Round and Round It Goes: The Epidemiology of Childhood Lead Poisoning, 1950–1990

BARBARA BERNEY

Boston University

"LEAD IS TOXIC WHEREVER IT IS FOUND, AND IT is found everywhere." The 1988 report to Congress on lead poisoning in children by the Agency for Toxic Substances and Disease Registry (1988) thus neatly summarized the last 25 years of epidemiological (and toxicological) studies of lead.

Lead has been a known poison for thousands of years. The ancient Greeks described some of the classical signs and symptoms of lead poisoning: colic, constipation, pallor, and palsy (Lin-fu 1980). Some historians suggest that lead acetate used by the Romans to process wine contributed to the fall of the Empire (Mack 1973). Despite its known toxicity, lead use in the United States increased enormously from the industrial revolution through the 1970s, especially after World War II. Between 1940 and 1977, the annual consumption of lead in the United States almost doubled. In the 1980s, largely as a result of regulation of lead in gasoline, lead use in the United States leveled off and began to decrease.

In this article I explore the interaction of epidemiology and social forces in the continuing evolution of knowledge about the effects of low-level lead exposure, the extent of the population's exposure, and the sources of that exposure. I will concentrate on the effects of lead on the central nervous system (CNS) of children.

The Milbank Quarterly, Vol. 71, No. 1, 1993

In the United States, the problem of lead poisoning and low-level lead exposure has been pursued with some consistency over the last 25 to 30 years. Research done in the 1950s and 1960s was generally confined to studies of the symptoms, diagnosis, and treatment of acute lead poisoning in low-income children suspected of eating the paint and plaster in their deteriorated housing. However, the questions raised by the research have expanded rapidly to broader political and epidemiological questions: What is the proper definition of an "adverse health effect"? What levels of exposure cause those effects? Is there a threshold below which no effects occur? Who is exposed to how much lead, and from what sources? What are the pathways of exposure?

Increasing attention to the epidemiology of lead coincided with what Vandenbrouche (1990) calls the "second wave of vocational epidemiology," which began in the 1950s. "Vocational epidemiology is epidemiology based on a profound, personally felt vocation to improve the fate of mankind by fighting the environmental and societal causes of disease" (Vandenbrouche 1990). Increasing interest in lead also coincided with epidemiology's transition to the study of noninfectious diseases. At the turn of the century, public health made a conservative turn away from the environment toward infected and infectious individuals as the source of disease. By the 1950s the trend was turning back toward the environment (Ozonoff 1988).

The problem of lead poisoning was perfectly suited to these developments in epidemiology. Lead poisoning had an environmental cause, exacerbated by social problems associated with poverty including slum housing, racial discrimination, and malnutrition. Its identified victims were children—poor black children—who continue to be at the greatest risk for lead poisoning even within the currently expanded definition of the population at risk. Not only did concerns about lead poisoning fit the emerging epidemiological agenda; they also were in tune with the emerging political agenda of the movement for civil rights and social justice. The problem of lead poisoning provided an opportunity for a generation of public health professionals to combine their professional, personal, and political goals. These public health professionals worked with community activists to establish and carry out screening programs. They presented the results of their research at public hearings, testifying for legislation and regulations aimed at preventing exposure to lead, and they argued in scientific and political arenas with the lead industry for increasingly stringent regulation of lead.

Lead epidemiology can be seen as a series of interactive "rounds" in which case finding or screening increased awareness of the disease and expanded the defined populations at risk. The increased numbers of children at risk led to more intensive study of the effects of lead and a lowering of the lowest observed effects level (LOEL). Lowering the LOEL, by definition, increased the population at risk. Because exposure of the enlarged population at risk could not be attributed only to deteriorating lead paint and pica (the tendency to eat nonfood items), epidemiologists began to look for additional potential sources of lead in the environment and considered normal behavior patterns, such as hand-to-mouth contact in toddlers. Augmented sources of lead, in turn, meant a larger population at risk of exposure, and so on. Each step in this process carried with it political implications related to both the cause and prevention of exposure. I have chosen as my topic the last 30 years of rounds.

The medical literature of the first half of the twentieth century contains dozens of articles on childhood lead poisoning. In fact, by 1934 nine countries and Queensland, Australia, had decided, based on the existing literature, to ban or restrict the use of leaded paint (Rabin 1989). Most of this literature consists of case reports or reports on series of cases of children hospitalized for acute lead poisoning. Several articles describe the effects of lead poisoning, especially the neurological effects (Rabin 1989). However, after the introduction of tetra-ethyl lead as a gasoline additive in the early 1920s, lead research in the United States was dominated by the lead industry, particularly by Robert Kehoe. Kehoe was "the nation's most vocal and influential scientist working on lead hazards" (Graebner 1988) from the 1920s to the mid-1960s. From 1925 to 1958, he was also the medical director of Ethyl Corporation—manufacturer of tetra-ethyl lead—and director of the Kettering Laboratory, which received funding from the lead and automobile industry (Graebner 1988).

Round One: Midcentury to the Mid-1960s

Until the mid- to late 1960s, childhood lead poisoning was viewed as an acute disease leading to encephalopathy and was diagnosed in its early clinical stages only by the suspicious and informed physician. Lead levels in diagnosed children generally exceeded 80 micrograms per deciliter (μg/dL), and were often well above 100 μg/dL. The epidemiology of

this period, which consisted largely of case finding and case summaries, accurately identified the population at greatest risk; described the symptoms, methods for diagnosis, and causes of lead poisoning; and suggested most of the neurological sequelae that were to be studied for the next 30 years. Researchers consistently found that prolonged and repeated exposure was more often associated with significant neurological damage than even large single exposures. Therefore, they frequently reiterated the need for eliminating lead from the environment of children in general and poisoned children in particular (Mellins and Jenkins 1955; Eidsvold, Mustalish, and Novick 1974; Jacobziner 1966).

In the 1950s, a few clinicians in children's hospitals and health departments in several eastern cities became concerned with the cases of childhood lead poisoning that they saw and treated. Their interest resulted in expanded case finding and in research based on case summaries of the long-term neurological sequelae of exposure to lead.

Extensive field studies were carried out in Baltimore, Chicago, and New York City (Blanksma et al. 1969; Specter and Guinee 1970; Guinee 1971). The Baltimore City Health Department, in cooperation with the department of pediatrics at Johns Hopkins and the University of Maryland medical schools, had the nation's largest program of case finding and research on the treatment and evaluation of lead-poisoned children. Julian Chisolm, one of its leaders, has been a prominent researcher on childhood lead poisoning for more than 30 years. As early as 1951, the Baltimore Health Department (1971) completed a study of 293 lead cases that occurred in children from 1931 to 1951. The results showed that most cases occurred in two-year-old children and that incidence was greatly increased during the summer months. The children "lived in old rented properties and ate paint flakes or chewed on windowsills" (Eidsvold, Mustalish, and Novick 1974). These demographics and case characteristics were confirmed in other cities with expanded case-finding programs and in the results of larger studies over the next 30 years. Thus the population at greatest risk, the source of the poison, and the seasonal variation of the poisoning were all identified in the early 1950s (Eidsvold, Mustalish, and Novick 1974).

Although this information was available in respected medical journals for more than a decade, few cities took action to prevent exposure of children to lead paint. The exception was Baltimore: presented with evidence from its health department, the city attempted primary prevention through legislation and succeeded, in 1951, in passing one of the country's earliest lead paint laws. The law prohibited the use of

paint with "any lead pigment" on interior surfaces, but, like many other lead paint laws, it was rarely enforced. In 1958, after recording its highest-ever number of lead-poisoning cases and deaths—133 cases and 10 deaths—the city passed another ordinance requiring that all lead-containing paint be labeled with a warning against its use on interior surfaces, furniture, toys, windowsills, or any place used for the care of children. This law also proved ineffective in preventing lead poisoning. Twenty-six years later, in 1984, 75 percent of Baltimore's housing was believed to contain lead-based paint. It was projected that some 6,000 Baltimore children had suffered lead-paint poisoning (Agency for Toxic Substances and Disease Registry 1988).

Although the clinical case finding done in the 1950s and early 1960s was based on identifying overt symptoms of lead poisoning, investigators began looking for neurological sequelae of lead exposure. Their studies included children with relatively mild symptoms of poisoning and their collected evidence suggested that asymptomatic levels of exposure might cause CNS effects. These findings led clinicians and public health officials to suggest that the cut-off between normal and elevated blood lead levels was too high. The Baltimore group consistently suggested that the upper limit of normal be lowered: first, in the 1950s, from 60 to 80 μg/dL down to 50 μg/dL; then, in the 1960s, from 50 or 60 down to 40 μg/dL (Lin-fu 1972).

The definition of "normal" blood lead levels was a political as well as a scientific controversy. During the late 1920s, and in the 1930s and 1940s, Robert Kehoe, supported by the lead industry, wrote extensively on the presence of lead in the environment and on its uptake and excretion by humans. He concluded that lead occurred naturally, including in human tissues and excreta. He further argued that the body did not store lead as a result of exposure to "naturally" occurring amounts of lead, but rather that it established an equilibrium between lead intake and elimination, and that "beyond the point of equilibrium absorption did not occur" (Graebner 1988). He argued further that these facts meant "there was no necessary relation between lead absorption and lead intoxication—no necessary connection between lead concentration in feces, urine, or tissues and lead poisoning" (Graebner 1988).

To refute these widely accepted arguments, much research carried out during the 1950s, 1960s, and 1970s was designed to show the following:

1. Lead in the environment was a result of human use of lead in industry.

2. Lead accumulated in the human body in proportion to the amount of lead found in the environment.
3. Lead was absorbed by the body from the environment.
4. Such absorption, measured in feces, urine, blood, and other tissues, was an indication of exposure and poisoning.

