Conferences and Reviews

Lead Poisoning

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Lead poisoning is the most common disease of environmental origin in the United States today. Adult lead poisoning results primarily from exposure by inhalation in the workplace. Pediatric lead poisoning results principally from the ingestion of lead from environmental media, including paint chips, dust, soil, drinking water, ceramics, and medications. Lead is toxic to many organ systems, among them developing erythrocytes, the kidneys, and the nervous system. Lead-induced toxicity to the central nervous system causes delayed development, diminished intelligence, and altered behavior. In young children, this effect has been demonstrated convincingly to occur at blood lead levels between 10 and 20 μg per dl. The Centers for Disease Control and Prevention has recommended that a blood lead level of 10 μg per dl or higher be considered evidence of increased lead absorption, and the National Academy of Sciences has concurred in that recommendation. Unresolved issues in need of further study include the frequency of screening young children for lead, the question of whether women should be offered screening for lead before conceiving a pregnancy, the role of x-ray fluorescence analysis in assessing lead in bone, and the appropriate legislative response of the United States government to lead-based paint abatement.

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Lead poisoning is the most common disease of toxic environmental origin in the United States today. It affects both children and adults. Lead is now known to be toxic to persons of all ages at levels that only a few years ago were considered to be safe. This realization has forced major revisions of strategies for the prevention and management of lead poisoning.[1,2]

In this article, we review recently developed information on the epidemiology and toxicology of lead exposure, particularly at low doses. In addition, we review recent research and current controversies in diagnosing and preventing lead poisoning.

Epidemiology and Sources of Exposure

Adult Exposure to Lead

Most adult exposure to lead occurs in the workplace. The usual route of occupational exposure is by inhalation. The potential for occupational exposure exists in hundreds of industries, and the National Institute for Occupational Safety and Health estimates that more than 3 million workers in the United States are at risk.[3] Industries classically associated with lead exposure include smelting, battery making, ship burning, soldering, stained glass manufacture, brass foundry work, and lead-based paint abatement. More recently construction and demolition work have become major sources of occupational lead exposure. Lead exposures in construction are intense, and although exposure to lead in most industrial sectors has been effectively controlled by standards promulgated by the US Occupational Safety and Health Administration (OSHA), construction work had until 1993 been exempted from those protections. Particularly severe exposures have occurred in construction workers involved in the demolition and renovation of painted steel structures such as bridges and elevated highways.[4,5]

Nonoccupational exposure to lead is also widespread in the adult population of the United States, but levels are generally lower than those in the workplace. Persons of all ages encounter lead in air, dust, soil, and drinking water.

Pediatric Exposure to Lead

Because of their normal oral exploratory behavior, children absorb most of their lead by ingestion.[6] The mean blood lead level in children in the United States today is about 5 μg per dl. This level is substantially higher than that found in preindustrial populations living in remote areas, but is substantially lower than that of American children in the mid-1970s, reflecting enormous decreases in the use of lead in gasoline.[7] An estimated 3 to 4 million American preschool children have blood lead levels above 10 μg per dl, a level now recognized to be associated with subclinical neurologic impairment.[6] Among poor, minority, inner-city preschool children, the prevalence of blood lead levels above 10 μg per dl is estimated by the Centers for Disease Control and Prevention to be as high as 68%.[8]

Lead-based paint. Lead-based paint continues to be

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the principal source of high-dose lead exposure for children in the United States. An estimated 57 million housing units in the United States contain lead-based paint, and in 3.8 million homes children are intimately exposed to deteriorating lead paint.[8]

Although lead-paint poisoning classically occurs among poor children living in deteriorated housing in inner cities, the disease does not respect geography or social station. Lead-paint poisoning has been reported in the children of affluent parents who have moved into cities as "urban homesteaders."[9] It has also been reported among children living in lead-painted houses in suburban and rural areas.[10] Household renovations are associated with particularly severe lead exposures.[9,10]

Children may absorb lead from paint directly by ingesting paint chips (pica), or they may absorb it indirectly by inadvertent ingestion of lead-contaminated house dust. The latter route is the more common.

