



COMMENTARY

On Childhood Lead Poisoning and Social Responsibility

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In the inaugural issue of this Journal, Editor-in-Chief Jennifer Leaning traced the history of Physicians for Social Responsibility (PSR) from its beginnings in 1961, described the changing political world in which it has evolved, and limned the "connection between disarmament and civilization that led PSR in 1990 to adopt a major focus on the environment. . . . [T]he implications of major environmental change" would now be examined as would "the role of physicians and scientists in the creation of knowledge and the development of technologies . . . that have profoundly affected our moral landscape" [1]. The problem of childhood lead poisoning is a paradigm of this intersection of technological development and "our moral landscape."

The technological development trajectory had its origin at the start of the industrial revolution when, for the first time, significant quantities of lead were loosed into the global environment. Despite the historical record of acute lead poisoning in early Greece [2], and the persistent speculation that lead may have figured in the fall of Rome [3], it is probable that the corpora of most early humans were virtually lead-free. Note here Needleman's statement: "Clair Patterson, the geochemist credited with dating the

age of the earth, . . . suggested that civilization had raised everyone's body lead burdens to 1,000 times that of our ancient ancestors (personal communication, 1992)" [4]. This possible three orders-of-magnitude increase is not unlike the increase in present-day blood lead levels over "natural" blood levels in preindustrial humans as estimated by Flegal and Smith [5]. These investigators derived a simple linear regression that describes the relationship between bone lead and blood lead in four populations: environmentally exposed humans, occupationally exposed humans, rats maintained under standard laboratory conditions, and rats maintained under conditions clean of trace metals. When the linear trend was extrapolated to an experimentally determined value for skeletal lead levels of preindustrial humans, the blood lead concentration could be read as 0.016 $\mu\text{g}/\text{dl}$ (0.8 nmol/L) (Figure). This value, which Flegal and Smith think is "conservatively high," is about 1/600 the 10 $\mu\text{g}/\text{dl}$ criterion lead level adopted in 1991 by the Centers for Disease Control (CDC) [6]. They suggest that future criteria for safe body burdens of lead should take the presumed "natural" level into account. Their value is also far lower than levels of 0.8 $\mu\text{g}/\text{dl}$ and 3 $\mu\text{g}/\text{dl}$ reported for twentieth century humans living in remote, relatively unpolluted regions of, respectively, the southern [7] and northern [8] hemispheres.

The 10 $\mu\text{g}/\text{dl}$ standard is the most recent revision downward (from 25 $\mu\text{g}/\text{dl}$ in 1985, 30 in 1975, and 40 in 1970) of the federal definition of an elevated blood

1051-2438/1993/0301-0003\$03.00/0

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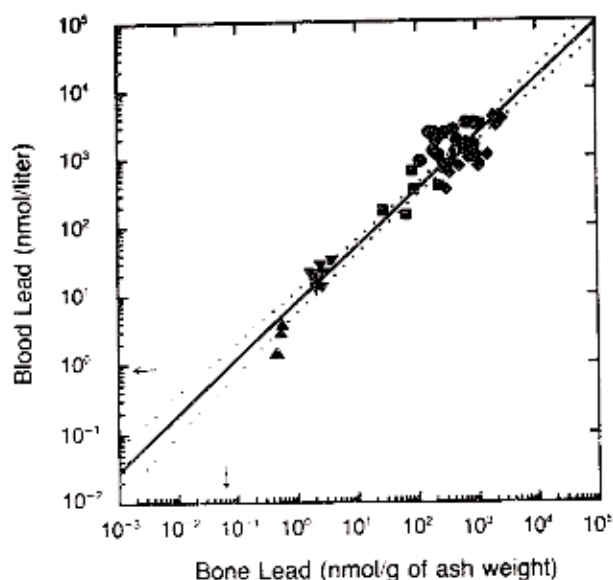