Additional controversy resulted from the fact that falling "hazardous" levels not only threatened the lead industry, but also increased the need for public health programs to find and treat poisoned children and to eliminate lead exposure. Resources required for such programs could be immense. For example, in 1957, Baltimore mounted the first large-scale screening for lead paint, one of the few programs ever to look first for lead in paint and only secondarily for lead in children. The program aimed to "assess the prevalence of lead paint in Baltimore homes . . with a view to its possible removal as a preventive measure" (Baltimore Health Department 1971). Mass screening of paint was possible because a rapid screening test had just been developed by the city's Bureau of Laboratories. Of 667 dwelling units tested in 1957, 70 percent had lead in excess of 1 percent.

In 1961, after testing thousands of dwellings and children, the health department suggested extensive removal of lead paint from housing in the areas where childhood poisoning was the highest. The department encountered tremendous opposition from landlords, who did not want to bear the expense of removing lead paint from rental units. After calculating that 100 person-years of sanitarian time would be required to enforce removal, the city rapidly abandoned the notion of preventive removal (removing paint before a child is poisoned in the dwelling unit) as too expensive (Shucker et al. 1965). Although some states and cities have legislation requiring lead removal prior to poisoning, it is hardly ever enforced. This means that, since 1961, virtually no jurisdiction has used primary prevention to eliminate lead paint poisoning. Instead, public health departments have relied on screening children's blood as the warning system for poisoning. Only in the last few years have cost-benefit studies been developed to show that the cost of damage done by lead paint outweighs the cost of its removal (Florini, Krumbhaar, and Silbergeld 1990; Szabo and Pollack 1987). These studies relied on 20 years of research that showed adverse health effects at blood lead levels from 10 to 15 μg/dL, or even less. Several programs to eliminate lead paint from dwellings have been proposed recently (Needleman 1989; Florini, Krumbhaar, and Silbergeld 1990).

Several researchers working in the 1950s studied the neurological sequelae of lead poisoning. Mellins and Jenkins (1955) studied the mental and emotional development of the children in a Chicago cohort. Their findings suggested that symptoms of CNS involvement preceded hospitalization. Furthermore, symptoms and effects that were noted either while the children were hospitalized for poisoning or while they were undergoing follow-up examinations suggested virtually all the subtle damage reported in much later studies of low-level exposure: speech problems, especially in the naming of objects and conceptualization, which would "limit the symbolic processes so necessary to mature verbal behavior" (Mellins and Jenkins 1955); problems with visual motor coordination, especially fine motor coordination; distractibility; and short attention span. The authors concluded that improved housing and the elimination of lead paint was "essential to prevention." The Chicago study and one by Smith (1954) came to virtually identical conclusions. These studies considered 50 to 60 μg/dL as normal blood lead levels and 70 to 80 μg/dL as levels indicative of frank poisoning.

In 1959, Byers, who worked at the Children's Hospital in Boston, and whose studies of childhood lead poisoning spanned 40 years at the time of his death a decade ago, published his classic review article on lead. He cited the known findings, but his work also suggested that lead poisoning might be a much bigger problem than previously indicated. He observed that some poisoning might not be attributable to pica, but rather to the normal mouthing behavior of children in environments where paint contained very high levels of lead. (This observation had been made in 1904, but had been lost or forgotten [Rabin 1989].) Byers noted that intact, as well as peeling, paint could represent a hazard. He reported that some researchers had noted that poisoning might occur at blood levels below 60 μg/dL, even at levels as low as 40 μg/dL, although he concluded that levels above 60 μg/dL were generally agreed to be pathological. He further reported the presence of lead in umbilical cord blood and in infants less than six months old. He suggested as well that chronic exposure or reexposure to lead after treatment appeared to result in greater risk of retardation than single, or short-term, high-dose exposure that was properly, adequately, and quickly treated (Byers 1959).

The field epidemiology and case finding of the 1950s laid the foundations for later work by indicating that increasingly intensive case finding or screening would uncover more cases and prevent death; by accurately describing the population at greatest risk; and by providing provocative data on the neurological sequelae of the disease that no

doubt formed the basis of the cross-sectional and prospective studies of low-level exposure reported from the mid-1970s into the 1990s.

In the 1940s titanium oxide began to replace lead as the pigment of choice for white paint; and, in 1955, the American National Standards Institute adopted a voluntary limit of 1 percent lead for paint. Although many people believed that these changes would eliminate the problem of lead paint poisoning, this was not to be. Paint stocks with a lead content greater than 1 percent continued to be produced. Furthermore, a 1 percent limit was not adequate to protect the health of children exposed to it. Moreover, although changes in the production of new paint represented effective toxic use reduction, they did nothing to remove highly leaded paint from existing housing. Thus, although interest in the problem ebbed at least until the mid-1960s, the problem persisted.

Round Two: The Mid-1960s Through the 1970s

In the 1960s lead poisoning was characterized as epidemic in scope and was named a national health problem. Lead paint in deteriorated housing continued to be seen as the major source of lead exposure. Politically active health professionals, in cooperation with community groups, began screening programs, which were later expanded and sponsored by city agencies and which provided evidence of a widespread problem. Civil rights and progressive political groups challenged the medical and governmental establishments to do something to abate the problem. In 1970, the surgeon general issued a formal statement on lead poisoning (U.S. Department of Health, Education and Welfare 1970). The following year Congress passed the Lead Paint Poisoning Prevention Act, which eventually provided funds for greatly expanded screening programs that offered further evidence of an even more widespread problem.

Lead was a perfect issue for the emerging social and political movements of the 1960s. Lead poisoning highlighted many problems of concern: the lack of preventive health care, the focus of medicine and public health on the individual to the exclusion of the environment, the lack of community services in low-income neighborhoods, and the rela-

tion between poverty, racial discrimination, and health. Because the population at greatest risk was poor and black, lead poisoning could be defined in terms of race and class.

Lead poisoning resulted from bad housing conditions. Blacks were forced into bad housing by discrimination. The housing was allowed to deteriorate by gouging landlords and city housing officials, who did little to eliminate the problem or protect the health of the children affected. Lead poisoning could be used to tie the emerging environmental movement (and the reemerging consciousness of the environment in public health) to civil rights issues.

Lead poisoning was preventable, but, if neglected, it could permanently disable or kill its victims. Prevention required a low-tech, community-level environmental intervention, whereas cure required painful, expensive treatment in a hospital. Prevention could be carried out by low-skilled members of the community who needed work. Cure involved overworked doctors and crowded hospitals. Well-designed prevention programs required concerned community workers to canvas door to door and raise people's consciousness about the connection between bad housing and children's health and to inform parents about available services.

Lead carried tremendous symbolic power: It was a poison. It was deceiving—hidden in sweet-tasting paint and plaster on the very walls of homes. Its victims were innocent and already disadvantaged. It could be used as a symbol of what was wrong with society: the indifference of landlords, government officials (especially health and housing officials), and industry (paint and gasoline makers). Articles appearing in popular magazines, such as *Time*, *Reader's Digest*, *Saturday Review*, and *Good Housekeeping*, during the 1960s and early 1970s made these points and used these symbols (*Time* 1969; *Scientific American* 1969; Block 1970; Craig 1971; Remsburg and Remsburg 1972; *Parents' Magazine* 1973).

After the War on Poverty was declared in 1964, Medicaid and community health centers made resources available for addressing the lead problem. Doctors and community activists, who were often employed by hospitals and new federally funded neighborhood health centers that served inner-city communities, began advocating and organizing screening programs for children with symptomatic lead paint poisoning in Chicago, New York, Philadelphia, and other cities. They were assisted by progressive organizations of scientists and professionals that were formed during this period to focus attention on the social and political

implications of scientific and health policy and research, among them Science for the People, Scientists for Public Information, Center for Science in the Public Interest, Medical Committee for Human Rights, and Physicians for Social Reform.

The issue of lead poisoning appealed to many staff members of health centers and hospitals in low-income communities. Many staffers were newly graduated physicians who chose to work in federal jobs in lieu of military service in Vietnam. Others were conscientious objectors to the war who had found alternative service jobs in health care. Still others were drawn to health care, especially in the inner cities, because they saw it as a means of pushing for social change and having a professional career at the same time. Departments of community and social medicine were established or expanded at many medical schools in the 1960s (David Rosner 1990, Benjamin Siegel 1990: personal communications). These departments provided a base for people concerned with environmental and community health issues and programs.

The push for expanded lead screening grew from the inference that if intensified case finding led to the identification of larger numbers of cases, then screening—more systematic and widespread case finding—would identify even more cases. The lead belts—old, deteriorated housing inhabited by children—had been defined. Organizers and selected health professionals in lead-belt communities first put together volunteer-based screening programs. Then, when those programs found high numbers and rates of cases, they went to local governments to demand funds for expanded, well-organized screening programs.

In the mid-1960s, in Chicago, after failing to convince the city council to establish a screening program, a group of health activists started one themselves, using volunteer professionals and concerned members of the community (Quentin Young 1990: personal communication). Shortly after this program began, two children were admitted to the hospital with acute lead poisoning and died. The newspapers picked up the story and, in 1965, responding to pressure from the Citizens' Committee to End Lead Poisoning, the American Friends Service Committee, and the Medical Committee for Human Rights, the Chicago Board of Health began the first large-scale screening for lead poisoning in the United States (Quentin Young 1990, Jane S. Lin-fu 1990: personal communications; Lin-fu 1979). This screening program found that from 5 to 15 percent of the children screened had excess body burdens of lead, defined as blood lead levels in excess of 50 μg/dL (Lin-fu 1990: personal

communication; Lin-fu 1979). The Chicago findings encouraged community and professional groups in other communities to pursue similar efforts.

