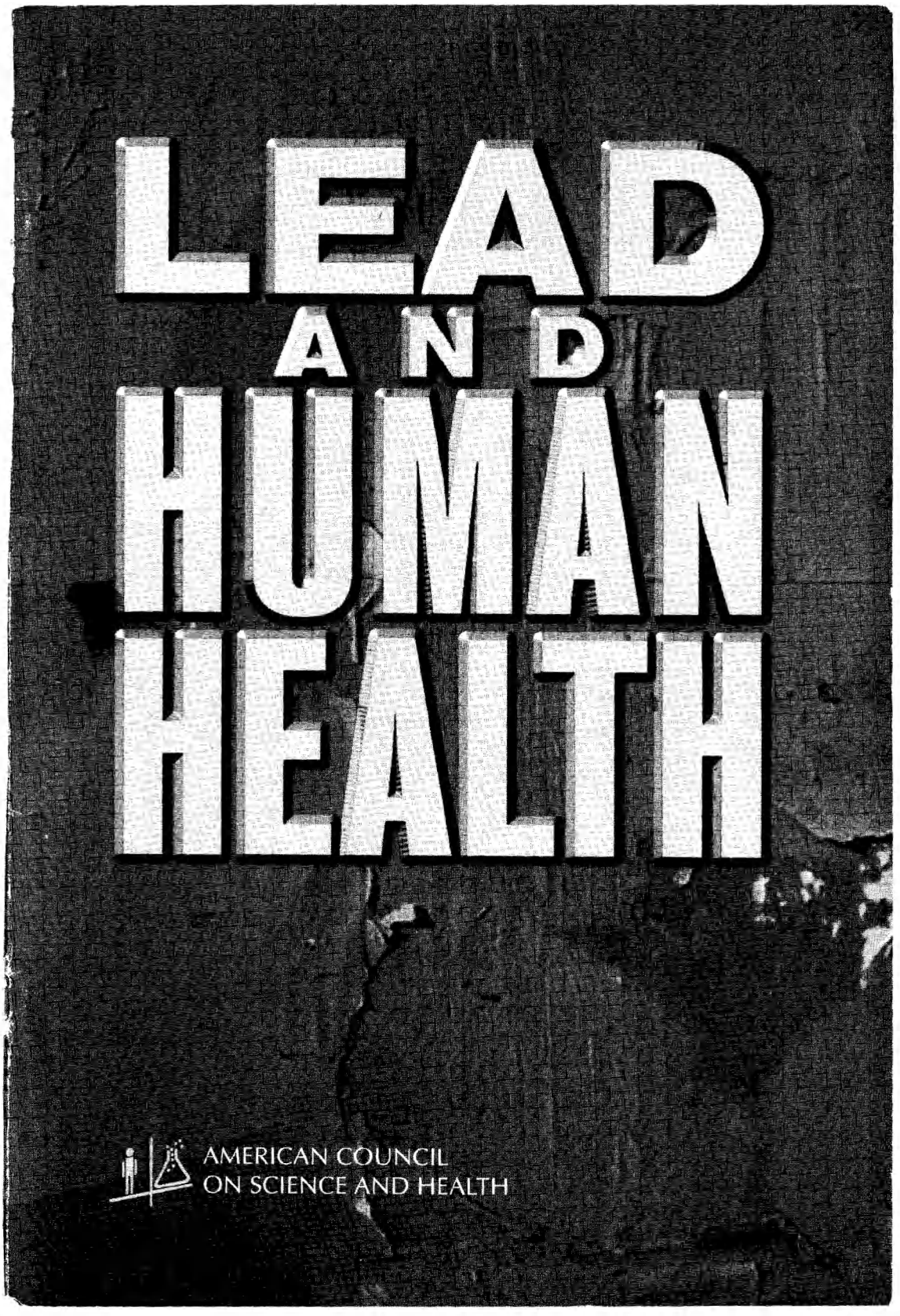
LEAD
AND
HUMAN
HEALTH
AMERICAN COUNCIL
ON SCIENCE AND HEALTH

Lead and Human Health

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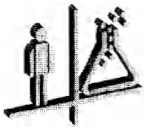

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Executive Summary

Concern over lead poisoning dates back many centuries. Today, the potential effects of exposure to lead continue to receive as much attention as any modern public health risk; lead is often cited as America's leading environmental health concern.