Contaminated dust and soil. Contaminated dust and soil are pervasive sources of lead exposure for children.[8,11] Concentrations of lead in dust and soil range from near zero to many thousands of parts per million. In urban and suburban areas, the principal sources of lead in dust and soil are flaking paint from buildings and bridges, automotive emissions (much less now than a decade ago), and industrial releases. In general, lead in dust and soil appears to be responsible for elevations in children's blood lead levels when the lead concentration exceeds 300 to 500 ppm.[12,13]

Airborne lead. Airborne lead is generally a low-dose source of exposure for children.[8] Automotive and industrial emissions are the major contributors to lead in air. Automotive emissions have diminished substantially since the phasedown of lead in gasoline,[7] and industrial lead emissions have been reduced in most areas since the passage of the Clean Air Act.

Although industrial emissions account for only a small fraction of airborne lead releases to the air, these stationary sources can produce concentrated zones of extremely high-dose exposure. The worst such situation in the United States existed in the vicinity of a large lead ore smelter in Kellogg, Idaho. In 1974, 98% of 1- to 9-year-old children living within a mile of this smelter had blood lead levels greater than 40 μg per dl, and 22% had levels above 80 μg per dl.[14]

Drinking water. Drinking water is a common source of lead exposure.[8] Although lead is seldom present in drinking water in high concentrations, it contributes widely to background exposure across a wide segment of the American population. At its source drinking water is almost always lead-free but can become contaminated as it passes through lead pipes or comes into contact with lead solder. Aggressive soft water of low pH poses the greatest hazards because it has the highest potential of leaching lead from pipes and solder.

Leadworkers' children. Children of leadworkers may be exposed to lead dust transported home from the workplace.[15] In an evaluation of lead exposure among workers' children, a close correlation was found between children's blood lead levels, the severity and duration of parental exposure, and the lead concentration of household dust. Of 91 workers' children, 38 (42%) had elevated blood lead levels and 10 required treatment for lead poisoning.[15] Under regulations set forth in 1978 by OSHA, industries using lead are now required to provide changing and showering facilities for their workers.[16] Although this measure has greatly reduced the incidence of take-home lead poisoning, such poisoning is still seen occasionally among the children of construction workers, reflecting the exemption until August 1993 of construction work from OSHA protections.

Home remedies. Home remedies can occasionally be a source of lead poisoning in both adults and children. Numerous case reports have documented this hazard,[17,18] and it appears to be especially common among ethnically isolated groups.

Body Burden of Lead

The lead burden, the total amount of lead in the human body, represents the difference between the cumulative lifetime absorption of lead from all sources and total excretion.[19] Lead is not distributed homogeneously in the human body. Experimental studies have shown instead that it is dispersed among several physiologically distinct compartments that differ from one another in their size and accessibility. These compartments and their interrelations can be portrayed by metabolic models that describe both qualitatively and quantitatively the absorption, distribution, deposition, accumulation, and excretion of lead.

A reasonable portrayal of the functional anatomy of the body lead burden is provided by the model of Rabinowitz and co-workers.[20] This model is based on stable isotope and metabolic balance studies. It proposes that the lead burden be described in terms of three dynamically interrelated compartments.

Lead in Blood

Lead in blood makes up about 1% of the body lead burden in the Rabinowitz model. Because it is conveniently accessible and is the fraction of the body burden that correlates most closely with recent environmental exposures, it is the component of the body burden most frequently measured. Approximately 90% of the lead in blood is found in the erythrocytes. The half-life of lead in blood is 36 ± 5 days.

Lead may be added to the blood compartment by inhalation, ingestion, or movement from the deeper compartments. The influence of lead from the deeper compartments on the blood lead level is proportional to the amount of lead in those stores,[21] although there exists considerable interindividual variability in the release of lead from bony stores. If the amount of stored lead is large, its effects on the blood lead level can be considerable; conversely, if skeletal lead stores are small, then lead recently inhaled and ingested becomes the dominant factor determining the blood lead level. Thus, in the early phases of exposure to lead, the blood lead level rises rapidly and provides a sensitive indicator of recent ab-

sorption.[14,20,21] By contrast, in persons with long-term heavy exposures to lead, such as workers with many years' employment in the lead industries, the blood lead level has been found to correlate poorly with current exposure; under those conditions, the blood lead level appears principally to reflect the release of stored lead.[21]

Soft-Tissue Lead

Compartment 2 in the Rabinowitz model is composed principally of soft tissue lead.[20] Lead in soft tissues such as the kidneys, bone marrow, and nervous system is responsible for most of lead's toxicity.