The screening programs that began in the 1960s resulted in the "discovery" of large numbers of cases. In New York, the number of reported cases grew from 116 in 1958 to 700 in 1968–1969. The screenings carried out in several large cities during the years 1967–1970 showed that from 25 to 45 percent of one- to six-year-old children living in high-risk areas had blood lead levels (pbB) exceeding 40 μg/dL (considered at the time to be the upper limit of normal).

> Most of these children had no symptoms of lead poisoning. Suddenly, undue lead absorption unassociated with overt clinical evidence of toxicity gained recognition as a phenomenon which required careful investigation because of the enormous number of young children involved. (Lin-fu 1979)

The data provided indisputable and overwhelming evidence that the lead-poisoning problem was immense.

Increasing evidence about the average lead levels in urban populations compared with levels in geographically remote and preindustrial populations defeated Kehoe's model of naturally occurring lead in the environment. Accumulating evidence both of adverse health effects at ever-decreasing blood lead levels and of lead being stored in the body from all sources similarly challenged Kehoe's argument that the human body established a natural equilibrium between lead intake and elimination. These changes in the definition of "natural" forced down the definition of the upper limit of normal blood lead levels.

Evidence on lead levels was carefully accumulated by researchers who wished to show that existing levels in urban populations were a result of increasing industrial use and pollution and not, as the lead industry argued, "naturally occurring" (Patterson 1965; Shapiro, Grandjean, and Van Neilsen 1980). Studies of levels of lead at various depths in the arctic ice showed them increasing with time, especially after World War II. Studies on the remains of ancient Nubians and Peruvians demonstrated levels of lead in bone and teeth 100 times lower than those found in current urban populations (Shapiro, Grandjean, and Van Neilsen 1980; Ericson, Shirahata, and Patterson 1979). These studies were constantly cited in articles about what blood lead level should be considered "nor-

mal" (Agency for Toxic Substances and Disease Registry 1988; Lin-fu 1980, 1985; Cohen et al. 1973; Environmental Protection Agency 1977, 1986).

The combination of the number of children found to have elevated blood lead levels and the evidence that elevated levels were not "normal" led to greatly increased interest in exploring the biological and behavioral effects of low-level exposure, and in determining whether or not these effects were adverse and at what level.

The 1970s: The Surgeon General's Statement, the Lead Paint Poisoning Prevention Act, and a Decade of Screening

In 1970, the surgeon general issued a statement that shifted the focus in lead poisoning from case finding and treatment of overt lead poisoning to its prevention through mass screening of young children and the termination of hazardous exposure for children with evidence of undue lead absorption. It defined "undue exposure" as a blood lead level of 40 μg/dL at a time when 45 percent of the children screened in New York City had blood leads above this level (U.S. Department of Health, Education and Welfare 1970). The burden on local health departments was immense. The justification for choosing this blood lead level, then considered to be asymptomatic, was that time was needed to remove a child from leaded surroundings after "undue exposure" had been noted and before poisoning occurred.

The agitation, epidemiology, and publicity of the late 1960s led to congressional hearings on lead poisoning and the passage of the Lead Paint Poisoning Prevention Act of 1970. This act marked the beginning of two decades of often ambivalent government investigation and regulation of lead. Under its provisions, the Centers for Disease Control (CDC) funded the screening of close to four million children from 1972 to 1981 (Lin-fu 1985). Thus, the 1970s became the decade of screening.

The early 1970s also brought dramatic changes in screening techniques. In 1973, erythrocyte protoporphyrin transformed testing for lead poisoning. The new tests were much cheaper in terms of equipment, consumable supplies, and skill required to take and process blood samples. They used finger stick rather than venipuncture techniques. Results of the tests could be obtained on site in a few minutes, thus lim-

iting loss to follow-up. Because the procedure did not test directly for lead, it was not subject to environmental contamination.

Screening Results Expand the Population at Risk

Data from the screening programs of the late 1960s through the 1970s had a profound effect on the understanding of who was affected by undue lead absorption. Surveys in the early 1970s indicated that the problem was not confined to large urban slums and areas east of the Mississippi River. Fourteen cities with populations ranging from 10,000 to 150,000 were found to have problems comparable to those in large cities (Lin-fu 1979). A Department of Health, Education and Welfare (DHEW) survey of 52 communities throughout the nation revealed that undue lead absorption among children was geographically widespread, occurring in cities of every size and in rural areas as well (Lin-fu 1979; Cohen et al. 1973). "The clearly defined borders of lead belts began to disappear when screening extended beyond them" (Lin-fu 1979). Although poor black children in the inner cities were still at highest risk for poisoning, excessive lead absorption affected urban middle-class and even rural children of every race, making it perhaps the largest preventable childhood health problem in the nation.

In the early 1970s, biological and epidemiological findings on lead absorption and enzyme and CNS effects led to greatly increased concern about low-level exposure in children. King et al. (1972) suggested that children might absorb lead from the gut more efficiently than adults. A year earlier, King (1971) had estimated the "maximum safe daily dose," assuming absorption of 10 percent of ingested lead. By 1974, at least one study had confirmed that children absorbed close to 50 percent of the lead they ingested (Alexander 1974). These new findings represented a quintupling of the absorption rate for children, and dramatically decreased the amount of lead that children needed to consume in order to raise blood lead levels to the level of concern. These findings were enough to change the thinking about the importance of pica in cases of undue lead absorption (Lin-fu 1973; Sayre, Charney, and Vostal 1974). The suggestion that normal hand-to-mouth activity in normal ambient environments could cause undue exposure was confirmed, which meant that many more children were at risk.

During the same period several studies found that inhibition of

amino levulinic acid dehydratase (ALAD), an enzyme involved in hemoglobin synthesis, showed a continuous dose–response to blood lead levels ranging from 5 to 95 μg/dL (Hernberg et al. 1970; Hernberg and Nikkanen 1970; Millar et al. 1970; Secchi, Erba, and Cambiaghi 1974). These findings indicated that lead might affect hemoglobin production at blood levels as low as 5 μg/dL. Retrospective studies suggested that mental retardation and learning disabilities occurred in children previously considered asymptomatic (de la Burde and Choate 1972, 1975; Perino and Ernhart 1974; Rummo 1974). In 1973 an article appeared in the *Journal of the American Medical Association* comparing blood lead levels in rural and urban populations and suggesting that 40 μg/dL might be too high for a definition of "undue absorption" (Cohen et al. 1973). The author referred to other studies of screened children and articles on hyperactivity in children with low-level lead exposure that had appeared in *Lancet* the previous year (David, Clark, and Voeller 1972). Although these findings were disputed and challenged, they increased the pressure for additional research on CNS effects at low exposure levels.

CNS Effects of Low-level Exposure

Byers and Lord (1943) and Byers (1959) had discussed neurological sequelae in the earlier literature, noting that continued exposure appeared to cause greater damage to the CNS than did even extremely high, but short term, exposures. A study by Perlstein and Attala (1966) examined 425 survivors of lead poisoning and found that 39 percent suffered neurological sequelae. Case-control studies of children with minimal brain dysfunction and hyperactivity found that children with these symptoms seemed to have higher levels of lead than controls (Cohen and Ahrens 1959; Thurston, Middlekamp, and Mason 1955; David, Clark, and Voeller 1972). These early analytical studies of lead and neurobehavioral effects (NBEs) were criticized because they did not account for a wide range of background variables that might affect outcome, especially race, class, and parental IQ and education (Rutter 1980; Ernhart, Landa, and Schell 1981; Harvey et al. 1984; Smith 1985). By incorporating the newest epidemiological methods, however, they had taken a step forward from earlier case reviews.

Other studies also found neurological dysfunction in children at progressively lower lead levels. De la Burde and Choate (1972) found dys-

function in children above 30 μg/dL, but with a mean blood lead of 59 μg/dL. In a follow-up study on the same children (1975), the authors found continuing evidence of neurological damage and a greatly increased rate of school failure and school problems. Although these and other case-referent studies done in the 1970s matched cases and controls by race and other social and economic variables (de la Burde and Choate 1975; Kotok et al. 1977; Rummo 1974), these studies were mainly criticized for their lack of control of confounding background variables (Rutter 1980; Ernhart et al. 1981; Harvey et al. 1984; Smith 1985). Perino and Ernhart (1974) also found neurobehavioral deficits in children whose lead levels ranged from 40 to 70 μg/dL; however, when these data were reanalyzed to control for various methodological problems, they failed to show statistically significant differences between high- and low-lead children. The most repeated and significant criticism of these studies was their failure to rule out the possibility that children of lesser intelligence either had habits, like an increased tendency to put nonfood items in their mouths, or came from social and physical environments that caused them to ingest more lead than smarter children. That is, the studies were criticized for failing to rule out the cases of lead exposure *resulting from* neurobehavioral problems rather than *causing* them. Nevertheless, the differences detected between high and low lead groups in these studies were consistent and lent credibility to the idea that asymptomatic children with lead levels of between 40 and 80 μg/dL were suffering adverse effects.

As a result of criticisms of these studies for their poor control of confounding variables, later studies turned to restricting the domain of the study base, first to whites and then to upper- and upper-middle-class whites, as a means of controlling ill-defined and hard-to-measure background variables. The study by Needleman et al. (1979) of schoolchildren in Chelsea and Somerville was a landmark examination of the NBEs of low-level lead exposure. Needleman's subjects were almost all white, working-class children. All of them came from English-speaking households. Needleman collected deciduous teeth from 2,146 children and analyzed them for lead content. He wanted to use tooth lead because he was interested in cumulative rather than recent lead exposure. None of the subjects in the study had a known history of lead poisoning. He tested 58 children in a high-lead group and 100 children in a low-lead group, using several cognitive and other measures of CNS function. He also asked the teachers of all the children from whom he

collected teeth to evaluate their classroom behavior. Controlling for 39 confounding variables, he found lead effects in IQ and adaptive classroom behavior. Needleman had few data available to correlate tooth lead level with blood lead level, although he was able to find blood lead levels for several subjects in his study. They indicated that his low-lead subjects probably had blood lead levels in the range of 12 to 37 μg/dL, whereas the high-lead group was in the range of 15 to 54 μg/dL (Rutter 1980).