FIGURE. The lead concentration in the blood of preindustrial humans [0.8nmol/L (0.016 $\mu\text{g}/\text{dl}$)] (arrow on the ordinate) was derived from the reported concentrations of lead in preindustrial human bone (arrow on the abscissa) by graphical extrapolation of the relation between paired bone and blood lead concentrations of environmentally exposed humans (squares), occupationally exposed humans (circles), laboratory rats maintained under standard conditions (diamonds), and rats maintained under conditions clean of trace metals (triangles and inverted triangles [unpublished data]). The dotted lines indicate the 95% confidence limits. Reprinted, by permission of The New England Journal of Medicine 1992;326:1293-1294.

lead level. Since its adoption, further evidence has supported the suspicion that lead is toxic at even lower levels. Thus, medical scientists are approaching consensus that lead, like ionizing radiation, is a non-threshold toxicant [6]. Bellinger, Stiles, and Needleman have recently reported a long-term neuropsychological follow-up study of relatively advantaged 10-year-olds. The data showed that a six point decline in Wechsler Intelligence Scales (WISC-R) full-scale IQ scores was associated with each 10 $\mu\text{g}/\text{dl}$ increase in the children's blood leads at age 24 months (over a range from 0 to >20 $\mu\text{g}/\text{dl}$), as was a nine point decline in similarly standardized achievement scores [9]. In a longitudinal study by Dietrich and colleagues of severely *disadvantaged* children, the decrement in WISC-R scores was seven points for 10 $\mu\text{g}/\text{dl}$ increases in blood lead [10]. Dietrich also reported adverse effects on central auditory processing [11] and (his group) on gross and fine-motor skills [12]. Statistical analyses of the data in these investiga-

tions were carefully adjusted for confounders and covariates. This point is emphasized because the debate about public policy implications of the findings arises in part from disagreement about the rigor of the study designs [13,14].

Single-digit decrements in IQ (or in other well validated measures of neuropsychological development) associated with toxic exposure to lead may not appear dramatic, and of course in individual children it cannot be determined that they are. But the "modest" six point median difference in IQ between the "high-lead" and "low-lead" children in Needleman's pioneering study of 1979 [15] had special significance: The frequency distribution of the scores was such that high-lead children were almost four times as likely as low-lead children to have IQs less than 80. And none of the high-lead children had IQs greater than 125 whereas 5% of the low-lead group did. It is sobering to consider that the child who would have been normal at an IQ of 98 is likely to be more disabled by a six or eight point IQ loss than is the child whose IQ might have been, for example, 128.

Although the circumstance of large populations of children functioning suboptimally must have social impact, even if it is not quantifiable, public and professional responses have traditionally been stimulated by episodes of acute lead poisoning. In the late 1960s, when neurotoxicity was still clinically evident, community groups organized "Get the Lead Out" committees. They were assisted in their fact-finding and their representations to government by scientists and physicians who viewed lead poisoning as an inner-city problem, evidence of what we now call "environmental racism." (That lead poisoning affects all economic strata, classes, and races—unequally, to be sure—had not yet been documented.)

The First National Conference on Childhood Lead Poisoning was held in 1969 at Rockefeller University. It concluded with Dr. Rene Dubos' prophetic statement: "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" [16].

Then in 1971, over the objections of the lead industry [17], and three quarters of a century after the toxicity of leaded paint had been established [18], Congress passed the first Lead-Based Paint Poisoning Prevention Act. During the following two decades, paint and related housing laws were revised, drinking water legislation was passed, and leaded gasoline

was effectively banned. It should be pointed out (to newcomers to this saga) that compliance with paint, housing, and drinking water legislation was indifferent or nonexistent [19], and that restrictions on leaded gasoline were adopted primarily because catalytic converters (to reduce hydrocarbon and carbon monoxide emissions from cars) couldn't function in the presence of lead. However, ambient air lead levels did drop markedly, and so did lead concentrations in children's blood.

