THE EXPOSURE OF CHILDREN TO LEAD FROM INDUSTRY EPIDEMIOLOGY AND HEALTH CONSEQUENCES

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A B S T R A C T

Particulate lead of industrial origin may reach children through various routes to cause increased lead absorption and occasional lead poisoning, including direct absorption as the result of inhalation or ingestion of lead particles, the transplacental fetal accumulation in offspring of women exposed to lead in industry, and trans-generational or vertical from exposure of parental germ cells to lead before conception. The first two epidemiological mechanisms have been well documented, the third is more speculative. Recent work indicates that there may also be subclinical correlations to the hematologic and neurologic effects in children as well as anemia, encephalopathy, and chronic renal disease.

Introduction

Particulate lead has recently been rediscovered as a potential cause of lead exposure and lead poisoning in children. Much attention lately has been directed to particulate lead from automotive exhausts, which accounts for over 98 percent of atmospheric lead emissions in the United States.[1] This brief review will consider the exposure of children to particulate lead from industrial sources.

It has been recognized, at least since the investigations at Broken Hill, Australia in the late 19th. Century,[2] that children who live in the vicinity of lead smelters and other lead works may be exposed to high concentrations of particulate lead. Especially intense exposures may occur when climate and geography minimize opportunities for particulate dispersal. It is, therefore, possible to investigate in detail the routes by which children are exposed to particulate lead in such localities. Several of these epidemiologic mechanisms as well as the hematologic, neurologic, and possible renal consequences of such exposure will be discussed.

Exposure to Emitted Lead

An estimated 2,300 tons of particulate lead are discharged into the atmosphere each year by industrial sources in the United States.[1] Depending primarily upon particle size, this lead may either be inhaled or ingested by children living in the vicinity of such sources. Respiratory lead abosrption is inversely proportional to particle size and at least 30 percent of particles below 2 µg in diameter are retained and subsequently absorbed in the lungs. Between 10 and 30 percent of the 2 to 5 µg particles and almost none of those above 5 µg are retained;

however, larger particles may be swallowed and contribute to gastrointestinal absorption.[6]

Our group has recently had the opportunity to collaborate with the City-County Health Department in El Paso, Texas, in an investigation of childhood lead absorption around a lead smelter.[3] This smelter has emitted over 1,100 tons of lead into the atmosphere in the preceding 3 years.[4,5] Concentrations of lead in the soil adjacent to the smelter were high, decreased with distance, and reached background values at approximately 6.5 miles, Figure 1. Similar patterns were noted for lead levels in air and dust. Increased lead absorption in children, as defined by a blood lead level $\geq$ 40 µg/100 ml,* was found in 69 percent of the 1-4 year olds living within 1 mile of the smelter and in 27 percent of those living between 1 and 4.1 miles. Children living within a 1-mile radius were exposed to high levels of lead in the air.

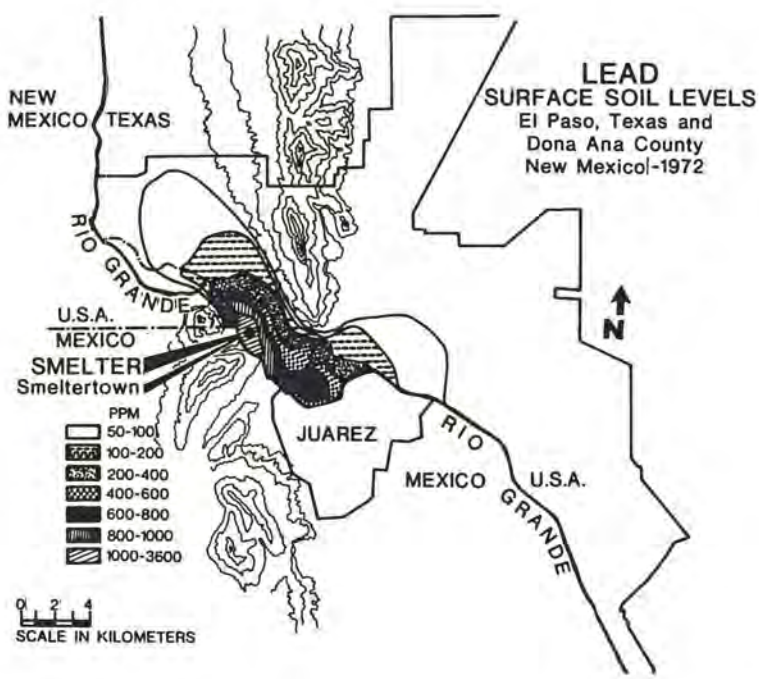

Figure 1 Map of El Paso, Texas and Dona Ana County New Mexico, showing lead surface soil levels