Because this was a cross-sectional study, the data could not be used to show that lead exposure preceded CNS effects. Nevertheless, the study was key to moving the discussion of low-level effects forward. It was a study of white, working-class children in the general public school population. None of the subjects had any overt symptoms of poisoning, yet the effects of lead on classroom performance were statistically significant across the entire range of exposure. Differences in IQ and attention were significant between the high- and low-exposed groups (Needleman et al. 1979). This study also took advantage of computer techniques and programs for multivariate analysis that came into common usage in epidemiology in the 1970s.

During the late 1960s and throughout the 1970s, researchers learned an enormous amount about the metabolism and biological effects of lead through the study of animals. These studies greatly advanced the understanding of the biochemistry of lead from subcellular effects to the impact of the inhibition of heme synthesis on various organs. Models were also developed for studying NBEs in animals, including hyperactivity and learning (Silbergeld and Goldberg 1973; 1974a,b). These studies provided plausible biological explanations of the human epidemiological data.

The Overlapping Debate: Where Does the Lead Come From? (1960–1988)

Lead in Air

Concern with low-level exposure and growing awareness of widespread exposure led researchers to examine sources of lead other than paint. Lead in air became an important subject of research and debate. In examining the relation of lead in air to lead poisoning, several questions

had to be addressed: Did lead in the air cause exposure? That is, was exposure to air with higher concentrations of lead associated with higher blood lead levels? If air lead contributed to the lead body burden, did it cause poisoning? How much of the lead body burden could be attributed to air lead? Where did the lead in air come from? These questions were complicated by the fact that much of the lead in air was not inhaled or absorbed directly, but rather was deposited on dust and soil and then ingested or inhaled.

Lead in Air and Gasoline

The initial battles over the health effects of lead in gasoline were fought when tetra-ethyl lead was first added to gasoline as an antiknock additive in the early 1920s. Leaded gasoline was actually banned in several cities while studies of its potential effects were carried out. By 1925, the public health forces had been soundly defeated by the lead industry, ensuring the "accumulation of tons of lead dust on every New York City street" and the streets of every city and town across America, just as Yale physiologist Yandell Henderson had predicted in 1925 (Graebner 1988).

In 1958, the Ethyl Corporation, manufacturer of tetra-ethyl lead, asked the surgeon general for advice on increasing the concentration of lead in gasoline. A committee, established by the surgeon general, reported back that "the proposed increase in lead apparently would pose no health hazard" (Graebner 1988), but asked for additional research on atmospheric lead. The surgeon general commissioned a study, managed by the Public Health Service, but conducted with the cooperation of the automobile industry, gasoline producers, and Kettering Laboratory. The resulting "Tri-City Study" concluded that levels of airborne lead were lower in 1961 and 1962 than they had been 25 years earlier. At the 1966 Senate hearings on air pollution Clair Patterson of the California Institute of Technology accused Robert Kehoe of conducting a whitewash in his analysis of the data, and pointed out that lead levels in U.S. cities were 100 times higher than they had been in the mid-1930s. He further challenged the role of industry in public health research. "It is not just a mistake for public health agencies to cooperate and collaborate with industries in investigating and deciding whether public health is endangered—it is a direct abrogation and violation of the duties and responsibilities of those public health organizations" (Graebner 1988).

In 1971, the year after Congress passed the Clean Air Act and the

Lead Paint Poisoning Prevention Act, the Environmental Protection Agency (EPA) put together a working seminar on lead. That year the working group received a position paper that reviewed the available research in order to determine "the contribution of atmospheric lead to the endangerment of public health" (Engel 1971). It stated that nonoccupational exposure to air lead might increase body burden, but that evidence from available studies was uneven. However, it concluded that settled lead in dustfall in the streets and soil "is sufficient to produce poisoning. . . ." The report also noted that heavy traffic increased lead dustfall significantly, suggesting that gasoline may be an important source of environmental lead contamination. Although it acknowledged that lead paint was the source of poisoning in most children, it noted that lead is accumulated from all sources and stored in the body. Therefore, the paper suggested, air lead could push children over the edge from a nonpoisoned to a poisoned state. Over the next 10 years, evidence accumulated to show that some 50 percent of children's blood lead could be attributed to lead in gasoline.

In 1971, Dr. Lin-fu of DHEW wrote to Irwin Billick, program manager for Lead-Based Paint Poisoning Prevention Research at the U.S. Housing and Urban Development Department (HUD). In her memorandum, Dr. Lin-fu outlined the nature and extent of lead-based paint poisoning in the United States by reference to several papers and screening data from several cities. She asked HUD to concentrate its efforts on finding methods to remove lead from residential housing as required by the act (Lin-fu 1971). While examining data collected by screening programs in New York City from 1970 to 1976, Billick noticed that blood lead levels were dropping and he turned to falling air lead levels for an explanation of these data. He obtained data from a single air-monitoring station in New York City and noted that blood lead levels tracked air lead levels very closely (Billick, Curran, and Shier 1979, 1982).

There were two main sources for lead in air: point sources, which were usually lead smelters, and mobile sources, which were cars that burned gasoline containing tetra-ethyl lead. A series of lead smelter studies in the 1970s looked at lead levels of children living at various distances from smelters (and at CNS effects of lead in those children). These studies showed that subjects who lived closer to the smelters had higher blood lead levels than children living farther away. The studies also measured air lead and lead in dust, soil, paint, water, and other media. They demonstrated that leaded air emissions from point sources contributed to lead in air, soil, and dust and to lead body burden and

that lead was absorbed both from the air and from dust (Yankel, von Lindern, and Walter 1977; Landrigan et al. 1975, 1976). In its 1977 *Air Quality Criteria Document*, the EPA states: "The conclusion to be drawn from (these studies) is that people who live in the vicinity of a major industrial source of lead are exposed to abnormally high lead concentrations" (Environmental Protection Agency 1977).

Several studies of the relation between lead isotopes in the environment and in blood were carried out to determine the sources of lead and the amount of body burden that could be attributed to each source (Manton 1977; Garibaldi et al. 1975). Manton determined that 7 to 41 percent of blood lead came from air (through gasoline). The isotope lead experiments showed that gasoline was responsible for 90 to 95 percent of lead in air (Agency for Toxic Substances and Disease Registry 1988). The combined effect of these studies was to defeat Kehoe's position that exposure to lead in air would not necessarily lead to either increased absorption or a greater lead body burden.

From 1975 to 1984, gasoline lead consumption fell by 73 percent because of EPA regulation of lead in gasoline and the introduction of catalytic converters that required the use of unleaded gasoline; lead levels in air fell by a similar amount over the same period (Agency for Toxic Substances and Disease Registry 1988).

From 1976 to 1980, the National Center for Health Statistics carried out the second National Health and Nutrition Examination Survey (NHANES II), collecting data on a stratified random sample of the U.S. population. Almost 10,000 blood lead determinations formed part of the data collected. The results were shocking.

Median blood lead levels for the U.S. population as a whole were 13 μg/dL. The median level in children (aged six months to five years) was 15 μg/dL; in black children it was 20 μg/dL. More important, the data collected for NHANES II exhibited significant time trends. From 1976 to 1980, blood lead levels dropped 37 percent, from 14.6 to 9.2 μg/dL. Regression models controlling for a large number of confounding variables showed that this reduction was almost entirely the result of decreased use of lead in gasoline. Similar time trends were observed in data from lead-poisoning screening programs (Environmental Protection Agency 1986; Billick et al. 1979, 1982; Annest et al. 1983; Schwartz, Janney, and Pitcher 1984).

The dramatic decline in the lead in gasoline between 1975 and 1980, which resulted from the introduction of catalytic converters that required unleaded gas, generated a natural experiment. Exposure data,

often the biggest problem in conducting environmental epidemiology, were extensive in the case of gasoline. The Department of Energy and the EPA had excellent records of how much lead was contained in gasoline sold on a monthly or quarterly basis by metropolitan area. The NHANES II survey and the various CDC screenings provided exact data on blood lead levels on given dates, both in the total population and in children. These data sets were combined and multiple regression analysis was used to determine the exact contribution of gasoline lead to blood lead (Billick et al. 1982; Billick, Curran, and Shier 1979; Annest et al. 1983). Isotope studies artificially varied the isotopes of lead in gasoline over time and found that the isotopes of lead in blood varied similarly (Garibaldi et al. 1975; Facchetti 1985). Time trend studies revealed that lead in blood decreased consistently with and shortly after the drop in leaded gasoline use. Taken together, these studies demonstrated conclusively that gasoline lead was responsible for perhaps half of children's blood lead on average and that decreasing lead in gas was extremely effective in reducing children's blood lead.

Using these studies, which were early examples of the use of large data bases for epidemiological studies, the EPA was able to determine the effects of gasoline lead on lead in blood and to predict the number and demographic characteristics of children and fetuses (and adults) who were at risk at different allowable levels of gasoline lead. The predictions were straightforward. They did not rely on complex and questionable models for tracking lead in air, nor on extrapolation either from animals to humans or from high doses to low doses, nor on highly uncertain mathematical models (Environmental Protection Agency 1985, 1986).

