PREVENTING LEAD POISONING IN CHILDREN

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ABSTRACT
Lead poisoning is the most significant and prevalent disease of environmental origin among US children. Despite over 100 years’ knowledge of the special hazards of lead exposure for young children, it has taken over a century for effective primary prevention to be adopted. Obstacles to primary prevention have included deliberate campaigns by industry to prevent restrictions upon such uses on lead as plumbing, paints, and gasoline additives; influence of industrial support of biomedical research at major US medical schools; lack of appropriate policy mechanisms to identify and control lead exposures; and opposition to investing resources in lead poisoning prevention. The removal of lead from gasoline, which began in the United States in 1972 and was completed in 1995, has resulted in almost fourfold reductions in median blood lead levels in US children from 1976 to 1991. Increased screening and interventions to identify and abate lead sources, such as lead in housing, also contributed to this major public health success. Nevertheless, lead exposures remain prevalent, although increasingly less generally distributed. Perhaps because of the renewed “ghettoization” of lead, support for lead poisoning prevention has waned. Objections to investing public and private resources in screening and source abatement have challenged the continuing commitment of public health officials to prevention. The demonstrable success and social benefits of preventing lead toxicity are cited in support of continued preventive health policies.

INTRODUCTION
Lead poisoning, for much of this century, has been the most prevalent and serious disease of environmental origin for young children in the United States. Its causes were described accurately by the mid-1920s, but not until the late 1970s were comprehensive and effective actions taken to prevent this disease
by interdicting major uses of lead in consumer products and industry. The fact that over half a century elapsed between knowledge and action indicates that any review of the struggle to prevent lead poisoning in children must attend to the political and economic forces that for 50 years interfered with the implementation of primary prevention policies. The struggle for prevention has involved almost every consumer and environmental protection statute and policy strategy extant in the United States; it has engaged communities, states, and the federal government; it has revealed the nature and scope of injustice in the distribution of environmental risks; it has elicited courageous acts and great mendacity. From the perspective of a century’s experience, this review not only describes this history but also provides some insights that may be useful in preventing other environmental diseases, avoiding both the social toll exacted by lead and the political delays involved. Since the author of this review has been personally involved in much of the policy debate on lead poisoning prevention at the local, state, and federal over the past 20 years, this review should be read in light of the strong opinions held and expressed (65).

No small part of the delay in recognizing the nature and scope of lead poisoning and seizing opportunities for disease prevention is due to the actions and expressed opinions of some biomedical researchers considered experts in their day. These scientists and clinicians—among them, Joseph Aub and Cecil Drinker of Harvard and Robert Kehoe of Cincinnati—were retained by the lead industry in its organized campaign to control the discourse on lead poisoning (see 29, 25, 55). For decades, from the late 1920s through the 1970s, the lead industry funded most of the research on lead toxicity at major institutions in the United States, including Harvard, the University of Cincinnati, and Johns Hopkins. Only with the rise of the National Institute of Environmental Health Sciences, under the leadership of David Rall, were other funds available, from a neutral source, and it is not coincidental that with this support a new generation of researchers began to publish new findings on the toxicity and sources of lead in the mid-1970s. Even then, the struggle was neither easy nor pleasant. Attacks on the integrity of researchers reached their nadir when allegations of scientific fraud were raised by consultants to the lead industry against an internationally recognized scientist (64). Although eventually rebutted, these charges were of a piece with earlier personal attacks by the industry and its consultants on Alice Hamilton and Randolph Byers.

The history of lead must induce careful reflection: The natural and laudable bias of scientists toward vigorous skepticism can be exploited to support inaction on the basis of uncertainty. In public health, giving the “benefit of the doubt” to a potentially toxic substance like lead runs the risk of denying a margin of protection to its victims. In the case of lead, the expressed uncertainties about low-level lead exposures (a relative term that changed over the course of this history, as described below) provided not only repeated excuses for inaction
but, more dangerously, justifications for new and expanded uses of this toxic metal. By the time these uncertainties were reduced to a level to support a new consensus on lead’s hazards in the early 1980s, thousands of tons of lead had been dispersed into the environment. The responsibility for this catastrophic mistake (to use the terms of Carl Shy) must be shared among industry, government, and academic researchers.

The Nature of Childhood Lead Poisoning

Lead poisoning is an entirely preventable disease, induced by exposures to lead. The toxicity of lead has been recognized for almost as long as this useful metal has been mined, smelted, and used by human societies (34, 42, 48). Rules prohibiting the use of lead additives in beverages were among the earliest food regulations in Europe and the American colonies (18, 75). Although it was recognized as early as the mid-nineteenth century that children might be among the most vulnerable to lead toxicity [for discussion of early opinions and observations, see Reference (50)], specific attention to children as a population at risk was only formalized in the 1920s. Hamilton (26) directed attention to the devastations of occupational and peri-occupational exposures for young children; she recommended that young children be excluded from employment in the lead trades, and that measures be taken to reduce the transport of lead dusts from the workplace to the home. In 1897, the Australian pediatrician Lockhart Gibson reported on cases of young children intoxicated by lead from lead-based paints used on porches and doors (24). The importance of lead paint as a cause of lead toxicity in children was soon reported in the United States, in Baltimore and in Boston [see (54) for a history of the recognition of lead paint poisoning in the United States].

The definition of lead poisoning—that is, at what level of exposure clinical toxicity occurs—has changed markedly over the century. Over this period, medical and public health opinion has shifted dramatically, from assuming that only high-dose, overtly encephalopathic exposures were significant, to the recognition that very low doses, without clear symptom presentation, are associated with measurable neurotoxicity.

