Pollution and People

Is low-level lead pollution dangerous?

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Present-day Americans are said to have about 500 times more lead in their skeletons than Peruvians of 1800 years ago.¹ Lead pollution has become widespread in developed countries, especially from the use of lead in industry and in petrol and from its contamination of canned foods and some water. Nevertheless, lead poisoning, which was first described by the Greek physician poet Nikander in the second century BC,² is very rare, being associated with high blood concentrations over 3.9 $\mu\text{mol/l}$ (80 $\mu\text{g}/100\text{ ml}$), or occasionally over 2.9 $\mu\text{mol/l}$ (60 $\mu\text{g}/100\text{ ml}$). The current debate centres on whether concentrations usually considered safe—under 1.9 $\mu\text{mol/l}$ (40 $\mu\text{g}/100\text{ ml}$)—may in fact cause subtle neurological and behavioural abnormalities.

Problems of method

Young children are at greatest risk from the effects of lead (see box). Children who recover from lead encephalopathy are left with cognitive deficits and abnormalities of behaviour.³ It is reasonable therefore to look for lesser impairments after a less extreme exposure to lead. Virtually all the studies in children, however, contain some defects, failing either to show or to exclude an effect without reservation.

One source of doubt arises from the measurement of lead.⁹ The WHO recommends the use of blood lead estimations for population studies, given that exposure is reasonably close to "steady state."¹⁰ But this cannot always be assumed. For example, a child may have acquired a considerable burden of lead as a toddler, particularly if he indulged in pica or even if he just played in a lot of dust and dirt; but his blood concentrations—reflecting current exposure—a few years later, perhaps after damage has been done, may be unremarkable. On the other hand, a single blood lead estimation may reflect a temporary and perhaps unimportant rise. Thus, in theory at least, studies based on blood lead may either miss real associations or suggest spurious ones—possibly the former is more likely. The ideal would be serial blood tests. Since lead accumulates in the skeleton, estimates of lead in the dentine of shed teeth may provide a better index of the body burden, including past exposure.⁹ Even here there are problems of method, including the question of whether concentrations in different teeth are comparable; but the method is promising. Hair would be an even more accessible material for assay but at present the data on lead in hair are difficult to interpret.⁹

Another set of problems concerns the assessment of intelligence and behaviour. For example, subtle cognitive effects might be missed unless a range of rather sophisticated tests is carried out; but this may not be feasible in a large population sample⁹—and how can such things as ability to concentrate and

The action of lead in the body

Lead is a neurotoxin that may cause encephalopathy and death. The other main target organs are the erythroid cells of the bone marrow and the kidney. It is both ingested and inhaled, being distributed via the red cells throughout the soft tissues and accumulating in the skeleton, though much is excreted. Biochemically, the action of lead includes inhibition of enzymes concerned with haem synthesis.³⁻⁵ It reduces concentrations of δ -aminolaevulinic acid dehydratase even at a blood concentration of 0.97–1.9 $\mu\text{mol/l}$ (20–40 $\mu\text{g}/100\text{ ml}$); but how important this is clinically is not known.³ Biochemical and morphological abnormalities of the brain can be produced by giving newborn animals lead in amounts not causing obvious toxicity.⁵ But, as with all animal work, extrapolations to man must be speculative. Young children are at the greatest risk. Not only is the developing brain especially vulnerable to toxins but, while adults absorb only 5–10% of their intake of ingested lead, children absorb up to half.³⁻⁵ The proportion of lead actually retained in the body varies widely between individuals; and both absorption and retention are affected by diet. In particular, a deficiency of calcium may increase the absorption, retention, and toxicity of lead; iron, copper, zinc, and selenium deficiency and an excess of fat may also have an adverse effect.⁴⁻⁶ Lead has recently been found to have a considerable affinity for fetal haemoglobin (HbF).⁷

perseverance be reliably compared in large groups? Again, assessments of behaviour by an outside research worker may not be sufficiently comprehensive; but parents' and teachers' assessments have their own limitations.

