Childhood Lead Poisoning: Man-Made and Eradicable

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Lead poisoning has been known about since antiquity. On many occasions, alarms about the disease have been raised and rapidly ignored. Recent data have shown that lead at doses not associated with symptoms are associated with lower IQ scores, impaired attention, speech and language deficits, and behavior disorders. The toxic threshold for lead has dropped regularly over time as new studies have shown effects at lower doses. Progress in eradicating lead poisoning has lagged behind scientific knowledge for a number of reasons. These include the beliefs that this is a disease only of poor children, that inferior mothering is a cause, and that it no longer is a public health problem, and the obstruction by the lead industry of attempts toward regulatory control. [PSRQ 1992;2:130–134]

A substantial portion of this nation's medical resources are directed at dramatic and expensive technological adventures. Far less notice is given to the prevention of disease through low-technology and effective but less spectacular activities such as immunizations, prenatal care, lifestyle education, and control of environmental pollutants.

The national response to childhood lead poisoning offers a clear example of this maldistribution of wealth and effort. Here is a disease recognized since antiquity, for which the steps to elimination can be prescribed, and whose appalling social costs become clearer each day. The reasons for the sluggish and hesitant approach to ending lead poisoning are as interesting as any of the biomedical and epidemiological riddles encountered in the study of the pathogenesis of the disease. Indeed, a full understanding of lead poisoning in the United States cannot stop with pathophysiology and pharmacology; it draws one inexorably to examine the economics and politics in which the illness is embedded.

Two thousand years ago, in the 2nd century B.C., Dioscerides, a Greek physician, noted that "Lead makes the mind give way." A little less than one hundred years ago, in 1897, childhood lead poisoning was described by A. J. Turner, a house officer at the Brisbane Children's Hospital in Australia [1]. Turner and his senior colleague, J. L. Gibson, noticed that children admitted with this diagnosis were nail biters or had just moved. By careful observation and logic, they established the source of the disease as

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white lead paint on the porches of the homes They then set out to have lead paint banned. This task brought them into direct conflict with commercial interests and, to their chagrin, some segments of the medical profession. In 1920, they succeeded in obtaining a ban on the use of lead in household paint. It took another 50 years for a similar law to be passed in the United States

The earliest cases of lead poisoning in childhood in the United States were reported in the first decade of the 20th century in Boston and Baltimore [2,3]. It was generally believed that, if a child did not die during the acute episode and if exposure ended, the child recovered without complications. In 1943, Randolph Byers followed up 20 children assumed to have recovered and reported that 19 had behavior disorders or learning disabilities. Byers asked, in that historic paper, how many cases of learning disabilities were in fact cases of missed lead poisoning [4]. The modern era of lead toxicology began with this report.

Until the late 1960s, a blood lead level of 60 μg/dl was considered the upper limit of normal for a child. Life was simple for pediatricians then; if a child had a blood lead level of 61 μg/dl, he or she was admitted to the hospital and chelated with calcium versenate. If the blood lead level was 59 μg/dl, the child was followed. When screening studies of ostensibly normal children showed that as many as 20% to 45% had blood lead levels in the range of 40 μg/dl, the question of silent lead toxicity was raised.

A few studies of silent lead exposure and children's cognition were published in the early 1970s [5–7]. Some showed an effect; some did not. The early studies generally evaluated small samples, used crude measures of outcome, classified children by blood lead level, and had limited control of other possibly confounding variables. I was interested in looking at children in the first grade, whose exposure was thought to have ended. The residence time of lead in blood is short and could misclassify children. Lead goes to bone, and it occurred to me that the shed deciduous tooth was a good way to estimate bone lead. Indeed, tooth lead was high in children who had been treated for lead poisoning, in children where lead poisoning is common, and in children who lived near a lead plant.

We collected 3,335 shed teeth from about 2,500 children from Somerville and Chelsea, Massachusetts, and classified children by tooth lead. We compared the children in the highest end of the distribution with those in the lowest end after controlling for covariates such as socioeconomic status, mother's IQ, family size, and mother's and father's education [8]. High-lead children had lower IQ scores, poorer speech and language function, and longer reaction times at long delay intervals (a measure of attention). Most interestingly, when we asked teachers to evaluate all of their children on an 11-item forced choice test, we found that, as tooth lead increased, the rate of poor function on each item increased in a dose-dependent function with no evidence of a threshold (Fig 1).

