Technology **Vol. 6, pp. 261-268, 1999**

COMMENTARY

CHILDHOOD LEAD POISONING AND TAINTED SCIENCE

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TF 9804-532 C (Received 1 April 1998; accepted 10 August 1998)

INTRODUCTION

Childhood mean blood lead levels (BPb) have plummeted fivefold, and prevalence of significant BPb levels (i.e., >20 μg/dL) has decreased by a factor of 60 among children in the United States during the past two decades. Government agencies are telling people that childhood lead poisoning is often named as the leading environmental threat to our children. This conclusion is not accepted by most practicing physicians, who almost never see a case of symptomatic lead poisoning. Most pediatricians who practice in a large medical group in an urban area see environmental threats daily. These include poverty, violence, homelessness, family dysfunction, abuse, teenage pregnancy, drugs, and alcohol--but they have not included symptomatic lead poisoning. Most physicians do not accept current proclamations about the importance of childhood lead poisoning: the nation's pediatricians did not comply with 1991 recommendations of the Centers for Disease Control and Prevention (CDC) for annual, universal childhood BPb screening (1), and the CDC recently reversed these recommendations (2). A recent report by the American Council on Science and Health (ACSH) provides an analysis of the current ACSH position (3).

HIGH CHILDHOOD BPb LEVELS: A BRIEF HISTORY

Before 1970, childhood lead poisoning was an important health

problem--particularly among the urban poor. Thousands of children annually suffered major brain damage from lead encephalopathy, and many died. As a pediatric house officer in Boston between 1949 and 1954, 1 commonly saw children who had symptoms of lead poisoning; some of these children were ill, and some died as a result. BPb levels in these ill children were astronomical by current standards: according to the National Research Council, the research arm of the National Academy of Sciences; the National Academy of Engineering; and, the Institute of Medicine (4, 5), symptomatic childhood lead poisoning was associated with a mean BPb level of 178 μg/dL, and fatal lead encephalopathy was associated with a mean BPb level >300 μg/dL. It is known on clinical grounds that a child was out of immediate danger if [begin p. 262] the BPb level was <100 μg/dL, and the CDC officially defined the lower threshold for lead poisoning at 60 μg/dL.

In Chicago during the 1960s, childhood lead poisoning caused about 100 deaths annually, leading to a classic study conducted by Henrietta Sachs between 1968 and 1971. Sachs assessed BPb levels in >200,000 inner-city Chicago children, mainly African Americans, and found that 8% of these children had BPb levels >50 μg/dL. Although only 8.9% of these children at high risk for lead poisoning had symptoms of the disease, lead poisoning was recognized to be a significant health threat to children and often necessitated chelation therapy for decreasing the body lead burden as well as intensive public health measures to eliminate lead exposure (6,7). Subsequently, a 20-year follow-up study (8) of 60 of 465 children with BPb levels >80 μg/dL indicated that socially and educationally, these adults did at least as well as their community peers.

Since the early 1970s, when regulations were promulgated eliminating lead from gasoline, paint, and other sources, mean BPb levels have rapidly and continuously fallen, and the threat of lead encephalopathy and related death has essentially disappeared in the United States. National Health and Nutrition Examination Survey reports have confirmed the rapidity of this decline in BPb levels (9, 10, 11): From a mean BPb 15.0 μg/dL reported in the 1976-80 survey, BPb levels fell more than fivefold (to 2.7 μg/dL) in the 1991-94 survey. These reports (9. 10, 11) also indicated that, among U.S. children. the prevalence of BPb levels > 10 μg/dL fell from 88% to 4.4%. a 20-fold decrease; and most significantly, prevalence of higher BPb levels (ie, levels >20 μg/dL) declined from 24.7% to 0.4%--a 62-fold fall. These remarkable results emphasize the reality of decreasing environmental exposure to lead.