Perhaps the most difficult problem, however, is the matching of groups to control for social and other factors—the problem of "cause, consequence, or correlate," as Rutter puts it.⁹ Retarded and disturbed children, for example, are especially prone to pica and their raised lead concentrations could be the result rather than the cause of their impairment. Again, low intelligence and attainment tend to go with the poor environment and social deprivation that hold so many risks of excessive lead exposure, including traffic and industrial pollution, dust and dirt, old and peeling high-lead paint, lack of salubrious places to play, and lack of supervision, with a poor diet to aggravate the effects. A current British study, using dentine lead estimations and elaborate psychological testing, is attempting to include more detailed assessments of social and family factors than has been usual.

Some studies in children

The studies of low-level lead exposure in children fall into five main categories: psychological investigations in children with raised lead concentrations without signs of toxicity, and in children treated with lead chelation; studies of lead concentrations in mentally handicapped and disturbed children; investigations of children living near lead smelters; and studies of tooth lead in groups of the general population.⁹ I refer here to just a few of the many papers to give some idea of the issues and the uncertainties.

CHILDREN WITH KNOWN LEAD EXPOSURE

Among the studies in children with moderately high lead concentrations, the work of de la Burd and Choate is interesting in including a follow-up.¹¹ Sixty-seven children who as toddlers had had either blood concentrations of at least 1.9 $\mu\text{mol/l}$ (40 $\mu\text{g}/100\text{ ml}$) or "lead lines" in their long bones on radiography had psychological tests at 4 and 7 years. Compared with 70 generally well-matched control children, the most striking difference at 7 years was in adverse "behaviour profile" ($p=0.001$); differences in IQ and visual motor and fine motor co-ordinations found at 4 years were also still present. At 7 years no difference in blood lead concentrations between the groups remained but mean tooth lead concentrations diverged widely. The authors point out that this suggests permanent damage by lead rather than continuing slight toxic effects. The main weakness of the study is that it did not control for pica,⁹ which had given rise to lead ingestion in the test group and which might have been the result of prior psychological abnormality rather than causing it. The authors, however, give reasons for doubting this.

One of the best studies in this group is that of Rummo *et al.*¹² Children with lead encephalopathy and with lesser long- and short-term lead exposure were compared with matched controls for psychological and neurological deficits and hyperactivity (90 subjects in all and 45 controls). The two intermediate groups were intermediate in the tests—that is, inferior to the controls; the differences fell short of significance but were consistent in their direction.

Rutter concludes in his review of the various studies of this type that blood lead concentrations persistently above 2.9 $\mu\text{mol/l}$ (60 $\mu\text{g}/100\text{ ml}$) were probably associated with a three- or four-point reduction in IQ, with doubtful pointers to adverse effects at 1.9–2.9 $\mu\text{mol/l}$ (40–60 $\mu\text{g}/100\text{ ml}$).⁹

CHILDREN TREATED WITH LEAD CHELATION

The psychological effects of removing lead from the body by chelation have been studied in small groups of children—for example, by David *et al* with reference to hyperactivity.¹³ Some striking improvements have been seen, but cause and effect cannot be taken for granted in view of the small numbers and limitations of study designs.⁹

MENTALLY HANDICAPPED AND DISTURBED CHILDREN

Some studies of mentally handicapped children (for example, by David *et al*¹⁴) have shown higher blood lead concentrations where the handicap is of unknown origin than where it has a probable cause or in control children; but, among other problems, the dividing line between unknown and probable cause is questionable. Once again, in some of the studies the pica giving rise to excessive ingestion of lead could reflect pre-existing mental abnormality; but work done in Glasgow, where high concentrations of lead are found in some drinking water, attempted to overcome this difficulty. In one study 17% of the retarded children had lived in houses with water containing over

38.6 $\mu\text{mol/l}$ (800 $\mu\text{g/l}$) lead but none of the control children; these were not perfectly matched, however.^{15 16} Also in Glasgow, phenylketonuria cards were used to compare retrospectively the neonatal blood concentrations in 41 mentally retarded and 36 control children.¹⁷ The means for the two groups were 1.25 and 1.02 $\mu\text{mol/l}$ (25.8 and 21.2 $\text{mg}/100\text{ ml}$), but there are some doubts about this study too—in particular about the analysis of samples.