Skeletal Lead

Lead in the skeleton is by far the largest component of the body burden.[20] This compartment contains about 99% of all lead in the human body. Skeletal lead is calculated to have a half-life of approximately 10,000 days.[20] Lead in the skeleton appears to exist in two physiologically distinct compartments. Lead in trabecular bone plus lead in the subperiosteal and subendosteal regions appears to constitute a relatively mobile fraction of the bone lead, whereas lead in deep cortical bone is relatively immobile.

Lead Poisoning

Lead is now well recognized to produce a wide range of toxicity. These toxic effects extend from acute, clinically obvious, symptomatic poisoning to subclinical effects.[1]

Acute Toxicity

Intense, acute exposure to lead, either by inhalation in the workplace or by the ingestion of paint chips among children, can cause acute symptomatic poisoning. Characteristics of this life-threatening syndrome are abdominal colic, constipation, fatigue, anemia, peripheral neuropathy, and in most cases, alteration of central nervous system function.[22] In severe cases, a full-blown acute encephalopathy with coma, convulsions, and papilledema may occur. In milder cases, only headache or personality changes may be evident.[22] In many instances, persons who have suffered from acute lead encephalopathy are left with permanent neurologic and behavioral sequelae.[23]

'Subclinical' Toxicity

Subclinical toxicity denotes the concept that relatively low-dose exposure to lead can cause harmful effects that are not evident on a standard clinical examination.[24] The underlying premise is that there exists a continuum of toxicity in which clinically apparent effects have their asymptomatic counterparts; these subclinical counterparts have recently been called "biologic markers" of toxicity.[25] Thus, clinically obvious manifestations of lead poisoning such as anemia, neuropathy, and renal failure lie at the upper end of the range, whereas such covert effects as impaired biosynthesis of heme, slowed nerve conduction, and altered excretion of uric acid are their subclinical correlates. It is important to note that these subclinical changes represent truly harmful outcomes and are not merely homeostatic or physiologic adjustments to the presence of lead.

The toxic effects of lead are evident principally in three organ systems: the erythrocytes and their precursors, the central and peripheral nervous system, and the kidneys. Lead has also been shown to have adverse effects on reproduction in both men and women.[26] It is a potent carcinogen in three animal species.[27]

Hematologic Toxicity

Anemia is the classic clinical manifestation of lead toxicity in the erythrocytes. The severity and prevalence of lead-induced anemia are correlated directly with the blood lead level. The anemia induced by lead may be either normochromic or hypochromic and may be associated with an increased reticulocyte count. It is caused primarily by an impairment of heme biosynthesis, but an increased rate of erythrocyte destruction may also occur. At blood lead levels as low as 10 μg per dl, lead begins to inhibit the cytoplasmic enzyme δ-aminolevulinic acid dehydratase in the heme biosynthetic pathway; the enzyme's inhibition is virtually complete at lead levels of 70 to 90 μg per dl.[28] Also at blood lead levels of 15 μg per dl in children and of 25 to 30 μg per dl in adults, lead begins to inhibit the mitochondrial enzyme ferrochelatase, which is responsible for catalyzing the transfer of iron from ferritin to protoporphyrin to form heme.[29,30] Ferrochelatase inhibition causes the metabolic intermediate erythrocyte protoporphyrin to accumulate to excess in erythrocytes. Erythrocyte protoporphyrin elevation is thus a measure of both lead absorption and toxicity.

Neurologic Toxicity

In the peripheral nervous system, the motor axons are the principal target of lead. Lead-induced pathologic changes in these fibers include segmental demyelination and axonal degeneration.[31] Extensor muscle palsy with wrist and ankle drop has since the time of Hippocrates been recognized as the classic clinical sign of this toxicity.

