POLICY FORUM: ENVIRONMENTAL HEALTH

The Paradox of Lead Poisoning Prevention

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Subclinical lead toxicity, defined as a blood lead level of 10 μg/dl or higher, is estimated to affect 1 in every 20 children in the United States (*1*). The preponderance of studies demonstrate that low-level lead exposure has serious deleterious and irreversible effects on brain function, such as lowered intelligence and diminished school performance, especially from exposures that occur in early life; hearing deficits and growth retardation have also been observed (*2*). Collectively, the results of these studies argue that efforts to prevent neurocognitive impairment associated with lead exposure should emphasize primary prevention—the elimination of residential lead hazards before a child is unduly exposed. This contrasts, paradoxically, with current practices and policies that rely almost exclusively on secondary prevention efforts—attempts to reduce a child's exposure to residential lead hazards only after a child has been unduly exposed. Furthermore, despite an abundance of recommendations about how to prevent lead exposure from residential hazards, there is a paucity of data demonstrating the safety or benefits of these recommended controls for children with blood lead levels below 25 μg/dl (*3*).

Although the mechanisms by which lead causes its toxic effects remain unknown, substantial progress has been made in reducing widespread lead exposure. Before 1970, lead poisoning was defined by blood lead levels greater than 60 μg/dl, a level often associated with acute symptomatic disease, including abdominal colic, frank anemia, encephalopathy, or death. Since then, the threshold for defining elevated blood lead levels has gradually been reduced. In 1991, the Centers for Disease Control (CDC) reduced the threshold even further, to 10 μg/dl (*4*). During the past two decades, average blood lead levels in U.S. children have fallen by over 90%, largely as a result of the elimination of lead from gasoline and dietary sources (primarily foods and beverages from lead-soldered cans) (*5*). It is currently estimated that 890,000 (4.4%) preschool children in the United States have a blood lead level of 10 μg/dl or higher (*1*). In some cities, especially in the northeastern United States, more than 35% of preschool children have blood lead levels exceeding 10 μg/dl from exposure to residential lead hazards (*6*).

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From Screening Children to Housing

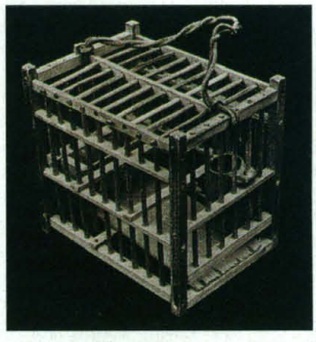
Universal screening of children for elevated blood lead levels in the United States is controversial. Blood lead levels in U.S. children vary greatly by age, poverty level, race, and condition and age of housing (*1*, *6*). Because lead exposure is so variable, few children are identified as having an elevated blood lead level in some communities. As a result, many pediatricians and public health officials are hesitant to support universal screening or vigorously oppose it. In addition, because lead exposure is cumulative and its detrimental effects are irreversible (*7*), any strategy that is limited to screening children after an exposure has occurred is flawed. Although there continues to be a need to refine screening strategies to target and identify children with undue lead exposure (*8*), it is more critical to expand our efforts to identify and eliminate residential lead hazards before children are unduly exposed.

Residential Sources and Standards

Paint appears to be the major source of childhood lead poisoning in the United States. Children with blood lead levels above 55 μg/dl are more likely to have paint chips that are observable in abdominal radiographs, and most preschool children with blood lead levels greater than 25 μg/dl are reported to have put paint chips in their mouths (*9*). In contrast, house dust contaminated with lead from deteriorated paint and from soil tracked in from outdoors is the major source of lead ingestion for children with blood lead levels between 10 and 25 μg/dl (*10*, *11*). More than 95% of U.S. children who have elevations in blood lead fall within this range (*1*).

Under section 403 of Title X, the U.S. Congress mandated that the Environmental Protection Agency (EPA) promulgate health-based lead standards and post-abatement clearance testing for house dust and residential soil. There are at least three reasons to develop residential lead standards. First, standards are necessary for screening high-risk housing to identify lead hazards before occupancy and before a child is unduly exposed. Our current strategy of identifying children only after they have been unduly exposed to lead rather than to screen high-risk housing before occupancy is analogous to the practice of sending a canary down a mineshaft to determine whether toxic gases have been released (see figures). Second, residential standards are critical to identify and eliminate lead hazards for children who already have elevated blood lead levels; major sources of lead will be neglected if dust and soil testing are not routinely done. Finally, standards serve as a benchmark and are necessary to compare the effectiveness and duration of various lead hazard controls.

A number of controversies have delayed the promulgation of residential lead standards, however. There was, for example, considerable debate about how to assess a child's risk of lead exposure. It has largely, but not entirely, been resolved that dust sampling should be done by using a wipe method, which typically measures lead loading in terms of micrograms of lead per unit of surface area (*12*). Most studies indicate that dust lead loading is a better pre-



E. DOTTER

Soul sacrifice. Canary's cage used to detect the release of toxic gases in a mine. The death of the canary serves as a warning that toxic gases are present. (Below) A canary cage in use.

dictor of children's blood lead levels than is lead concentration (that is, micrograms of lead per gram of dust). Moreover, as compared with measurements of the lead concentration of paint, measurements of dust samples are a better indicator of whether a lead hazard exists (that is, whether lead is accessible to a child) (*10*, *11*). The EPA, however, has continued to rely on a mechanistic model based on dust lead concentration.