PARADOXICAL RESPONSE TO DECREASED BPb LEVELS

Paradoxically. in the past decade as symptomatic lead poisoning has disappeared, the attention and expenditures devoted to childhood lead poisoning have multiplied. In 1991, the CDC issued a report (1) decreasing the threshold of concern about BPb levels in children from 25 μg/dL to 10 μg/dL, thus increasing the number of children considered to be at risk from childhood lead poisoning from 250,000 to over 3 million, creating an "epidemic by edict" (12). The CDC also recommended that all U.S. children should first have lead testing done during the second half of their first year and then annually until age 5 y (1). These recommendations would have required testing for as many as 8-16 million U.S. children annually at a mean cost of about $20 per test, or $320 million annually for laboratory costs alone. Further regulations by the U.S. Environmental Protection Agency (EPA) and the U.S. Department of Housing and Urban Development (HUD) brought the total cost of lead testing and abatement programs to billions of dollars annually. The CDC issued a report referring to childhood lead poisoning as "the leading environmental threat to U.S. children" (13).

QUESTIONABLE SCIENTIFIC VALIDITY OF POLICY

What caused this flurry of expenditures and concern despite rapidly-diminishing childhood BPb levels? The answer: controversial studies showing that BPb levels far lower than those causing symptoms were responsible for subtle neurobehavioral defects in children, including decreased IQ and learning disabilities.

Concern about these supposed defects was largely the result of a study by Herbert Needleman and colleagues, who published an article in 1979 (14), which showed diminished 1Q in children who had elevated lead levels in dentine. Further work by Needleman (15) and other investigators indicated a possible decrease of 4-8 IQ points for every 10 μg/dL rise in BPb level (16, 17).

However, in the nearly 20 years since Needleman's original report. multiple studies have shown contradictory results (18, 19, 20). A meta-analysis by Pocock et al. (2) indicated that any neurobehavioral effect of low BPb levels, if it exists at all, is minor--in the range of 1-2 IQ points--and not clinically significant in any individual child. Further, even this slight potential effect might be explained by confounders as well as by reverse causality. Western European authorities have not

lowered the threshold of concern from 25 μg/dL to 10 μg/dL (as has been done in the U.S.) and have [begin p. 263] not recommended universal childhood lead screening. Moreover, the estimates of the 1991 CDC Committee on Childhood Lead Poisoning concerning the prevalence of BPb levels > 10 μg/dL in the U .S. proved to be grossly exaggerated, and the CDC in 1997 reversed its recommendation for universal childhood lead screening after hundreds of millions of dollars were spent assessing BPb levels in low-risk children. Instead, the CDC recommended targeted screening of children at high risk of lead exposure--mainly poor children living in old, dilapidated housing (2).

The validity of Needleman's original studies and the scientific work and statements of some others involved with the 1991 CDC report have been questioned, as have other actions taken by the CDC, the EPA, and HUD. As early as 1983, the methodology and validity of Needleman's 1979 studies were challenged and were the subject of an investigation, as was the work of Claire Ernhart, whose studies on childhood lead poisoning did not support Needleman's conclusions (22). The findings of the investigatory board confirmed Ernhart's results but raised questions about inconsistencies in Needleman's work which were never resolved (22). In spite of this, Needleman, with support from federal grants and environmental advocacy groups, assumed an increasingly influential role as chairman and member of the CDC advisory committees and as consultant to government agencies--including the EPA. He played an important role in the CDC Advisory Committee on Childhood Lead Poisoning in 1977, 1991, and 1993, during which time the official CDC threshold of concern about PBb was lowered from 60 μg/dL (before 1970) to 40 μg/dL (1970-75) and then progressively to 30 μg/dL (1975-85), to 25 μg/dL (1985-91), and to]0 μg/dL (1 991). It seems logical to empirically set the BPb level of concern lower than the reported symptomatic mean BPb (178 μg/dL) by a reasonable multiple. Indeed, the BPb level (25 μg/dL) which the CDC set as a cutoff for concern prior to 1991 was seven times lower than this mean symptomatic value and twice as low as the generally-accepted minimum symptomatic level (50 μg/dL). However, lowering the level of concern further to 10 μg/dL in 1991 at the behest of Needleman and other low-lead crusade protagonists at the CDC has unjustifiably resulted in a tenfold increase in "abnormal" results, thus creating parental anxiety, lack of acceptance among practitioners, and exorbitant costs--all based on contradictory evidence.