Recent studies of the peripheral nerves in persons with lead exposure have used electrophysiologic probes to determine whether lead causes covert abnormalities in function. In the first of these studies, evidence of asymptomatic slowing of motor nerve conduction velocity was found in workers whose blood lead levels had never exceeded 70 μg per dl.[32] Most recently, in a prospective study of new entrants to the lead industry, slowing of ulnar nerve conduction velocity was noted at blood lead levels as low as 30 to 40 μg per dl.[33]

In the central nervous system, extensive research has sought to determine whether lead causes asymptomatic impairment in function at doses insufficient to produce clinical encephalopathy. The best-designed studies have been carried out in children. In an early investigation, it was found that clinically asymptomatic children with elevated body lead burdens (as defined by dentine lead levels) had a 4.5-point deficit in mean verbal IQ scores compared with children with lower lead burdens.[34] This finding was still strongly evident after correcting for a

wide range of socioeconomic, behavioral, and biologic factors. Similar results were reported in other early studies.[35-37]

This early work was corroborated by a long-term follow-up of the children in the original study group to age 18.[38] At that time the children who had had higher lead burdens in early life were found to have a higher frequency of reading difficulty, of failure to graduate from high school, and of criminal behavior.[38]

Most recently, evidence for the neuropsychologic toxicity of lead at low doses has been provided by a series of prospective studies of newborn children.[39-41] In each of these investigations, correlations have been sought between intellectual performance in young children and umbilical cord blood lead levels at birth. All of these studies have found subclinical but apparently irreversible decrements in central neurologic function. This dysfunction is characterized by diminished intelligence, shortened attention span, and slowed reaction time. These findings are highly credible. They have been accepted by the Centers for Disease Control and Prevention as the basis for revamping the national strategy for preventing lead poisoning in the United States,[6] and they have been accepted also by the National Academy of Sciences in their recent authoritative review of childhood lead poisoning.[42]

An unexplored implication of the finding that lead causes insidious asymptomatic injury to the central nervous system is that some as-yet-unknown fraction of cases of presenile dementia, motoneuron disease, or other chronic neurologic and psychiatric conditions may possibly be caused by long-term exposure to lead.[43] Such long-term exposure may result in an accelerated attrition of neurons that becomes clinically evident with age. Epidemiologic and clinical studies to assess previous lead exposure in persons with chronic neurologic disease such as Parkinson's disease, motoneuron disease, and dementia will be needed to assess this possibility.

Renal Toxicity

Chronic nephropathy, which may progress to kidney failure, is the classic renal manifestation of lead toxicity. It appears to result from long-term, relatively high-dose exposure to lead.[44] The cells lining the proximal tubules appear to be the tissue in the kidneys most highly sensitive to lead.[29] In these cells, at blood lead levels of 40 to 80 μg per dl, lead induces the formation of dense intranuclear inclusion bodies consisting of a lead-protein complex.[29] Hyperuremic gout, apparently resulting from increased reabsorption of uric acid by the tubular cells, is a metabolic correlate of lead-induced renal impairment.[29]

The evolution of lead nephropathy is usually silent. The central event appears to be the progressive destruction of tubular cells and their replacement by fibrosis.[45] Clinical manifestations of impairment, consisting of elevations in blood urea nitrogen or serum creatinine levels, do not ordinarily become evident until 50% to 75% of the nephrons have been destroyed. The later stage of lead nephropathy is characterized pathologically by interstitial fibrosis with atrophy and dilation of the tubules and relative sparing of the glomeruli; in this stage, intranuclear inclusions are infrequent.[29]

An excess mortality from renal disease has been observed in epidemiologic studies of lead workers.[46-49] In each of these investigations, a twofold to threefold increase has been noted in the number of deaths from chronic nephritis. In one study, an association was observed between the duration of employment in a lead smelter and death from nephritis.[49]

Lead and Hypertension

Long-term, high-dose exposure to lead was reported early in this century to be associated with an increased incidence of hypertension and cerebrovascular accident.[50] With the reduction in lead exposure that has occurred in most industries, these overt associations are now noted less commonly. Several recent epidemiologic studies, however, have found evidence that lead absorption, even at relatively low levels, is associated with a substantial elevation in blood pressure.[51] Toxicologic studies have also documented an association between increased lead absorption and hypertension.[52] These effects appear to be mediated both through the toxic effects of lead on the kidneys and by direct action on vascular smooth muscle.

Reproductive Toxicity of Lead

A body of experimental evidence indicates that lead at high doses is toxic to reproductive function in both male and female laboratory animals.[26] Also, clinical reports, most of them from the first half of this century, described reproductive toxicity in workers of both sexes with high-dose exposure to lead; the incidence of spontaneous abortion was reported to be increased in female lead workers and in the wives of male lead workers.[53]

In male workers with heavy exposure to lead (mean blood lead level, 74.5 μg per dl) and also in men with moderately increased lead absorption (mean blood lead level, 52.8 μg per dl), decreased sperm counts and an increased prevalence of morphologically abnormal sperm have been reported.[54] Two more recent studies also noted sperm count depression at relatively high blood lead levels (>60 μg per dl).[55,56] Studies are needed to assess the possible toxicity of lead to male reproduction at lower levels of exposure and using state-of-the-art biologic markers of sperm function.

