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onstrate a positive effect following the removal of toxins. The rule is to remove hazardous material if it exists. Furthermore, if there is an imminent threat to humanity, the hazardous material must be removed within a year. All of this is done whether or not there is any evidence of exposure or whether there is any consistently strong evidence of human health effects. For example, there is currently a \$45 million Superfund project in Holbrook, Massachusetts, to remove contaminants that have no comparable immediate impact on human health. Yet the Boston City Health Department could not get adequate funds to remove lead—a proven contaminant—from the most contaminated residential areas in Boston. In fact, \$45 million would cleanup *all* of the highest risk areas of Boston. And to aggravate matters, the Boston pilot project is being held up ostensibly as a result of the failure of the EPA and the Commonwealth of Massachusetts to agree on the method of evaluating the pilot project and their fail-

ure to provide the necessary level of funding to implement it.

Racial discrimination affects the life chances of blacks—especially the more critical aspects of health and safety. It would seem that the manner in which lead poisoning is being handled in Boston provides an example of the real meaning of racism. Young black children are being poisoned by lead every day in known lead-contaminated areas, yet no major cleanup efforts are underway!

(For more on lead poisoning, see the article below.)

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Lead Poisoning: A Health Epidemic in the Black Community

by
Wornie L. Reed

Lead poisoning in humans has been identified as a cause of high blood pressure, heart disease, birth defects, complications in pregnancies and developmental problems in infants. It is a health problem of epidemic dimensions in the black community. This serious health problem is yet another example of the production of “illth” in the modern society. As the means of production create wealth for some sectors of society, they also create illth. As Lamont C. Cole wrote in 1970:¹

At the present time refuse produced in this country is estimated to be increasing about four percent per year; . . . about the same as the yearly increase in the Gross National Product.

It is apparent that lead in the environment can be considered as undesirable refuse. Just as the health and wealth of society accrue to some groups more than others, so does the illth. The black community—as usual—gets a disproportionate share of the latter. And undoubtedly, the fact that this health

hazard is centered in the black community is the reason more is not done to eliminate and prevent it. Society shows little concern for those who are the most likely victims of lead poisoning—small black children from poor and minority families living in old housing in dilapidated inner city areas. In affluent and middle-class suburbs only 3% of white children have dangerous levels of lead in their blood, compared to 30% of inner city black children.

Background^a

As a result of industrialization, lead is ubiquitous in the human environment. Having no known physiologic value, lead can only produce harm. Children are particularly susceptible to its toxic effect. Excessive absorption of lead is one of the more prevalent and preventable childhood health problems in the United States today.

Since 1970 medical opinion regarding lead tolerance has changed substantially. Before the mid-1960s a level below 60 micrograms of lead per deciliter (ug/dl) of whole blood was not considered dangerous enough to require intervention.² By 1975, as a result of more experience with this phenomenon, the level at which intervention is suggested declined 50%—to 30 ug/dl.³ In that year the Center (now Centers) for Disease Control (CDC) published the study, *Increased Lead Absorption and Lead Poisoning in Young Children: A Statement by the Center for Disease Control*. Since then new evidence has indicated that lead is toxic at levels previously thought to be nontoxic. Now the elevated blood level at which intervention is recommended is 25 ug/dl or greater.

Furthermore, it is now generally recognized that lead toxicity is a widespread problem—one that is neither unique to inner city children nor limited to one area of the country.

Progress has been made. Average blood levels for the U.S. population have been established by the Second National Health and Nutrition Examination Survey (NHANES II), and lead-contaminated soil and dust have emerged as important contributors to blood lead levels, as has leaded gasoline through its contribution to soil and dust lead levels. An increasing body of data supports the view that lead, even at levels previously thought to be “safe,” is toxic to the developing central nervous system, and screening programs have revealed the extent of lead poisoning in target populations.

Obviously, a major public health objective would be the *prevention* of lead poisoning. A major advance in primary prevention has been the reduction of lead in gasoline. It is probably responsible for the findings of reduced average blood lead levels in children nationwide⁴ and in two major cities.⁵ In addition, lead is no longer allowed in paint for residential dwellings, furniture and toys.