These data were soon to be followed by a number of studies from the United States, the United Kingdom, Europe, New Zealand, and Australia showing effects of lead at similar or lower doses [9]. In response to this, the Environmental Protection Agency (EPA) and the Agency for Toxic Substances and Disease Registry stated that the effect level for lead toxicity was between 10 and 15 μg/dl and might be lower [10,11].

Lead crosses the placenta and can be measured in the umbilical cord blood before an infant has a chance to breathe or ingest very much lead. We measured umbilical cord blood lead levels and showed that umbilical cord blood lead was related to the risk of minor malformations [12]. When we followed these children at six-month intervals, we found that intrauterine exposure was related to IQ score as late as two years of age, after adjustment for other factors [13]. Investigators from Cincinnati and Australia found similar outcomes [14,15]. A few studies failed to find a relationship. The Cincinnati group also found that intrauterine lead exposure and exposure during the first year of life were associated with decreased linear growth [16].

In 1990, we followed the children that we had classified by dentine lead level in 1979 into adulthood. The findings were troubling: an elevated dentine lead level in 1979 was associated with a sevenfold increase in the risk for failing to graduate from high school and a sixfold increase in the risk for reading disability. Subjects who had high lead levels also had lower vocabulary scores, lower class standing, and disturbances in fine motor function [17]. The impact of early, asymptomatic lead exposure is permanent and associated with life success (Figs 2 and 3).

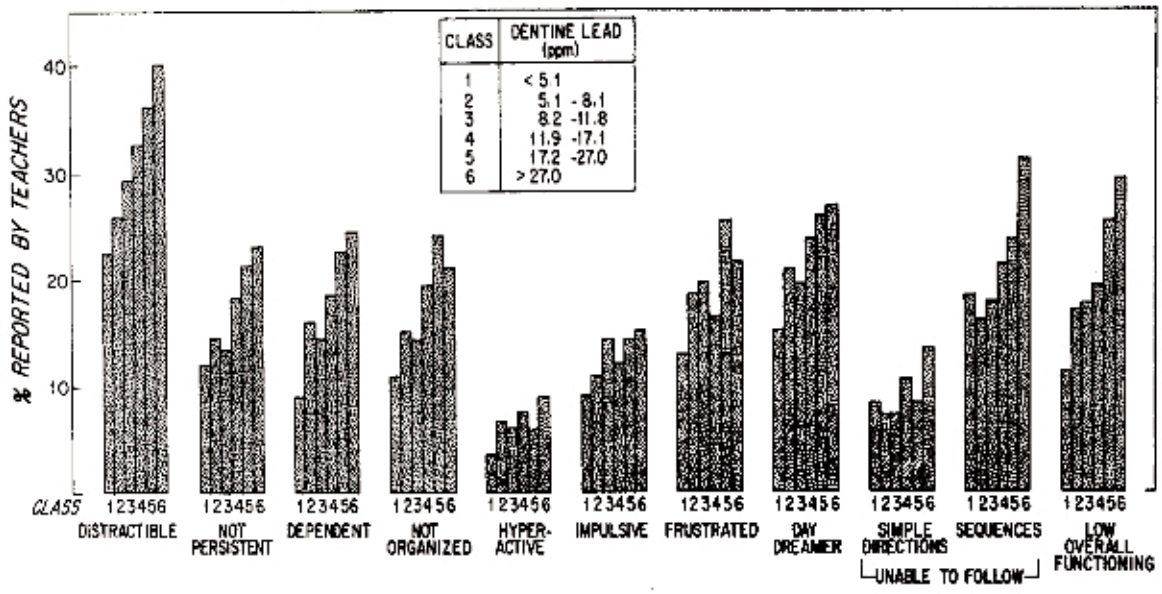

FIGURE 1. Teachers' Rating Scale. Distribution of teachers' ratings of 11 classroom behaviors. The group sizes were chosen to achieve a symmetrical distribution around the median (N = 2146 subjects) Reprinted with permission from the *New England Journal of Medicine* 1979;300:689–695.

