Commentaries

The Persistent Threat of Lead: A Singular Opportunity

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Abstract: Recent data have demonstrated health effects of lead in children at doses previously believed to be harmless. Data from epidemiological studies in many countries, and from experimental studies of animals given lead, demonstrate psychological impairment at blood lead concentrations of 0.5–0.7 μmol/L. Current estimates are that 17 per cent of American children (3–4 million) exceed the level of 0.7 μmol/L. Lead exposure is not a problem for urban poor children alone, but inner-city minorities have a higher rate of exposure. The overabundance of lead coexists in the same area with two serious shortages: affordable housing and jobs. It is argued that a program to train unemployed inner-city residents in safe de-leading, while expensive, makes hygienic, economic, and common sense. (*Am J Public Health* 1989; 79:643–645.)

Introduction

Childhood lead poisoning is a man-made disease. Unlike other important illnesses, e.g., AIDS (acquired immune deficiency syndrome), cancer, or Alzheimer's Disease, its nature is clear. Few mysteries surround it; the greater enigma is why lead has been permitted to persist in the human environment in the face of a mass of convincing data about where it is, what it does, and what is needed to get rid of it.

A number of reasons exist to explain the sluggish pace of control. Many clinicians believe that lead afflicts only poor American children and that, in some way, inferior parental care is at the root.[1] While the poor and minorities have much higher rates of lead exposure, the middle class does not escape. The National Health and Nutrition Examination Survey II (NHANES II) estimated that 4 per cent of all American children had blood lead levels above 1.5 μmol/L.[2] For poor Blacks, the rate was 18 per cent. Equally pervasive is the belief that legislation banning the use of lead in household paint, and the removal of lead from gasoline have virtually eliminated the major sources for humans. While it is true that newly made paint is almost lead-free, many homes have paint applied before 1970. The removal of lead from gasoline has resulted in a 99 per cent decrease in the tonnage of alkyl lead inserted into our atmosphere. At the same time, newer studies of lead's effects have shown central nervous system, growth, endocrine, and perceptual changes at levels previously held to be safe.

Less than a year ago, a group of studies reported at the Sixth International Symposium on Heavy Metals in the Environment[3] painted a comprehensive picture of the broad band of lead toxicities. The rate of acquisition of new knowledge in this area is jarring; the newer studies have both lowered the perceived threshold for observed health effects and demonstrated toxic effects in new areas. Only 25 years ago, the toxic level of blood lead was 2.9 μmol/L: some hospital laboratory slips still list this as the normal level. The current definition from the Centers for Disease Control of the lead toxicity threshold is 1.2 μmol/L. Careful epidemiological studies in Scotland, Denmark, and Greece[4–6] have shown IQ changes in children at blood levels as low as 0.5–0.7 μmol/L. These studies have identified and controlled for the relevant non-lead covariates that are potential confounders. The populations sampled were middle class children. The difference in mean IQ scores between exposed and control groups generally is about 4–7 points.

This 4–7 point difference in means has been taken by some as a small effect. This is deceptive. The cumulative frequency distribution for IQ, typical for many distributions, is sigmoid. When cumulative distributions between groups are plotted and compared, a shift in the curve resulting in a difference in medians of 6 points results in a four-fold increase in the rate of severe deficit (IQ <80). In addition, the same shift in distribution truncates the upper end of the curve, where superior function is displayed, by 16 points.[7] This means that 5 per cent of lead-exposed children are prevented from achieving truly superior function (IQ>125). The costs of this effect at the high end of the distribution have received no attention; they may be extraordinarily important to our society.

Adding conviction to the conclusions of these epidemiological investigations are behavioral studies in experimental animals at doses comparable to those experienced by children. Among the most compelling work is that of Rice and colleagues.[8] These investigators gave lead to monkeys at two low-dose levels from birth until about 100 days of life. Peak blood levels in the animals were 1.2 μmol/L and 0.7 μmol/L for the experimentals, and 0.1 μmol/L for controls. Steady state levels were 0.7, 0.6, and 0.1 μmol/L, respectively. When later evaluated as juveniles and young adults, both the high- and mid-level exposure groups showed statistically significant deficits in discrimination learning. The nature of the deficit clearly was a disturbance in attention. These findings stand in close parallel to the reports in children of impaired reaction time under varying intervals of delay, and teachers' reports of increased distractibility, impulsiveness, aggressiveness, and hyperactivity in the classroom. These latter findings, first reported in 1979,[9] have been replicated in England and Greece.[6,10]