Based on a review of the current scientific literature, ACSH concludes:

- that lead is an important toxicant that can exert adverse effects, given sufficient exposure and accumulation in the body;

- that there exist many federal regulatory standards and mandates that serve to minimize or eliminate the amount of lead in consumer products, occupational settings, and the environment;

- that elimination of lead-based gasoline and the reduction of lead in consumer products (e.g., paints); the home (e.g., plumbing systems); and food-packaging applications (e.g., soldered cans) have, in conjunction with other federal and state programs, served to significantly reduce blood lead levels in the U.S. population over the past decade;

- that symptomatic childhood lead "poisoning," seen often until the 1970s, has essentially disappeared (such lead poisoning no longer constitutes a widespread public health threat in the U.S.; problems in localized areas continue to exist, however);

- that trace amounts of lead contained in calcium supplements and other FDA-approved nutritional aids and beneficial foods are not toxicologically significant and do not pose a health risk to humans;

- that children are more sensitive than adults to the effects of lead and that precautions should be taken to limit childhood exposure;

- that targeted, rather than universal, screening is indicated in order to identify children and other susceptible individuals with elevated blood lead levels; and

- that elimination or minimization of exposure to lead can be successfully achieved through adjustments to personal habits, public education, and improvements in living conditions, particularly among certain population groups.

1. Introduction

Lead can have severe and lasting effects on human health, given sufficient exposure. The general public remains confused about the relative health risks associated with lead, however. This is due, in part, to the conflicting and often biased information disseminated by government agencies, industries, and activist groups, among others. Additionally, the public often receives opposing—and skewed—views from the news media: reports that fail to distinguish real environmental hazards from minor, perceived human health risks.

The intent of this review is to convey what is known about the toxicity—the hazard—of lead and to stress that "hazard" alone does not equal risk. Hazard weighed in combination with exposure and absorption are appropriate measures for the evaluation of human health risk. It has been well documented that if sufficient exposure occurs, lead may cause serious toxicity in humans. The actual threat of lead exposure to human health remains remarkably controversial, however, given the sensitivity of today's technology and our ability to quantify contaminants in the environment. Accordingly, this review will also put into perspective how low levels of lead affect our everyday lives and whether we should be concerned about such exposures.

Finally, this report provides recommendations from the American Council on Science and Health (ACSH) concerning the need to identify those individuals and populations at highest risk of exposure to lead. Billions of dollars are spent annually in the United States on lead-related regulations, abatement projects, and surveillance programs. In an age of shrinking public financial resources we must use our dollars wisely to insure that public health protection efforts focus on those risks that are of highest concern.

2. Lead in the Environment

Lead is one of the most ubiquitous of the heavy metals. Lead has been detected in virtually all areas of the environment (air, water, and soil) and in biological systems. Lead in the environment occurs both naturally and as a consequence of human activities.

Concentrations of lead in the environment are quite variable.[1] In surface water, lead is likely to form insoluble complexes with other substances in the water. In soil and sediment, lead binds with other particles, thereby reducing its bioavailability (the amount of lead that can be absorbed by the body) to organisms living in those environments. Plants may contain small amounts of lead as a result of atmospheric deposition or root absorption from soil. Lead is not as pervasive in the environment as it once was, due principally to the commercial introduction of lead-free gasoline and the reduced use of lead in manufacturing processes and consumer products.

3. Human Exposure

3.1. Routes of Exposure

The scientific and medical consensus is that a child's primary route of lead exposure is through ingestion of lead-based paint and, to a lesser extent, through lead-contaminated soil and the ingestion and inhalation of lead-containing dust. For some adults the most significant route of exposure is the inhalation of lead-containing dusts and fumes in occupational settings, particularly during mining, smelting, and refining operations or during battery manufacturing and reclamation operations.[2] Exposure to lead may also occur through eating or smoking in a lead-contaminated environment.[1,3]

Today, lead-based paint remains the most common source of lead exposure for young children. White lead paint containing up to 50 percent lead was in widespread use in the United States through the 1940s. In later years, up to the 1970s, binder paints containing approximately 5 percent lead were more common.

In 1978 the Consumer Product Safety Commission banned the manufacturing of paint containing more than 0.06 percent lead by weight for use on interior and exterior residential surfaces, toys, and furniture.[4] It has been estimated, however, that 83 percent of privately owned housing units and 86 percent of public housing units in the U.S. built before 1980 still contain some lead-based paint.[4]

Homes in close proximity to either lead smelters or industries involved in the manufacture of lead products may contain elevated concentrations of lead in

their surrounding soil, thus providing a potential source of exposure to children. The bioavailability of lead, once in the body, is quite limited, however.[5] Drinking water can also serve as a source of lead exposure to humans, because of leaching from lead-containing pipes and fixtures; but most researchers agree that lead-containing water only rarely results in cases of human toxicity.[6] Dermal (skin) exposure is much less important as a significant pathway of lead exposure than is either ingestion or inhalation.