This research also showed that while poor children in inner cities were no doubt at the highest risk of lead poisoning, all children were exposed to lead from gasoline. Combined with a sophisticated cost-benefit analysis showing that the benefits of removing lead in gas outweighed the costs by more than 5 to 1 (Environmental Protection Agency 1985), these studies greatly facilitated government actions to lower allowable levels of lead in gasoline and provided the basis for defending those actions.

Other Sources of Lead

Once researchers had demonstrated that all sources of lead combined to elevate the body burden measured in blood or tooth lead, it became im-

portant to estimate the relative contributions of various sources of lead in the environment to total internal exposure; the contribution of different sources was directly relevant to debates about controlling lead, whether in gasoline, in emissions from stationary sources, or in house dust. Information on the contribution of different sources could be used to estimate both the effect (and effectiveness) of various control measures in reducing body burden and the number of people a particular control measure was likely to bring below a given "level of concern."

Studies were done to show that blood lead levels varied with the amount of lead on children's hands and that environmental contamination was a better predictor of blood lead levels than sociological or demographic factors (Milar et al. 1980). One study sent in "dust reduction teams" to wet mop dusty areas to control dust. This quasi-experimental study did show that the intervention lowered blood lead levels somewhat, but only during the period when the "teams" were working. Blood lead levels rose when the intervention ceased (Charney et al. 1983). At least some of these studies were part of a debate over whether childhood lead levels were caused by the degree of contamination of the environment or by parenting and housekeeping practices. Such arguments related directly to public policy prescriptions. Was lead poisoning to be blamed on poor education and parenting and controlled through education campaigns, or was it to be attributed to environmental conditions and controlled by abating those conditions?

Studies of soil and dust showed that they were contaminated by lead from both gasoline and paint. Although the studies revealed that increasing soil and dust lead levels were correlated with increasing blood lead levels, the percent of blood lead attributable to these sources was unclear. In 1986, Superfund provided for demonstrations and studies of the effect of removing heavily contaminated soil. The results of these studies were not published until 1991 and are beyond the scope of this article.

Data on sources of exposure, their occurrence in the environment, and their effect on blood lead levels allowed the Agency for Toxic Substances and Disease Registry (1988) and others (Florini, Krumbhaar, and Silbergeld 1990) to pinpoint the population at risk and to describe a series of overlapping exposure gradients that ranged from inner city to rural populations; from black to white; from old, deteriorated housing to new housing. The accumulated information on population blood lead levels and sources of exposure allowed the government and private organizations to estimate the number, percent, and location (by city) of chil-

dren with various blood lead levels by age, race, income, and location in or out of the center city.

Round 3: The 1980s

In 1970 Congress passed not only the Lead Paint Poisoning Prevention Act, but also the Clean Air Act, the Clean Water Act, and the Occupational Safety and Health Act. By 1980, the National Institute of Occupational Health and Safety (NIOSH) had produced a number of evaluations of the lead literature and at least two recommendations for setting standards. The Occupational Safety and Health Administration (OSHA) had promulgated an occupational standard for lead, lowering the allowable levels by 75 percent. The EPA had produced an air-quality criteria document for lead, set an ambient air standard for it, begun regulating lead in gasoline, and published documents related to regulating lead in drinking water. The Consumer Product Safety Commission had limited lead in paint to 0.06 percent. None of these agencies even existed before 1970. All of these activities required extensive review and evaluation of the epidemiology and other health literature on lead.

Several publications summarized the research on neurobehavioral effects of lead at the turn of the decade. In 1979 the Office of Maternal and Child Health of DHEW sponsored a conference entitled "Management of Increased Lead Absorption in Children: Clinical, Social and Environmental Aspects" and published its adapted proceedings in 1982 (Chisolm and O'Hara 1982). In 1980, Herbert Needleman edited *Low Level Lead Exposure: Clinical Implications of Current Research* and Michael Rutter published a careful review of the literature on the CNS effects of low-level lead exposure.

These reviews and compilations of the literature noted several problems and data gaps. In addition to the difficulty of controlling confounders, studies completed before 1980 shared a number of problems in demonstrating NBEs:

1. They lacked a single accepted indicator of dose or lead exposure.
2. The age, duration, and intensity of exposure that might cause NBEs was not known.
3. Outcome measures were not standard and were often not very sensitive.

4. Retrospective or cross-sectional studies could not demonstrate that NBEs occurred after exposure.

Several prospective studies carried out or reported in the 1980s were designed to remedy these problems. Longitudinal prospective studies were conducted, using birth cohorts whose blood lead levels were tracked from birth (from cord blood) or even before (using maternal blood lead levels as a marker for prenatal exposure). Neurobehavioral functioning was also tracked from birth, thereby enabling researchers to show that lead exposure preceded neurobehavioral deficits. Because blood lead was tested every three to six months from birth, a fairly reliable history of exposure was obtained, greatly reducing the risk of misclassification by exposure.

Repeated testing also gave some indication of the importance of timing, duration, and dose of exposure. Several of these studies used the same standardized measures of neurobehavioral functioning: the Bayley Mental Development Index, Stanford–Binet IQ, the Wechsler Preschool and Primary Scale of Intelligence (WPPSI), and the McCarthy Scales. Using multiple reliable, validated tests reduced the risk of misclassification by outcome, thus enhancing the studies' ability to find differences between the exposed and unexposed groups.

Confounding was controlled in two ways. First, information was collected on many confounding variables such as socioeconomic status (SES), home environment, parental IQ, perinatal disease, parenting practices, trauma, and family size. These covariates were controlled by statistical techniques. Second, studies were designed to limit the domain, so that confounders would not vary greatly across the population included in the study. For example, Needleman and Bellinger (Bellinger et al. 1984; 1985; 1986a,b; 1987a,b; 1989) and their colleagues studied a cohort of white upper- and upper-middle-class infants in Boston. Dietrich and Bornschein (Bornschein et al. 1989; Dietrich et al. 1986; 1987a,b) and their colleagues studied a cohort of inner-city children in Cincinnati. Control of confounding through limitation of the study domain, rather than through matching, was desirable because potential confounders were not only numerous, but were also difficult to define and to measure. Limiting variability reduced the possibility of errors caused by poor measurement of confounders such as SES.

Prospective studies also allowed researchers to address the issue of the persistence of lead's effects. By 1989, children had been followed in sev-

eral studies for approximately five years (Bellinger et al. 1987b; Ernhart and Morrow-Tlucak 1987). In 1990, Needleman published an 11-year follow-up study of the Chelsea/Somerville cohort he first reported on in 1979 (Needleman et al. 1990). Needleman found that effects such as school dropout rates and rank in class persisted into young adulthood. The birth cohort studies showed mixed results. Exposure at birth was inversely related to neurobehavioral performance up to 24 months of age (Bellinger et al. 1984; 1985; 1986a,b; 1987a,b; 1988; 1989) in some studies and up to six months in others (Dietrich 1986; 1987a; Bornscheinet al. 1989). In the Boston and Cincinnati studies, postnatal exposure was associated with longer-lasting deficits. In reviewing these studies, the EPA (1989) pointed out that, because of the low power of the particular studies involved, the positive results were more telling than the negative ones.

The prospective studies of birth cohorts in Boston, Cleveland, Cincinnati, and Port Pirie, Australia, were all of low-level exposure. The highest exposure levels were 25 μg/dL. Effects were observed in groups with exposure levels below 10 μg/dL. The medical community and the federal government concluded that 10 to 15 μg/dL was a level at which adverse NBEs occurred. In 1991, the CDC lowered the "level of concern" to 10 μg/dL to reflect this new information.

By the late 1980s, the epidemiological studies of lead and neurobehavioral deficits had finally met the requirements, summarized by Hill in 1965, for showing that an association is causal: the association was *strong* at high levels of exposure and was *consistent*, even at low levels of exposure. Deficits of four to six points on various intelligence scales were observed at exposures in the range of 10 to 25 μg/dL. The effects of lead were often, although not always, *specific*. (Hill was careful to point out that specificity should not be stressed too much as even bacteria may cause more than one effect; streptococcus, for example, can cause sore throats, heart disease, and skin infections.) Although the NBEs of lead, as measured by standard development and IQ tests, may not always be the same, its biochemical effects were well documented and specific. The *temporal* relation between lead exposure and NBEs has been demonstrated: exposure precedes effect. A *dose-response* relation was evident: extremely high exposures cause encephalopathy and death, lower doses cause severe retardation, and lesser doses lead to school problems, small but significant shifts in IQ, and other measures of CNS function. Huge numbers of in vitro and animal studies demon-

strated not only the *biological plausibility* of the observed effects, but also many of their physiological mechanisms. Finally, the evidence was *coherent*.

Although research on the health effects of lead continued apace in the 1980s, other aspects of lead-poisoning prevention changed substantially. Following Ronald Reagan's election in 1980, federal programs in lead-poisoning prevention were cut back. The CDC's Lead Poisoning Prevention Program, which had distributed $89 million in the decade preceding 1981, was subsumed, along with many other categorical programs, into maternal and child health block grants given to the states. The total amount of the block grants was less than the amounts previously provided for all the programs they replaced. States made their own programming decisions and the reporting, data collection, and federal technical assistance aspects of the individual programs were lost. Most states, in fact, continued to do some lead screening, but federal programs were dismantled. In 1982 HUD ended its lead research program.

Social activism decreased notably in the 1980s around all issues, not just lead, partly in response to decreases in funding for community organizations and public information and partly in response to growing conservatism. Political energy was concentrated in defending public programs eroded both by the Reagan administration and by increasingly cash-starved state and local governments.

Summary and Conclusion

In the 40 years from 1950 to 1990, lead epidemiology and public policy based on it made enormous strides. Exposure levels that caused concern in the medical and public health community fell from 80 to 10 μg/dL of blood. In the space of 20 years, beginning in 1970, first the surgeon general and then the CDC lowered the official "level of concern" from 50 or 60 to 10 μg/dL. The public health community has turned its attention from the prevention of poisoning that results in encephalopathy, mental retardation, and death to the reduction of exposure to avoid subtle neurobehavioral deficits that are detectable only in fairly large epidemiological studies.

