6000 years, were recognised by Egyptian, Greek, and Roman physicians. Hippocrates in 370 BC described a severe attack of colic in a man extracting metals, and Pliny reported cases of lead poisoning between AD23 and 79.

In more recent history Thackrah in 1831 first drew a direct association between "miners sickness" described by Paracelsus in the 16<sup>th</sup> Century: a conclusion supported by a contemporary study of the health of lead miners in Derbyshire in 1857. The effects of the metal on the body were by this time becoming better understood. In 1774

Thomas Percival had reviewed the current knowledge in his *Observations and Experiments on the Poison of Lead*.

However, little was done for the protection of its workers. Not until the publication in 1912 of the definitive text *Lead Poisoning and Lead Absorption* by Legge and Goadby did the whole problem of industrial exposure in mining, the potteries, the paint industry and other areas of lead use in Great Britain receive serious, comprehensive attention.

In 1900 there were 1058 cases of lead poisoning listed in the *Annual Report of the Chief Inspector of Factories*: 36 of these cases proved fatal. In 1958 the number of cases had fallen to 55, none of which was fatal.

By 1966 the disease ceased to be classified as a separate cause of death. With some relief one may tend to conclude, therefore, that lead no longer presents a threat to health. Wrong! While cases of industrial poisoning have been controlled, the extent of the metal's effects on the population as a whole has increased over the past 50 years and is only now becoming recognised.

As a natural constituent of the earth's crust, lead is present in water and soil, in the latter at an estimated average concentration of 16ppm. It thus enters the food chain of both plants and animals, becoming incorporated into the structural tissue.

In an elegant study in 1965, Patterson calculated contributions from this natural source to the body burden of man as 2mg Pb/70kg of body weight, and a natural blood-lead concentration of 0.25µg/100ml of blood. Yet ready acceptance as "normal" is now given to blood lead levels in the range 10-30µg/100ml, despite, for example, the fact that the activity of the enzyme for delta-amino laevulinic acid, used in heme biosynthesis, is affected at levels of the order of 10µg Pb/100ml of blood in (*Air Quality Criteria for Lead, 1977*)

This poses 2 questions: first, what is the source of the additional body burden of lead, and second, why is the source not eliminated? We propose that the answer to both questions is, in part, encompassed by what we suggest is the lead sandwich syndrome; both literally and figuratively.

Occasionally sections of the community are exposed to adventitious sources of lead, perhaps because of their locality of the culture. Such sources are multifarious and very widely in extent: inhalation of outfall from a lead works, pica in children exposed to lead paints in old properties or on imported toys, poorly glazed or soldered domestic utensils, lead water pipes and, a particularly serious threat, certain Asian and Arabic medicinal or cosmetic preparations with a high lead content (Aslam *et al*, 1979; Aslam *et al*, 1980). Adventitious sources give rise to the majority of cases of overt lead poisoning recognised each year, predominantly among

children.

While each case is of special concern, such sources are not the prime cause of an increase of the body burden of lead in the general population. Equal attention must be directed at the possibilities of subclinical lead poisoning of the total population deriving in the main from road side emissions from leaded petrol. Consider the figures.

World wide, approximately 4.5 million metric tons of refined lead are consumed annually; about 300,000 tons in Britain alone (Hilburn, 1979).

The battery industry remains the principal user with a consumption which has remained relatively stable. In Britain in 1951 62,000 tons (metric) of lead went to battery production, while in 1977 68,000 tons were used for this purpose. Contrast this figure with that for lead alkyl production over the same period: in 1951, 5000 tons; in 1977, 55,000 tons, of which 16,000 tons were used in Britain, the remainder going for export.

Tetraethyl lead (TEL), the original lead-alkyl used, was first introduced into petrol in America in February 1923 following a discovery of its anti-knock qualities by the chemist Midgley in 1921. However, its introduction was not without cost, as it caused the death or illness of 139 workers involved in its production.

In May 1925 its sale was stopped and an investigation was begun by the US Public Health Service. In 1926, following of introduction of stringent regulations governing the manufacture and handling of TEL, its sale in petrol was resumed in the summer of that year.

Although introduced in petrol in this country during the 1930's, TEL manufacture in Britain did not start until 1940.

Tetramethyl lead (TML) was introduced as an anti-knock agent in 1960, and currently a variety of mixed alkyl compositions are used. Within the United Kingdom there has been a phased reduction in the lead content of petrol from the 1971 maximum limit of 0.84g/litre to the current figure introduced on January 1<sup>st</sup> of this year of 0.4g/litre. However, this decrease has coincided with an increase in traffic density and fuel consumption, so the overall lead emissions have not decreased.

According to the Department of Transport's own data (1979) some 7 000 tons of lead are emitted *each year* via petrol into the atmosphere of the United Kingdom. The adage, 'what goes up must come down,' is clearly true here. Now arises the dichotomy.

Government regulations restrict the lead content of paint used on toys to a maximum of 2 500ppm, and yet values in the region of 5 000ppm were reported in 8% of samples of household and street dust measured in a survey in Birmingham (Dept. of the Environment, 1978). Other studies

indicate that such values are by no means the exception.

Recently a close correlation has been shown between lead on the hands of children (mean age 11 years) and blood lead levels (Roels *et al.*, 1980). Levels of several hundred ppm of lead in dust were found by us in a more rural environment where gardens extended to within four metres of the roadside (Healy and Aslam, 1980).