CHILDREN LIVING NEAR LEAD SMELTERS

Children living near lead smelters have been the subject of six studies. One carried out by Landsdown *et al* in the East End of London found no correlation between IQ and either distance from the factory or blood lead concentration¹⁸; indeed, the high-lead group had slightly higher IQs—presumed to be the result of more socially deprived families moving into the outskirts of the area. But there are reservations about aspects of this study.^{9 19} A study at El Paso, Texas, compared matched groups of children with blood lead concentrations of 1.9–3.9 $\mu\text{mol/l}$ (40–80 $\mu\text{g}/100\text{ ml}$) and below 1.9 $\mu\text{mol/l}$ and found significantly lower performance IQ and slower wrist tapping in the former²⁰; but again there are unanswered questions about this work.⁹ Rutter concludes that four of the smelter studies are consistent with an IQ deficit of one to five points in children with blood lead concentrations of 1.9–3.9 $\mu\text{mol/l}$.⁹

CHILDREN IN THE GENERAL POPULATION—STUDY OF DENTINE LEAD

Probably the best study so far is the one by Needleman *et al* based on the dentine lead in teeth from Massachusetts school-children without clinical signs of lead poisoning (see box).²¹ IQ scores were about four points lower in the high-lead than low-lead group (a significant difference), and these children also did significantly worse in some of the other tests—notably those measuring attention and verbal and auditory processing.

A psychological study in children

In the study by Needleman *et al*²¹ dentine lead concentrations were measured in teeth from 2146 school-children. The 58 "high-lead" and 100 "low-lead" children in the highest and lowest percentiles of the total group (lead > 24 and < 6 ppm) were studied in most detail; about half had had blood samples screened a few years before and the means were $1.7 \pm 0.5\text{ }\mu\text{mol/l}$ ($35.5 \pm 10.1\text{ }\mu\text{g}/100\text{ ml}$) for the high-lead group and $1.1 \pm 0.3\text{ }\mu\text{mol/l}$ ($23.8 \pm 6.0\text{ }\mu\text{g}/100\text{ ml}$) in the low-lead group, with 2.6 $\mu\text{mol/l}$ (54 $\mu\text{g}/100\text{ ml}$) as the highest value in the former. These two groups had psychological and neurological tests and, together with the total group (divided into six subgroups on the basis of dentine lead concentrations), received teachers' ratings of behaviour. These covered such items as distractible, not persistent, disorganised, hyperactive, impulsive, easily frustrated, day dreamer, does not follow a sequence of directions, and low overall functioning. The high- and low-lead children were compared for physical, medical, socioeconomic, and family variables, and also for parental attitudes (on the basis of a questionnaire)—namely, aspirations for the child, home learning environment, attitudes to the school and to the child, and restrictiveness.

Child on verandah with peeling paint: old, poorly maintained houses with high-lead paint are a source of danger for children, especially if they develop pica. (Reproduced by courtesy of the Lead Industries Association, New York.)

Moreover, the teachers' ratings of behaviour were mostly significantly less favourable for the high-lead children, though they are described by Rutter as "crude" measures. The two aspects in which the groups were not significantly different were disorganisation and hyperactivity. Could these results be explained in terms of, say, home environment? Comparison of the parents' attitudes showed no significant differences between the two groups. The fathers' social class and level of education did differ significantly but these and several other variables were allowed for in the analysis. It is, however, difficult to say how well the groups were matched in more subtle respects. Pica was much commoner in the high-lead group but there was no evidence that it could explain the findings.⁹

In the group as a whole, for each item in the teachers' ratings the trend was for adverse ratings to increase with increasing lead concentration. Again, "not organised" and "hyperactivity" were the only two characteristics where the differences fell short of statistical significance. The significance of the trends was reported not in the paper but in a personal communication to Rutter⁹; the Conservation Society's report⁴ appears to assume that all were significant and incorrectly chastises the Lawther Committee²² for its comments on hyperactivity with respect to this paper. (In fact "hyperactivity" is a contentious concept,⁹ a label used more in the United States than in Britain; and there is some doubt about how widely it applies as a clinical entity.) For the other aspects of behaviour, the trends suggest a dose-related deterioration in general functioning with increasing dentine lead; but one reservation is that social factors were not looked at in the six subgroups.