The ingestion of food represents an additional route of lead exposure. Lead occurs in and on food, both naturally and as a result of human activity. Lead may also be introduced into food inadvertently during harvesting, transportation, processing, packaging, or preparation. Sources of this lead in food include dust; metals used in grinding, crushing, or sieving; solder used in packaging; and water used in cooking. Between 1973 and 1978 the food industry made intensive efforts to remove sources of lead from infant food items. Much of the reduction was achieved by the discontinuation of the soldered cans formerly used in infant formula packaging. Since then, can manufacturers have stopped producing soldered cans for the food industry.

Daily dietary intake of lead has decreased since the 1940s, when some estimates ranged as high as 400–500 μg (micrograms) of lead per day per individual for the U.S. population. Present levels are under 20 μg per day.[7] The United Nations Environment Program has estimated that the global average daily intake of lead is 80 μg/d from food and 40 μg/d from drinking water.[8] This continuing decline in dietary intake of lead over the years has contributed to the general decline in blood lead levels.[9]

Because lead, like most chemicals and substances, is toxic only at some defined dose, the critical issue is the determination of the specific body burden level at which lead begins to exert adverse health effects on humans. The mere detection of lead in bone, blood, or other organ systems is not a sufficient indicator of toxicity. For the vast majority of humans, chronic low-level lead exposure from environmental sources does not cause overt toxicity or lead "poisoning." Lead toxicity is observed primarily in cases of occupational overexposure and in some children with high environmental lead exposures.

3.2 Current Blood Lead Levels

Blood lead level (BLL), reported as micrograms of lead per deciliter of blood (μg/dL), is considered the principal biomarker (an indicator of exposure) for lead exposure. The test to determine blood lead is widely used, reasonably easy to perform, of low cost, and more reproducible and sensitive than other indirect measures of lead exposure.

Screening of children in major U.S. cities in the early 1960s revealed that

20 percent to 45 percent of children evaluated had elevated BLLs on the order of 40 μg/dL.[10] In the 1970s federal regulatory and legislative efforts were undertaken to reduce lead exposure. These efforts included actions to limit the use of lead in paint, gasoline, and soldered cans.[5]

The second National Health and Nutrition Examination Survey (NHANES II, 1976 to 1980) conducted by the Centers for Disease Control and Prevention (CDC) established the first set of baseline BLLs for the U.S. population and demonstrated the pervasiveness of lead exposure across race, urban and rural residence, and income level.[11] Data from the NHANES II survey also showed a decline in BLLs that was closely correlated to declines in the use of leaded gasoline during those years.[12]

A subsequent comparison of the NHANES II survey (in which 9,832 persons were evaluated), with data from the third National Health and Nutrition Examination Survey (NHANES III, phase I, 1988 to 1991; 12,119 persons evaluated) and the Hispanic Health and Nutrition Examination Survey (HHANES, 1982 to 1984; 5,682 persons evaluated) found that the mean BLL of persons aged 1 to 74 had dropped 78 percent, from 12.8 to 2.8 μg/dL, during the interval between the surveys.[12]

Importantly, the mean BLLs of children aged 1 to 5 years declined 77 percent (from 13.7 to 3.2 μg/dL) for non-Hispanic white children and 72 percent (from 20.2 to 5.6 μg/dL) for non-Hispanic black children. The prevalence of BLLs of 10 μg/dL or greater for children aged 1 to 5 years declined from 85.0 percent to 5.5 percent for non-Hispanic white children and from 97.7 percent to 20.6 percent for non-Hispanic black children. It is important to note that phase I of NHANES III (1988–1991) reported that national estimates of children 1 to 5 years of age indicated that 8.9 percent, or approximately 1.7 million children, may still have had BLLs of 10 μg/dL or greater.[13]

The Third National Health and Nutrition Examination Survey (NHANES III) specifically addressed BLLs in the U.S. and their correlation with sociodemographic factors. Notably, BLLs were consistently higher for younger children than for older children, for older adults than for younger adults, for males than for females, for blacks than for whites, and for central-city residents than for non–central-city residents. Other correlates of higher BLLs included low income, low educational achievement, and residence in the Northeastern region of the United States.[14]

Most recently, phase II of NHANES III, as reported by the CDC, continues to indicate declining BLLs in the U.S. population.[9] This recent update has confirmed that BLLs among children aged 1 to 5 years were more likely to be elevated among those who were poor, non-Hispanic black, and living in large metropolitan areas or in older housing.