But those were the years when the subtle cognitive and behavioral effects of lower levels of body lead burdens were first being observed, and clearly it felt to some guardians of the public health that coping with the lead poisoning problem was akin to running in place [20]. In truth, the recognition that low-level lead exposure has adverse neurobehavioral effects in children, and that these are measurable in populations but not in individuals, has shifted the issue of childhood lead poisoning from exclusively medical governance into the arenas of public health and social policy. At the present time, the importance of these nonclinical effects, and the public policies that should be adopted in response to them, is under conscientious and sometimes contentious debate [13,14]

It is here that humankind's technological advances intersect with our moral landscape. Did Dr. Dubos have it right? If lead poisoning is a "social crime" and its causes and cures are known, are we as a society culpable if we do not move to eliminate it?

It depends.

No one doubts that preventable diseases are "social crimes." The 1991 CDC Strategic Plan for the Elimination of Childhood Lead Poisoning [21] and its Statement on the Prevention of Lead Poisoning in Young Children later that year [6] were welcomed unambivalently by the child advocacy community and more guardedly by pediatricians: child care providers in private practice, academe, community medicine, and/or public health have differing perspectives on the feasibility and advisability of the recommendations.

The Statement was prepared by an Advisory Committee whose expert members and consultants were drawn from a variety of institutions and disciplines. Since its publication, its conclusions and recommendations have received additional research [9-12,22] and vigorous evaluative [14,23,24] support. Nevertheless, approval has not been unanimous.

The most visible contributions of the Statement, i.e., the lowering of the criterion lead level to 10 $\mu\text{g}/\text{dl}$,

and the stated goal of screening all children between ages nine months and six years, have provoked controversy. Critics of the lowered value have characterized the more-than-a-dozen studies of neurobehavioral sequelae of low-level lead exposure as scientifically flawed. The studies are faulted for failure to control rigorously for an array of confounders and covariates. Thus, the observed differences between "high-lead" and "low-lead" children, and the changes observed along a continuum of body lead burdens, are thought to reside not in lead itself but in quality of parenting, mother's education, prenatal maternal nutrition, family income, or numerous other "more complex influences on our children." The CDC judgment to lower the criterion lead level is criticized for "not [being] adequately supported by available research" [13]. Recommendations on the basis of that judgment are therefore resisted [25].

There is also concern that, though the body of work on which the report's recommendations are based is scientifically solid, the 10 $\mu\text{g}/\text{dl}$ criterion level is open to misinterpretation or perhaps overinterpretation. This is due in part, as Piomelli has pointed out, to the inadequacy of current technology: "measurements of [blood lead] levels by present techniques carry an inherent error of + 4 $\mu\text{g}/\text{dl}$ " [26].

In point of fact, the Committee report presents careful and conservative discussion of its decisions. In consideration of the practical problems attendant upon its recommendations, it identifies "priority groups for screening" starting at six months of age and determined by environmental exposure.

Central to the Statement is the definition of classes of children according to their blood lead levels. Class I includes children whose blood lead concentration is $\leq 9 \mu\text{g}/\text{dl}$. These children are "not considered to be lead-poisoned," and no follow-up activity is recommended. For each class above the criterion level, follow-up activities are recommended. For example, Class II A is 10 $\mu\text{g}/\text{dl}$ - 14 $\mu\text{g}/\text{dl}$, and detection of many children with blood lead in this range "should trigger community-wide childhood lead poisoning prevention activities....Children in this range may need to be rescreened more frequently." Note that no action with respect to individual children is suggested. Class II B is 15 $\mu\text{g}/\text{dl}$ - 19 $\mu\text{g}/\text{dl}$, and these children "should receive nutritional and educational interventions and more frequent screenings. If the blood lead level persists in this range, environmental investigation and intervention should be done." The schedule continues to Class V, with blood lead ≥ 70

µg/dl, a lead poisoning emergency that requires immediate "medical and environmental management"