This study provides some evidence of adverse effects on children's cognitive functioning and behaviour from concentrations of lead that have been considered safe, under $1.9 \mu\text{mol/l}$ ($40 \mu\text{g}/100 \text{ ml}$). The Conservation Society report concludes that the threshold of harm cannot be set higher than 5 ppm.⁵ This figure is suggested by the fact that the second of the six subgroups (with a dentine lead concentration not of 5 ppm but of $5.1\text{--}8.1 \text{ ppm}$) generally had more adverse teachers' ratings than the children with the lowest lead concentrations; but the inference is questionable, especially as we do not know how comparable the groups are socially. More generally, there are several uncertainties in the study—for example, somewhat insensitive psychological tests were used, and the large intermediate group was not studied in any detail. Commentators differ in the weight they attach to them. Rutter concluded that the findings are suggestive and the most impressive evidence so far; and that the reservations and queries, which apply even more strongly to most other investigations, are not sufficient to

invalidate the results. Nevertheless, he points out that the difference of about four IQ points is much the same as has been found after more severe exposure to lead, commenting that such similarity of deficit associated with blood lead concentrations of $1.9\text{--}3.9 \mu\text{mol}$ ($40\text{--}80 \mu\text{g}/100 \text{ ml}$) and under $1.9 \mu\text{mol/l}$ would be odd, though not impossible. He emphasises the importance of further studies.

Since then electroencephalographic (EEG) findings on some of the original two groups of children have been published,²³ and these are claimed to support the earlier findings. Computer analysis of the EEG data showed significantly increased low-frequency delta waves over the central parietal and occipital cortices and significantly decreased alpha waves over the parietal and occipital regions in the high-lead group (22 children, compared with the 19 low-lead children). The study suggested that the high- and low-lead children could be distinguished much more effectively by using the EEG and psychological data in combination than by using either alone ($p = 0.0002$).

CONCLUSIONS ABOUT EFFECTS OF LOW-LEVEL LEAD IN CHILDREN

The inferences drawn from the published work about children's susceptibility to the effects of modest concentrations of lead differ widely. The Conservation Society's report⁴ concludes that there is probably no threshold for toxicity and that neurotoxic effects are liable to occur above a lead concentration of 5 ppm in dentine (on the basis of the study by Needleman *et al.*²¹) and $0.24 \mu\text{mol/l}$ ($5 \mu\text{g}/100 \text{ ml}$) in blood (on the basis, for instance, of a study in young rats²⁴). The Lawther Committee²² was in no doubt that there were toxic effects above $3.9 \mu\text{mol/l}$ ($80 \mu\text{g}/100 \text{ ml}$) and that there was no convincing evidence of effects below about $1.7 \mu\text{mol/l}$ ($35 \text{ mg}/100 \text{ ml}$). But it was unable to come to a conclusion, from the evidence it had reviewed, about the effects of small or moderate amounts of lead, in the range $1.7\text{--}3.9 \mu\text{mol/l}$ ($35\text{--}80 \text{ mg}/100 \text{ ml}$)—though it recommended that action should be taken when a child had a concentration over $1.7 \mu\text{mol/l}$. Rutter's final conclusion⁹ is that, although the research findings are "somewhat contradictory," the evidence suggests slight cognitive impairment (an average of one to five points' reduction) and possibly behavioural difficulties as a result of persistently raised blood lead concentrations over $1.9 \mu\text{mol/l}$ ($40 \mu\text{g}/100 \text{ ml}$); with concentrations below this, he says, there are pointers to psychological risks but the evidence is inconclusive. He also observes that a reduction in IQ of five points for the general population would mean a doubling of the number of mentally retarded children, with an IQ under 70.