Controversy over the levels of lead in house dust and residential soil that are associated with undue lead exposure also has delayed the promulgation of residential lead standards. In a rule proposed 1 June 1998, the EPA defined their level of statutory concern as between 1 to 5% probability of a child having a blood lead level in excess of 10 μg/dl and proposed a floor lead standard of 4.6 μg/m^2 (50 μg/ft^2) (*13*). Scientists have estimated from epidemiologic data that 5% of children will have a blood lead level ≥10 μg/dl at a floor lead level of 0.46 μg/m^2, or one-tenth of the proposed EPA floor standard (*10*, *14*). At a floor standard of 4.6 μg/m^2, 20% of children are estimated to have a blood lead level ≥10 μg/dl (*10*, *14*). Children who are exposed to floor dust lead levels ≥3.7 μg/m^2 (40 μg/ft^2) are at 10 times the risk of having blood lead levels ≥10 μg/dl as compared with those exposed to levels below 0.92 μg/m^2 (10 μg/ft^2) (*10*, *14*). Thus, the floor standard proposed by EPA is inconsistent with their definition of blood lead levels that "pose a threat."

Prevention of Lead Poisoning

Lead poisoning is often regarded as a preventable disease. In practice, however, the safety and benefits of measures intended to control or reduce residential lead hazards are uncertain. Interventions to prevent or control childhood lead exposure (called lead hazard controls) have far too often been shown to result in an increase in children's blood lead levels (*15*). There is some evidence that lead hazard controls, including abatement (that is, complete removal of lead-based paint by scraping and replacement of doors and windows) and stabilization (that is, repair of deteriorated painted surfaces) of lead-based paint in residential dwellings, can reduce lead exposure for children who have blood lead levels ≥30 μg/dl (*16*). In contrast, for children who have lower blood lead levels, there is little evidence that these interventions are beneficial; in some cases they have caused a rise in children's blood lead levels (*15*). Presumably, this rise in blood lead levels is due to dispersion of lead resulting from removal or scraping of leaded paint (*17*). It is likely that lead hazards caused by lead hazard controls can be overcome by promulgating effective health-based dust standards and requiring that clearance tests be conducted after any renovation or abatement is completed (*12*–*14*).

A comprehensive strategy for the primary prevention of childhood lead poisoning should include several components. First, the promulgation of effective health-based residential lead standards is essential for use both as a screening tool and after lead hazard controls or major renovation. If the final EPA lead standards are set too high, because of either economic considerations or uncertainties about what lead levels are feasible to attain, children will continue to be used as biologic indicators of lead hazards (as canaries in mines)—especially children who are black or impoverished (*1*, *4*–*6*, *10*, *14*). In contrast, if these standards are set too low, it may be difficult to find adequate and affordable housing for families with children. Second, it is critical to identify and target housing containing lead hazards, especially those causing lead poisoning. National, state, and community surveys of housing need to be conducted to identify and prioritize the elimination of lead hazards before occupancy by children. Residential screening could be done on a routine basis, depending on the age and condition of the house, or terminated if certain lead hazard controls are conducted and the housing is certified to be free of lead hazards. Third, once residential hazards are identified, it is critical that safe and effective methods to eliminate lead hazards be available. Lead hazard controls need to be assessed in trials that are experimental in design or, at a minimum, include a control group to account for potential confounding variables, such as seasonal variation and the predictable decline in children's blood lead levels as they mature. Finally, it is necessary to develop a plan for the gradual elimination of lead hazards during renovation or demolition of older housing.

Rhetoric or Responsibility?

The costs of eliminating childhood lead poisoning from residential hazards are substantial. It has been estimated, for example, that the first-year cost of reducing residential lead hazards in federally owned or federally assisted housing would be $458 million. The overall estimated benefit, defined as increase in lifetime earnings of children who are protected from the detrimental effects of lead exposure, was $1.538 billion—a net benefit of $1.08 billion (*18*). This estimate does not include other anticipated advantages, such as reduction in cardiovascular disease, behavioral problems, and delinquent behaviors.

Despite a strong federal commitment to children's health, it is unlikely that EPA's final residential lead standards will adequately protect urban children from undue lead exposure. The current lead poisoning prevention strategy largely ignores existing scientific evidence, which indicates that our efforts should emphasize primary prevention. Most federal agencies involved in the prevention of lead poisoning acknowledge that primary prevention is preferable, yet our efforts continue to be focused on screening children for elevated blood lead levels and controlling lead hazards after a child has been unduly exposed. For too long, policy decisions about lead poisoning have ultimately favored the lead industry or economic concerns over children's health (*19*). The lead industry has left a toxic legacy comparable with that of the tobacco industry—yet it has contributed nothing to its resolution. It is time to establish a scientifically based strategy to eliminate subclinical lead toxicity by controlling residential lead hazards; it is within our grasp.

References and Notes

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