Numerous advances in technology and analysis have facilitated progress in lead epidemiology. In order to show that intellectual deficits

were related to lead exposure rather than to such confounding variables as parental education, parental IQ, income, or parents' age at time of birth, researchers performed extensive regression analyses of fairly large sample populations, controlling for as many as 39 confounding variables. These analyses would have been virtually impossible but for the development of computer software programs that became available beginning in the late 1960s and early 1970s. Handling of large data bases, such as NHANES II, also required access to computer hardware and software not generally available earlier.

The existence of the data depended on other technological innovations that made screening of large populations inexpensive and relatively simple. In 1973, erythrocyte protoporphyrin screening transformed testing for lead poisoning. With the advent of atomic absorption spectroscopy and rapid improvement in equipment for blood lead analysis, obtaining blood lead levels became less expensive and easier and produced more accurate results. Increasing attention both to preventing environmental contamination of samples and to controlling laboratory quality also improved the accuracy of data collected. Advances in screening and analytical technology made the mass screening of the 1970s possible—both the programs coordinated by the CDC and NHANES II.

NHANES II showed the extent of the problem of lead exposure to be even greater than previously thought. It was estimated that in 1980 almost 2 percent of all children aged six months to five years had blood lead levels over the CDC level of concern of 30 μg/dL. More than 13 percent of black children had levels above 30 μg/dL. Lead was certainly the most widespread threat to child health in America. In 1985, when the CDC lowered the level of concern to 25 μg/dL, the population defined to be at risk tripled.

NHANES II, which represented the first population-wide data on internal lead exposure, combined with detailed data on leaded gas sales, allowed analysis of both the extent of the population's exposure to lead and the impact of lead in gasoline on that exposure. The NHANES II data set also allowed for analyses of lead in relation to many demographic and health variables such as housing, income, geographic location, race, nutritional status, anemia, blood pressure, and hearing.

Advances in research on the metabolism of lead and its subcellular effects and the development of animal models for the study of CNS and other effects provided important information that enhanced understanding of possible mechanisms of action and demonstrated the biolog-

ical plausibility needed to argue for the causal relationship between low-level lead exposure and CNS effects.

Lead is an example of how changing science policy actually redefined adverse health effects. Until the 1970s, only clinical manifestations of disease such as encephalopathy, frank anemia, wrist drop, or kidney damage were considered adverse health effects of lead exposure. As the ability to study more subtle changes improved, accompanied by a better understanding of their significance, researchers came to recognize that interference with heme synthesis and the production of other proteins, as well as subtle changes in neurobehavioral functioning, were adverse effects. The change in the definition of adverse effects reflected a policy change as well as a change in the ability to observe and measure biological changes and to understand their biological importance. These policy changes were facilitated by a combination of advances in scientific understanding of the mechanisms and natural history of disease and attention to risk factors that accompanied the rise of chronic disease epidemiology. Policy shifted also partly in response to the growing demands of the environmental and occupational health movements for a more preventive approach to exposure to environmental toxins.

The population at risk to the effects of lead has certainly affected the degree of interest in and attention to the problem of lead exposure. Lead epidemiology captured the public eye and the interest of scientists in the 1960s in part because the poor black children who constituted lead's most obvious victims were at the center of a growing movement for civil rights, economic justice, and social change. Some of the scientists and public health professionals who participated in this movement were attracted to the study of lead because of its political implications. Professionals working on lead were drawn into the political debates surrounding the implications of their work. Lead epidemiology rose on a crest of vocational epidemiology and has been carried forward by several groups of researchers committed to studying effects at ever-decreasing levels of exposure.

The differential exposure of poor and black children to lead (see table 1) has also affected the epidemiology of low-level lead exposure because being poor and black are considered confounders of intellectual achievement and appropriate school behavior—the outcomes of interest in studies of low-level lead exposure. Thus, much of the task of the last 20 years of lead epidemiology has been to show that lead exposure and absorption cause—rather than result from—neurobehavioral problems.

TABLE 1
Estimated Distribution of Elevated Blood Levels in U.S. Children, 1984

Children	Blood lead level		
	>15 μg/dL	>20 μg/dL	>25 μg/dL
All children[a]			
Number (%)	2,380,600 (17)	715,000 (5.2)	200,000 (1.5)
Poor black children in urban areas[b] (%)	(68)	(31)	(11)

Sources: Agency for Toxic Substances and Disease Registry (1988); Mushak and Crocetti (1990).
[a] Children aged six months to five years living in standard metropolitan statistical areas (SMSAs).
[b] Black children aged six months to five years living in poverty (annual family income below $6,500) in the central areas of SMSAs with a population greater than one million.

The counterargument—that neurobehavioral problems cause children to eat more lead—implied that poor, stupid black children, whose parents neither care for them properly nor keep their homes clean, eat more lead than "normal" children. When stated so baldly, the underlying racial and class prejudice of the argument becomes apparent.

Attention has increasingly focused on the effects of low-level lead exposure in part because of overwhelming evidence—especially data from NHANES II—of the size of the population exposed. The number of children (and adults) exposed to lead at levels that resulted in blood lead levels above 25, 15, and 10 μg/dL was so great that even relatively small shifts in such measures as IQ carried enormous social costs in terms of the number of children who would fall into the below-normal category or who might fail to achieve brilliance (Needleman 1990).

Lead has come to be recognized as a ubiquitous toxin presenting a hazard in soil, dust, and air as well as in more traditional sources such as paint. As the country grew more conservative in the 1980s and public concern with civil rights and social justice diminished, interest in the lead problem and in regulation to abate it was enhanced by the discovery of the importance of gasoline lead, which affected all children, not just those living in old housing who were disproportionately black and poor (see table 2). Although action to remove lead from gasoline was not swift, it was complete and effective.

TABLE 2
Estimated Numbers of Children Potentially Exposed to Sources of Lead—United States, 1984

Source	Date of concern	No. of children[a] (millions)
Lead-based paint	Hazards reported in the international literature since the 1920s. Reestablished and accepted in U.S. by the end of the 1960s. Banned (.06%) by CPSC in 1978. Still present in housing units.	12.0[b]
Lead from gasoline	First regulated in U.S. in 1973. Controversy dates to 1925 when lead additives were introduced. Analysis of New York City screening (1979) and NHANES II (1982 and 1983) data confirmed contribution of gasoline lead to blood lead.	5.6[c]
Stationary/industrial sources	1970s	0.2
Dust/soil	Some work done in 1977. Follow-up, increased concern in 1984.	5.9–11.7
Water/plumbing	Ban on lead solder in water pipes and systems in 1986. Proposed reduction of maximum contaminant level from 50 μg/L to 20 μg/L in 1988.	10.4
Food	In 1970, 90% of food cans contained lead solder; in 1986, 20% of cans contained lead solder.	1.0

Source: Mushak and Crocetti (1989).
[a] These numbers are not additive because children are usually exposed to multiple environmental sources of lead.
[b] Number of children under seven estimated to be living in unsound lead-painted housing in 1984.
[c] Number of children under seven living in the 100 largest SMSAs in 1984.

The falling observed-effects level, combined with evidence of significant dispersion of lead in the environment and its bioavailability from practically any source—paint, air, soil, dust, ceramic glaze, water—has expanded the population at risk to include some 17 percent of Ameri-

TABLE 3
Children Whose Blood Lead Is Above a Certain Level, by Main Source of Lead

	No. of children		
	Paint[a]	Gasoline[b]	Water[c]
>15	1.2 million	8.1 million	241,000
>20	0.5 million	2.6 million	
>25	0.2 million	0.8 million	

Source: Agency for Toxic Substances and Disease Registry (1988). Paint: tables VI-4 and VI-6; gasoline: table VI-8; water: page VI-44.

[a] Estimated number of U.S. children under seven years of age living in unsound, lead-based painted housing in 1984. This is an annual figure. Children move through this housing. Although there is overlap from year to year, each year additional children are exposed. The number of exposed children will not decrease over time without deleading.

[b] Estimated numbers of U.S. children aged six months to 13 years falling below indicated pbB (μg/dL) levels as a result of lead-gasoline phaseout. This number is the sum of children falling below indicated blood lead levels each year from 1985 to 1990. Decreasing numbers of children are exposed as lead is phased out.

[c] Estimated number of children under six years of age whose blood lead is above a certain level (241,000 > 15; 230,000 between 15 and 30) because of lead in their water as a result of the action of corrosive water on aged plumbing.

can children under six years, making lead the health problem that affects the largest number of American children (tables 2 and 3). Lead in food has been largely eliminated (Mushak and Crocetti 1990); the EPA has lowered allowable levels of lead in drinking water, but existing lead paint lingers, exposing millions of children—mostly poor and disproportionately black—annually. Regulatory efforts to eliminate lead paint from the environment of young children have been a dismal failure. Massachusetts, with one of the best state laws on deleading, deleaded less than 0.5 percent of its leaded housing stock from 1982 to 1986 (Mushak and Crocetti 1990). At that rate complete deleading would take 800 years. Although deleading homes is technically more difficult and expensive than deleading gasoline, certainly the public policy failure is related to the class and race of the affected population as well as the difficulty and cost of the task. It remains to be seen whether 25 years of intensive study resulting in the increased understanding of the dangers of lead will lead to effective demands to eliminate exposure and protect our most vulnerable citizens.

References

Agency for Toxic Substances and Disease Registry. 1988. The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress. Document no. 99-2966. Atlanta: U.S. Department of Health and Human Services/Public Health Service.