Recent Research Developments

A major research need in lead poisoning is a method to assess long-term lead exposure. A newly applied technology that holds promise for the accurate, noninvasive assessment of long-term lead exposure is the measurement of the bone lead concentration by x-ray fluorescence analysis. This technique offers a relatively rapid approach to the individual assessment of body lead burdens.[57,58] It takes advantage of the fact that the half-life of lead in bone is approximately 10,000 days (25 years).[18] Moreover, the radiation dose is minimal, amounting to only about 0.2% of the radiation delivered by dental x-ray studies.

X-ray fluorescence analysis may be undertaken using either K or L x-rays, and instruments of both types have been developed.[59,60] The K instrument has several intrinsic advantages over the L instrument.[61] First, the K instrument samples lead across the entire transverse section of bone, in contrast to the L instrument, which obtains 90% of its signal from only the most superficial 2 mm. If there is variation in the concentration of lead across the bone, the L measurement will fall victim to this variation, but the K will not. In addition, the K instrument does not require a measurement of the thickness of the overlying skin, whereas the L instrument is sensitive to any error in the required measurement of skin thickness. Third, the K instrument is relatively insensitive to movement of the subject during the sampling period, whereas the L instrument is very sensitive to movement; given that the typical sampling time is about 15 minutes, this consideration is not trivial. Finally, the L x-ray fluorescence instrument has not been well validated. In defense of the L instrument, there may be detailed modeling studies in which it will be desirable to examine lead content simultaneously in several bone compartments. In such circumstances, the combined use of the K and L instruments might offer uniquely valuable information on the kinetics of lead.

The future use of x-ray fluorescence analysis in workers and children exposed long term to lead will refine current knowledge of the relationships between cumulative lead exposure and its toxic effects on the nervous system, kidneys, cardiovascular system, and reproductive organs.[62]

Current Issues and Controversies

How Frequently Should Young Children Be Screened for Lead?

The Centers for Disease Control and Prevention and the American Academy of Pediatrics have recommended that all preschool children in the United States be screened for lead.[6] We concur in this recommendation. Although screening for blood lead in children is somewhat costly, the price per test is coming down. Moreover, the analysis can be done on a carefully obtained fingerstick blood specimen; if state-of-the-art instruments are available and a child's finger is scrupulously cleaned, venipuncture is not required. The public health benefits of the early detection of lead intoxication in children are so great that the performance of at least a single blood lead measurement on all American children is well justified.

If only a single blood lead determination is to be done on a child, the most logical time to do it is between the ages of 1 and 2 years. Most studies of the distribution of blood lead levels have shown that the highest levels are seen in 1-, 2-, and 3-year-olds. Moreover, detecting an elevated blood lead level at an early age increases the opportunities for intervention.

If in the judgment of a pediatric practitioner a child is at high risk of lead poisoning, then the child will need to be screened more frequently. The Centers for Disease Control and Prevention has developed a series of indicators of high risk (Figure 1).[6] If a child is found through a brief history to have one or more of these risk factors, testing ought to proceed more frequently, with the exact frequency being determined by the pediatric practitioner, possibly in consultation with the state or local health department. When repeated screening is targeted to those children at highest risk, the direct effects of lead testing programs on other public health programs such as childhood immunization will be minimized.

Prepregnancy Screening for Lead

It has become common practice in obstetrics in the United States to screen women for rubella and hepatitis B antibody status before the conception of a planned pregnancy. It is not standard at the present time to measure the blood or bone lead level in a woman who intends to become pregnant. This is an issue that appears worthy of investigation.

The underlying hypothesis is that lead in the bone may become mobilized during pregnancy and could then cross the placenta to reach the fetus. Now that relatively inexpensive diagnostic methods exist for determining blood and bone lead levels and given that bone lead instruments will likely become widely available in the years ahead, research should be undertaken to assess whether prepregnancy bone lead screening is worthwhile in women who may have had a past exposure to lead. Hypothetically, if the bone lead concentration is found to be elevated, chelation therapy could be prescribed before the pregnancy is conceived to reduce the body lead burden. Chelation therapy is clearly contraindicated during pregnancy because the possibility exists that the chelating agent may enhance the movement of lead across the placenta.

