HEALTH EFFECTS OF ENVIRONMENTAL TOXINS IN DEFICIENT HOUSING*

PHILIP J. LANDRIGAN, M.D., M.SC.
Chairman
Department of Community Medicine
Mount Sinai School of Medicine
New York, New York

HOMELESSNESS AND PRECARIOUS housing have become widespread in New York City and across the United States.[1] Estimates of the number nationwide who lacked adequate housing ranged in 1987 from 350,000 to more than 3 million. The one-year increase from 1986 to 1987 in the number requiring shelter was 20%.

Although traditionally the homeless population consisted principally of single adults, most of them male, that group accounts at present for only about 20% of the homeless. Approximately 20% of the population in shelters are now women. Families with children represent more than 33% of the homeless population nationally, and in New York City they comprise more than 75%.

People without adequate housing lack ready access to health care, and what care they receive is often fragmented. They are subject to a broad range of acute and chronic illnesses, including trauma, respiratory disease, AIDS, tuberculosis, infestations by scabies and lice and peripheral vascular disease (Table 1). These are common illnesses, magnified by disordered living conditions, exposure to extremes of heat and cold, lack of protection from rain and snow, bizarre sleeping conditions, and overcrowding in the shelters.[2,3]

Homeless children, 50% of whom are younger than five years of age, are at increased risk of malnutrition, anemia, neurologic disorders, developmental delays, and seizures. They are at high risk of abuse.[1] Between 25 and 50% of homeless children in New York City are not adequately immunized against measles, diphtheria, pertussis, tetanus, or poliomyelitis (Table II).[4,5]

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Address for reprint requests: Department of Community Medicine, Mount Sinai School of Medicine, 1 Gustave L. Levy Place, New York, NY 10029

TABLE I. MEDICAL PROBLEMS OF THE HOMELESS

Trauma	— violence		
	— blunt trauma		
	— burns		
Respiratory Infections		— colds	
		— influenza	
		— pneumonia	
Tuberculosis			
Infestations	— scabies		
	— lice		
Peripheral vascular disease with leg ulcers			
Inadequately treated chronic disease			— diabetes
			— psychiatric illness
AIDS			

LEAD: A TOXIC HAZARD OF DEFICIENT HOUSING

Lead is the principal environmental toxin encountered by children living in deficient housing. Although great gains have been made in understanding and controlling lead poisoning, including substantial reductions of lead in paint, food, milk, water, air, and gasoline, increased lead absorption continues to be widespread in the United States, particularly among poor children in large cities.[6,7]

Lead paint is the most important source of high-dose, concentrated lead exposure for urban children.[8] Exposure to lead-based paint has decreased since the Lead-Based Poisoning Prevention Act in 1970. Prior to that time, some interior paints in the United States contained more than 50% lead by

TABLE II. MEDICAL PROBLEMS OF HOMELESS CHILDREN

Anemia (primarily from nutritional deficiencies)		
Seizures		
Poor teeth		
Gastrointestinal illness		
Skin disorders		
Infestations		
Psychological problems	—	developmental delays
	—	depression and anxiety
	—	learning difficulties
Inadequate immunization		
Lead poisoning		

weight. By contrast, today the legally mandated limit for the lead content of new paint is 0.06%. However, old lead-based paint remains widespread. Most lead-based paint is encountered in housing units built before 1950. Additionally, the flaking and weathering of leaded paint contributes to the lead content of urban dust and soil.[8] As childrens' exposure to lead from other sources continues to be reduced, the distribution of childhood lead poisoning in the United States will increasingly reflect the legacy of lead paint used in urban areas.

Inadequately housed children are at particularly high risk of encountering lead-based paint, because they typically move from housing unit to housing unit under poorly supervised conditions; in their migrations, inadequately housed children may encounter housing units that have not been deleaded. Also, there is danger that children may encounter lead-based paint in shelters; most shelters were built for purposes other than the housing of small children, and they may not have been subjected to the same sort of scrutiny for lead hazards as have housing units.

Inadequately housed children may also be exposed to lead if they are present while abatement of lead-based paint is proceeding. Paint abatement may generate substantial quantities of lead-containing dust and fumes that may be either ingested or inhaled by young children.[9]

In New York City an estimated 422,800 (60.1%) of 703,500 children, one through five years of age, live in pre-1950 houses potentially contaminated with lead-based paint. This is by far the largest number of children in such housing in any city in the United States.[10]

Lead in soil and dust is another source of lead exposure for inadequately housed children.[6] Lead in soil and dust is derived primarily from the weathering and flaking of lead paint on houses and from the burning of leaded gasoline and from industrial emissions. Lead is very persistent and relatively immobile in soil under normal conditions of acidity. Young children absorb lead from dust and soil by ingestion as a consequence of their normal oral exploratory behavior.