Alexander, F.W. 1974. The Uptake of Lead by Children in Different Environments. *Environmental Health Perspectives* 7:155–70.

Annest, J.L., J.L. Pirkle, D. Makuc, et al. 1983. Chronological Trends in Blood Lead Levels Between 1976 and 1980. *New England Journal of Medicine* 308:1373–7.

Baltimore Health Department. 1971. *Baltimore Health News: Chronology of Lead Poisoning Control. Baltimore 1931–71.* (December): 34–40.

Bellinger, D.C., H.L. Needleman, A. Leviton, et al. 1984. Early Sensory-Motor Development and Prenatal Exposure to Lead. *Neurobehavioral Toxicology and Teratology* 6:387–402.

Bellinger, D., A. Leviton, C. Waternaux, et al. 1985. A Longitudinal Study of the Developmental Toxicity of Low-Level Lead Exposure in the Prenatal and Early Postnatal Periods. In *International Conference: Heavy Metals in the Environment*, ed. T.D. Lekkas (vol. 1, 32–4). Edinburgh, U.K.: CEP Consultants.

Bellinger, D., A. Leviton, H.L. Needleman, et al. 1986a. Low-Level Lead Exposure and Infant Development in the First Year. *Neurobehavioral Toxicology and Teratology* 8:151–61.

Bellinger, D., A. Leviton, M. Rabinowitz, et al. 1986b. Correlates of Low-Level Lead Exposure in Urban Children at 2 Years of Age. *Pediatrics* 77:826–33.

Bellinger, D., A. Leviton, C. Waternaux, et al. 1987a. Longitudinal Analyses of Prenatal and Postnatal Lead Exposure and Early Cognitive Development. *New England Journal of Medicine* 316:1037–43.

Bellinger, D., J. Sloman, A. Leviton, et al. 1987b. Low-Level Lead Exposure and Child Development: Assessment at Age 5 of a Cohort Followed From Birth. In *International Conference: Heavy Metals in the Environment*, eds. S.D. Lindberg and T.C. Hutchinson (vol. 1, 49–53). Edinburgh, U.K.: CEP Consultants.

Bellinger, D., A. Leviton, C. Waternaux, et al. 1988. Low-level Lead Exposure, Social Class, and Infant Development. *Neurotoxicology and Teratology* 10(6):497–503.

Bellinger, D., A. Leviton, C. Waternaux, et al. 1989. Low-level Lead Exposure and Early Development in Socioeconomically Advantaged Urban Infants. In *Lead Exposure and Child Development: An International Assessment*, eds. M.A. Smith, L.D. Grant, and A.I.

Sors (International Workshop on Effects of Lead Exposure on Neurobehavioral Development, September 1986, Edinburgh). Lancaster, U.K.: Kluwer.

Billick, I.H., A.S. Curran, and D.R. Shier. 1979. Analysis of Pediatric Blood Lead Levels in New York City for 1970–1976. *Environmental Health Perspectives* 31:183–90.

Billick, I.H., et al. 1982. *Predictions of Pediatric Blood Lead Levels from Gasoline Consumption*. Washington: U.S. Department of Housing and Urban Development.

Blanksma, L.A., H.K. Sachs, E.F. Murray, and M.J. O'Connell. 1969. Incidence of High Blood Lead in Chicago Children. *Pediatrics* 44:661–7.

Block, J.L. 1970. My Family is Dying. *Reader's Digest* 96 (April):171–2.

Bornschein, R.L., J. Grote, T. Mitchell, et al. 1989. Effects of Prenatal Lead Exposure on Infant Size at Birth. In *Lead Exposure and Child Development: An International Assessment*, eds. M.A. Smith, L.D. Grant, and A.I. Sors (International Workshop on Effects of Lead Exposure on Neurobehavioural Development, September 1986, Edinburgh). Lancaster, U.K.: Kluwer.

Byers, R.K. 1959. Lead Poisoning: Review of the Literature and Report on Forty-Five Cases. *Pediatrics* 23:585–603.

Byers, R.K., and E.E. Lord. 1943. Late Effects of Lead Poisoning on Mental Development. *American Journal of Diseases of Children* 66:471.

Charney, E., B. Kessler, M. Farfel, and D. Jackson. 1983. Childhood Lead Poisoning: A Controlled Trial of the Effect of Dust-Control Measures on Blood Lead Levels. *New England Journal of Medicine* 309:1089–93.

Chisolm, J.J., and D.M. O'Hara. 1982. *Lead Absorption in Children: Management, Clinical, and Environmental Aspects*. Baltimore: Urban & Schwarzeneberg.

Cohen, G.J., and W.E. Ahrens. 1959. Chronic Lead Poisoning: A Review of Several Years' Experience at the Children's Hospital, District of Columbia. *Journal of Pediatrics* 54:271–84.

Cohen, C.J., G.N. Bowers, and M.L Lepow. 1973. Epidemiology of Lead Poisoning. A Comparison Between Urban and Rural Children. *Journal of the American Medical Association* 226:1430–3.

Craig, P.P 1971. Lead, the Inexcusable Pollutant. *Saturday Review* 54 (Oct. 2):68.

David, O., J. Clark, and K. Voeller. 1972. Lead and Hyperactivity. *Lancet* 2(783):900–3.

de la Burde, B., and M.S. Choate, Jr. 1972. Does Asymptomatic Lead Exposure in Children Have Latent Sequelae. *Journal of Pediatrics* 81:1088–91.

———. 1975. Early Asymptomatic Lead Exposure and Development at School Age. *Journal of Pediatrics* 87:638–42.

Dietrich, K.N., K.M. Krafft, et al. 1986. Early Effects of Fetal Lead Exposure: Neurobehavioral Findings at 6 Months. *International Journal of Biosocial Research* 8:151–68.

———. 1987a. The Neurobehavioral Effects of Early Lead Exposure. In *Toxic Substances and Mental Retardation: Neurobehavioral Toxicology and Teratology*, ed. S.F. Schroeder, 71–95. Washington: American Association on Mental Deficiency (monograph no. 8).

———. 1987b. Low-level Fetal Lead Exposure Effect on Neurobehavioral Development in Early Infancy. *Pediatrics* 80:721–30.

Eidsvold, G., H. Mustalish, and L. Novick. 1974. The New York City Department of Health: Lessons in Lead Poisoning Control Program. *American Journal of Public Health* 64:956–62.

Engel, E.E. 1971. Health Hazards of Environmental Lead: A Position Paper. Paper presented at the Working Seminar on Lead to the Assistant Administrator for Research and Monitoring, Environmental Protection Agency, May 4.

Environmental Protection Agency. 1977. *Air Quality Criteria for Lead.* Pub. no. EPA-600/8-77-017. Washington: Office of Research and Development.

——— 1985. *Costs and Benefits of Reducing Lead in Gasoline. Final Regulatory Impact Analysis.* Pub. no. EPA-230-05-85-006. Washington: Office of Policy Analysis.

———. 1986. *Air Quality Criteria for Lead.* Pub. no. EPA-600-8-86-028aF. Research Triangle Park, N.C.: Environmental Criteria and Assessment Office.

———. 1989. *Supplement to the 1986 Air Quality Criteria for Lead* (Vol. 1, addendum A1–A67). Pub. no. EPA-600/8-89/049A. Washington: Office of Health and Environmental Assessment.

Ericson, J.E., H. Shirahata, and C.C. Patterson. 1979. Skeletal Concentrations of Lead in Ancient Peruvians. *New England Journal of Medicine* 300:946-51.

Ernhart, C.B., B. Landa, and N.B. Schell. 1981. Subclinical Levels of Lead and Developmental Deficit: A Multivariate Follow-up Reassessment. *Pediatrics* 67:911–19.

Ernhart, C.B., and M. Morro-Tlucak. 1987. Low Level Lead Exposure in the Prenatal and Early Preschool Years as Related to Intelligence Just Prior to School Entry. In *International Conference: Heavy Metals in the Environment*, eds. S.E. Lindberg and T.C. Hutchinson (vol. 1, 150–2). Edinburgh, U.K.: CEP Consultants.

Facchetti, S. 1985. Isotope Lead Experiment—An Update. Paper presented at a workshop on Lead Environmental Health: The Current Issues, Durham, N.C., Duke University, April/May.

Florini, K.L., G.D. Krumbhaar, Jr., and E.K. Silbergeld. 1990. *Legacy of Lead: America's Continuing Epidemic of Childhood Lead Poisoning.* Washington: Environmental Defense Fund.

Garibaldi, P., S. Facchetti, A. Quagliardi, et al. 1975. Petrols Additivated with Isotopically Differentiated Lead: Proposal of an Experiment to Estimate the Incidence of Traffic on the Environment of Pollution by Lead—First Experimental Results. In *Proceedings of an International Symposium: Recent Advances in the Assessment of the Health Effects of Environmental Pollution* (vol. 3, 1287–99). Luxembourg: Commission of the European Communities.

Graebner, W. 1988. Private Power, Private Knowledge, and Public Health: Science, Engineering, and Lead Poisoning, 1900–1970. In *The Health and Safety of Workers*, ed. R. Bayer, 1–15. New York: Oxford University Press.

Guinee, V.F. 1971. Lead Poisoning in New York City. *Transactions of the New York Academy of Sciences* 33:539–51.

Harvey, P.G., M.W. Hamlin, et al. 1984. Blood Lead, Behavior and Intelligence Test Performance in Pre-school Children. *Science of the Total Environment* 40:45–60.

Hernberg, S., and J. Nikkanen. 1970. Enzyme Inhibition by Lead Under Normal Urban Conditions. *Lancet* 1:63–4.

Hernberg, S., J. Nikkanen, G. Mellen, and H. Lilius. 1970. Delta-Amino-Levulinic Acid Dehydratase as a Measure of Lead Exposure. *Archives of Environmental Health* 21:140–45.