This issue will require further research and assessment

Does your child

- live in or regularly visit a house built before 1960 with peeling or chipping paint? This could include a day-care center, preschool, or the home of a babysitter or a relative.
- live in or regularly visit a house built before 1960 with recent, ongoing, or planned renovation or remodeling?
- have a brother or sister, housemate, or playmate being observed or treated for lead poisoning—that is, blood lead level ≥15 μg per dl?
- live with an adult whose job or hobby involves exposure to lead?
- live near an active lead smelter, battery recycling plant, or other industry likely to release lead?

Figure 1.—The questions in this figure were developed by the Centers for Disease Control and Prevention[6] to assess the risk of high-dose exposure to lead.

in the years ahead. No recommendation except further study is justified at present.

Should Workplace Protection Be More Stringent?

Data developed over the past 10 to 15 years clearly indicate that lead causes toxicity among workers at levels of exposure below the current permissible exposure limit established in 1978 by OSHA.[1,2,16] This exposure limit allows blood lead levels in American workers to be as high as 50 μg per dl. Hypertension and neurologic impairment are documented to occur at blood lead levels below 50 μg per dl, and it is possible that long-term exposure at this level may result in renal impairment.[63]

A most difficult problem in occupational lead exposure is raised by the recently reported finding that lead causes neurologic damage to fetuses at blood lead levels as low as 15 to 20 μg per dl—levels substantially below current workplace exposure standards. This finding has been noted in three separate prospective studies and appears highly credible.[39-41] Lead passes virtually unimpeded across the placenta, and the neurologic impairment that it produces in fetuses appears to be irreversible.

The central problem here is a profound mismatch between current occupational standards for lead and human biology.

We strongly recommend on the grounds of preventing systemic toxicity in workers and toxicity to fetuses that the permissible blood lead level in workers be lowered to 10 μg per dl.[2]

Should X-ray Fluorescence Assessment of Lead in Bone Be Used Routinely in Leadworkers?

At present the only medical screening of leadworkers that is required is periodic determination of the blood lead level. Industrial managers have become skilled at maintaining workers' blood lead levels below permissible exposure limits. This goal has been achieved by a combination of reducing workplace exposures plus rotating workers out of heavily contaminated jobs if and when their blood lead levels begin to approach the permissible exposure limit. The problem with this approach is that although blood lead levels today seldom exceed the biologic limit value, workers continue to be at risk of depositing substantial amounts of lead in bone. Thus, the bone lead level can rise steadily even while the blood lead level is kept within legal limits. This situation may increase the risk of long-term toxicity.

In the future, as x-ray fluorescence instruments become more widely available, it may be necessary to consider incorporating the determination of lead in bone by this method into the standard medical monitoring of leadworkers. Annual or biennial determinations of bone lead content in any leadworker would supplement the periodic determination of blood lead levels. If such a requirement were to be developed, it would need to be linked to the provisions for medical removal protection and rate retention that currently are included in the OSHA occupational lead standard.[14]

How Should the Federal Government Deal With the Problem of Lead Abatement?

Because millions of housing units in this country are contaminated with lead paint, it is clear that the prevention of lead-paint poisoning in current and future generations of American children will require massive abatement of this material. A major need is the development of a large cadre of workers who have been properly trained and certified in lead abatement. Such a cadre exists already for the abatement of asbestos. These workers have been trained under the provisions of the Asbestos Hazard Emergency Response Act. Similar legislation and similar training requirements will be necessary for leadworkers.

An unresolved issue is the source of funding for lead abatement. The cost will be enormous. It is not fair to require that landlords pay these costs in their entirety, since in many cases they were unaware of the lead hazard when the buildings were purchased. Tenants also should not be expected to pay the cost. Governmental bodies will inevitably be required to pick up a portion of the costs and perhaps also to provide tax write-offs for renovations. An innovative proposal has been made to impose a stiff excise tax on new lead as a means of financing lead-paint abatement.[64] The attractiveness of this proposal is that it will penalize the corporations that profited in the past from sales of lead-based paint at a time when the hazards of this paint were already widely known. This proposal deserves serious consideration.

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