**A whole blood lead level of 40 µg or more per 100 ml is considered by the Surgeon General to represent increased lead absorption, while a confirmed level $\geq$ 80 µg per 100 ml represents lead poisoning.*[6]

In addition, a highly significant relationship was found in these children between lead levels in blood and levels of lead in household dust; a similar, though less striking relationship was noted between blood lead and soil lead levels, shown in Fig 2. These data were taken to indicate that chronic exposure to particulate lead deposited in air, dust, and soil by the smelter had been the major cause of increased lead absorption in the children living near the El Paso smelter.

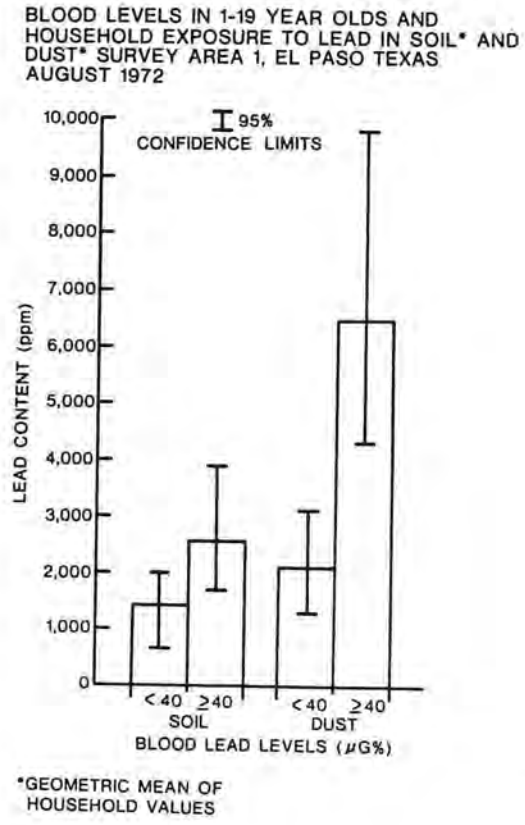

Figure 2 Blood Levels in 1-19 Year Olds and Household Exposure to Lead in Soil* and Dust* Survey Area 1, El Paso, Texas, August, 1972

Similar situations in which increased lead absorption and lead poisoning in children have been traced to industrial lead emissions as reported in Australia,[2] previously noted, in Toronto,[7] in Idaho,[8] in Montana,[9] in Italy,[10] in Britain,[11] and in Chile.[12]

Exposure of Lead Workers

Exposure of lead workers constitutes a second mechanism by which children may be exposed to industrial particulate lead. Recent surveys in Britain of blood lead levels in children living near lead smelters have shown that workers' children were more likely than their peers to have blood lead levels $\geq$ 40 µg/100 ml.[11] It was hypothesized that the workers had carried lead particles home to their children on their persons and clothing. The same phenomenon was observed 60 years ago in the wives of lead workers who, being unliberated, hand-washed their husbands' clothes and developed wrist-drop.[2] These situations are reminiscent of those noted elsewhere among the children of asbestos, beryllium, and trichlorophenol workers in whom mesothelioma,[13] berylliosis,[14] and chloracne[15] resulted, apparently from exposure to the parents' clothing. Such episodes can clearly be prevented by the enforcement of such work practices as changing and showering at the factory at the end of each work-day.

Exposure *in utero*

A third possible mechanism of exposure to industrial lead is exposure *in utero*. In the earlier literature, it was reported that antenatal lead exposure produced increased numbers of miscarriages and stillbirths in female lead workers, and that their live-born children were highly susceptible to neonatal convulsions.[2] High concentrations of lead have been demonstrated in the placenta, liver, and brain of infants born to lead workers.[16] It seems likely that such events would occur with less frequency in the United States today, given the general improvement that has occurred in industrial hygiene. Nevertheless, it does not seem reasonable, even today, to simply dismiss those older reports. Longitudinal studies of the outcome of pregnancy in female lead workers are needed to assess whether *in utero* exposure to lead remains a problem.