In 1990, Needleman cited his 1979 studies when he testified for the EPA in a case against a steel company (23). The validity of these investigations was challenged by Ernhart and Sandra Scarr, a psychology professor from the University of Virginia. Because his work was financed by federal grants, Needleman was ordered to reveal his original data (23). Partial review of these data by Ernhart and Scarr unearthed questionable data and methodology and resulted in an inquiry of Needleman's work by his own university, the University of Pittsburgh (24); and by the Office of Research Integrity (ORI) (25). The findings of these investigations--released in 1993 and 1994--were critical of the quality of Needleman's scientific methodology, but the multiple misrepresentations in his work fell short of the rigid current ORI definition of scientific misconduct: fabrication, falsification. and plagiarism (FFP).

The report of the University of Pittsburgh Hearing Board (24) found Needleman's studies to consist of a "pattern of errors, omissions, [and] contradictions" going back for many years.

In regard to a 1979 article by Needleman (14), the University of Pittsburgh Hearing Board unanimously believed that Needleman was deliberately misleading, stating that "if the paper had contained all the caveats it should have contained regarding subject selection and model selection, it might not have been published, and it certainly should not have been a basis for federal policy" (24). Nonetheless, this study was published and Needleman became a consultant for the federal 1991 CDC recommendations (1), which introduced universal childhood lead screening as well as lower BPb levels of concern.

A subsequent review by the ORI (25) seconded the University of Pittsburgh findings, confirming the "pattern of errors, omissions, [and] contradictions," and discovering additional defects. Needleman was found to have misplotted graph points in a way that was "difficult to explain ... [as] honest error" and to have ignored the pleas of Gunnoe, coauthor of the 1979 article (14), to correct known methodological errors before submitting the article to the journal. However, like the University of Pittsburgh, the ORI [begin p. 264] concluded that Needleman's scientific deficiencies could not be defined as FFP and thus did not constitute scientific misconduct. Commenting on this "fuzzy verdict," Taylor questioned the Pittsburgh Board's decision "for what most scientists would consider a reprehensible act: deliberately misrepresenting procedures used in a study to enhance the
study's perceived value or its chances of publication" (26).

With these questions about validity of his scientific methods coming from both his own university (24) and the ORI (25), one would have thought that Needleman would have opted to moderate his views. A more realistic expectation was that medical journals, federal granting agencies, and the scientific community would have hesitated to support Needleman's work. Very little has happened, however. Because his multiple scientific infractions were not found to be FFP, Needleman claimed that he was "vindicated." He instituted lawsuits against his university (27) and the ORI (28), and preemptively published an article in *Pediatrics* attacking his critics and claiming he was a victim analogous to the Salem witches (29). Although Needleman was directed by the University of Pittsburgh to submit a correction to the *New England Journal of Medicine* indicating that his studies "were not as originally reported and did not meet scientific standards of reproducibility," he initially failed to do so. When he finally submitted a "correction" (30), his statement did not reflect the true nature of his errors as the University had directed (24). He was successful in gaining the support of environmental advocacy groups and a strong activist organization--which, however, he helped to found--the Alliance to End Childhood Lead Poisoning.

In 1996, after the reports of his university and the ORI had been released, Needleman published a paper claiming that increased delinquency was
related to elevated bone lead (31). This study was again criticized for methodological irregularities (32. 33. 34), consistent with earlier demonstrated
patterns of substandard science and contradictions. The article contained a major contradiction: that African-American boys with high bone lead levels
not only had a higher rate of delinquency but had a greater mean IQ (31). In other words, according to the study. elevated bone lead levels resulted in smarter delinquents. When this contradiction was pointed out to him, Needleman's response was that it was "puzzling" (35).