Similarly, concepts related to treating lead poisoning have also undergone profound change. Prior to 1940, it was generally assumed that if the child’s exposure was promptly reduced, then the effects of lead were reversible unless severe toxicity had been induced (such as coma and convulsions). After the introduction of the chelating agents ethylenediamine tetraacetic acid (EDTA), d-penicillamine, and British anti-Lewisite (BAL) into therapeutic practice in the 1950s and 1960s, drug treatment was added to reduction of exposure as the means of medical management (13). Although there is a large medical literature associating chelation treatment with reductions in blood lead levels and increased excretion of lead in urine, there is much less evidence that treatment
actually affects outcome. Silbergeld & Chisolm (67) reported that children given EDTA showed reductions in blood lead and urinary catecholamine excretion, a potential marker of neurotoxicity; Rosen et al (56) reported that chelation lowered bone lead and improved children’s performance on measurements of cognitive function; and David et al (17) reported that chelation reduced hyperactivity in children with past lead exposure. However, these studies involved children with relatively high exposures to lead (in excess of 45 \( \mu g/dL \)).

The reversibility of lead toxicity has been questioned since Byers & Lord (9) suggested in 1943 that lead could induce persistent damage in children, even after their external exposures were reduced and clinical indicators of toxicity were no longer detectable. Needleman and colleagues (45) demonstrated through a careful prospective study that early lead exposure affected children’s behavior and intellectual attainment for at least 10 years. Thus it is not clear how much benefit, in terms of reversing toxicity, any postexposure treatments bring. Chelation is undoubtedly valuable to lower high blood levels of and prevent acute neurotoxic effects, such as convulsions, as well as reducing effects of lead on kidney from prolonged high-dose exposure. Extended chelation can reduce overall body burdens of lead as well, including lead in bone (51, 57). Nutrition may play a role in reducing absorption of lead (27), but there is little evidence that essential elements, such as calcium or iron, can counteract the presence of lead at target sites of action once it has been absorbed.

These findings have had profound impacts upon strategies for prevention. It is now the general foundation for public health policy in the United States, France, Australia, and other countries that childhood lead poisoning, or toxicity, is associated with blood lead levels as low as 10 mcg/dL (or 0.5 mmol/L), and that the prevention of toxicity requires prevention of exposure through the identification and control of lead sources in the environment, in air, food, water, and dusts and soils (see 50, 65 for reviews of national policies). The considerable decreases in what have been considered “acceptable” levels of lead exposure for children have driven an expanded consideration of opportunities to prevent exposures through more comprehensive identification and control of potential sources. Thus, the struggle to prevent lead poisoning has been transformed from tertiary/secondary prevention to primary prevention. Nevertheless, current public health policies remain imperfect instruments for achieving progress in primary prevention. In large part, this relates to the complexity of source reduction and to continuing controversies over the value of such strategies as universal screening.

The Demographics of Lead Poisoning

The prevalence of childhood lead poisoning is largely determined by two factors: age and proximity to environmental sources or media contaminated by
lead. Other factors that have been associated with increased levels of lead in blood—sex, income, race, place of residence—are mostly predictors of exposure. Children’s blood (PbB) lead levels are generally highest between 9 and 18 months of age (see 2, 8). This is because children in this age range tend to explore their immediate environments intensively, with a good deal of hand-to-mouth activity (11, 12), and as compared to adults they are also more efficient, by five- to tenfold, at absorbing lead taken in orally (71). Some children have an abnormal pattern of ingesting non-food items, often soils (pica), which, if these soils are contaminated by lead, can result in very high doses of lead. Boys have slightly higher levels of PbB than girls, which is thought to be behavioral, related to a greater frequency of exploratory behaviors in early childhood. Nutrition and genotype play a very slight role in modifying PbB (27, 77).

The variables of income, race, and residence signify the social tragedy of lead poisoning in the United States. The poor and disadvantaged are more likely to live in lead-contaminated environments, especially in dilapidated housing with flagrant lead paint hazards; they are more likely to live in urban neighborhoods where years of traffic have left tons of lead deposits from leaded gasoline; they are more likely to live near point sources of lead, such as smelters, or hazardous waste sites (41).

The fact that lead poisoning is not evenly or randomly distributed among children in the United States continues to raise tremendous obstacles to its prevention. Until the late 1970s, it is fair to say that this disease was “ghettoized”, considered to be a risk exclusively to the urban minority poor (35, 43). This assumption was based on very limited data and highly skewed attempts to identify children with elevated blood lead (PbB) levels. Even in Baltimore, the city with the longest history of public health surveillance for lead exposures, few children were tested or evaluated for lead exposure, and hardly any testing was conducted outside the well-defined “lead belt” of inner-city housing, first described by Huntington Williams in the 1930s (78). Concerns for lead poisoning waxed and waned with social concerns for the disadvantaged until the results of the first representative national survey of lead exposures in US children began to be published in 1982 (36). Coupled with the newer experimental and clinical studies demonstrating toxicity at lower levels of lead exposure (42), these national prevalence reports substantially transformed lead poisoning as a public health issue by making it an environmental concern for the general public (for instance, Environmental Defense Fund). Although the median PbB of poor urban black children measured in NHANES II (1976–1980) was the highest of all persons (nonoccupationally exposed), a significant proportion of all children had PbB > 15 µg/dL, and the median PbB for affluent white children was 13 µg/dL.

