
Childhood lead poisoning prevention, the CDC experience

This communication reviews the past experience with and the current status of childhood lead poisoning prevention efforts in the United States, especially as it relates to the work of the Centers for Disease Control and Prevention (CDC). The CDC is one of several federal agencies who have a role in controlling lead exposure in the United States population. CDC's responsibility has mainly concerned the public health response to childhood lead poisoning and has emphasized screening, surveillance, and interventions directed at individual children with elevated blood lead levels.

Other key federal agencies include the Environmental Protection Agency (EPA) for regulating lead emissions into the environment, the department of Housing and Urban Development (HUD) for control of lead exposure in federally-subsidized housing, the Food and Drug Administration (FDA) for limiting lead exposure from food and food containers, the Consumer Product Safety Commission (CPSC) for regulating lead exposure from new house paint and other consumer products, the National Institute of Health (NIH) for research into the health effects of lead and mechanisms of toxicity, and the Occupational Safety and Health Administration (OSHA) for regulation of occupational lead exposure.

The main topics which will be covered include recent research findings with an emphasis on studies in which CDC was involved and their impact on policies and programs in the following areas :

- health effects of lead ;
- surveillance of blood lead levels in the population ;
- screening ;
- assessment of residential lead exposure ;
- abatement of residential lead hazards.

Health effects

Serious lead poisoning prevention efforts in the United States started in the 1950's and 1960's when severe clinical lead poisoning was epidemic in most large cities. Hospital admission for frank lead encephalopathy was common and case fatality rates were high. As a result, prevention efforts were largely developed in the context of the medical model : symptomatic children were

treated with chelating agents, asymptomatic children were screened and treated if their blood lead levels were very high, and efforts were made to remove lead paint from dwellings of poisoned children.

It had been recognized that symptomatic lead poisoning could leave children with permanent neurologic impairment. Starting in the 1970's, researchers began to investigate the relation between blood lead levels and neurodevelopment in children who did not have symptoms. The culmination of that work came in the 1980's with several large, carefully designed studies that related levels of lead in blood of asymptomatic children to IQ (Intellectual Quotient), other cognitive measures, and behavior. The results of several such studies were synthesized in a meta-analysis by Schwartz (Schwartz, 1994). Schwartz estimated that a blood lead increase from 100 to 190 $\mu\text{g/l}$ is associated with an estimated IQ loss of 2.6 points, adjusted for multiple confounding factors. The weight of evidence supports a subtle effect on intellectual development at blood lead levels that had been thought safe.

All of the other animal and human research data concerning low-level lead toxicity will not be reviewed here. Suffice it to say that, based on this and similar information, in 1991, CDC issued new guidelines for preventing lead poisoning in young children (CDC, 1991). Among other recommendations was a reduction to 100 $\mu\text{g/l}$ in the blood lead level at which some action should be taken.

Trends in lead exposure

During the same period during which research lowered the threshold for known health effects of lead, lead exposure in the population was changing and these changes were identified through surveys and surveillance. The most important source of data on blood lead levels in the United States population comes from the National Health and Nutrition Examination Survey, or NHANES. This is an ongoing series of national examinations, conducted by CDC, of the health and nutritional status of the noninstitutionalized US population.

There was a dramatic decline in the geometric mean blood lead level among preschool children between the NHANES II survey, conducted in the late 1970's, and the NHANES III survey, conducted in two phases spanning the years 1988-1994. The geometric mean level in the early 1990's, 27 $\mu\text{g/l}$ (CDC, 1997a), is less than one-fifth the level in the 1970's.

This decrease in lead exposure has benefited all demographic subgroups of the population. However, lead exposure remains quite unevenly distributed in the population. Using 100 $\mu\text{g/l}$ as the threshold for an elevated blood lead level, the prevalence varies substantially by race, income, urban status, and the age of the dwelling in which the child lives. For example, for low income children

living in housing built prior to 1946, the estimated prevalence is more than 30 times higher than for middle income children living in housing built after 1973 (CDC, 1997a).

This pattern most likely reflects two major sources of lead exposure in the United States : deteriorated leaded paint and soil and dust contaminated by paint and by past leaded gasoline emissions. The three housing age categories, while not designed for assessing lead exposure, roughly correspond to three important milestones in the use of lead in paint. Around 1950, the paint industry in the United States voluntarily began to phase out the use of lead pigment in paint, though they continued to add lead to paint in smaller amounts for other purposes. Prior to that time, high levels of lead were found in most residential paints. In 1978, the addition of lead to housepaint was banned by the CPSC.