During the 1991–1994 survey period, the mean BLL of the U.S. population older than 1 year was 2.3 μg/dL, down from the 2.8 μg/dL reported for the time period 1988–1991. It is encouraging that the average BLL of the most susceptible individuals—children—continues to decline, with the most recent data reporting that among those aged 1 to 5 years, approximately 4.4 percent (890,000) had BLLs in excess of 10 μg/dL, down from 8.9 percent (1.7 million) of those surveyed during the 1988-1991 survey period. For children with blood lead levels greater than 20 μg/dL (levels at which children may be at greater risk), prevalence has declined from 24.7 percent in 1976–1980, to 1.1 percent in 1988–1991, to 0.4 percent in 1991–1994 (see Table 1, below).[9] Despite these encouraging findings, however, the most recent data indicate that BLLs continue to vary considerably by age, sex, race/ethnicity, urban status, income, and other sociodemographic factors.

Collectively, the NHANES survey results demonstrate a significant decline in BLLs for the U.S. population and for selected subgroups of the population. The NHANES data provide convincing evidence that BLLs for the vast majority of those assessed are below the level considered by the CDC to be the bench-

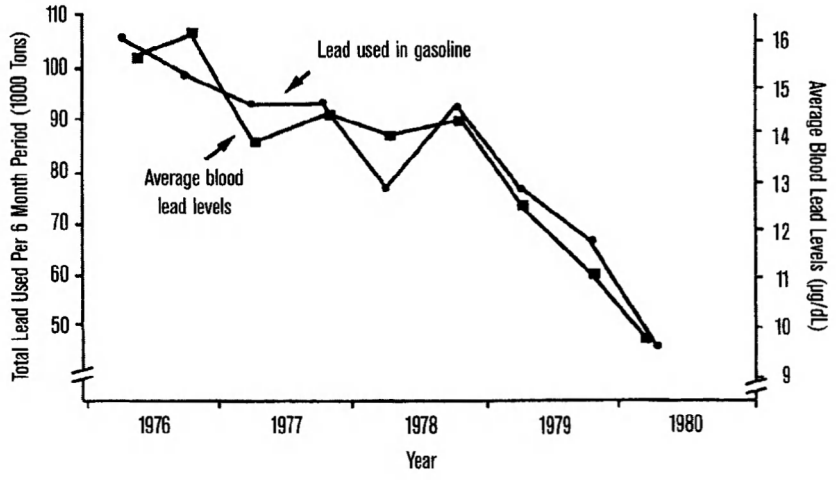
Table 1. Decline in Blood Lead Levels (BLL) of Children Aged 1–5 Years from 1976 to 1994 (National Health and Nutrition Examination Survey–NHANES)

	Mean BLL (μg/dL)	Prevalence BLL ≥ 10 μg/dL	Prevalence BLL ≥ 20 μg/dL
NHANES II 1976–1980	15	88%	24.7%
NHANES III Phase 1 1988–1991	3.6	8.9%	1.1%
NHANES III Phase 2 1991–1994	2.7	4.4%	0.4%
Amount of Decline	X5.6	X20	X62

mark for intervention (10 μg/dL). The major causes of the observed decline are likely attributable to the removal of more than 99 percent of the lead from gasoline (see Figure 1, below) and the removal of lead from paint, soldered cans, and plumbing systems. But while the NHANES data demonstrate a major success in reducing human lead exposure, they also indicate that certain sociodemographic factors (e.g., young age, race/ethnicity, and low income level) continue to be associated with higher BLLs.

Because of the overall decline in BLLs among the U.S. population, the CDC's 1997 lead-screening guidelines call for targeted screening of at-risk children. This recommendation differs from the previous (1991) recommendation of virtually universal screening of children aged 12 to 72 months. The CDC noted recently that "[m]any children, especially those living in older housing or who are poor, need screening and, if necessary, appropriate interventions to lower their BLLs. At the same time, children living where risk for lead exposure has been demonstrated to be extremely low do not all need to be screened."[15]

Figure 1. Change in Blood Lead Levels in Relation to a Decline in Use of Leaded Gasoline, 1976-1980



Source: Annest, J.L., et al. (1983). Chronological trend in blood lead levels between 1976 and 1980. *New Engl. J. Med.* 308, 1373-1377.
Reprinted in: CDC. (1991). *Preventing Lead Poisoning in Young Children.* U.S. Department of Health and Human Services.