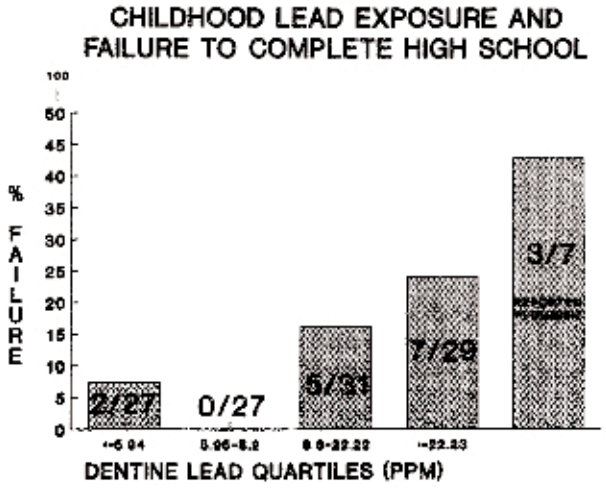

FIGURE 2. The proportion of subjects who failed to graduate from high school classified by past exposure to lead. Asymptomatic subjects are classified by lead quartile. Seven of 10 subjects who were reported to have had clinical plumbism are also displayed. Reprinted with permission from the *New England Journal of Medicine* 1990;322:83–88.

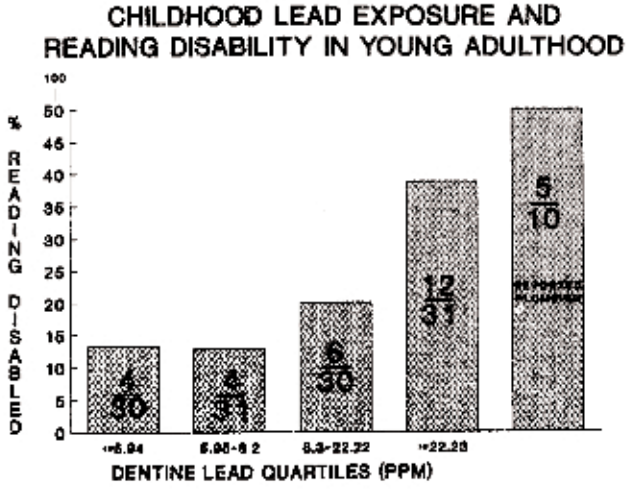

FIGURE 3. The proportion of reading-disabled children classified by past exposure to lead. Asymptomatic subjects are classified by lead quartile, and 10 children with a past history of clinical plumbism are displayed. Reading disability is defined as reading at two or more grades below expected. Reprinted with permission from the *New England Journal of Medicine* 1990;322:83–88.

With the accumulation of these studies and an impressive body of elegant research in animals that supported the epidemiological data, attitudes in the Public Health Service (PHS), and certain parts of the EPA, toward the definition of lead toxicity began to change. In 1972 the PHS definition of lead toxicity in children was 40 μg/dl. This was revised downward to 10 μg/dl in 1991 by the Centers for Disease Control (CDC) after review of the current studies. The prevalence statistics are impressive. Estimates from the Agency for Toxic Substances and Disease Registry are that 17% of all American children have blood lead levels in excess of 15 μg/dl. The estimate for economically favored white children is 7%, for poor whites 25%, and for economically favored black children 25%. For black children in poverty,

the estimate is that 55% have blood lead levels exceeding 15 μg/dl. This means that half of the black children in poverty enter the first grade with a potentially handicapping condition. This is why Dr. Louis Sullivan has declared that lead poisoning is the "most serious environmental disease for American children" [18].

In 1991, Assistant Secretary of Health James Mason, after study of the question, commissioned the CDC to draft a plan to eliminate the disease. In February 1991, the CDC announced the Strategic Plan to Eliminate Childhood Lead Poisoning [19]. This plan calls for increased surveillance, decreased entry of new lead into the environment, and large-scale abatement of housing. It also includes a cost-benefit analysis of total housing abatement. The document shows that the social costs for lead exposure of medical care and remedial education are conservatively placed at $4,600 per lead-exposed child. The net benefits to society for abating all of the housing is estimated at $28 billion. CDC characterizes this as a conservative estimate, since it does not include such costs as health effects other than central nervous system damage and excludes benefits such as increased employment.

In 1991, the federal legislature began to respond to the new information. Four bills in Congress, two in the Senate and two in the House, were written. These bills would decrease the nonessential uses of lead, encourage recycling, and step up abatement and case finding. Increasing numbers of lawsuits against landlords and insurance companies have been settled for large sums. In Massachusetts, a suit was brought against six major paint companies, claiming that there was documentary evidence that the paint companies were aware of the toxic properties of their product in the 1920s, but covered it up. Rapidly, similar suits were filed in Philadelphia, New York, and New Orleans.