Other critics question the advisability of universal screening, even as a goal. The first reason (again) is the practical deterrent that at present there is no satisfactory mass screening technique. The measurement of erythrocyte protoporphyrin in capillary blood, thought to be dependable until recently, is now obsolete with respect to lead because it does not reliably identify concentrations of blood lead between 10 µg/dl and 25 µg/dl. Capillary blood for direct lead determination is susceptible to contamination, though it is widely used for initial screening; elevated blood lead results must then be confirmed by analysis of venous blood. Measurement of lead in venous blood is more replicable, but as a screening tool it is time-consuming, expensive, and not patient-friendly. The 1991 CDC Statement recognized the need for better testing methods, and reported that "work on developing easy-to-use, cheap, portable instruments . . . is ongoing." More than a year later, we know that at least two "easy-to-use, cheap, portable," and highly sensitive enzymatic methods have been developed, but their availability depends upon regulatory approval and commercial development (a series of personal communications, 1991 - 1993, with Dr. Bernard Davidow, former Director of the Bureau of Laboratories, New York City Department of Health, and Dr. Ellen Silbergeld, Program in Toxicology, University of Maryland School of Medicine and Environmental Defense Fund).

A second reason that universal screening is criticized is that, once a child has been found to have an elevated blood lead level, the frightened and frustrated parents are confronted by the inadequacy of technical and financial resources for safe environmental remediation. A third is the fear that a child who is identified as lead poisoned will be stigmatized as cognitively affected, simply because neurobehavioral deficits have been measured in *populations* of children. A fourth concern about the screening recommendation is the elusiveness of mechanisms to ensure its universality; how will the children not now receiving regular medical care (and probably most at risk) be brought into the process?

In addition, there are advocates of primary-prevention-only who believe that limited resources will be used more productively if they are applied to the reduction or elimination of lead in the environment, or perhaps to other "proven health-care problems of

US children" [25]

The American Academy of Pediatrics is still formulating its revised policy with respect to childhood lead poisoning [27]. The reservations summarized above have widened and deepened the discourse. There are presumptive answers to some of them: A highly appropriate screening instrument is due soon; the Residential Lead-Based Paint Hazard Reduction Act enacted by the 102nd Congress and signed by then-President Bush will, as its title says, provide funding for training and safe abatement of residential sources of lead; enforceable standards for lead in drinking water will be reintroduced during the 103rd Congress if support and advocacy continue; a mass media educational campaign on childhood lead poisoning, promised for early 1993 by the President's Commission on Environmental Quality, is being designed to alert and inform, but not alarm, a very wide public; and if President Clinton's "proposal to Congress [guaranteeing] the immunization of every child" [28] were delivered, screening for lead could be incorporated into the structure that its implementation would necessitate.

On primary prevention, the CDC Statement identified as one of the most important of its themes "the need to identify and remove sources of exposure to lead before children are harmed." A relevant justification for screening programs is that "[d]ata generated [from screening] can be used in targeting interventions to places with children at high risk for lead poisoning" [6].

Two final points: The cost-effectiveness of lead poisoning prevention programs was analyzed for the Environmental Protection Agency in 1986 [29], and the data were cited by the CDC in 1991 in its Strategic Plan for the Elimination of Childhood Lead Poisoning [21]. It was calculated that every dollar spent on reducing the prevalence of lead poisoning would be significantly exceeded by savings in educational remediation and medical care, and increased productivity of less handicapped young people. As for the chronic underfunding in the health and human services sector, child advocates may seize this pivotal movement to force reallocation of the nation's resources: better health care, housing, education, jobs, and nutrition—all are critically needed and all would militate against accumulation of toxic concentrations of lead in children's bodies.

These comments opened with excerpts from the editorial in this Journal's first issue and close with a passage from the seventh:

Discovery can relate to small questions as well as large, with impact felt only in ratified circles, at least for many years. What makes a discovery have significant impact on social policy may have less to do with its potential revolutionary content than with the doggedness and creativity with which those who recognize its relevance to an environmental, social, or economic problem keep it before the public view [30].

The elimination of childhood lead poisoning is possible [6,26,31]. Wherever this preventable disease is placed in the hierarchy of threats to children's health, the exercise of social responsibility dictates that it be kept "before the public view" until it no longer intrudes upon our landscape—medical or moral. ■

ACKNOWLEDGMENT

Jennifer Morgan, staff assistant to the Public Health Program at Natural Resources Defense Council, was unfailingly helpful in the preparation of this manuscript.

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