FAILURE OF CURRENT STANDARDS FOR SCIENTIFIC CONDUCT

The Needleman case is one of several which have pointed out the ineffectiveness of the 1989 federal regulations limiting scientific misconduct to FFP. Under these regulations, it has been virtually impossible to gain a verdict of scientific misconduct without either a frank confession by a conscience-stricken investigator or a battery of evidentiary "smoking guns." Moreover, the

regulations did not protect whistle blowers. As a result, the NIH Rehabilitation Act of 1993 established a Commission on Research Integrity (the Ryan Commission) which was asked to consider revising the definition of scientific misconduct as well as to offer protection for whistle blowers. In a report issued in 1995 (36), this commission recommended a new definition of scientific misconduct--misappropriation, interference, or misrepresentation (MIM)--to replace the FFP standard. However. this recommendation has not yet been accepted: Many scientists worry that the proposed new regulations are too strict; that they reflect an overreaction to the malfeasance of very few researchers; and that they could restrict free scientific thought. In addition, despite a strong Congressional and public movement for scientific reform, the outlook for new regulations seems bleak in the face of academic and scientific opposition. Today, virtually the only protection against scientific misconduct is the belief that scientists are honest; no effective oversight is imposed over anyone. The Needleman case is an example of this lack of control.

Pessimism about the likelihood of imminent oversight is fostered by the clash between ethical and legal standards. In a number of publicized cases in which scientists were found guilty of scientific misconduct by the rigid FFP standards, the verdicts were overturned on appeal. Whistle blowers and investigatory boards are made up of scientists--not district attorneys or judges--and misconduct decisions are made on scientific--not legal--grounds. Increasingly, those found guilty of scientific misconduct are discovering that an experienced defense attorney can have virtually any misconduct decision [begin p. 265] overturned on legal grounds, usually on the basis of due process. Confessions and negotiated settlements currently appear to be the only viable means of maintaining any control over unprincipled science. This quandary was pointed out by John Dingell, the then Chairman of the Subcommittee on Oversight and Investigation of the U.S. House of Representatives in a lecture on misconduct in 1992 (37). On the basis of many years of knowledge and experience with scientific misconduct, Dingell was extremely critical of scientists, research institutions, and medical journals, and he gave examples of cases in which well-considered verdicts of scientific misconduct were overturned on legal appeal. Although Dingell's appeal for reform was followed by the Ryan Commission's 1995 report (36), it failed--and the scientific community is still in misconduct limbo.

These cases illustrate problems with editors of medical journals: To be aware of an author's previous scientific misdeeds, journal

editors and reviewers need inside knowledge leading them to review the investigative reports about questionable studies, even though these investigative reports may not be easily accessible. Even more difficult to explain are the actions of *The New England Journal of Medicine*, which printed the 1979 study (14) later found to consist of substandard science and to contain misrepresented data which did not meet scientific standards of reproducibility (24): the journal accepted Needleman's "correction" (30) without question or comment, even though this "correction" did not reflect the true nature of his errors. Moreover, Needleman is currently listed as a reviewer for the journal (38), indicating that at least one investigator whose own work was found not to meet proper scientific standards is now reviewing the scientific work of others.