These data prompted efforts to prevent lead poisoning in children on a national scale.
LEAD POISONING PREVENTION FROM 1900 TO 1970

In the absence of national legislation or suitable government institutions, attempts to prevent lead poisoning over the first 70 years of this century (and earlier) were undertaken only by states and cities. Certain uses of lead, such as lead adulterants to cider and wine, were recognized to pose risks of excess exposure, and these were restricted on a local basis in Europe and the American colonies (18, 75). Other proposals—such as banning lead in plumbing, proposed as early as the eighteenth century in England (32)—were resisted by the lead industry, which in 1922 organized itself to fight attempts to restrict its products (55). The Lead Industries Association succeeded for years in stopping many regulations on lead, including blocking the United States from signing the International Labor Organization (ILO) convention prohibiting use of white lead in paint, an international treaty that the United States has never signed (48) (Table 1).

Most attention until 1970 focused, with reason, on occupational lead poisoning. As documented by Hamilton (26), thousands of workers annually were

Table 1  Signatures to White Lead Paint Convention, International Labor Organizationa

<table>
<thead>
<tr>
<th>Year</th>
<th>Country/Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>1923</td>
<td>Czechoslovakia, Sweden</td>
</tr>
<tr>
<td>1924</td>
<td>Austria, Poland, Spain</td>
</tr>
<tr>
<td>1925</td>
<td>Bulgaria, Chile, Romania</td>
</tr>
<tr>
<td>1926</td>
<td>Belgium, France, Greece</td>
</tr>
<tr>
<td>1928</td>
<td>Cuba, Luxembourg</td>
</tr>
<tr>
<td>1929</td>
<td>Finland, Norway, Yugoslavia</td>
</tr>
<tr>
<td>1933</td>
<td>Columbia, Nicaragua, Uruguay, Venezuela</td>
</tr>
<tr>
<td>1936</td>
<td>Argentina</td>
</tr>
<tr>
<td>1938</td>
<td>Mexico</td>
</tr>
<tr>
<td>1939</td>
<td>Afghanistan, The Netherlands</td>
</tr>
<tr>
<td>1952</td>
<td>Italy</td>
</tr>
<tr>
<td>1953</td>
<td>Vietnam</td>
</tr>
<tr>
<td>1956</td>
<td>Hungary, Morocco, Tunisia</td>
</tr>
<tr>
<td>1959</td>
<td>Guinea</td>
</tr>
<tr>
<td>1960</td>
<td>Benin, Burkina Faso, Cameroon, Central African Republic, Chad, Congo, Gabon, Ivory Coast</td>
</tr>
<tr>
<td>1961</td>
<td>Mauritania, Niger</td>
</tr>
<tr>
<td>1962</td>
<td>Algeria</td>
</tr>
<tr>
<td>1964</td>
<td>Lao People’s Democratic Republic</td>
</tr>
<tr>
<td>1966</td>
<td>Iraq</td>
</tr>
<tr>
<td>1969</td>
<td>Democratic Kampuchea</td>
</tr>
<tr>
<td>1970</td>
<td>Panama</td>
</tr>
<tr>
<td>1976</td>
<td>Suriname</td>
</tr>
<tr>
<td>1978</td>
<td>Comoros, Djibouti</td>
</tr>
<tr>
<td>1988</td>
<td>Malta</td>
</tr>
</tbody>
</table>

a Source: United Nations Environment Programme, 1989 (country names used are those shown in the source cited).
poisoned by lead in industries ranging from ceramics through battery manufac-
ture. She also reported on the poisoning of children through their employment
in the lead trades, and through the uncontrolled movement of lead dusts from
the workplace to the worker’s home, which remains a continuing problem (37).
Some children were screened, starting in Baltimore in 1935, with the develop-
ment of laboratory methods at Johns Hopkins (31). But from 1935 to 1951,
fewer than 2000 children were tested with the dithizone method (78).

No actions were taken during this time on lead in environmental media such
as air or water, in the absence of any comprehensive environmental legislation.
An attempt was made in the 1930s to reduce allowable residues of lead in food
(lead arsenate was widely used as a pesticide), but this was defeated by heavy
industry lobbying. The production and consumption of lead in the United States
continued to grow. Worse, in 1925, a new use of lead was approved, and this
was to become the largest single source of lead contamination in history.
The decision to permit the use of alkyl lead additives in automobile fuels
represents the greatest single failure in preventing lead poisoning. Described
as “the public health catastrophe of the 20th century” (63), this decision caused
the release of thousands of metric tons of lead into ambient air, with both
long- and short-range impacts. As shown in Figure 1, the greatest increase in
human use of lead occurred at this point in time (note log scale on y-axis).
This increase was quickly followed by rises in lead concentrations measured
in stable ecological indicators, such as Greenland ice (42, 30). How the lead,
gasoline, and automobile industries convinced the US Public Health Service to
approve lead for gasoline has been eloquently described (58, 47). A sample of
the industry propaganda of the time is shown in Figure 2.

Despite the objections of Hamilton and others, a US government commission
decided in 1925 to permit tetraethyl lead (TEL) use in gasoline, with a promise
to reconsider the issue later (58). Unfortunately for the nation’s health, “later”
took 50 years to occur.