Exposure to high levels of lead in deteriorated paint and the house dust it contaminates probably accounts for much of the income and race gradients in blood lead levels. The association with urban status likely is due to the past deposition of lead from gasoline in more densely populated areas with more vehicles.

The decline in blood lead levels was not unexpected. In the late 1970's, the use of leaded gasoline began to decline with the introduction of catalytic converters for controlling auto emissions. The NHANES II survey data showed that the decline in use of leaded gas was paralleled by a decline in average blood lead levels (Annest, 1983).

Data from ongoing screening programs also tracked the decline in blood lead levels during the 1980's. The health department in the city of Chicago has been screening children with venous lead testing since the early 1970's and keeping records of test results. These records have proven useful for answering a number of questions about lead exposure and lead screening. We analyzed these data in conjunction with data from routine monitoring of air lead levels. Again we found a steady decline in the median blood lead level in this high-risk population, paralleling the decrease in airborne lead levels (Hayes et al., 1994). This is one example of how routinely collected blood lead testing data can be used for answering important questions about lead exposure in the population. Because of this, CDC provides funding and technical assistance to state health departments to develop and maintain systems for laboratory-based reporting of blood lead test results. This data is taking on increasing importance as states move to develop recommendations for targeted screening of high-risk populations.

The important role that phasing out leaded gasoline played in reducing lead exposure has already been mentioned. However, because of the many sources and pathways of lead exposure, a number of changes have probably contributed to the downward trend in blood lead levels (Pirkle et al., 1994). The other major factor, we believe was the virtual elimination of lead-soldered

food and beverage containers from the United States. Prior to that, leaching of lead from can solder was a major contributor to lead exposure in the general population. This change was achieved voluntarily by food canneries, with encouragement, but not regulation, by the FDA.

Lead exposure from paint is generally due to deteriorated older paint ; therefore the banning of leaded paint in 1978 did not have a major effect on lead exposure in the short run. However, as older housing has been demolished and newer housing built, the proportion of children living in housing with leaded paint has decreased substantially. Between 1980 and 1990, for example, nearly 4 million pre-1950 dwellings went out of existence.

Lead levels in drinking water are falling for two reasons. First, in 1988 lead-containing solder was banned from use in household plumbing ; prior to that, it had commonly been used to join copper pipes. Second, in the early 1990's, new clean water regulations were implemented, requiring public water systems to test for lead-contamination at household taps and treat water to reduce corrosiveness where high levels were found.

Ongoing screening programs and public education have almost certainly contributed to reducing lead exposure on the upper end of the blood lead distribution. Higher birthrates in low-income populations, increasing immigration, changes in government food programs have probably had a minor role changes in population blood lead levels.

Lead screening

The main function of the lead poisoning prevention program of CDC is to provide funding and technical assistance to state and local lead poisoning prevention programs. The CDC currently provides grant support to 30 state and 11 local health departments. These grantees were responsible for screening 1,6 million children in Fiscal Year 1997.

The recognition of health effects at low blood lead levels coupled with the reduction in lead exposure has had major implications for CDC's policies and programs concerning lead screening. In 1991, CDC issued new guidelines recommending that all children be screened for lead poisoning unless it could be demonstrated that a community did not have a childhood lead poisoning problem. Because health effects were recognized at levels below which chelation would be given, a multi-tiered approach to intervention was also recommended. Children with relatively high levels would receive medical evaluation, and possibly chelation, while lower levels would trigger education and environmental interventions. These recommendations for the interpretation of lead test results are still current.

414 Screening and medical treatment are key roles of pediatric providers, but CDC recommends that they play other roles as well. Most importantly, we

recommend that pediatric clinicians provide so-called « anticipatory guidance », that is, counseling about lead exposure and how to avoid it at routine well-child visits, even before children reach age 1, when screening is recommended.

Because of the decline in blood lead levels and their uneven distribution, pediatricians and health departments in some areas with a very low prevalence of elevated blood lead levels began to question the utility of universal screening in all areas. Our own analysis indicated that universal lead screening in very low risk communities would not be cost effective. In 1997 CDC issued revised guidance to state and local health departments concerning how to define low high risk areas and populations where universal screening would be most appropriate (CDC, 1997a and b). We are just in the process of working with health departments to help them implement this guidance and to evaluate its impact.

At the same time, our laboratory has been working with the private sector to encourage the development of portable, reliable, lead testing devices for use in the field or physician's office. This effort bore fruit this past year with the introduction of the LeadCare(device (Zink et al., 1997). This instrument has performance characteristics that compare quite favorably with laboratory-based devices. It is too soon to know how widely accepted this instrument will be in clinical practice, but it has the promise to make screening much more efficient by reducing the need for follow-up visits to confirm elevated blood lead levels.