The U.S. Public Health Service has developed estimates of the distribution of blood lead levels among preschool children in the United States.[10] These estimates are based on the Second National Health and Nutrition Examination Survey (NHANES-II), a nationwide health survey conducted from 1976 to 1980. The principal finding in these estimates was that in 1984, the most recent year for which data are available, approximately 2.4-million urban children in the United States (approximately 17% of all urban children) had blood lead levels of 15 μg/dl or more (Table III).

TABLE III. ESTIMATED DISTRIBUTION OF ELEVATED BLOOD LEAD LEVELS IN ALL URBAN CHILDREN SIX MONTHS TO FIVE YEARS—UNITED STATES, 1984[10]

		Blood lead levels	
Population	*>15 μg/dl*	*>20 μg/dl*	*>25 μg/dl*
13,840,000	2,380,600 (17%)	715,500 (5.2%)	200,700 (1.5%)

Among black children living in poverty (annual family income below $6,500.) in the central areas of large cities with populations greater than one million, the estimated distribution of blood lead levels was as shown in Table IV.

The principal finding here is that 68% of preschool children in this highly disadvantaged group, a group which includes many inadequately housed and homeless children, had blood lead levels of 15 μg/dl or more. Although precise information on the distribution of blood lead levels in inadequately housed children in New York City is not available, clearly the prevalence of elevated blood lead levels in inadequately housed children must be very high.

Health Effects of Lead Exposure

Epidemiologic and clinical studies using increasingly sensitive biological markers have established that lead is toxic to children at levels that were considered safe only a decade ago. Lead at low levels is now recognized to cause "subclinical poisoning," a syndrome that affects erythrocytes, kidneys, reproductive organs, and, most important, the nervous system of children. All of these effects may occur in children who have no clinical symptoms of lead poisoning at blood lead levels as low as 15 μg/dl and possibly even lower.

Effects on erythrocytes. Anemia is the most serious hematologic effect of lead toxicity. Lead impairs heme biosynthesis and increases erythrocyte destruction. In young children, effects on hematocrit first become evident at blood lead levels of 20-30 μg/dl and become more pronounced about 40 μg/dl.[11] Inhibitory effects on heme biosynthetic enzymes are observed at still lower levels. Thus, inhibition of ferrochelatase with resultant elevation of free erythrocyte protoporphyrin (FEP) begin at blood lead levels of 15-20 μg/

TABLE IV. ESTIMATED DISTRIBUTION OF ELEVATED BLOOD LEAD LEVELS IN BLACK CHILDREN BELOW THE POVERTY LINE IN CITIES LARGER THAN ONE MILLION—UNITED STATES, 1984[10]

		Blood lead levels	
Population	*>15 μg/dl*	*>20 μg/dl*	*>25 μg/dl*
346,000	234,900 (68%)	106,700 (31%)	36,700 (11%)

dl in young children.[12] Inhibition of delta-amino levulinic acid dehydratase (ALA-D) begins at blood lead levels below 10 μg/dl, and inhibition is 50% complete at blood lead levels of 30 μg/dl.[13] Iron deficiency, a common phenomenon in inadequately housed children, increases vulnerability to lead-induced anemia.[14]

Effects on the nervous system. At high doses, lead damages the peripheral nervous system, principally affecting large myelinated motor fibers. Patients with severe lead neuropathy show wrist or ankle extensor muscle palsy (wrist or ankle "drop"). Recently, sensitive electrophysiological studies in both adults and children have detected subclinical dysfunction in the peripheral nerves at progressively lower levels of lead exposure. One series of studies of lead workers with blood lead levels under 40 μg/dl found evidence for slowed motor nerve conduction velocity.[15] Other studies have complemented these observations, finding subclinical alterations in neuromuscular performance such as reductions in grip strength and eye-hand coordination.[16] In children with increased lead absorption, slowed nerve conduction velocity has also been observed and appears to reduce the ability to perform rapid, highly coordinated hand movements.[17]

Blood lead levels above 120 μg/dl in adults and above 100 μg/dl in children have been associated with acute central nervous system toxicity. Manifestations include coma, convulsions, irreversible intellectual and behavioral impairment, and even death.

In children, lower exposures, characterized by blood lead levels of 15-40 μg/dl, cause chronic, clinically invisible dysfunction of the central nervous system, characterized by subtle, but apparently irreversible deficits in intelligence, behavior, and school performance. One carefully controlled cross-sectional epidemiologic study found that, after correction for socioeconomic factors, clinically asymptomatic children with elevated body lead burdens had a 4.5-point mean deficit in verbal I.Q. scores compared to children with lower lead burdens. These shifts in mean I.Q. scores are associated with substantial reductions in the number of children with I.Q.s above 125 and with a trebling in numbers with I.Q.s below 70.[18]

Most recently, a series of prospective epidemiologic studies of children exposed to lead *in utero* and followed longitudinally through early childhood noted that depressions of I.Q. scores were correlated with lead levels in umbilical cord blood at birth, even for cord blood lead levels as low as 15-20 μg/dl.[19-21] These findings indicate that the subclinical effects of lead on the central nervous system of young children occur at even lower levels than previously recognized. These findings suggest that there may be no safe level for lead in blood in children.