In response to these new threats, the industry responded in traditional style. Representatives claimed that the evidence on the health effects of lead was unclear and imposed their idiosyncratic interpretation on the body of studies published to date.

They particularly relied on the 1981 study of Dr. Claire Ernhart, an industry grantee, who reported in 1979 that low-level lead exposure was associated with lowered IQ scores [20]. In a 1981 paper, Ernhart followed up 63 of the original 80 subjects, who were then in the first grade. She measured the association among preschool blood lead level, school-age blood lead level, and IQ score [21]. Ernhart found that school-age level was significantly related to IQ but dismissed that as a chance finding. Preschool blood lead level was dismissed as nonsignificant. The actual P value of the association was $P = 0.08$. The weak power in this study to find an effect of this size is readily apparent; with 63 subjects, the power to find an effect is 0.52. This study had a 50-50 chance to miss an effect at the 0.05 level. This tactic, of using studies of weak statistical power or inferior design to argue that there is no effect, is characteristic of the lead industry.

Another characteristic method employed by the industry is to personalize their attack on investigators who argue that lead is hazardous. When Byers published his paper in 1943, he was visited by lead industry representatives who threatened him with a $1 million lawsuit [22]. When Clair Patterson wrote his landmark paper on the worldwide contamination of the biosphere with lead [23], the industry attempted to block its publication and have him removed from the faculty at California Institute of Technology. Most recently, lead industry spokespersons have alleged to the Office of Science Integrity of the National Institutes of Health (NIH) that my studies violated the canons of scientific integrity. This is the second time that this has been attempted. In 1983, during the drafting of the Air Lead Standard (which eventually determined that lead would be removed from gasoline), these charges were investigated by the EPA. After thorough investigation of my data and reanalyses carried out under the EPA's suggestions, the Clean Air Science Advisory Committee, the EPA's highest advisory group, recommended the following statement, which was published in the final version of the Air Quality Criteria for Lead, 1986 [24]:

> A pioneering general population study was reported by Needleman et al (1979). . . . Significant effects ($P < 0.05$) were reported for full scale WISC-R scores, WISC-R verbal IQ scores, for 9 of 11 classroom behavioral scale items, and several experimental measures of perceptual motor function. . .
> Reanalyses carried out in response to the Committee's recommendations have been reported by Needleman (1984), Needleman et al (1985) and U.S. EPA's Office of Policy Analysis (1984) as confirming the published findings on significant associations between elevated dentine lead levels and decrements in IQ, after cor-

recting errors in data calculations detected in earlier published analyses and using alternative model specifications that incorporated better control for potentially confounding factors.

Eight years later, under new legislative assault, the industry has revived these charges of lack of scientific integrity.

Industrial activity explains some but not all of the reasons that it required 50 years after Australia's actions for the United States to pass a lead paint poisoning prevention act. There are other factors that deserve scrutiny. The lack of interest of the academic medical community is important. This disease simply does not enjoy the fascination or prestige of molecular biology or transplant surgery, and it is at best a break-even financial proposition for hospitals. Many academic pediatric departments give only lip service to lead poisoning, and some do not even bother to offer this. Private pediatricians rarely screen for lead. Many still believe that this is a disease of poor minorities alone, and some go further to blame the mother's care. The poor rarely receive their share of resources or priorities, and, once the mother has been blamed, authority is separated from the obligation to act. Many believe that, with the passage of the lead paint act, the problem was solved, and, with the removal of lead from gasoline, it has disappeared. At the same time, confronted with data showing that perhaps 30 million homes have lead paint, many say that the problem is too big to handle. This paradox can exist within the single person: "the problem is gone, why bother; the problem is too big to handle."

The removal of lead from the human environment presents an extraordinary opportunity to address a number of important problems. Lead paint is not a problem for poor children alone, but the poor do see much more lead. The overabundance of lead coexists in the same places where there is a shortage of employment and of decent housing. A comprehensive abatement plan could provide jobs where they are most needed and return houses to decency.

Only through analysis of these false myths, and by confronting the vested forces who have never missed a chance to obstruct the reduction of lead in the environment of children, can we hope to end this silent killer of children's brain cells, truncator of their futures, and continuing hazard to the lives of babies yet to be born. ❧

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