LEAD POISONING PREVENTION SINCE 1970

This phase of the prevention struggle has been source directed, rather than case
oriented. For the first time, dedicated efforts were made to identify and prevent
lead exposures prior to exposure of children. These efforts took two forms:
setting enforceable standards for environmental media and drinking water, and
specific restrictions on certain uses of lead. Such actions were possible because
of the creation of new government institutions, the Environmental Protection
Agency (EPA) and the Occupational Safety and Health Administration (OSHA).
These actions are summarized in Table 2 (50). Sometimes these actions flowed
together. For instance, the EPA’s review of the National Ambient Air Quality
Standard (NAAQS) for lead, begun in 1981 and concluded in 1986 (71), did
not result in any change in the NAAQS [although the EPA Science Advisory Board did recommend a reduction from 1.5 \( \mu g/m^3 \) to 0.5 or 0.75 \( \mu g/m^3 \) (72)]; rather, its four volumes provided substantial weight to public campaigns to ban the use of lead in gasoline, a goal finally achieved in December 1995, after the passage of the Clean Air Act Amendments of 1990. In a similar fashion, the EPA’s decision to reduce the drinking water standard for lead from 50 ppb to 5 ppb, in 1986, resulted in bans on use of lead in plumbing solders and fixtures to avoid exceedances due to leaching of lead into drinking water.

These actions to improve environmental standards and to restrict lead uses had major effects on the nature and extent of lead poisoning in US children.
Ethylized Gasoline

For one year and nine months ethylized gasoline has been on sale. It is now being distributed through about 20,000 filling stations covering one-third of the territory of the United States. About 200,000,000 gallons have been used by more than one million motorists, with complete safety and satisfaction.

Recently a distressing accident occurred at an experimental plant, where a new process for the manufacture of tetra-ethyl lead, one of the constituents used in ethylizing gasoline, was under development.

Tetra-ethyl lead is a poison, as are many raw materials which enter into the manufacture of harmless compounds. Ethylized gasoline consists of 1,300 parts of ordinary gasoline containing less than one part of tetra-ethyl lead.

This statement is issued to make plain the all-important difference between tetra-ethyl lead, the raw material, and ethylized gasoline, the commercial product.

Ethylized gasoline is more than an improved fuel, giving smoothness to the motor and eliminating knocks; it is a scientific discovery which, in its ultimate development, will contribute largely to the conservation of the world’s supply of gasoline.

The dangerous character of tetra-ethyl lead having been recognized from the outset, exhaustive tests have been conducted which have established the safety of ethylized gasoline when used properly as a motor fuel.

These tests have been confirmed by the United States Bureau of Mines, which is making additional studies to determine whether any possible injury can result from continued contact when used for other than motor purposes. Scientific data based on these studies will be submitted to any health commissioner or other public health official on request.

Figure 2 Advertisement placed by the Ethyl Gas Corporation, in the Baltimore Sun, in 1925, in the midst of controversy related to the risks of lead additives to gasoline.

over the 1980s. The reduction in lead used in gasoline (greater than 100-fold, from 1976 to 1986) was associated with average reductions in PbB of over 40%, as shown in Table 3 (66). Controls on lead in other products, such as increased vigilance by the FDA and CPSC over imported foods and toys, also probably contributed to these substantial reductions (2, 53). Use of lead in house paint had been restricted by regulation in 1977, but, for reasons discussed below, this action has had little short- or medium-term impact on lead exposures.

The most dramatic and creative effort at primary prevention was an economic disincentive against lead consumption, introduced as legislation by Congressman Ben Cardin of Maryland in 1993. Inspired by a suggestion
Table 2

<table>
<thead>
<tr>
<th>Environmental Sources</th>
<th>Environmental Media</th>
<th>Products</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drinking water</td>
<td>Air</td>
<td>Soil</td>
</tr>
<tr>
<td>Surface waters</td>
<td>Ambient air</td>
<td>Agriculture</td>
</tr>
<tr>
<td>Soil</td>
<td>Soil</td>
<td>Foodstuff</td>
</tr>
<tr>
<td>Ambient air</td>
<td>Ambient air</td>
<td>Gasoline</td>
</tr>
<tr>
<td>Ambient air</td>
<td>Ambient air</td>
<td>Baticides</td>
</tr>
<tr>
<td>Soil</td>
<td>Agriculture</td>
<td>Pesticides</td>
</tr>
<tr>
<td>Agriculture</td>
<td>Agriculture</td>
<td>Hunting/ Fishing</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Countries</th>
<th>Austria</th>
<th>Canada</th>
<th>Finland</th>
<th>Iceland</th>
<th>Japan</th>
<th>Mexico</th>
<th>New Zealand</th>
<th>Norway</th>
<th>Switzerland</th>
<th>Turkey</th>
<th>United States</th>
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<tr>
<td></td>
<td>x</td>
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<td>x</td>
<td>x</td>
<td>x</td>
</tr>
</tbody>
</table>

Table 3  Relationship between lead in gasoline and blood lead levels in the US population

<table>
<thead>
<tr>
<th>Year</th>
<th>Lead used in gasoline (10^6 kg)</th>
<th>Median blood lead level (µg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1976</td>
<td>186.47</td>
<td>14.6</td>
</tr>
<tr>
<td>1980</td>
<td>51.59</td>
<td>9.2</td>
</tr>
<tr>
<td>1990</td>
<td>0.47</td>
<td>2.8</td>
</tr>
</tbody>
</table>

*Data from References 2, 53.

from the Environmental Defense Fund (21), Cardin proposed a tax on lead, which would serve two purposes: Encourage product substitution by “leveling the playing field” for substitutes for lead, and provide funds dedicated to the screening of children and abatement of past uses of lead, primarily lead paint in housing. This initiative failed in an anti-tax climate, but it represents something of a high-water mark in public health strategies in primary prevention.