Exposure Before Conception

A fourth, still more speculative mechanism by which children may be exposed to industrial lead, is through exposure of their parents' germ cells before conception. While it would be impossible in female workers to distinguish this mechanism from exposure *in utero*, earlier studies of the offspring of males employed in the lead industry[2] showed fetal loss rates (miscarriages plus stillbirths in wives) as high as 50 to 80 percent. The relevance of those data is unclear; however, cohort studies in the families of lead workers would seem a reasonable means of assessing the current risk of this mode of exposure.

Health Consequences of Childhood Lead Exposure

Anemia, encephalopathy, and interstitial nephritis have for many years been recognized as the end-results of increased lead absorption in childhood. The major focus of interest today, however, is on the possibility that there may be subclinical antecedents to each of these easily recognized, but often irreversible, end-results.

Hematologic Consequences

The hematologic effects of lead absorption, both clinical and subclinical, have probably been the most thoroughly studied. It has been demonstrated that lead causes enzymatic inhibition of heme biosynthesis at several points in the metabolic pathway, with resulting increase in the blood levels of free erythrocyte protoporphyrin (FEP), increased urinary excretion of coproporphyrin and uroporphyrin, and decreased levels in red blood cells of the enzyme, delta aminolevulinic acid dehydratase.[1] This work has contributed to the emerging concept that there may really be no threshold for the metabolic and physiologic effects of lead on children, but rather that each atom of lead which is absorbed has the capacity to inhibit some enzymatic interaction.[17] Such a conclusion is suggested by extrapolation from data relating blood lead levels in children near smelters to levels of FEP, Figure 3.

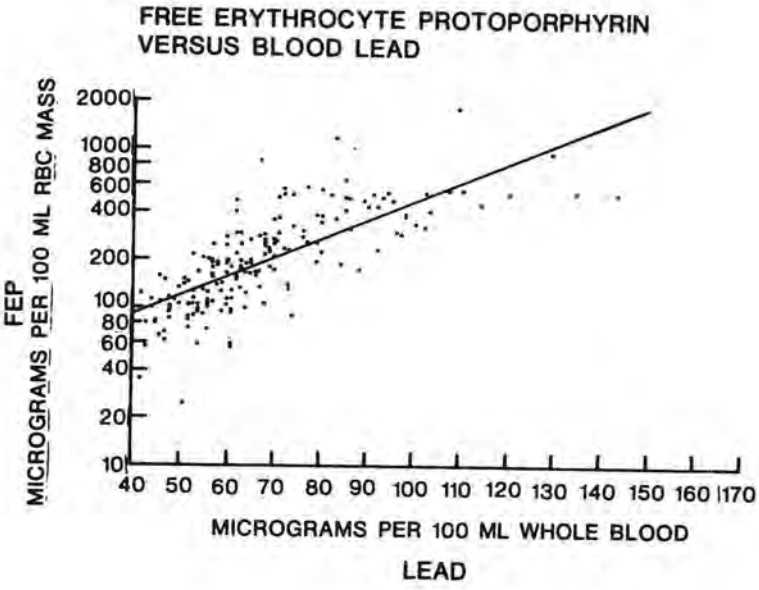

Figure 3 Free Erythrocyte Protoporphyrin *versus* Blood Lead in Children Living Near Smelters

Neurologic Consequences

Perhaps the most controversial topic today in the entire dialogue on lead in childhood is whether asymptomatic increased absorption of lead can cause subclinical alteration of neurologic or psychologic function. The central methodologic problem here, I suspect, is a lack of simple parameters which can be measured simply. While decreased enzyme levels

tell the whole story of increased lead absorption in the red blood cell, straight forward neurologic measurements, such as the well-documented abnormalities of peripheral nerve conduction observed in children with increased body lead burden,[18] do more than hint at the possible effects of lead on higher neural function. At the same time, attempts to measure higher function directly in children with increased lead exposure are subject to all the difficulties of physiologic adaptation and observer bias inherent in such testing. Yet, despite these difficulties, a trend may be emerging.