Assessment of lead exposure in housing

The CDC and others have long called for moving towards primary prevention of lead exposure, rather than relying on secondary prevention. There has certainly been success in this area with respect to ongoing sources of lead exposure that can be reduced or eliminated by regulation. Examples include lead in gasoline and drinking water. However, it has proven far more difficult to move from secondary prevention to primary prevention of lead hazards associated with housing.

It is useful to review some of the limitations of the secondary prevention approach. First, because lead accumulates in bone stores from where it is released slowly over time, reducing blood lead levels in children who have had long term exposure can be difficult. Second, it is not clear to what extent effects of lead exposure on neurodevelopment can be reversed with reduction in exposure or other interventions. Third, in older, more mobile children, identifying sources of current lead exposure can be more challenging. Fourth, removing lead hazards from homes occupied by children can be more difficult and costly than at other times, such as unit turnover. Fifth, treatment of lead

toxicity has side effects and risks. Finally, and perhaps most important are the ethical problems with using children as « lead detectors » before responding.

In order to move to primary prevention of lead hazards in housing, we need better information concerning the assessment of risk in housing and how to most cost-effectively reduce the risk of residential lead exposure.

It was long thought that lead poisoning required pica for paint chips. However, in a study of the relation between finding evidence of paint chip ingestion and the severity of lead poisoning, recent paint chip ingestion was rare among children with blood lead levels less than 500 µg/l, but much more common for children with higher levels (Mc Elvaine et al., 1992). Fortunately, such cases represent a small proportion of children with high blood lead levels today.

It has now been established that lead in housedust, contaminated by deteriorated paint, tracked in soil, or both, is the most important vehicle for lead exposure in most young children. Bornschein et al. (1986) at University of Cincinnati used structural equation modeling to demonstrate the pathway from paint to dust to hand to blood lead. Many questions have remained, however, concerning the dose response relation for house dust lead and blood lead and concerning the methods used to sample housedust. More recently, Lanphear et al. (1996a) at the University of Rochester addressed some of these questions. (figure 1). The authors selected children in Rochester (a small city in the northern part of the state of New-York) who were 12 to 31 months of age and who had lived at the same residence since at least age 6 months. They found that housedust lead level was the most important predictor of blood lead, along with paint lead level and condition and soil lead level. They also established that the loading of lead in dust (weight of lead per unit area) was a better predictor of blood lead than was mass concentration of lead in dust and that a simple wipe method performed just about as well as a more cumbersome vacuum method.

Lanphear et al. (1998) recently completed a combined reanalysis of 12 studies to examine the dust lead-blood lead relation in children and better define safe levels of exposure (figure 2). Prior to this work, standards for acceptable levels of lead in housedust on floors had been set at 200 µg/ft² and were recently lowered to 100 µg/ft². The statistical methods accounted for the inevitable bias in regression slopes due to error in the dust lead measurement. The reanalysis showed a strong gradient in blood lead level occurs with floor dust lead levels far below these standards. Lead in exterior dust or soil also has an important effect at any given level of lead in housedust. Even at an interior dust lead loading of 20 µg/ft², the model predicts more than 10 % of children will have blood lead levels above 100 µg/l if the exterior soil lead level is near the residential average for the nation. On this basis, the department of HUD is proposing reducing the standard to 40 µg/ft², a compromise between health considerations and feasibility in urban areas.

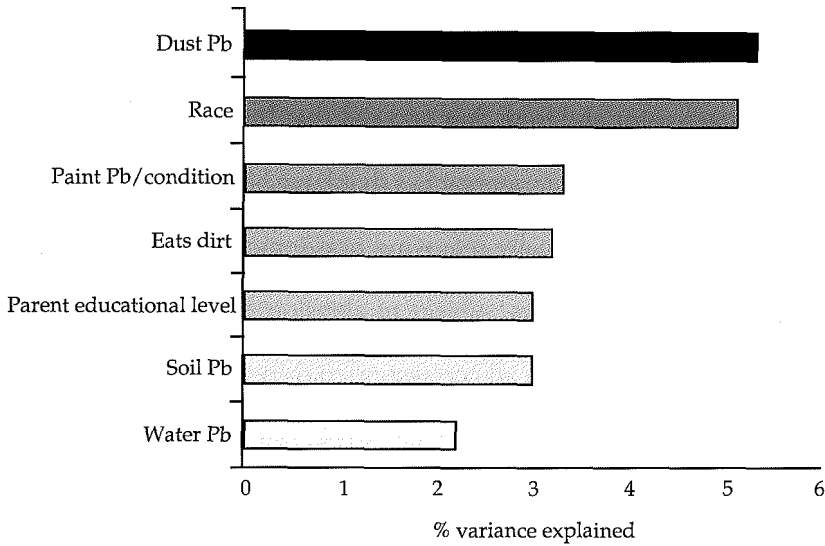


Figure 1 : Predictors of children's blood lead levels. Rochester, New-York (Lanphear et al., 1996a).