One question that has been raised about the observed relation between lead and behavioral deficit is whether the direction of causalty is reversed, and lead is simply a marker for preexisting impairment. Do developmentally impaired children mouth more lead-containing substances? That pos-

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sibility has been convincingly refuted by studies of lead exposure before birth (as measured by concentrations in the umbilical cord blood), and later intellectual development. These reports clearly show that umbilical cord blood levels are predictive of Bayley Developmental scores at 6, 12 and 24 months of age.[11,12] The effect level is 0.5 μmol/L.

Lead's toxic expression is not limited to the central nervous system. Intrauterine and early infant exposure to lead at low dose interferes with growth of the infant during the first year of life. Blood lead levels are inversely correlated with linear height and chest circumference.[13] Hearing deficits have been measured in association with blood lead levels; no threshold was found.[14]

These data draw a convincing picture of lead's broad impact on children's intelligence, growth, ability to hear and perceive language, and to focus, maintain, and shift attention. They certify, to the satisfaction of all but representatives of the lead industry, that lead is a potent, versatile, and widely distributed toxicant. In recognition of this new data, a recent paper by US Environmental Protection Agency officials set the defined threshold for neuropsychological impairment in infants and children at 0.5–0.7 μmol/L.[15] A recent report to Congress by the Agency for Toxic Substances and Disease Registry, written by Mushak and Crochetti, a toxicologist and a demographer, using NHANES II data, estimates the number of children who have blood levels above 0.7 μmol/L to be 3–4 million. Like most man-made risks, this one is not borne equally by all strata of society. Approximately 17 per cent of White children above the poverty level have blood levels in this range. But, for poor Black children, the rate is 55 per cent.[16]

Most studies of lead's psychological impact have focused on psychometric intelligence, perception, and learning. Considerable speculation, but little data, exist on lead's impact on higher order behaviors which condition the child's social adaptation. What clues exist are troubling. Lead poisoning produces hyperactivity and aggression, and studies of low-dose exposure show an increased incidence of those behaviors subsumed under the attention deficit syndrome.[9] Attention deficit and learning disorders are well-established risk factors for antisocial behavior.[17] The rate of later delinquency in children who early display attention deficit syndrome and conduct disorder is .58. The attributable risk for hyperactivity in children with elevated lead levels is .55. Multiplying the lower 95 per cent confidence limits for these two proportions leads to a joint probability of .2 for delinquency, given excess lead exposure. Whether there is a causal link between lead and delinquency has not been subject to systematic study, but the clues are a subject for troubled conjecture.

There are a number of parallels between risk factors for criminality and lead. Wilson and Herrnstein, in *Crime and Human Nature*,[18] argue that criminality has constitutional roots. They ground their position on seven findings:

1) criminal behavior shows itself early in life;
2) criminality is commoner in males;
3) the rate of criminal behavior is higher in Blacks;
4) the rate is higher in urban areas;
5) criminals have lower IQs, with particular impairment on verbal scores;
6) criminals have a high incidence of hyperactivity in early childhood; and
7) the families of criminals are disorganized and aggressive, and their homes are ill kept.

All of the above associations also exist either as effects of lead or risk factors for lead exposure. Lead exposure occurs early in life; it is commoner in males, in Blacks, and in city dwellers; it lowers IQ, particularly verbal IQ scores; and dirty, disorganized households have children with higher lead levels.

This is not to say that lead is the solitary cause of delinquency; life is more complex than that. It can be argued that these associations are correlational, not causal. But it is at the same time a reasonable conjecture that the disordered thinking, impaired impulse control, reduced verbal skills, and the demonstrated increase in school failure that are a known product of lead exposure increase the probability that some individuals will adopt antisocial responses to the challenges of society. This proposition is currently under systematic study.