Screening is an important aspect of disease prevention, through the early identification of increased exposures and the prompt delivery of therapeutic interventions. Screening not only identifies individuals at risk, but it can also permit health authorities and others to identify sources of lead and reduce or remove them prior to other children being exposed. Screening for lead poisoning involves the measurement of lead in blood, or an appropriately sensitive and specific biomarker of exposure. For lead, the biomarker of erythrocyte protoporphyrin (EP), a biological precursor in the cellular synthesis of heme, was widely used to diagnose lead exposures. This test was highly successful in facilitating screening because it was relatively cheap and, most importantly, with the hematofluorometer, results could be quickly obtained under clinic or field conditions (10, 52). Screening increased in the 1970s in many cities and states until the early 1980s, when screening decreased in the face of efforts to defund public health programs and roll lead screening into underfunded block grants to the states.

When medical consensus caused CDC to lower the guideline defining toxic exposures to lead, first to 25 µg/dl (1985) and then to 10 µg/dl (1991), the EP test was not sufficiently sensitive to identify children with PbBs in excess of these levels. The only recommended methods for screening involved careful collection of blood by venupuncture and lead analysis at a technically competent laboratory (11). The costs increased and logistics become more complicated.

LEAD POISONING PREVENTION IN THE 1990S

The successful regulation of certain lead sources substantially lowered average PbBs in children in the United States, from 1976 to 1991. As shown in Figure 3,
the distribution of PbBs has shifted dramatically. The role of removing lead from gas in this reduction was clear from a trend analysis of the NHANES II data collected from 1976 to 1980, a period that coincided with the first major phasedown of lead in gasoline (Figure 4). While this change must be considered a public health success, this very success has reduced the momentum to fully eliminate lead poisoning. The disease has to a large extent become “re-ghettoized”. Now, children with PbB > 10 are much more likely to be black, live in large cities, and to be poor (Table 5) (8). For these children, lead paint is the overwhelming source of exposures (61, 73), followed by exposure to highly contaminated urban soils that contribute to household dusts (39). However, as found by Ashley et al (3) when lead-painted surfaces are present in a home in poor condition, the paint source dominates in house dust and children’s blood.

Lead-Based Paint

Lead-based paint in housing has been the most significant source of high-level lead exposure for most of this century. As shown in Table 4, it is the most significant source of lead exposures for young children (1). Despite early recognition, restrictions on the use of lead in paint in the United States were successfully thwarted by the concerted actions of the organized lead industry (55). The city
Figure 4 Relationship between lead used in gasoline and median blood lead levels in the US population, 1976–1980 (NHANES II). Although not conducted for this reason, the NHANES survey of national lead exposures serendipitously coincided with the first major reductions in lead usage in gasoline. Later analyses, by Annest and others at CDC, demonstrated a high degree of correlation between monthly lead usage and blood lead (see Reference 2 for details).

of Baltimore, under the inspired leadership of Huntington Williams, banned the use of lead-based paint in public housing, but it was not until 1977 that the US Consumer Product Safety Commission finally issued regulations limiting the concentration of lead in paint to 0.06%. This was a substantial reduction, given that some earlier paint formulations were as much as 45% lead (by weight). However, the regulations covered only paints on indoor surfaces of

<table>
<thead>
<tr>
<th>Category</th>
<th>Construction date</th>
<th>Lead-based paint homes number</th>
<th>Children number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peeling paint</td>
<td>Pre-1975</td>
<td>1,972,000</td>
<td>567,000</td>
</tr>
<tr>
<td>Broken plaster</td>
<td>Pre-1975</td>
<td>1,594,000</td>
<td>458,000</td>
</tr>
<tr>
<td>Holes in walls</td>
<td>Pre-1975</td>
<td>2,602,000</td>
<td>747,000</td>
</tr>
<tr>
<td>Totals</td>
<td>Pre-1975</td>
<td>6,168,000</td>
<td>1,772,000</td>
</tr>
</tbody>
</table>

Data from HUD.
Table 5 Blood lead levels in the US population, 1988–1991

<table>
<thead>
<tr>
<th>Population</th>
<th>Blood lead, µg/dL</th>
<th>Percentage above 10 µg/dL (geometric mean)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Hispanic white</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1–2</td>
<td>3.5</td>
<td>8.5</td>
</tr>
<tr>
<td>3–5</td>
<td>2.9</td>
<td>3.7</td>
</tr>
<tr>
<td>1–5 low economic status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>high economic status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1–2</td>
<td>6.3</td>
<td>21.6</td>
</tr>
<tr>
<td>3–5</td>
<td>5.9</td>
<td>20.0</td>
</tr>
<tr>
<td>1–5 low economic status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>high economic status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mexican-American</td>
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<td></td>
</tr>
<tr>
<td>1–2</td>
<td>4.2</td>
<td>10.1</td>
</tr>
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<td></td>
<td>8.8</td>
</tr>
<tr>
<td>high economic status</td>
<td></td>
<td>*</td>
</tr>
</tbody>
</table>

*Insufficient numbers for estimation. Data from Reference 8.

Residential structures, and articles (such as toys and furniture) designed for use by children.

More seriously, these steps could not deal with the lead paint that had been used in housing before the 1977 ban. As documented by the Department of Housing and Urban Development, over 40 million residential units in the United States are estimated to contain lead-painted surfaces because they were built before 1960 (Table 4). Of the 42 million units, some 26 million are in dilapidated condition (73), such that lead paint hazards are more likely to present risks of exposure through contamination of house dust and presence of paint flakes and chips (14, 15). Their distribution varies with the age of communities; Massachusetts, Illinois, New York, New Jersey, and Pennsylvania have millions of these units.