DISCUSSION

The tragedy of the continuing epidemic of childhood lead poisoning is that certain consequences, especially the subclinical neurologic sequelae, are irreversible. No therapy can replace neurons destroyed by chronic absorption of lead or lost intelligence. Thus, the major effort of physicians and policy makers must be directed at prevention.

Approaches to the prevention of lead exposure in childhood include: removing all lead from gasoline, banning lead from all pigments, including ink and paint; abating lead paint in dwellings; developing a safe alternative to the lead storage battery; stringently enforcing existing standards or instituting a new standard based on recent understanding of the toxicity of low doses of lead, to reduce workplace exposure; and more strictly regulating lead levels in soils, surface dusts, and drinking water.

For inadequately housed children, the major lead hazard is lead-based paint. Exposure to lead-based paint is far too common among poor young children in large cities, including inadequately housed children. In such children, the loss of intelligence induced by lead constitutes an additional impediment to normal development, an impediment that will compound and intensify the effects of the numerous social and behavioral traumas suffered by these children.

Lead exposure is, however, a specifically indentifiable and preventable cause of toxic injury in inadequately housed children. It should, therefore, be specifically prevented.

Questions and Answers

DR. ULLMANN: How quickly do you expect this problem of lead exposure in inadequately housed children to become somewhat less significant? Do you expect major reductions in this problem?

DR. LANDRIGAN: We are not going to make much progress until we reduce the number of housing units that contain lead. We have virtually taken care of the problem of leaded gasoline. Also, because of the deindustrialization of New York, we have pretty well taken care of industrial sources of lead in this city. The major residual source of lead exposure for children is paint and, to a lesser extent, contaminated soil and dust.

DR. ULLMANN: But that is primarily a function, then, of failure to maintain property, isn't it? After all, lead paint isn't made any more, is it?

DR. LANDRIGAN: The problem may secondarily be a function of failure to maintain property. However, going back to first causes, it has been docu-

mented that in the 1930s and 40s the lead industry went around to state legislators throughout this country and persuaded the state legislators that lead paint should not be banned for domestic use. The industry argued that if lead paint were used safely, it would be safe. In consequence of that campaign, the industry was able to delay for many years the banning of lead-based paint; in the intervening years, many thousands of additional housing units were painted.

Today we are faced with the legacy of that industry campaign. I would argue there is a need for direct targeted governmental action today—either expenditure by government or government-mandated expenditure by landlords and homeowners to remove the lead. There is no other way. We can't just talk about keeping clean houses and thereby preventing children from getting into lead paint. That won't work, because the paint is already there.

DR. ULLMANN: But lead paint is still made then?

DR. LANDRIGAN: Yes, but no longer for home use. The only place where lead paint is applied *de novo* today is in marine applications and for yellow paint on highways.

PARTICIPANT: You really didn't mention any data specific for the issue of lead in homeless children. You talked more generally about inner city blacks. However, there are some data; they are not public, but come from the Bellevue group, who looked at free erythrocyte protoporphyrin (FEP) and the prevalence of anemia in children coming from various welfare hotels. The children from the welfare hotels were compared with a group of inner city kids coming to the Bellevue clinics. Although the incidence of anemia was the same in the two groups, the trend was to higher levels of FEP in the homeless children. Perhaps this trend is a reflection of higher lead burden in the kids from the welfare hotels. These data were presented at the ambulatory pediatric meetings a couple of years ago.

DR. LANDRIGAN: That is interesting information. Thank you.

PARTICIPANT: In Hudson County, New Jersey, we are seeing an increase, not a decline, in lead poisoning in children. Even though we are not sure what is causing it, renovation of houses—improper abatement—may be factors. We are not seeing a decline.

DR. LANDRIGAN: That is interesting. Two causal factors may be operating here. One would be relocation of families into homes which have not previously been occupied by young children and are contaminated with lead-based paint. The only way to assess this possibility would be to actually interview some of the children and their families and to test the homes. And then, the second possible cause could very well be the hazards associated with improper paint abatement.

Improper paint abatement can be a very serious problem. Indeed, we had a family seen at Mt. Sinai about a year ago. They were not economically disadvantaged; the father was a university professor and the mother a magazine editor. They had bought a farmhouse in the Hudson Valley near Poughkeepsie, and they were having it renovated. They did not consider the possibility of exposure to lead. Workers were sanding walls and torching door frames—a very hazardous practice, because it creates a lead fume. The first clue that something was wrong was the death of the family dog. The veterinarian obtained a postmortem blood lead level in the dog. It was elevated. The children were then tested. They too were elevated, and they required intravenous chelation therapy. Really, quite a disaster.

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