A wide variety of neurologic and psychologic abnormalities have been reported in recent studies of children exposed to lead. They have included slowing of motor nerve conduction velocity,[18] peripheral muscular weakness,[19] decreased fine-motor function scores on the Stanford-Binet intelligence test,[20] decreased scores on the McCarthy scale of intelligence in children,[21] and behavioral hyperactivity.[22] Also in our own blind studies of neuropsychologic function in El Paso,[23] we evaluated 46 asymptomatic children, ages 3-15 years, with blood lead concentrations of 40-68 µg/100 ml (mean 48 µg/100 ml) and 78 ethnically and socio-economically similar controls with levels <40 µg/100 ml (mean, 27 µg/100 ml). All children lived within 6.6 kilometers of the smelter and, in many cases residence there had been lifelong. Mean age in the lead group was 8.3 years and in the controls 9.3. Testing with Wechsler intelligence scales for school children and preschoolers (WISC and WPPSI) showed age-adjusted performance I.Q. to be significantly decreased in the group with higher lead levels (mean scores, WISC plus WPPSI, 95 vs 103, $p<0.01$). Children in all ages in the lead group also had significant slowing in a finger-wrist tapping test. Full-scale I.Q., verbal I.Q., behavior, and hyperactivity ratings did not differ.

In few of these instances have individual test results been frankly pathologic. Indeed, study design usually excluded children with such findings. Nevertheless, the gradient of abnormalities from exposed to unexposed children has been consistent and often statistically significant. Taken as a group, the results suggest that increased absorption of lead during childhood does cause a neurologic lesion, but that the lesion is diffuse and subtle. The histologic substrate may be a diffuse degeneration of neuroglia and other supporting cells, as has been suggested in several animal models.[24]

Of course, there have also been studies which have failed to demonstrate any neurologic or psychologic abnormalities in children with increased lead absorption. Two general sorts of criticisms may, however, be directed toward those studies: either the tests used, such as the Denver Developmental Evaluation, were relatively insensitive,[25] or the study and control groups were so constructed that there was little difference between them in degree of exposure to lead. For example, in the studies of Lansdown[26] in England and of McNeil and Ptasnik[27] in

El Paso of neuropsychologic function in children living near lead smelters, a high proportion of the children in the exposed groups had blood lead levels below 40 µg/100 ml and thus appear to have differed rather little from control subjects.

Renal Consequences

The effects of lead absorption upon the kidneys have been more difficult to study in children than the hematologic and neurologic effects. End-stage renal disease has been recognized in Australia among young adults who, as children, consumed high doses of lead in paint.[28] Also, acute, generally self-limited renal injury in the form of the Fanconi Syndrome, has been seen in a large proportion of children hospitalized in this country with acute lead encephalopathy. It is, however, not known whether chronic exposure to lower doses of lead in particulate form has any adverse effect upon the kidneys. Studies of this problem should perhaps be undertaken among groups of children who have lived near lead smelters or other lead works and have been chronically exposed to particulate lead.

Conclusions

To summarize, I have discussed several epidemiologic mechanisms by which children may be exposed to particulate lead of industrial origin, and I have described some of the possible ill consequences to health that might result from such exposure. Clearly, the best documented epidemiologic mechanism for the transfer of particulate lead from workplace to child is through exposure to airborne emissions or through exposure to lead workers. Of the health effects, the hematologic are supported by the firmest evidence. The rest should be taken with the cautions offered.

While any extrapolation is dangerous, it seems reasonable to extrapolate from the studies of lead absorption among children living near smelters to children living in cities and near highways who are exposed to particulate lead from automobiles. While children near roadways are generally exposed to lower levels of lead in air and dust than children living around smelters, there is no *a priori* reason to suspect that these children avoid inhaling and ingesting the particulate lead in their environment or that they escape the metabolic consequences of such absorption. The concept of a socially acceptable level of absorption must certainly be considered in a situation of low-level of exposure. That concept must, however, be weighed against the thought that the most deleterious effects of lead on children may be precisely those subtle but far-reaching and possibly irreversible aspects which have been most difficult to demonstrate.

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