Dealing with existing lead paint in housing has proved difficult because of the complex of issues related to poverty, housing, and disease prevention. Housing is an unusual vector of disease: It cannot simply be eradicated without provision of adequate substitutes. When the most dangerous housing is in poor neighborhoods, the social concerns of providing any affordable housing for the poor can come into conflict with preventing disease.

The provision of lead-safe housing has been estimated to require major public or private investments, costing billions of dollars (73). Public policy, where it has existed (usually on the local level), has placed the burden on the private
sector to abate lead hazards when identified. For example, by 1980, Baltimore had developed, on paper, the most rigorous legal standards to require landlords to remove all lead paint from homes where children with elevated PbB were found.

Ford & Gilligan (22) were among the first to document the concerns about the practical problems with this policy. They evaluated the economic resources for private sector lead-paint abatement in Baltimore and concluded that landlords could not recover the costs of adequate abatement from the revenues that could be generated from rental housing in poor neighborhoods. They warned that existing laws, if fully enforced, would result in large-scale abandonment of housing and a loss of affordable housing for the poor. Their analysis was quickly taken up by property owners’ organizations to attack lead poisoning prevention programs in Baltimore, New York, and elsewhere. Their concerns were shared, for different motivations, by some advocates for housing and community development, such as the Enterprise Foundation. The controversy was considerably heightened by the success of attorneys for some children and their families in winning large judgments and settlements against property owners.

Despite applied research projects demonstrating that adequate hazard reduction could be achieved for substantially lower costs than first estimated (19, 20), the conflict remained. It came to a head after 1992, when legislation related to lead-paint poisoning prevention had been passed through the efforts of Senators Alan Cranston of California and Paul Sarbanes of Maryland, among others. The Housing Act attacked the lead-paint problem in three ways. First, it closed a loophole in occupational health, whereby abatement and construction workers had not been covered by the 1978 OSHA lead standard (68). Second, it enlisted market forces to encourage abatement by requiring disclosure of lead paint in private real estate transactions. Third, it required HUD to develop policies for dealing with private and public rental housing, particularly for poor and low-income families.

The real estate disclosure program, finally implemented with regulations in 1996, may encourage source reduction, but these are likely to occur only in those housing markets able to sustain the investment required, that is, where the value of property can absorb an abatement “penalty” borne by the seller (either as reduced sale price or investment in abatement prior to sale). The success of disclosure and notification to prevent lead poisoning in children is unknown. Some anecdotal evidence suggests that it has worked in Boston in terms of increasing the amount of abatement (S Pollock, personal communication). But it has served to separate the lead poisoning problem of the affluent from that of the poor. The poor are more likely to be renters than owners, and their housing is in many cases insufficiently valued to absorb the costs of abatement without public subsidy.
The problem of low-income housing was given by HUD to a national task force to resolve. This task force, advisory to HUD, was chaired by two advocates, for housing (C. Dolbeare) and lead poisoning prevention (D. Ryan). The task force noted the scope of the lead-paint poisoning problem and attempted to resolve the conflicts between housing affordability and lead poisoning prevention by two strategies: lowering the standards for abating lead paint hazards, and inducing property owners to take preventive actions in return for insulation against civil litigation. The Task Force endorsed a policy of managing lead paint in place, rather than requiring actual paint removal or replacement of certain structural elements, such as door and window frames [as recommended by Farfel & Chisolm (20)]. Property owners were to be immunized from litigation if they undertook certain hazard-reduction steps. These recommendations were not universally endorsed. A minority of the Task Force (of which this author was one) rejected the concept of managing hazards in place without an enforceable system of ongoing inspection and maintenance, and they criticized the legal immunity proposal on the ground that it stripped away one of the few protections, admittedly imperfect, available to children at risk.

Several states have adopted this strategy in law or practice. In Maryland, the state legislature passed a law that replaced Maryland’s rigorous program with the elements of the HUD Task Force recommendations. Similar proposals have been debated in other states.

**Lead-Contaminated Soil**

A similar retreat occurred in another arena where the expenses of remediation challenged the ambitious goal of source control. ATSDR had identified lead-contaminated soils as a significant source of lead exposures for children in the United States (1). Under the Superfund hazard ranking system, the presence of lead in soil tended to place a site on EPA’s National Priority List based on the assumption that lead in soil was significantly correlated with lead in children’s blood. Some 400 sites are on this list because of lead contamination. However, some studies of children living in communities with high soil-lead concentrations did not confirm this association. As reported by Freeman et al (23) and others (see 5), in some communities where the source of lead in soils was related to mining and smelting, no children with PbB above 10 µg/dL were found. This was in contrast to experience in smelter and mining communities in El Paso, Texas, (33) or Port Pirie, Australia (the site of the world’s largest lead mining and smelting operations in the 1960s and 1970s) (38). Once again, an aspect of social class seemed to play a role, as in the housing controversies. When the citizens of Aspen, Colorado, and Park City, Utah, two affluent resort towns in the Rockies, balked at being designated as Superfund sites, one citizen activist in Aspen remarked that “everyone knows that lead poisoning is a disease of poor Black kids and we don’t have any of [them] here” (New Yorker).
Studies on the association between urban soil lead and children’s PbB have been inconclusive. The EPA sponsored studies in Boston, Baltimore, and Cincinnati to test the effects of reducing soil lead on children’s PbB. Relatively small changes were observed (76). The cost of cleaning up lead-contaminated sites was clearly very high, because only soil removal could work. If communities with high soil-lead levels were added to the Superfund list, it was not clear who would or could pay for abatement, since in some cases the source was not an industry but rather the use of lead in gasoline (39).

