Lead Control

Readers of *Science* who have wondered why the pace of lead control has been so halting in the face of an extraordinary body of knowledge about its toxicity can find instruction in Joseph Palca's article "Get-the-lead-out guru challenged" (News & Comment, 23 Aug., p. 253). Lead has been known to affect children's brains for at least 80 years, but regulation has lagged far behind the science, primarily because of the unrelenting activity of the lead industry and its spokespersons. The result was that we did not pass a Lead Paint Poisoning Prevention Act in this country until 1971, 50 years after Australia and 45 years after Great Britain, Sweden, Greece, and Poland (*1*).

Recoiling from a new spate of lawsuits against smelters, landlords, and paint companies and the publication by the Public Health Service in February 1991 of the historic "Strategic Plan to Eliminate Childhood Lead Poisoning," the lead industry is once again attacking the data base that drew the federal government to certify that blood lead concentrations above 10 micrograms per deciliter (μg/dl) are neurotoxic. They have ignored growing literature (*2*) and have focused largely on my 1979 paper (*3*), which showed lead-induced deficits in "normal" children and successfully responded to many of the methodologic questions that had vexed the question of low-dose lead toxicity.

The case for human lead toxicity is strong, but it does not stand by itself. Ignored in Palca's article are the massive experimental data on the biology of lead at low doses (*2*) showing biochemical, physiological, and behavioral alterations in rodents and primates that had received internal doses within the ranges of those received by millions of children. The Environmental Protection Agency's (EPA's) Air Lead Criteria Document (*4*) is the most comprehensive source to review this literature.

The activities of EPA's Clean Air Science Advisory Committee (CASAC) were reported in *Science* in 1983 (News and Comment, 25 Nov. 1983, p. 906), and the current article is largely a restatement of that piece. At that time, the International Lead Zinc Research Organization seized on the opportunity provided by the distribution to reviewers of an uncorrected preliminary EPA draft report (*5*) that contained some criticisms of my work and hired a public relations firm to send printed copies, minus the caveat "This document . . . should not at this stage be construed to represent Agency policy," to science editors and investigators around the world, representing it as EPA's position on my study. In fact, after reviewing my comments on the error-ridden draft and examining the reanalyses of the data that we did according to EPA's suggestions, the EPA committee read and responded to my corrections and stated in the final Air Lead Criteria Document:

> The Committee also recommended that the Needleman data set be reanalyzed. Reanalyses carried out in response to the Committee's recommendations have been reported by Needleman (1984), Needleman *et al.* (1985), and the U.S. EPA's Office of Policy analysis (1984) as confirming the published findings [*4*] on significant associations between elevated dentine lead levels and decrements in IQ. . . .

The EPA docket (*6*) shows clearly that I provided raw data and access to the tapes to EPA's statisticians and that Hugh Pitcher, EPA biostatistician, examined these data with particular attention to the issues raised by the industry spokespersons (confounding variables, exclusion of observations, and errors in variables) and reported to CASAC that "our analyses, or our reactions to the results of Needleman's reanalysis is essentially that the results that we found are robust to all of the checks, the empirical checks that were made on these issues" (*6*, p. 66).

After the CASAC reviewed the Pitcher

report, examined my revisions, and recommended rejection of the subcommittee's critical report, a lead industry executive wrote to members of the subcommittee on his own and attempted to elicit unfavorable comments on my work.

This kind of activity and the misrepresentation of EPA's position, repeated many times in many settings, has led me to the conclusion that the lead industry and its representatives do not qualify as disinterested scientists.

The central question clearly is not whether my 12-year-old study has flaws; of course it does. But the work has survived what I believe to be among the most thorough scrutinies in environmental health and has been judged by many critics as valid. In attempting to make their case the lead industry has ignored the animal data, the 17 studies of children published since my 1979 paper (all showing effects at lower lead levels than I did), and three published meta-analyses of cross-sectional low-level lead studies (*2*) which show a strongly significant lead effect. It is a defensible proposition that there are no independently funded researchers of lead effects in children who do not believe that lead at low doses is neurotoxic. If there are, they have neither published their comments in the open literature nor have they given them at meetings of scientific societies.

I would have ignored this chronic relapsing industrial food fight had it not occurred at a time when critical public health decisions are in the balance. The Public Health Service, having recognized that childhood lead poisoning is one of the most serious problems for the children in the United States and that it is an eradicable disease, has drafted a Strategic Plan that, if implemented, will begin the process of breaking the exposure link by getting lead out of the environment before it finds its way into children's brains. It now appears that the White House has decided that increased federal participation will not take place (*7*). This would be a tragedy and cannot be allowed to happen without a complete discussion of the costs to our society.

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Palca's piece on the effects of low-level lead on IQ leaves me bewildered. The only recourse in case of doubt is replication. Here $63 million was involved in one settlement. It appears that a British study is at odds with the conclusions of Needleman *et al.* One would think that there would be honest scientists who could be interested in resolving a matter so important to both health and the economy. It seems reasonable that a new study could be carried out for less than the cost of further compliance with unreasonably low levels, if, indeed, the levels set by EPA are unreasonably low. Yet, all we hear is denial, controversy, and name-calling. Is a resolution of important scientific issues through careful replication of the valid features of Needleman *et al.*'s study impossible in this country? Impractical? Must we continue in doubt?

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Palca's excellent article touches on one of the sorriest and most vexing aspects of modern toxicology, namely the large number of disputed and contradictory results. In many cases, the normal self-cleansing mechanism in science is short-circuited when regulatory agencies pick disputed results as their favorites. All of this has contributed to the unfortunate image of toxicology as a discipline where any kind of result can be obtained and published and any kind of toxicological view can be heard in the courts. Only sunlight and better science will ultimately resolve this and other controversies.

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Cancer Risk and Behavior Change

I couldn't agree more with the sentiments of many of the researchers interviewed for Jean Marx's Special Report "Zeroing in on individual cancer risk" (9 Aug., p. 612): prevention (and early diagnosis) is the way to go! I find it surprising, however, that the most effective prevention technique we have—behavior change—is scarcely mentioned. Stopping smoking, reducing dietary fat, getting more exercise, taking precautions against too much sun exposure, and following recommended screening procedures, such as having annual mammographies, pap smears, and skin examinations (where appropriate), would go a long way toward reducing cancer incidence and mortality in this country.

Molecular approaches do indeed appear to hold greater promise for identifying which individuals are at risk than do currently available epidemiological methods. But once individuals have been "identified," many may elect to change their behavior rather than use such "ultimate" remedies as chemoprevention, which almost certainly would entail nonnegligible risks of potentially serious side effects. As far as I know, no study has yet shown that changing behavior to reduce the risk of cancer has serious physiological side effects.

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Electrochemical Sensor: Prior Concept

In our report of 3 May 1991, "Molecular self-assembly of two-terminal, voltammetric microsensors with internal reference" (p. 1991) (*1*), we described a pH microsensor with detection based on measurement of the potential difference between cyclic voltammetric peaks for two electrode-confined redox reagents: a ferrocenyl alkyl thiol with a pH-insensitive redox potential and a hydroquinone alkyl thiol with a pH-sensitive redox potential. We regret that at the time of publication we were unaware of an existing paper by I. Rubinstein (*2*) that had previously demonstrated voltammetric pH sensing with detection based on the peak potential difference between two electrode-confined redox couples, and we wish to credit Rubinstein with priority for this concept. He also mentioned that his systems "are ideal candidates for miniaturization," that a "full voltammetric" operating mode "requires a two- (rather than three-) electrode assembly, which translates into simpler construction and application," and that