The primary sources of lead are air, water and food. Despite the 1977 ruling by the Consumer Product Safety Commission, which limits the lead content of newly applied residential paints, millions of housing units still contain previously applied leaded paints. Older houses that are dilapidated or that are being renovated are a particular danger to children. In many urban areas lead is found in soil⁶ and house dust.⁷ Consequently, screening programs—a form of secondary prevention—are still needed to minimize the chance of lead poisoning developing among susceptible young children.

A nationwide survey, conducted from 1976 to 1980, showed that children from all geographic areas and socioeconomic groups are at risk of lead poisoning.⁸ Data from that survey indicate that nearly 3.9% of all U.S. children under the age of five years had blood lead levels of 30 ug/dl or more. Extrapolating this figure, an estimated 675,000 children six months to five years of age had blood lead levels of 30 ug/dl or more. There are, however, race and class differences in lead poisoning. Two percent of white children had elevated blood lead levels; 12.2% of black children had elevated levels. The levels for some black children are even higher: among black children living in the cores of large cities and in families with annual incomes of less than \$6,000, the prevalence of levels of 30 ug/dl or more was 18.6%. Among white children, those in lower as opposed to higher income families had eight times the prevalence of elevated lead levels.

In the past decade, knowledge of lead toxicity and its effects increased substantially. Previously, medical attention focused on the effects of severe exposure to lead and clinically recognizable signs and

symptoms of toxicity. It is now apparent that lower levels of exposure may cause serious behavioral and biochemical changes. Results of a growing number of studies indicate that chronic exposure to low levels of lead is associated with altered neurophysiological performance; the young child is particularly vulnerable to this effect.⁹

Many factors can affect the absorption, distribution and toxicity of lead, factors that tend to put children more at risk than adults. Children are more exposed to lead than older persons because their normal hand-to-mouth activities introduce many nonfood items into their bodies.¹⁰ Once absorbed, lead is distributed throughout soft tissue and bone. Young children absorb and retain more lead on a unit-mass basis than adults. Their bodies also handle lead differently: higher mineral turnover in bone means that more lead is available to sensitive systems in children. Since lead accumulates in the body and is only removed slowly, repeated exposures to small amounts over many months produce elevated blood lead levels. In fact, this is the most probable means of acquiring lead poisoning from soil and dust.

Children are particularly susceptible to [lead's] toxic effect. Excessive absorption of lead is one of the most prevalent and preventable childhood health problems in the United States.

Lead toxicity is mainly evident in the red blood cells, the central and peripheral nervous systems and the kidneys. Lead also has adverse effects on reproduction in both males and females,¹¹ and recent data¹² suggest that prenatal exposure to low levels of lead may be related to minor congenital abnormalities. In fact, the margin of safety for lead is very small compared with other chemical agents.¹³

The effects of lead toxicity are nonspecific and not readily identifiable. Any number of behavioral and biochemical changes may result. Parents, teachers and clinicians may identify altered behaviors in children that result from lead toxicity as attention disorders, learning disabilities or emotional disturbances. Because of the large number of children susceptible to lead poisoning, these adverse effects are a major cause for concern.

Some of the symptoms of lead toxicity are fatigue, pallor, malaise, loss of appetite, irritability, sleep disturbance, sudden behavioral change and developmental regression. More serious symptoms include clumsiness, muscular irregularities (ataxia), weakness, abdominal pain, persistent vomiting, constipation and changes in consciousness due to early encephalopathy (disease of the brain). Children who

display these symptoms need thorough diagnostic evaluations, and, should the disease be confirmed, they need prompt treatment.

Lead Poisoning as a Child Health Problem^b

Lead poisoning among children has changed over the past decade. Previously, it was a disease often presented as encephalopathy associated with children ingesting peeling old lead paint. Now lead poisoning has become a largely “asymptomatic” condition, characterized by an elevated blood lead level linked with many sources of exposure and affecting a broader range of children.