Specifically, the CDC's current recommendation is for statewide targeted screening based on an assessment of local data and an inclusive planning process, including the formation of an advisory committee. In the absence of a statewide plan, universal screening as called for in the 1991 guidelines is advised.

4. The Toxicology of Lead

4.1. Absorption and Distribution

Humans may begin to accumulate lead in their bodies either during prenatal development (from placental transfer resulting from maternal exposure) or following birth as a result of trace-level exposure from a variety of sources. Adults absorb 5 to 15 percent of ingested lead and generally retain less than 5 percent of what is absorbed.[16] Young children absorb substantially more ingested lead (approximately 30 to 40 percent) than do adults because of physiological and metabolic differences.[16]

Once lead is in the blood, it is distributed primarily among three compartments: the blood; the soft tissues (kidney, liver, and brain); and the mineral tissues (bones and teeth). The fractional distribution of lead in bone (as contrasted with other body stores) increases with age from about 70 percent of body lead in childhood to as much as 95 percent with advancing age. Lead that is not absorbed by the body is excreted, primarily through the feces.

While some information—derived largely from laboratory animal studies done under controlled conditions—is available concerning the absorption of lead following direct inhalation, oral ingestion, and dermal exposure, there is less information regarding the absorption of lead by humans exposed to environmental lead. This is particularly true of information relating to the lead that children may ingest from lead-contaminated soil.

In assessing potential exposures of children to lead in soil, the Environmental Protection Agency (EPA) has generally relied on a computer model to predict BLLs, assuming that 30 percent of ingested lead will be absorbed. This may be an approximate estimate for certain soils, but studies of soil ingestion in children in several mining communities have revealed BLLs much lower than those predicted by the EPA model.[17] This suggests that the EPA's theoretical estimates of lead absorption may not be reflective of absorption under real-life conditions.

Recently, researchers have confirmed that lead absorption is highly dependent on the form of lead ingested and on the matrix (in soil, in dust, or as elemental lead) in which it is consumed.[18] Thus, models that evaluate the uptake and absorption of lead should incorporate such data, particularly if those models are to be used by regulatory agencies to predict human exposure and health risk.

With respect to the potential health effects of lead in soil, the EPA undertook a study of three cities (Baltimore, Boston, and Cincinnati) to determine the effect of soil abatement (decreasing the amount of lead in soil) on children and to quantify the relationships between the lead content of soil or dust and blood lead.[19] The researchers found that soil lead abatement, by itself, has minimal impact on blood lead status. They concluded that the lead in soil is not very bioavailable.

Overall nutritional status and eating behavior appears to influence the absorption and toxicity potential of lead in several ways. Lead taken in from water and other beverages tends to be absorbed to a greater degree than lead taken in from food. Lead ingested under fasting conditions is absorbed to a greater extent than lead ingested during food consumption.[20,21]

Lead interacts with and competes with several essential elements, principally calcium, iron, and zinc; dietary deficiencies of both calcium and iron are known to enhance the absorption of lead.[16] As indicated above, lead released from bone in humans is important relative to BLLs, particularly since various physiological and pathological conditions (e.g., osteoporosis, chronic disease, pregnancy, and lactation) may cause mobilization of lead stored in bone into the bloodstream.[16,22]

The CDC has appropriately recommended proper nutrition among children, particularly the insurance of an adequate daily supply of iron and calcium, as a measure to reduce lead absorption.[23]

4.2 Health Effects of Lead

The toxic effects of lead involve several organ systems within the body and vary from subtle biochemical effects (biomarkers of exposure without evidence of adverse effects) to clinical or overt effects such as lead poisoning (plumbism).[24] Subtle biochemical changes may occur at lower levels of exposure, but the significance of these changes is often not known. Such biochemical effects are not at present known to be adverse, however (unlike anemia, which may occur at a BLL of 80 μg/dL), but are rather indicators (biomarkers) of exposure. For example, BLLs approaching 10 μg/dL are associated with altered blood enzyme levels or changes in heme synthesis intermediates (components of red blood cells); but these changes may simply be indicators of, or adaptation to, exposure, and not toxic effects.