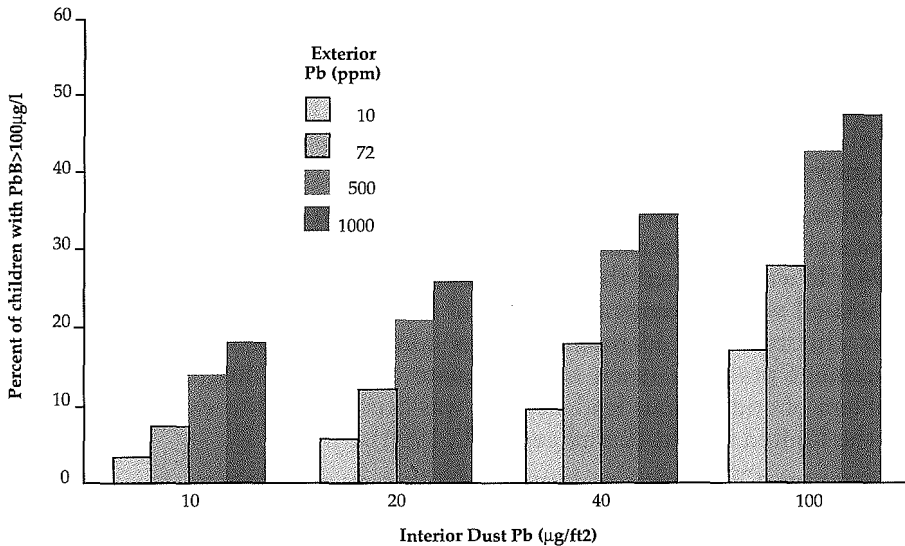


Figure 2 : Impact of lead in dust and soil on blood lead (Lanphear et al., 1998).

Reduction of lead hazards in housing

With a standard defining a hazardous level of lead in housedust, it will be possible to begin testing dwellings before children are lead poisoned by them,

perhaps before such children are even born or move into them. The next step in primary prevention is to take some measures to reduce the level of lead exposure in the home. Since we now know that children are exposed to lead from non-intact paint and from dust and soil that has been contaminated by paint and other sources, we must consider these pathways in reducing lead hazards. A listing of the basic approaches that have been proposed to reducing lead exposure in housing range from stabilization of non-intact painted surfaces containing lead (basically removing non-intact paint and repainting), removing paint, covering leaded paint, replacing lead painted components, especially those, like windows, subject to abrasion of paint, and floor treatments to make floors smooth and cleanable. There are many variations on each basic approach, but they all correspond to things that are done routinely during building maintenance and remodeling. However, special precautions are required before, during, and after such work, to ensure that lead exposure, especially from dust, is reduced rather than increased.

Since that time there has been a great deal of debate about the most effective, cost-effective, and safest approaches to lead paint hazard reduction. A full discussion of the technical aspects of lead hazard reduction are beyond the scope of this paper. Instead some recent studies in this area in which CDC has been involved will be reviewed.

The systematic study of this question did not really begin until the mid-1980's when investigators Farfel and Chisholm at Johns Hopkins in Baltimore began to examine why many children's blood lead levels did not come down, or indeed increased after their homes were deleaded. Based on their work, it was concluded that extensive on-site paint removal was difficult to perform without increasing lead exposure.

A retrospective study of lead paint abatement in St. Louis (where limited paint stabilization was their standard of practice) was undertaken (Staes et al., 1994). It was observed that, while some reduction in blood lead level was associated with abatement, blood lead levels were still quite high one year after children were identified. It was concluded that limitations of their abatement practices (such as inadequate cleanup of dust) along with the problem of endogenous lead stores it was been alluded to earlier, accounted for the limited benefit (Lanphear et al., 1998).