The most severe effects of lead (acute encephalopathy, seizures, coma and death) occur at blood lead levels 80 to 100 ug/dl and over. However, even moderately elevated blood lead levels (as low as 25 ug/dl) have effects on central nervous system functions. These less obvious effects occur in such central nervous system functions as intelligence, behavior control, fine motor coordination, neurological dysfunction and motor impairment. Further, metabolic effects occur in children with blood lead concentration as low as 10 to 15 ug/dl. Recent studies strongly suggest that, at even subclinical levels of lead intoxication, children sustain permanent cognitive and behavioral damage that manifests itself in poor school performance and a variety of learning disabilities.

Subclinical lead intoxication is especially troublesome because it is asymptomatic. Parents find it difficult to understand lead hazards when their children do not appear to be “sick”; long-term exposure is cumulative over time so that toxicity occurs without the parents’ recognition. Probably the most significant implications of lead’s distribution in the body are its high degree of accumulation with repeated exposure and its slow rate of removal after exposure occurs.

Sources Of Lead Exposure

Children may be exposed to lead from a wide variety of sources—tap water, canned food, air and paint. All children in the United States are exposed to lead in the air, in soil and dust and even in the normal diet. While lead may come from such sources as water from piping and water distribution systems and from lead leaching from the seams of soldered cans, probably the most critical sources are lead-based paint, airborne lead and soil and dust.

Lead-Based Paint

Direct ingestion of lead paint—the most concentrated source of lead—is most often the cause of high risk symptomatic or asymptomatic lead poisoning. Lead-based paint is the major source of high-dose lead exposure and symptomatic lead poisoning for children in the United States. The interiors

of about 27 million households in this country are contaminated by lead paint that was produced before the amount of lead in residential paint was controlled. Since 1977, household paint must contain no more than 0.06% lead. However, before 1977, some interior paints contained in excess of 50% lead. And a further complicating factor is that lead-based paint is still available for industrial, military and marine usage. Occasionally, this paint is used in homes.

Quite often lead poisoning occurs in children under six years of age who live in deteriorated housing built before World War II. Children in this age group often mouth and/or swallow peeling paint chips. This practice of pica, the ingestion of nonfood substances, is normal behavior for young children. It is not race or class based. However, poor families and black families are the principal occupants of such housing. Therefore, children in these families have more adverse health effects from normal childhood behavior. In recent years this kind of lead poisoning has been reported among “urban homesteaders” who are moving back into the cities and rehabilitating old houses.

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Airborne Lead

Inhalation of airborne lead is also a means of poisoning children. Although inhalation is a minor means, airborne lead that gets deposited in soil and dust is a major source of lead poisoning. This airborne lead is produced by automotive and industrial sources. Studies have shown that children living within 100 feet of major roadways have higher blood levels than those living farther away.¹⁴ Low income families and black families tend more than others to live near such major roadways.

Soil and Dust

Soil and dust that contain lead are also extremely important sources of lead exposure for children. Lead in soil and dust comes from particles of airborne lead produced by automotive, industrial and similar sources. Flaking lead paint also plays a part in contaminating the soil around homes. The potential health effects of lead in dust and dirt are increased by their absorptive properties and by their ubiquitous presence in children’s environments. In particular, hand-to-mouth transfer of lead-contaminated dust and dirt enters a child’s system through normal play.¹⁵ This activity often produces subclinical chronic lead intoxication, which constitutes over 90% of all childhood lead poisoning cases.

Prevention

Current childhood lead poisoning prevention programs have a case finding and treatment focus. These programs were established in the late 1960s and early 1970s, when the accepted threshold levels for toxic effects of lead were much higher than today. It was believed that if lead levels did not reach these thresholds, the children would not suffer any

Recent studies strongly suggest that, at even subclinical levels of lead intoxication, children sustain permanent cognitive and behavioral damage that manifests itself in poor school performance and a variety of learning disabilities.

serious or permanent health effects. At that time the emphasis was on screening children for lead poisoning, providing medical treatment and removing the sources of lead (i.e., lead paint). This approach was based on the assumption that with early detection and intervention lead encephalopathy and the resultant brain damage would be prevented. The early detection and intervention would keep children's lead levels from becoming too high. This approach was successful insofar as many of the more severe consequences of lead poisoning—death and mental retardation—were reduced. On the other hand, the lower levels of lead continued to cause serious, but asymptomatic, health effects. Now it is known that screening and medical and environmental treatment—important secondary prevention methods—do not provide primary prevention.

