Children and lead: some remaining doubts

Three questions about environmental lead and its possible effects on children have perplexed and worried paediatricians (and the wider public). Firstly, does an increased amount of lead in the body, short of that necessary to cause frank symptoms and signs of lead poisoning, nevertheless have adverse effects on children's intelligence and behaviour? Secondly, what is a safe amount of lead in the blood? Thirdly, does the lead from petrol fumes make an important contribution to the body lead of urban children, sufficient to have adverse effects?

Two recent reports summarise the available evidence on these questions. *Lead and health* is the report of the DHSS Working Party on lead in the environment, which was set up 'to review the overall effects on health of environmental lead from all sources, and in particular, its effects on the health and development of children, and to assess the contribution lead in petrol makes to the body burden'. The report is careful, clear, and thorough—the working party studied nearly 3000 published papers. The second important publication is a review by Professor Michael Rutter of the evidence that raised lead levels may affect children's cognitive functioning and behaviour. Rutter was a member of the DHSS Working Party, to whom his review was submitted as a working paper. He has a superb ability to analyse critically the conflicting data on a subject of this nature.

Since the subject of children and lead has become so controversial, we have decided to summarise some of the important conclusions of these comprehensive and authoritative reports, to indicate some remaining areas of doubt, and to encourage our readers to study the reports themselves.

Do small increases of body lead affect intelligence and behaviour?

'The possibility that exposure to lead at levels below those producing symptoms of poisoning can result in impaired intellectual functioning and related problems' (in children) was one of the main concerns of the working party, and Rutter's review is entirely devoted to this question. It is difficult to answer, for methodological reasons which are well dealt with in both reports. There are problems in measuring chronic exposure to lead, and in measuring behaviour; and children with high body lead contents frequently suffer from other adverse social and environmental circumstances whose effects may be difficult to disentangle from those attributed to lead. These difficulties have long been recognised, and because of them most of us were sceptical of the numerous reports suggesting that moderately raised body lead levels caused intellectual or behavioural impairment, until the study of Needleman and his Boston colleagues appeared. This showed that children with relatively high amounts of lead in the dentine of their shed deciduous teeth—which was thought to be a measure of long-term lead exposure—had slightly lower intelligence and more disturbed behaviour than children with less lead in their teeth. Both the working party and Rutter gave particular attention to the Boston tooth study, confirming that it is the most important evidence so far of a possible link between moderate body lead burdens and neuropsychological impairment, but indicating that it is not free of methodological problems. It is also difficult to equate the dentine levels in this study with the more familiar measurements of blood lead levels.

Neither report is able to draw a firm conclusion about whether moderately increased body lead burdens, insufficient to cause obvious poisoning, may cause psychological impairment. If the numerous studies are taken at face value, ignoring the methodological criticisms, more of them suggest that there is such an adverse effect than not. The working party have summarised this in a diagram which shows that 13 out of 19 matched studies of groups of children suggested some lowering of intelligence in the higher lead group, usually by about 5 points in IQ. On the other hand, both Rutter and the working party emphasise that these studies do not show the dose/effect relationship which might be expected if the increased lead burden were really having adverse psychological effects.

Though the working party has evidently drawn heavily on Rutter's paper, one detects a slight difference of emphasis in the way their conclusions are put, Rutter being occasionally more inclined to
acknowledge a possible effect of lead where the working party will simply say 'not proven'. Both advocate more and better research, the working party pointing out that most of the relevant studies have not been carried out in the UK.

Do UK children have too much lead in their blood?

The working party give data on current blood lead levels which are generally reassuring. Since an EEC directive in 1977, blood lead surveys were carried out in the UK in adults from urban areas and in children particularly likely to be exposed to lead. (One wonders, incidentally, if the part of this very useful study which made blood lead measurements on children required ethical approval. An EEC directive should not override the need for this.) Most of these surveys (including those among children of lead workers or living near very busy roads) showed blood levels well within the distribution regarded as acceptable by the EEC directive,* the great majority being below 20 μg/100 ml (0·97 μmol/l). This suggests that there is no cause for alarm about inapparent lead poisoning being widespread among our children. However it does not necessarily justify complacency about environmental lead. If the studies referred to in the previous section really do indicate an association between moderately increased body lead and impaired psychological functioning—and this is certainly open to doubt—it is difficult to define a safe upper limit for blood lead. The Boston tooth study might suggest that it is even lower than 30 μg/100 ml (1·45 μmol/l). The opening paragraph of the working party report indicates that lead is a toxic substance which has no known physiological function (the Archives has yet to report the first case of hypoplumbism), and therefore the less lead there is in the blood the better.

What should we do about lead in petrol?

Lead is added to petrol to increase its octane rating—that is, its efficiency as a motor fuel. In 1978 the petrol used in the UK contained 10 300 tonnes of added lead, of which about 7000 tonnes were emitted in exhaust fumes. The question of whether this lead may damage children's health and behaviour has been one of the most bitterly controversial aspects of the lead debate.

The working party analyse with great care and much numerical detail the various contributions to the daily intake of lead for people of different ages and in different environments. The calculations suggest that the average city child derives 55–95% of his blood lead from food, 0–40% from water, and only 3–10% from the air. Inhaled lead therefore appears relatively unimportant. Few people are likely to be exposed to long-term average concentrations of lead in the air above 1 μg/m³, and a typical urban background value would be about 0·5 μg/m³. The EEC directive regards levels up to 2 μg/m³ as acceptable, and studies suggest that exposure to an average concentration of 1 μg/m³ in the inspired air only raises blood lead levels by about 2 μg/100 ml (0·1 μmol/l).

However, there are again some doubts which the working party report may leave in the mind of the nonexpert reader. For most people in the UK, the main source of lead intake is food. The working party does not identify where this lead comes from, and though they do not believe airborne lead makes a substantial contribution, by settling on growing crops or on soil, they acknowledge the lack of quantitative data on this point. In the absence of such data, some may continue to suspect a connection between the 7000 tonnes of lead which disappear each year from petrol fumes to an unknown destination, and the lead which appears in food from unidentified sources to constitute the major human intake of this substance. There are similar uncertainties about the sources of lead in indoor and outdoor dust, which may be ingested from children's hands even in the absence of obvious pica.4

A further cause for doubt is that two studies cited by the working party—from New York and from Australia—suggest a closer relationship between the amount of lead in air and the blood lead levels of children than would be expected from the calculations quoted above.

Though the working party's answers to the 3 questions asked in this Editorial are reasonably reassuring, in each case there are some remaining doubts. Many, relying as much on instinct as on evidence, will share the view of the speaker5 at the 1980 BPA Meeting who asked 'Isn't it time we took the lead out of petrol?'. The working party must be right to give priority to removal from old houses of paint containing lead, and to a reduction of lead in tap water in those areas where plumbosolvent water is drawn through lead pipes. However, their conclusion that steps should be taken progressively to reduce emissions of lead into the air from petrol also deserves wide paediatric support.

References


*The 1977 EEC directive required that 50% of the group examined should have blood lead levels not exceeding 20 μg/100 ml, 90% levels not exceeding 30 μg/100 ml, and 98% levels not exceeding 35 μg/100 ml.
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Alexander F W. Isn’t it time we took the lead out of petrol? Paper read at the British Paediatric Association Annual General Meeting, York, 15–19 April 1980.