Two recent CDC-supported studies looked at dust control measures as a possible short-term, low cost measure where lead paint hazards could not be fully corrected. Both were randomized controlled trials. The Childhood Lead Exposure Assessment and Reduction study (CLEAR) in Jersey City involved education plus regular visits by cleaning crews and showed a 17 % reduction over a 1 year period (Rhoads et al., 1996). The Lanphear study involved only education and the provision of cleaning supplies to occupants, but found no benefit (Lanphear et al., 1996b). Both studies were « secondary prevention » studies and so may underestimate the benefits that similar interventions could achieve if begun before children accumulate lead burdens.

The largest study yet conducted of residential lead hazard reduction is an ongoing evaluation of a Department of Housing and Urban Development Grant program for correction of lead hazards in low and moderate income housing (National Center for Lead Safe Housing, 1998). The CDC helped to design the study and has collaborated in quality control and the analysis of results. In addition to the large and diverse sample of dwellings involved (more than 3 000 in 14 cities and states), the evaluation includes the collection of detailed data on the specific treatments used on each building component and their costs. While the final results of this evaluation will not be available until the millenium, some useful data are emerging.

The state and local governments contracting to have the work done were given flexibility to vary the approach to lead paint hazard reduction depending upon their budget and other considerations. The common interior approaches can be grouped into four broad categories : interim controls (spot paint stabilization and cleaning) only ; interim controls plus complete repainting ; the addition of window treatments to reduce friction ; the addition of window replacement. Median costs vary form several hundred dollars for interim controls only to more than \$8 000 for interventions that included window replacement (figure 3).

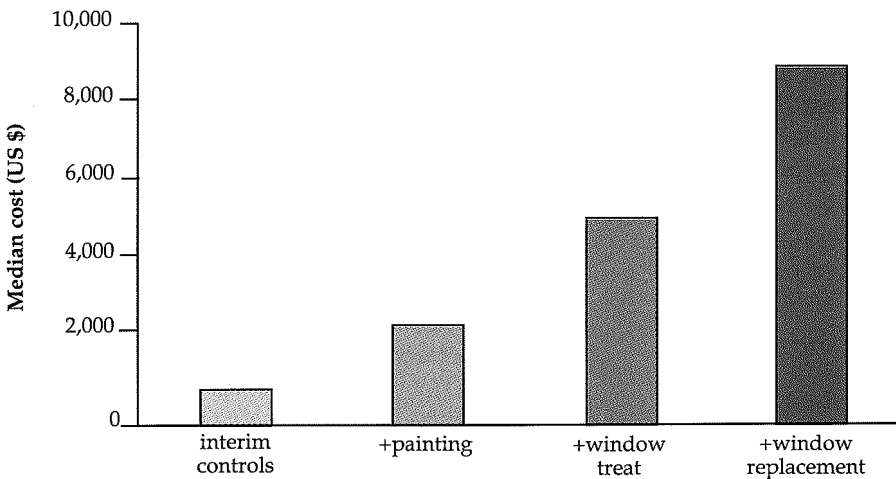


Figure 3 : HUD evaluation – Cost by interior strategy – Multi-family.

The ultimate objective of the analysis will be to assess the relation of health benefits to the costs and durability of various strategies and treatments. Overall, dust lead loadings are reduced by the interventions, but that the largest effect is seen in dwellings with high baseline levels. Dust lead levels for floors, window sills and windows troughs have shown similar patterns, but

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larger declines for window sills and troughs. There is also a tendency for some recontamination to occur with time, but not back to baseline levels.

Blood lead levels are also declining, on average and there are very few children showing increases that might suggest some contamination by the abatement process. Further analyses will involve adjustment for seasonal factors and aging of the children as well as relating the changes to the types of interventions carried out.

As a result of research carried out over the last several years, our knowledge base concerning how to prevent lead poisoning in housing is starting to catch up with our knowledge of the health effects of lead at low level. Looking ahead, we see the need for continued research to refine our knowledge in a number of areas, especially : the effectiveness of lead hazard reduction in housing ; the effectiveness of education and other low cost interventions ; the effectiveness of lead chelation on neurodevelopment (an ongoing NIH clinical trial will provide information on this a few years hence) ; the sources of lead in outside dust and how to remediate it ; and how to implement lead hazard reduction measures in housing in a primary prevention mode.

The Centers for Disease Control's work in prevention of childhood lead poisoning is very important and progressively bear fruit ; but a lot of questions and problems still stay. That is why these efforts will be carried on, in collaboration with the others agencies concerned.

To conclude, while efforts to reduce lead exposure in the U. S. population represent a public health success, additional efforts, especially related to the lead hazards in housing are required. CDC will continue its work with other federal agencies until childhood lead poisoning is eliminated as a public health problem.

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