Neurotoxicity and Chronic Kidney Toxicity

Neurotoxicity and chronic kidney toxicity are the chief concerns for adults with excess occupational exposure to lead. Nerve conduction is reversibly slowed in peripheral nerves at BLLs of approximately 30 μg/dL. Overt effects on the ner-

vous system, such as wrist drop (weakness of the wrist and finger extensors caused by the compression of a nerve), require BLLs of 60 µg/dL or greater.[16] The adverse effects of acute lead poisoning on the kidney (i.e., functional and morphological changes in the proximal renal tubular lining cells) have been well documented.[25] These changes may progress to diffuse nephropathy (generalized kidney disease), which is characterized by reductions and enlargements of the tubular structures due to disrupted function. It has been suggested that chronic and excessive lead exposure may result in end-stage renal disease.[26]

It is important to note, however, that chronic kidney effects require relatively high and prolonged exposure to lead.[7] Researchers have found that BLLs in the range of 40–80 µg/dL are associated with biological changes in the kidney that are largely reversible.[27] The results of occupational studies indicate that maintaining BLLs below 60 µg/dL will prevent biologically relevant renal changes in the majority of lead-exposed workers. Subtle alterations in kidney biomarkers have been observed below this level, although there is no evidence of renal dysfunction at these BLLs.

Bone Function and Vitamin D Metabolism

Bone is a major organ for lead deposition, and skeletal lead has been used as a measure of cumulative lead exposure.[28] Lead has also been suggested to affect bone function by altering growth and stature, and by perturbing vitamin D metabolism. Researchers have reported associations between blood lead and decreasing levels of vitamin D metabolites over BLLs ranging in concentrations from 12 to 120 µg/dL.[29,30] No threshold for this effect has been conclusively demonstrated. It has been hypothesized that lead at low exposure levels may alter vitamin D metabolism, with possible adverse effects on bone growth in children. Other studies, however, have reported that there is no effect on vitamin D metabolism, calcium and phosphorous homeostasis, or bone mineral content in children whose nutritional status is adequate and who experience low to moderate lead exposure.[31]

Reproductive Health

The effect of toxic levels of lead exposure on reproductive health has been recognized for some time. Severe lead intoxication is associated with sterility, abortion, stillbirth, and neonatal morbidity and mortality from exposure in utero.[32,33,34] The evidence for low-level exposure, however, is less suggestive. Current research has focused on the prenatal effects of lead at low exposure levels. A review of the literature indicates that prenatal lead exposure can alter the developing brain of some experimental animals, but it has proved difficult to demonstrate similar effects on neurodevelopment in infants and children.[35] There has been limited evidence that high prenatal exposures (resulting in

maternal BLLs in excess of 15 μg/dL) are associated with reduced birth weight[36] or increased risk of preterm delivery.[37,38] Most data on these outcomes are either contradictory or demonstrate no effect. Spontaneous abortions are not apparent at maternal BLLs less than 30 μg/dL and the weight of evidence suggests that lead does not cause congenital anomalies.[35]

Male Reproductive Function

Despite the large number of studies conducted on male reproductive function in workers occupationally exposed to lead, definitive statements on the effects are difficult to make. The available data indicate that extremely high lead exposure can have a marked adverse impact upon semen quality. Aberrant sperm morphology, decreased sperm count, and decreased sperm density have all been demonstrated in heavily exposed individuals.[39,40,41] It is not possible, however, to define precisely the levels of exposure at which these effects will occur. General population and occupational BLLs less than approximately 50 μg/dL appear to have little, if any, impact on sperm parameters. Despite some evidence in earlier studies,[42] current data suggest that the effects on semen quality are most likely when the BLLs are consistently elevated to 50–60 μg/dL or higher.[43] While the precise mechanism by which lead impairs spermatogenesis is not known, most studies assessing male exposure to lead have observed no impact on male fertility.[39,44,45]

Cancer

Cancer resulting from lead exposure is not considered a toxicological endpoint of concern. The carcinogenicity of lead has been of less interest due to a general consensus that human epidemiological data is, at best, inconclusive and that other health effects from overexposure are well defined.

Experimental animal studies in rats and mice have investigated the carcinogenic potential of several lead compounds following long-term administration of very high doses.[46] In most studies, the kidney of the male rat has been most susceptible to the induction of cancer. Prevailing hypotheses for mechanisms of lead carcinogenicity have thus tended to focus upon the susceptibility of the male rat kidney to nephropathy.[47]

Cancer studies of lead-exposed humans have found little relationship between occupational exposure and the subsequent occurrence of cancer. Some studies have been inconclusive because of concurrent exposure to confounding substances (e.g., chromium and arsenic) and inadequate control for smoking.[47,48,49,50,51,52] While a possible relationship with kidney cancer has been suggested by some investigators,[50] other studies have failed to confirm this relationship or have even reported a decrease in the rate of kidney cancer.[48] The col-

lective weight of evidence suggests that there is no association between cancer of any type and low-level lead exposure in humans.