Hill, A.B. 1965. The Environment and Disease: Association or Causation? *Proceedings of the Royal Society of Medicine; Section of Occupational Medicine* (Jan. 14):295–300.

Jacobziner, H. 1966. Lead Poisoning in Childhood. *Clinical Pediatrics* 5:277–86.

King, B.G. 1971. Maximum Daily Intake of Lead Without Excessive Body Lead-burden in Children. *American Journal of Diseases of Children* 122:337–40.

King, B.G., A.F. Schaplowsky, E.B. McCabe, et al. 1972. Occupational Health and Child Lead Poisoning: Mutual Interests and Special Problems. *American Journal of Public Health* 62 (August):1056–9.

Kotok, D., R. Kotok, et al. 1977. Cognitive Evaluation of Children With Elevated Blood Lead Levels. *American Journal of Diseases of Children* 131:791–3.

Landrigan, P.J., E.L. Baker, R.G. Feldman, et al. 1976. Increased Lead Absorption with Anemia and Slowed Nerve Conduction in Children Near a Lead Smelter. *Journal of Pediatrics* 89:904–10.

Landrigan, P.J., S.H. Gehlbach, B.F. Rosenblum, et al. 1975. Epidemic Lead Absorption Near an Ore Smelter: The Role of Particulate Lead. *New England Journal of Medicine* 292:123–9.

Lin-fu, J.S. 1971. Memorandum of May 5 to Dr. Irwin H. Billick: Information Pertinent to Title III—Federal Demonstration and Research Program of PL 91-695, The Lead-Based Paint Poisoning Prevention Act. Washington: Department of Health, Education and Welfare.

———. 1972. Undue Absorption of Lead Among Children—A New Look at an Old Problem. *New England Journal of Medicine* 286: 702–10.

———. 1973. Vulnerability of Children to Lead Exposure and Toxicity. *New England Journal of Medicine* 289:1229–33, 1289–93.

———. 1979. Lead Poisoning in Children. What Price Shall We Pay? *Children Today* 8:9–13,36.

———. 1980. Lead Poisoning and Undue Lead Exposure in Children: History and Current Status. In *Low Level Lead Exposures. The Clinical Implications of Current Research*, ed. H.L. Needleman, 5–16. New York: Raven Press.

———. 1985. Historical Perspective on Health Effects of Lead. In *Dietary and Environmental Lead: Human Health Effects*, ed. Mahaffey. New York: Elsevier.

Mack, R.B. 1973. Lead in History. *Clinical Toxicology Bulletin* 3:37–44.

Manton, W.I. 1977. Sources of Lead in Blood: Identification by Stable Isotopes. *Archives of Environmental Health* 32:149–59.

Mellins, R.B., and C.D. Jenkins. 1955. Epidemiological and Psychological Study of Lead Poisoning in Children. *Journal of the American Medical Association* 158(1):15–20.

Milar, C.R., S.R. Schroeder, P. Mushak, et al. 1980. Contributions of the Caregiving Environment to Increased Lead Burden of Children. *American Journal of Mental Deficiency* 84:339–44.

Millar, J.A., R.L.C. Cummings, V. Battistini, et al. 1970. Lead and Delta-Amino-Levulinic Acid Dehydratase Levels in Mentally Retarded Children and Lead-Poisoned Suckling Rats. *Lancet* 2:695–8.

Mushak, P., and A.F. Crocetti. 1989. Determination of Numbers of Lead Exposed American Children as a Function of Lead Source: Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning. *Environmental Research* 50(2):210–29.

——— 1990. Methods for Reducing Lead Exposure in Young Children and Other Risk Groups: An Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning. *Environmental Health Perspectives* 89 (Nov.):125–35.

National Center for Health Statistics. 1984. Blood Lead Levels for Persons Ages 6 Months to 74 Years: U.S. 1976–1980. Data from the Second National Health and Nutrition Examination Survey (NHANES II), series 11, no. 233. Washington.

Needleman, H.L. Ed. 1980. *Low Level Lead Exposure: Clinical Implications of Current Research*. New York: Raven Press.

———. 1989. The Persistent Threat of Lead: A Singular Opportunity. *American Journal of Public Health* 79:643–5.

———. 1990. Low Level Lead Exposure: A Continuing Problem. *Pediatric Annals* 19(3):208–14.

Needleman, H.L., C. Gunnoe, A. Leviton, et al. 1979. Deficits in Psychologic and Classroom Performance of Children with Elevated Dentine Lead Levels. *New England Journal of Medicine* 300:689–95.

Needleman, H.L., A. Schell, D. Bellinger, et al. 1990. The Long-Term Effects of Exposure to Low Doses of Lead in Childhood. An 11-Year Follow-up Report. *New England Journal of Medicine* 322(2):83–8.

Ozonoff, D. 1988. Failed Warnings: Asbestos-related Disease and Industrial Medicine. In *The Health and Safety of Workers*, ed. R. Bayer, 139–220. New York: Oxford University Press.

Parents' Magazine. 1973. Does Lead Poisoning Threaten Your Child? 48 (August):59–60.

Patterson, C.C. 1965. Contaminated and Natural Lead Environments of Man. *Archives of Environmental Health* 11:344–60.

Perino, J., and C.B. Ernhart. 1974. The Relation of Subclinical Lead Level to Cognitive and Sensorimotor Impairment in Black Preschoolers. *Journal of Learning Disorders* 7:26–30.

Perlstein, M.A., and R. Attala. 1966. Neurologic Sequelae of Plumbism in Children. *Clinical Pediatrics* 5:292–8.

Rabin, R. 1989. Warnings Unheeded: A History of Child Lead Poisoning. *American Journal of Public Health* (December):1668–74.

Rabinowitz, W.B., and H. Needleman. 1983. Petrol Lead Sales and Umbilical Cord Blood Lead Levels in Boston, Massachusetts. *Lancet* 1:63.

Remsburg, C., and B. Remsburg. 1972. Youngest Victims. *Good Housekeeping* 48 (August):59–60.

Rummo, J.H. 1974. Intellectual and Behavioral Effects of Lead Poisoning in Children. Unpublished Ph.D. dissertation, University of North Carolina, Chapel Hill.

Rutter, M. 1980. Raised Lead Levels and Impaired Cognitive/Behavioral Functioning. *Developmental Medicine and Child Neurology* 42 (suppl.):1–26.

Sayre, J.W., E. Charney, and J. Vostal. 1974. House and Hand Dust as a Potential Source of Childhood Lead Exposure. *American Journal of Diseases of Children* 127:167–70.

Schucker, G.W., E.H. Vail, E.B. Kelley, and E. Kaplan. 1965. Prevention of Lead Paint Poisoning Among Baltimore Children. *Public Health Reports* 80:11:969–74.

Schwartz, J., A. Janney, and H. Pitcher. 1984. *The Relationship Between Gasoline Lead and Blood Lead*. Washington: Environmental Protection Agency, Office of Policy Analysis.

Scientific American. 1969. Silent Epidemic. 220 (May):54.

Secchi, G.C., L. Erba, and G. Cambiaghi. 1974. Delta Amino Levulinic Acid Dehydratase Activity of Erythrocytes and Liver Tissue in Man. Relationship to Lead Exposure. *Archives of Environmental Health* 28:130–2.

Shapiro, I.M., P. Grandjean, and O. Van Neilsen. 1980. Lead Levels in Bones and Teeth of Children in Ancient Nubia: Evidence of Both Minimal Lead Exposure and Lead Poisoning. In *Low Level Lead Exposure: Clinical Implications of Current Research*, ed. H.L. Needleman. New York: Raven Press.

Silbergeld, E.K., and A.M. Goldberg. 1973. A Lead-induced Behavioral Disorder. *Life Sciences* 13:1275–83.

——— 1974a. Hyperactivity: A Lead-induced Behavior Disorder. *Environmental Health Perspectives*. 7:227–32.

———. 1974b. Lead-induced Behavioral Dysfunction: An Animal Model of Hyperactivity. *Experimental Neurology* 42:146–57.

Smith, H.D. 1954. Lead Poisoning in Children. *American Journal of Nursing* 54(6):736–8.

Smith, M. 1985. Recent Work on Low Level Lead Exposure and its Impact on Behavior, Intelligence and Learning: A Review. *Journal of the American Academy of Child Psychiatry* 24:24–32.

Specter, M.J., and V.F. Guinee. 1970. Epidemiology of Lead Poisoning in New York City—1970. Paper presented at the American Public Health Association, 98th Annual Meeting, Houston, Texas, October 26.

Szabo, N.B., and S. Pollack. 1987. *A Silent and Costly Epidemic: The Medical and Educational Costs of Childhood Lead Poisoning in Massachusetts*. Boston: Conservation Law Foundation of New England.

Thurston, D.L., J.N. Middlekamp, and E. Mason. 1955. The Late Effects of Lead Poisoning. *Journal of Pediatrics* 47:413–23.

Time. 1969. Deadly Lead in Children. 93 (April 4):42.

U.S. Department of Health, Education and Welfare. 1970. Medical Aspects of Lead Poisoning. Statement of the Surgeon General, November 8.

Vandenbrouche, J.P 1990. Epidemiology in Transition: A Historical Hypothesis. *Epidemiology* 1(2):164–7.

Yankel, A.J., I.H. von Lindern, and S.D. Walter. 1977. The Silver Valley Lead Study: The Relationship Between Childhood Blood Lead Levels and Environmental Exposure. *Journal of the Air Pollution Control Association* 27:763–7.

Address correspondence to: Ms. Barbara Berney, Department of Environmental Health, Boston University School of Public Health, 80 East Concord, T3C, Boston, MA 02118-2394.