This overabundance of toxic lead exists in exactly the same places as two shameful and dangerous shortages: housing and jobs. Many factors contribute to the increasing numbers of homeless Americans. Among them is the dire shortage of housing at rates affordable for people of low income, and the large number of unemployed or underemployed citizens. Although lead in household paint was banned by statute in 1972, as many as 24 million homes still have surfaces that were painted well before then. Of these, 2 million have deteriorated lead surfaces, and are inhabited by young children. This paint contains as much as 50 per cent lead by weight; it blisters and flakes, or simply powders and becomes part of the household dust, forming the dangerous residue which awaits the daily explorations of the curious child.

In this mix of excess and shortage is a remedy waiting to be seized. It is reasonable to ask why unemployed men and women cannot be trained to de-lead and rehabilitate houses under safe conditions and at adequate pay, and why some of their labor cannot be used to purchase equity in these houses once they are made safe to live in. De-leading and repainting an average-sized house costs about $5,000. This is no simple undertaking: de-leading houses is dangerous business. Children must be removed from the premises, workers must be trained and supervised in the use of safe techniques, and the property must be scrupulously cleaned after de-leading. Careful clean-up is required; lead in dust left behind after abatement is a serious hazard. But lead does not possess the same degree of hazard as an infectious agent or asbestos. Reasonable reductions in dust lead levels can be obtained with reasonable assiduousness at reasonable costs. To get the last few micrograms of lead out of a house can drive the costs of lead abatement to prohibitive levels. Data from lead screening programs show that reasonable care will result in children's lowered blood lead levels. This is, after all, the final goal.

The costs of de-leading are maintained by the use of an antiquarian technology; new R & D is urgently needed. To de-lead 2 million homes by today's technology, which differs not at all from that used at the turn of the century, would cost $10 billion. If this money were to be allocated over the next 10 years, 20,000 jobs could be created at $20,000 per year (with a 5 per cent yearly cost of living increase). A substantial proportion of the money paid out in salaries would be spent in poor neighborhoods, and its impact multiplied. This effort would cost $5.7 billion and leave $4.3 billion for training, materials, insurance, and administrative costs. If this sounds impractical, one should be reminded that the proposed allocation for new prisons is $11.6 billion.

Rarely do problems come so neatly assembled. Rarely is

the boundary between Utopian thinking and pragmatism so narrow and blurred. A serious attack on lead paint could be a powerful weapon in a simultaneous assault on unemployment, homelessness, and relentless brain damage: three, not one, man-made and therefore man-curable diseases.

Twenty years ago, René Dubos observed the national problem of lead paint poisoning, and issued the following warning:

> The problem is so well defined, so neatly packaged, with both causes and cures known, that if we don't eliminate this social crime, our society deserves all the disasters that have been forecast for it.[19]

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NIH Consensus Conference Announced: Sunlight, UV Radiation and the Skin

A Consensus Development Conference on Sunlight, Ultraviolet Radiation, and the Skin is being sponsored by the National Institutes of Health, May 8–10, 1989, in Bethesda, Maryland.

Overexposure to sunlight and artificial sources of ultraviolet light has the potential for acute and chronic adverse effects on the skin and its functions. These effects include some forms of skin cancer, immune system alterations, damage to blood vessels in the skin, and premature aging of skin. In recent years, the ability to measure the effects of ultraviolet radiation on the skin has improved greatly; we have also seen a dramatic change in the ability to protect the skin from ultraviolet radiation, and preliminary evidence suggests reversibility of some chronic effects through the use of prescription medications.

Considerable controversy remains, however, and this conference will address a number of questions and seek a consensus on the most appropriate strategies for prevention and, if possible, treatment of adverse effects of sunlight exposure and UV radiation on the skin.

The conference will bring together dermatologists, photobiologists, immunologists, oncologists, epidemiologists, pharmaceutical scientists, other health care professionals, and the public. Conference sessions will be held in Masur Auditorium, Building 10, National Institutes of Health, 9000 Rockville Pike, Bethesda, MD. There is no charge for registration and the conference is open to the public. Hotel rooms at area hotels should be reserved in advance. Individuals must arrange their own accommodations. To obtain further information, contact Andrea Manning, Prospect Associates, at (301) 468-MEET.

The NIH has certified this conference as meeting the criteria for 14 credit hours in category I of the Physician's Recognition Award of the American Medical Association.