BIASED ADVOCACY: INERTIA, SELF-INTEREST

Needleman--although the most prominent scientist responsible for developing the current over-concern with childhood lead poisoning despite plummeting childhood BPb levels--was not alone. The recommendation for universal childhood lead screening and lowering the level of concern about BPb came from the 1991 report of the CDC Advisory Committee on Childhood Lead Poisoning (1). This committee was dominated by members who, like Needleman, had long been committed to reporting low lead damage. Certain members and consultants of the CDC Advisory Committee were active in the Alliance to End Childhood Lead Poisoning, an influential activist lobbying organization of which Needleman was founder and Chairman of the Board. Chairman Rosen published an editorial in 1992 which exaggerated the prevalence of BPb levels > 10 μg/dL threefold to sevenfold (39). Goldman, another member of the CDC Advisory Committee, published faulty data which overstated the prevalence of elevated BPb in high-risk children in Oakland, California by using a method with >50% false-positive rates (40), but this important information was selectively omitted in a Morbidity Mortality Weekly Report (41). Silbergeld and Pollack have coauthored an Environmental Defense Fund publication which refers to the "current lead epidemic," and calls Needleman's 1979 report a "landmark study" (42).

This advocacy has prompted recommendations for a multibillion-dollar screening and abatement program which, according to Needleman, would have a societal as well as a medical benefit by helping to alleviate homelessness and joblessness (43,44). Being acknowledged as heroic initiator of such a program can be quite a stimulus for researchers to find

detrimental effects of low BPb levels.

THE OPPOSING STANDARD OF CREDIBLE BPb RESEARCH

Needleman and his supporters have characterized critics as being representatives of the lead industry. Emhart and Scarr, both respected scientific researchers, called attention to Needleman's errors, selection bias, and misrepresentations after reviewing his data for a legal case (23). The studies of both Ernhart and Scarr have adhered to the highest scientific standards and have never been found to contain evidence of substandard science or multiple methodological defects. Although Ernhart had a research grant from the lead industry years ago, this grant made up a small part of her research and academic support, and there is no evidence that it affected the high quality of her investigations. Scarr, the research psychologist, is an expert on [begin p. 266] scientific methodology but never worked in the field of lead research. Any claims that Scarr was part of the lead industry are based on her role as an expert witness who testified against Needleman's work after reviewing his data and then questioned his credibility.

The current effect of low BPb levels is slight, if any, as distinguished from the symptomatic high BPb values of the pre-1970 era. The results of multiple investigations (16, 17, 18, 19, 20), including prospective studies, are controversial, with some studies showing a slight decrease in IQ test scores and others showing none. One study (31) even associated higher IQ with elevated bone lead levels. The most thorough meta-analysis--that of Pocock et al (21)--found a very slight decrease in IQ (1-2 points) when BPb level was doubled from 10 to 20 mcg/dL. Such a minimal IQ decrease would be immeasurable and meaningless in any individual child and is only applicable to large population groups, and, as mentioned, could be explained by confounders or reverse causality. Claims of other neurobehavioral changes due to low BPb levels, such as learning disabilities and behavioral abnormalities, are anecdotal and are not based on credible evidence.

Inherent flaws in BPb research

There are many reasons for the almost impossible experimental task of demonstrating neurobehavioral damage caused by low levels of BPb. Elevated BPb level is a marker of a disadvantaged child and is associated with poverty, low parental IQ, dysfunctional families, violence, and other confounders. For example, abused children seen in an emergency department were

shown to be 27 times more likely to have elevated BPb levels as controls (45). Assessment of damage caused by low BPb levels is complicated by small effect size, imprecise outcome measures, and selection bias. When substandard science and contradictory results are added to this equation and encouraged by inadequate oversight, the conclusions lack credibility.

NEED TO PRIORITIZE COMMUNITY HEALTH RESEARCH

Society cannot afford to divert scarce resources into an ill-conceived, misrepresented universal childhood lead program when we have such critical
child health care needs as elimination of violence, teen pregnancy, premature birth, child abuse, drug and alcohol use, pediatric AIDS, and lack of immunizations and basic health care for a large segment of our childhood population. The recent decision by the CDC to replace the wasteful universal BPb screening program with targeted screening aimed only at high-risk children reflects belated recognition of this fact.

***Acknowledgment*--The Medical Editing Departrnent Kaiser Foundation Research Institute, provided editorial assistance.**

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