Blood Pressure

During the past two decades the relationship between lead and blood pressure has been examined in the general population as well as in occupational and experimental studies. Several recent reviews have generally concluded that there is only a weak association between lead and elevated blood pressure in studies with BLLs above 45 μg/dL.[53,54,55,56] This association has been inconsistent across studies because of potential confounders and the inability to establish a clear dose-response relation (the observance that a change in the amount of exposure is directly related to a change in response).

In order to evaluate the association between low-level lead exposure and blood pressure, one study randomly sampled a population in Belgium from 1985 to 1989 and reexamined the group from 1991 to 1995.[57] Numerous confounding variables were controlled for. The researchers found that the BLLs studied (in excess of 30 μg/dL) were not consistently associated with increased blood pressure or with increased risk of hypertension in the general population.

Although plausible mechanisms have been suggested for lead-related blood pressure effects on the basis of animal studies, it is difficult to determine whether extrapolation of animal data to humans is appropriate. Given these limitations, it is unlikely that weak positive epidemiological associations give rise to measurable excesses of cardiovascular morbidity and mortality in the general population. The International Programme on Chemical Safety (IPCS) has concluded that "despite intensive efforts to define the relationship between body burden of lead and blood pressure or other effects on the cardiovascular system, no causal relationship has been demonstrated in humans and the mechanisms remain obscure."[56]

4.3. Effects on Child Development

Summary of Studies

The issue that has received the most attention and discussion is the potential effect of lead overexposure on the nervous system of children.[46,58,59] More specifically, studies have associated lead overexposure with decreased intelligence; reduced short-term memory; reading disabilities; and deficits in vocabulary, fine motor skills, reaction time, and hand-eye coordination. During the last two decades, there have also been a number of epidemiological studies relating BLLs at the time of birth, during infancy, and through early childhood with measures of psychomotor, cognitive, and behavioral outcomes. A 1994 report in the *British Medical Journal* reviewed the epidemiological evidence concerning environmental lead and the effects on children's intelligence. The researchers sought

to quantify the magnitude of the relationship between full scale IQ in children aged 5 years or older and their body burden of lead.[60] To assess this potential relationship, 26 epidemiological studies published since 1979 were reviewed.

The researchers concluded that while low-level lead exposure may cause a small IQ deficit, other explanations or influencing factors need to be considered, including: (a) Are the published studies representative? (b) Is there sufficient control for confounders? (c) Are there selection biases in recruiting and following? and (d) Do children of lower IQ adopt behavior which makes them more prone to lead exposure and uptake (reverse causality)? Because of these uncertainties, experts have recommended that public health priority and attention be devoted to reducing moderate increases in children's BLLs while reviewing other social and biological detriments that impede intellect and influence behavior in children.[61]

Additional Considerations Concerning Lead and IQ

Associations between blood lead and effects on IQ have been reported for moderate to high BLLs, typically in excess of 30 to 40 μg/dL.[61] It has been more difficult to conclude, however, that low-level blood lead is associated with adverse effects on intelligence and neurobehavioral development. Given the complex nature of intelligence, it is important to consider and evaluate known factors that influence IQ before establishing a causal role for lead in effecting deficits in IQ performance.

Reverse Causality: It has been suggested that lead exposure in children is associated with lower IQs, attention-deficit hyperactivity disorder (ADHD), and other negative behaviors such as impulsiveness, hyperactivity, or poor attention span. It is also plausible, however, that lower IQ or behavioral disorders contribute to elevated BLLs. Children who demonstrate hyperactive, impulsive, inattentive behavior are more likely to ignore warnings to avoid eating lead-contaminated dirt or paint chips (reverse causality).