All children diagnosed as having lead poisoning require continual medical treatment, environmental assessment and educational monitoring. Obviously, lead-poisoned children must be treated. It should be noted that medical treatment, which is essential, is painful for the child and distressing for the family. But the failure to treat a child subjects that child to permanent damage. Environmental evaluation and deleading are necessary prior to returning an already poisoned child to the home environment. The child should also be carefully monitored and evaluated by the educational system, as the effects of lead poisoning on the central nervous system may cause such problems as attention disorders, learning disabilities and emotional disturbances.

Substantial preventable costs result from continual neglect of primary prevention. Treating already poisoned children is a costly way of dealing with the problem. Primary prevention of lead poisoning would protect children *before* they are poisoned;

however, the problem continues to be dealt with only after children are lead poisoned.

The Race Effect

Boston provides an example of how blacks are affected disproportionately by lead poisoning. Lead poisoning, while occurring throughout this city, is to a surprising degree concentrated within very limited geographic areas. Four neighborhoods—Dorchester, Roxbury, Jamaica Plain and Mattapan—rate highest in the number and percentage of children poisoned. These neighborhoods account for 87% of the City's lead-poisoned children and only 56% of the at-risk population (nine months to six years of age). Further, 16 of the census tracts in these neighborhoods—containing less than 18% of the City's at-risk children—account for 41% of Boston's lead-poisoned children. These neighborhoods contain a major portion of the black population of Boston. Although blacks make up only 20% of the population of the City of Boston, they are 78% of Roxbury, 81% of Mattapan and over 20% of Dorchester.

Conclusion

The Boston Childhood Lead Poisoning Prevention Program oversees the abatement of lead paint hazards and monitors the blood lead levels of young children throughout the City. The deleading of poisoned children's homes appears to contribute significantly to the reduction of their blood lead levels. However, even six to 12 months after deleading, about 50% of the children tested still had lead levels over 30 ug/dl (59% of those originally with levels over 50 ug/dl and 46% of those initially under 50 ug/dl).

Clearly then, for many children, lead-based paint is not the only significant source of lead. Because lead-contaminated soil has been found to be a major contributor to elevated lead levels in children, it is quite likely that for many of the children whose lead levels do not steadily decline to safe levels, soil is a significant source of lead exposure. The pattern of sustained toxicity almost certainly undermines cognitive and central nervous system development in these children. Consequently, there is a pressing need to remove lead contaminated soil and to do so in a preventive manner.

As stated by Ronald Jones, the Director of the Office of Environmental Affairs:

As long as we rely solely on education, screening and treatment programs, and reject active preventive measures, even in highly lead-contaminated environments that guarantee high rates of lead poisoning, we embrace a policy that stamps this violence to our children as acceptable public policy.

The Special Commission on Lead Poisoning Prevention for the Commonwealth of Massachusetts has made a number of recommendations for improving the prevention of lead poisoning. Perhaps the two most important are:

1. The addition of inspections and, where appropriate, removal of leaded soil, and
2. The initiation of a program of primary prevention in geographic areas with extremely high rates of lead poisoning.

To accomplish these objectives, Jones's office identified 28 "hot spots" in Boston and worked with others in a long fight to get the EPA Superfund money to remove lead-contaminated soil from the City's hot spots. Obviously, a lot hinges on this path-breaking demonstration project. There is every reason to believe that a correctly executed cleanup of this toxic substance will serve to reduce the lead poisoning of children in these neighborhoods.

NOTES

^aThis section is taken substantially from the Center for Disease Control. (1985, January). *Preventing Lead Poisoning in Young Children*. Atlanta: U.S. Department of Health, Education and Welfare.

^bThis section is taken substantially from Office of Environmental Affairs. (1985). *Boston Child Lead Poisoning: Request for Immediate Cleanup of Lead-Contaminated Soil in Emergency Areas*. Boston: Department of Health and Hospitals.

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