In the face of this controversy, the primary prevention campaign again faltered. The EPA, charged with issuing standards for lead in soil, retreated to promulgating “advisory” levels that are not enforceable. They were not clearly based on any health-based concept of the potential contribution of lead in soils to lead in blood.

**Screening**

As the struggle for prevention failed in the case of housing and soils, another element in lead poisoning prevention also came under attack in the 1990s. Screening has always been an element of preventive health policy and early detection of lead exposure can prevent further exposures that result in irreversible toxicity and can also assist in preventing exposures of other children to the same source of lead. But debate has arisen on the meaning of screening, and the reasonableness of measuring lead in blood of all children in the United States.

In 1991, the Centers for Disease Control had recommended that all children under the age of two years should be screened at least once by blood lead testing (11). This recommendation was predicated on the NHANES II data that reported widespread prevalence of lead exposures (see Figure 3) and on the inadequacy of postexposure treatment as a means of preventing or reversing toxicity. At the “high-water mark” of screening, in 1990, some 3 million children <6 years were tested. However, for screening to fulfill the purpose for prevention (primary or secondary) it must be part of an integrated program to identify and control the child’s lead sources, as well as to manage children with elevated levels. According to a survey conducted by the Association of State and Territorial Health Officials (ASTHO) in 1992, only 21 states had implemented or were planning to implement the 1991 CDC guidelines. Less than half of the 48 states responding had a state system to monitor and follow up children with PbB > 10 µg/dL (4). In no state were all children with excess PbB followed up, with identification of exposure sources.

Soon after the 1991 recommendation by CDC, criticisms were expressed from several sources. Physicians in California and Washington criticized the recommendation of universal blood lead screening as inflexible and unresponsive to local conditions (6). They argued that lead poisoning was unknown
in many communities where risk factors were low, and that in these situations requiring universal screening was an unacceptable waste of valuable health resources for the public and private sectors. While some localities reported that increased screening efforts were revealing a greater than expected prevalence of lead exposures in children (for instance, in Rhode Island), others found few if any children with PbB $> 10$ µg/dL (many of these studies were published in Pediatrics ref 1994). The experience of Maryland in 1993 is illustrative. As shown in Table 6, the highest rates of elevated PbB were found in Baltimore City, with the lowest median family income in the state and the highest proportion of minority children, and a preponderance of housing built before 1950. In contrast, in Montgomery County, where median family income is among the highest in the United States, African American children comprise less than 25% of the population, and where the housing stock is newer, the prevalence of PbB $> 10$ among screened children was less than 1%.

In 1993, the American Academy of Pediatrics endorsed universal screening (16). But in 1994, Birt Harvey, of the Academy, recommended that universal screening should be abandoned (Harvey, 1994). He disputed the benefits of screening for children with PbB $< 20$ µg/dL; he pointed to the fact that in many communities few children were detected with PbB greater than 20 µg/dL; and he expressed concerns over laboratory resources and competence.

Under pressure, CDC formally reopened its recommendation for universal screening in 1995. While it is probably true that in some communities, few if any children are exposed to lead such that their PbB exceeds 10 µg/dL (e.g. 7), the practical challenge is to develop a method that accurately defines these communities without missing children who are at risk of lead exposure (69).
Moreover, if such a strategy could be developed and validated, it is not clear that it would save health resources. The costs of blood lead screening can be calculated; the costs of alternate screening methods have not been calculated. Yet the methods proposed by CDC recommend that health care providers administer and interpret an individualized questionnaire, in the context of information on those characteristics of the community that indicate the presence of lead exposure risks. This process has costs in terms of the time required by experienced health care providers and public health officials.

Other criticisms have been leveled against screening of any type, notably by the US Preventive Services Task Force (74). This group concluded that:

There is relatively little convincing evidence that these interventions [screening and environmental or medical interventions based on screening] improve health, however. One issue is that most available studies in asymptomatic children evaluate the effects of various interventions on blood lead levels rather than on clinical outcomes. Second, blood lead levels typically decline with the passage of time. On average, blood lead levels in childhood decrease with age after peaking at about 2 years of age, even without intervention. Longitudinal studies of asymptomatic children with elevated lead levels have shown reductions in blood lead levels after short- and long-term follow-up in the absence of any intervention, a result attributable at least in part to regression to the mean, random variation, and laboratory error.

Evidence is not available to demonstrate that universal screening for blood lead results in better clinical outcomes than either screening targeted to high-risk persons or individualized testing in response to clinical suspicion. Several older studies reported that, compared to historical results from individualized testing, intensive screening programs targeted to children in high-risk neighborhoods reduced case fatality rates, mortality rates, and proportions of children detected with very high blood lead levels or who developed symptomatic lead poisoning. In the absence of concurrent controls, it is not clear whether the reported reductions in mortality and case fatality rates were due to screening, or to improvements in medical care over time. Reductions in mean lead levels may also have been due to secular trends, changes in screening tests, and to screening greater numbers of children, including many at low risk for severe lead poisoning. Thus, the available evidence regarding the efficacy of screening programs is weak (74).