It is reasonable to assume that the vegetables and fruit observed growing in these gardens was both lead laden and for consumption.

A number of studies (Rabinowitz, 1972; Tjell *et al.*, 1979) have clearly demonstrated that fall-out from exhaust emissions, and not natural soil lead, is the primary source of high concentration of the metal in food crops used by farm animals and humans. Thus a large contribution to the increased body lead must come from ingestion of contaminated food.

The *Lead in Food regulations (1979)* stipulate one ppm of lead as a maximum for fresh foods, with 0.2ppm as the maximum for infant foods. However, one can hardly rush into the kitchen and check the lead content of the freshly gathered lettuce or strawberries and so on.

It is not even possible successfully to monitor 'fresh' vegetables and fruit at the local greengrocer's because of the scale of the task. Where such produce is on display in front of the shop which may border a busy roadway, there is an additional hazard from deposition of the local dust which may add substantially to the level of lead the produce contains from the growing area. In our own work we attempted to determine the distribution of lead in a particular roadside environment and, while sampling was in progress, we observed people collecting large quantities of blackberries which grow in profusion along our chosen site.

Analysis of this fruit showed concentrations of lead in excess of 6ppm. The highest value was recorded at sites adjacent to lay-bys where, unfortunately, the fruit was also particularly accessible.

While direct eating of the fruit presents an unnecessary health hazard, because of the relatively short season for the crop this risk is limited.

However, following a standard recipe, preserves containing up to 4ppm of lead could readily be prepared from such fruit. This is of particular concern, for not only is this value 20 times the legal maximum for infant foods, but where such jams are fed to children (a not unlikely eventuality) the danger of the cumulative effects associated with lead toxicity must predominate. Nor does thoroughly washing the fruit remove the lead, as we found that 65%-80% of the

lead remained after stringent cleaning of the blackberries. It is of little surprise to find the danger is not restricted to blackberries. Even higher concentrations have been found in other roadside fruit (Fowles, 1976); almost 7ppm in elderberries and nearly 24ppm in hawthorn berries. Also, one can not ignore the indirect hazard such deposition of airborne lead can present.

Examination of the grassland adjacent to our study site showed lead levels in the region of 30-45 ppm. The metal appears to have a particular affinity for plant surface-tissue and is only partially removed from grass by rain or washing.

Use of such grass for grazing appears to provide an important source of the metal in the organs and flesh of the animals which are then consumed by man.

The contribution from various dietary substances to the daily lead intake in man has been calculated (Table 1) (on page 3). It can be clearly seen from the values obtained that fruits and preserves make up a significant portion of the total.

The evidence points strongly towards airborne lead from leaded petrol as a primary source. The arguments put forward against its removal from petrol are principally economic (although frequently propounded behind a mist of technicalities).

Scientists can only state the facts and allow the public to judge. However, in this debate the public at present appears spread between industry and g, but instead of jam in the sandwich the filling is lead.

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Table 1.

Food type	Mean lead Content (mg/kg)	Weight of food eaten (kg)	Lead intake per person (µg/day)
Cereals	0.12	0.27	32
Fruits and preserves	0.11	0.25	28
Meat and Fish	0.16	0.18	29
Green Vegetables	0.19	0.11	21
Root Vegetables	0.09	0.21	19
Milk	0.02	0.40	8
Fats	0.08	0.08	6
<b>Totals</b>	<b>0.09*</b>	<b>1.50</b>	<b>143</b>

\*Weight according to proportion of food type eaten

Table 1. *The estimated average daily intake of lead from food.* (from: Survey of Lead in Food (1970). Ministry of Agriculture, fisheries and Food. HMSO.)

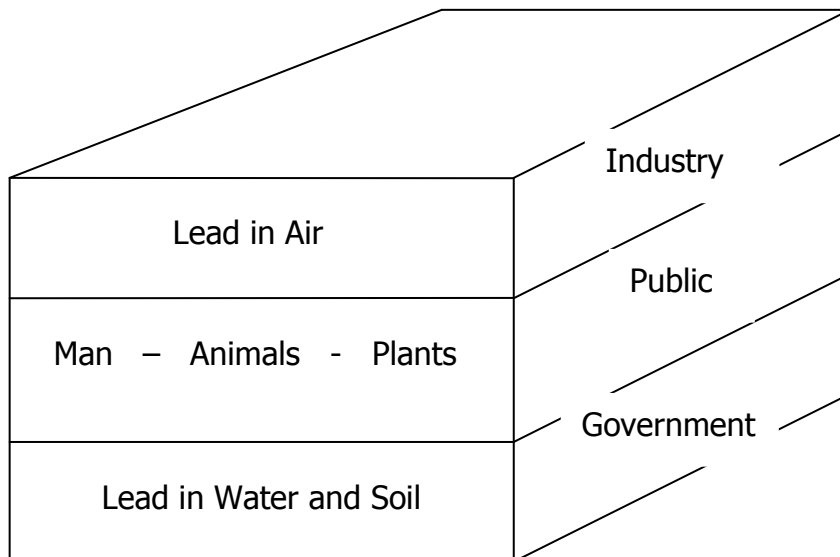


Fig.1 *The Lead Sandwich*