Studies in the infant and fetus

Lansdown and others point out that their negative results might have been different had they been able to study younger children.¹⁸ Special sensitivity to lead would be expected in the infant and fetus. Lead crosses the placenta and increases in the fetus through pregnancy; concentrations in cord blood at birth are correlated with those of the mother, usually being a little lower.²⁵ In a study of placental lead in stillbirths and normal births a much higher proportion of stillbirth than of normal placentas had concentrations over $0.72 \mu\text{mol/l}$ ($15 \mu\text{g}/100 \text{ ml}$).²⁶ But the authors suggest that this may reflect increased lead concentration in times of fetal stress. One report, however, suggests that maternal-fetal blood lead concentrations are inversely correlated with gestational age at birth—a finding claimed to imply that exposure to greater concentrations of lead in utero leads to earlier birth,²⁷ though there is too little information for this to be conclusive. Raised lead concentrations have also been found in the bones of stillborn and malformed infants, together with increased cadmium; but again this result must be regarded as tentative.²⁸

The Conservation Society report⁵ discusses animal studies

suggesting that raised (but not toxic) lead concentrations in the mother may cause learning difficulties and abnormalities in the offspring. In one of these, lambs whose mothers had had mean blood lead concentrations of 1.6 $\mu\text{mol/l}$ (34 $\mu\text{g}/100\text{ ml}$) throughout gestation themselves had a mean of only 1.2 $\mu\text{mol/l}$ (24 $\mu\text{g}/100\text{ ml}$) at 2-4 weeks; but later they needed significantly longer to learn visual discrimination problems than other groups of lambs.²⁹ The Lawther Report does not deal with fetal exposure but comments that extrapolation to human infants is difficult, particularly as the period of maximum brain growth varies from species to species.²² Nevertheless, the evidence points to the need for greater caution with pregnant women. Another possible pointer comes from the retrospective study in Glasgow referred to earlier.¹⁵ This found that mentally retarded children were significantly more likely than others not only to have had a high lead concentration in their drinking water in their first year of life but also to have mothers who had been exposed to such concentrations in pregnancy. The imperfect matching of groups, however, prevents a definite conclusion.¹⁶

Clearly more definite evidence is needed—the Lawther Report points out that studies of prenatal exposure to lead so far have not been of the standard of work on children.²² A case has indeed been made, since limits should be set for the fetus and infant rather than the older child, that such research would be preferable to further large-scale and expensive studies in children.³⁰

In my next article I will discuss the sources of exposure to lead.

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Has it been shown that blue light treatment in treating physiological jaundice in newborn infants reduces circulating bilirubin concentrations more quickly than without? Is it a matter of concern to reduce bilirubin concentrations quickly in these cases: can brain damage occur or is it simply the evident increased drowsiness of the jaundiced body that is the problem? How does blue light break down the bilirubin, and what are the chemical changes in the blood—in simple general practitioner terms? Are there optimum periods of phototherapy—that is, time spent under the lamps—and any known dangers?

Jaundice is common and usually transient in the newborn child requiring no specific treatment. Severe jaundice, particularly in the ill preterm infant, carries the risk of specific brain damage described as kernicterus. Unconjugated bilirubin is deposited mainly in the basal ganglia and hippocampus and may be associated with choreoathetosis, high-tone frequency deafness, and severe mental retardation. At the time of the intense jaundice the newborn infant may appear drowsy and floppy and may develop convulsions. Unfortunately, there are no definite limits as to what are acceptable degrees of jaundice, and what levels are associated with significant

risk of kernicterus. Phototherapy is often used to prevent severe jaundice and subsequent kernicterus. It has been known since 1958 that white light reduces the concentration of unconjugated bilirubin, but visible blue light of 460 nm wavelength is most effective in degrading this compound and controlling neonatal jaundice.¹ Unconjugated bilirubin is insoluble in water, and phototherapy degrades it to water-soluble diazo compounds of bilirubin, which may then be excreted in the urine. A small amount of biliverdin is also produced by this reaction, and these breakdown substances, although not fully analysed, appear to be non-toxic to the developing brain or other systems. There is a direct dose-response effect to phototherapy, and the more intense the light source the more effective will be the role of breakdown of unconjugated bilirubin. The infant's eyes are always covered to protect the retinas from the light. Several suggested hazards of phototherapy include increased loss of fluid, temporary malabsorption, and abnormalities of gonadotrophin secretion, and for these reasons the use of phototherapy must be strictly controlled.

¹ Raethel HA. Wavelengths of light producing photodecomposition of bilirubin in serum from a neonate with hyperbilirubinaemia. *J Pediatr* 1975;**87**:110-4.