BENEFITS OF PREVENTING LEAD POISONING

Despite the controversies over how best to prevent lead poisoning, by screening or environmental interventions, several analyses have shown significant net benefits to reducing lead exposures for children. The first of these studies was conducted by Schwartz and colleagues at EPA, in an analysis to support regulating lead in gasoline (See 46 for a review; 62 for the original analysis). As shown in Table 7, a correlation can be drawn between PbB and IQ in children based on several studies conducted worldwide (43). IQ is then correlated...
Table 7  Meta-analysis, studies of the lead IQ relationship

<table>
<thead>
<tr>
<th>References*</th>
<th>Year</th>
<th>n</th>
<th>Effect size</th>
<th>Power to detect small effect</th>
<th>p (df)</th>
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<tr>
<td>Perino et al (29)</td>
<td>1974</td>
<td>80</td>
<td>0.6</td>
<td>0.2</td>
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<tr>
<td>Needelman et al (9)</td>
<td>1979</td>
<td>73</td>
<td>0.35</td>
<td>0.47</td>
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<td>Yule et al (30)</td>
<td>1981</td>
<td>82</td>
<td>0.573</td>
<td>0.42</td>
<td>0.021</td>
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<tr>
<td>Winneke et al (31)</td>
<td>1982</td>
<td>26</td>
<td>0.26</td>
<td>0.18</td>
<td>0.15</td>
</tr>
<tr>
<td>Smith et al (19)</td>
<td>1983</td>
<td>185</td>
<td>0.17</td>
<td>0.7</td>
<td>0.12</td>
</tr>
<tr>
<td>Winneke et al (32)</td>
<td>1983</td>
<td>115</td>
<td>0.351</td>
<td>0.25</td>
<td>0.4</td>
</tr>
<tr>
<td>Harvey et al (21)</td>
<td>1984</td>
<td>48</td>
<td>0</td>
<td>0</td>
<td>0.025</td>
</tr>
<tr>
<td>Shapiro &amp; Maracek (33)</td>
<td>1984</td>
<td>193</td>
<td>0.46</td>
<td>0.48</td>
<td>0.66</td>
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<tr>
<td>Lansdown et al (34)</td>
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<td>162</td>
<td>0.07</td>
<td>0.48</td>
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<td>0.5</td>
<td>0.34</td>
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<td>0.64</td>
<td>0.25</td>
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<td>Schroeder et al (36)</td>
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<td>0.5</td>
<td>0.33</td>
<td>0.005</td>
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<tr>
<td>Fulton et al (12)</td>
<td>1987</td>
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<td>0.4</td>
<td>0.52</td>
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<td>Hatzakis et al (13)</td>
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<td>509</td>
<td>0.4</td>
<td>0.52</td>
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\[ \sum x = 109.13 \]
\[ p = 2.97 \times 10^{-12} \]

*See Reference 45 for original citations.

with academic attainment, from other literature, and academic attainment with lifetime earnings. By these calculations, Schwartz (60) estimated that $6.9 billion would accrue in benefits if children’s PbB were reduced by 1 \( \mu \)g/dL across the population. Salkever (59) updated these estimates to an increased benefits amount of $2.5 billion per birth cohort. [The other neurobehavioral sequelae of lead toxicity—antisocial behavior, school dropout, and criminal activity—were not monetized, largely because data are not sufficient to support a marginal analysis associating these outcomes in a dose-response relationship with incremental changes in PbB. Nevertheless, as pointed out by Needelman (43), these costs may be very great.] Large benefits of lowering blood lead levels were found for reducing lead in gasoline, lowering the drinking water standard, and abating lead paint in housing (60–62). However in some cases net benefits (exceeding costs) were found only when the value of reducing the effects of lead on blood pressure in adults and on materials damage (damage to spark plugs and plumbing) was added into the analyses.

It is more difficult to calculate the benefits of screening. Briss and Schwartz have undertaken several cost benefit analyses, in order to support a rational basis for universal or less than universal screening (CDC, unpublished data).
A major problem lies in the controversy over the role of screening in lowering PbB as discussed above (74).

CONCLUSIONS

The prevention of lead poisoning achieved significant successes in the 1980s when new data on the nature and extent of lead toxicity provided political support for major actions in primary prevention, notably the removal of lead from gasoline. That action undoubtedly reduced environmental levels of lead in soils and air (42) and reduced blood lead levels across the US population (53).

However, the campaign for prevention foundered by the end of the 1980s for three reasons. First, the “easy” tasks of source reduction (removing lead from gasoline, paint, and plumbing) were accomplished [it should be noted that the United States is the only country where these “easy” tasks have been achieved (50).] Second, these accomplishments reduced the urgency of lead poisoning as an issue among the US public as a whole, because lead poisoning, once again, was viewed as a disease affecting only a segment of the population, the disadvantaged of society, in a time when the political climate had largely turned against the “liberalism” of a welfare state. Third, the remaining sources of lead exposures for children—lead in housing and soil—presented daunting economic and technical costs for their solution. Even the relatively small costs of universal screening were considered by some to be too high.

Yet lead poisoning has not disappeared. A disease with a national prevalence of 5% cannot be considered to have been eradicated, as the US government once pledged to eradicate childhood lead poisoning (70). Moreover, a disease with such disparate prevalence, affecting those beset by other conditions of prejudice and disadvantage at a rate of over 25%, or five times more than advantaged children, should not be tolerated in a humane society. The costs of this neglect are born by all, even if the risks of lead poisoning are not equally shared.

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Literature Cited