In support of this hypothesis, two researchers recently reported that children with pervasive developmental disorders (PDD) have a tendency to exhibit pica behavior (an appetite for unfit foods or nonfood substances), which may place them at a greater risk for lead exposure than children without such a disability.[62] While the reverse causality hypothesis cannot be studied prospectively, most studies that have related BLLs to IQ have used statistical procedures that do not permit drawing definitive conclusions about causality.[63,64,65]

Confounder Considerations: Despite the attempt by investigators to control for all confounding variables within a study, there are many additional variables—among them socioeconomic status, childhood disease, parenting skills, styles of child rearing, parental time spent with the child, and skills and styles of key caretakers other than parents—that remain uncontrolled in most lead–IQ studies. For

example, otitis media (ear infections), in young children may be a relevant variable that is often overlooked. Infants or toddlers prone to repeated ear infections are likely to have verbal IQs that are lowered by several points when they reach school age.[66] Few studies, however, have controlled for this variable. One must consider a child's health history when evaluating whether an environmental factor such as lead contributes to a lower verbal IQ. Additionally, given the known relationship between maternal substance abuse or inadequate nutrition during pregnancy and a child's cognitive ability and behavior, it is unfortunate that relatively few lead–IQ studies have accounted for these factors.[67]

Parental IQ, a heritable trait that is believed to be an important influencing factor in the development of a child, has not been adequately considered in studies relating BLLs to IQ. Paternal IQ is almost always discounted or ignored. The failure to control for key confounding variables such as parental IQ, a child's medical history, or quality of prenatal care presents a challenge to the interpretation of lead–IQ studies. It is more appropriate to conclude that any effect on IQ performance observed in lead–IQ studies should be attributed to lead and all other potential confounders, known or unknown, that were not identified or controlled for in a study.

Nutrition is particularly important in neurobehavioral development and represents another confounding variable that must be carefully assessed in studies of lead and IQ. In 1955 it was first reported that urban women of lower socioeconomic status who were given vitamin and mineral supplements during pregnancy gave birth to children who performed better on IQ tests than did their peers.[67] More recently, research has concluded that poor nutrition may adversely affect the behavior and intelligence of children.[68,69] In total, studies by two research groups, one in the UK and one in the U.S., have found a positive correlation between diet supplementation and nonverbal IQ scores.[69,70,71] The increases in IQ observed in these studies were substantial.

Two separate research groups have demonstrated the particular importance of iron in influencing cognitive development and performance.[72,73] One of the groups has stated that "the most important systemic abnormality produced by iron deficiency in infancy is the alteration in cognitive performance."[72] Importantly, these same symptoms of altered cognitive performance are observed in lead-affected children who may also be iron deficient. The other investigative group followed children who as infants had had moderately severe iron-deficient anemia.[73] The researchers reported that the motor and mental functioning performance of the affected children remained below that of children who were not iron deficient.

Although early upbringing, social interactions, and education are often cited as likely causes of higher IQ, nutrition—a critical factor influencing cogni-

tive performance—is seldom considered as an important variable. The results of these studies are further intriguing because nutritional deficiencies thought to impact intellectual development would also be expected to affect the mechanism of lead absorption and increase the efficiency of lead uptake from the gastrointestinal tract (stomach and intestines). Thus, the negative impact of improper nutrition on IQ might contribute to a modest rise in blood lead, resulting in a statistical correlation in which the negative impact on IQ might be erroneously attributable to lead. This may be an important research area for the future; studies in this area may help us to understand some of the inconsistencies and anomalies of the current epidemiological research involving lead.

There are numerous statistical, toxicological, and methodological questions that must be evaluated before the relationship between low BLLs and children's IQ can be better defined. Intelligence is a complex concept influenced by so many variables that, at present, it is difficult to determine what effect low-level lead exposure exerts on human intelligence. Further research in animals and improved sensitive and specific methods that examine numerous aspects (i.e., neuromotor, cognitive, and electrophysiological aspects) of brain function are needed to better determine threshold effects of lead.

5. Lead in Consumer Products: Reason for Worry?

Humans are exposed daily to trace amounts of lead, along with other trace-level contaminants, through ingestion of food and water. From time to time a public health "scare" may arise because of a newly identified source of lead exposure. These scares often involve widely used consumer products found to contain trace amounts of lead.

In order to determine whether a true health risk exists, however, each "scare" must be evaluated on a scientific basis. In recent years the detection of lead in miniblinds, in men's hair dyes, and in calcium supplements has, each in its turn, become a topic of concern. These "scares" undoubtedly have left consumers confused and questioning the potential health risks they run from using these products.

Our ability to detect ever-smaller amounts of trace elements or contaminants in our environment has spawned the belief that any detectable concentration of a chemical in the environment constitutes a health risk. Clearly, lead can be toxic to humans; but we must not forget that the level of exposure and absorbed dose are critical determinants in the potential occurrence of adverse effects.

5.1. Lead in Miniblinds

Windows—long a focus of household safety concern—became the object of renewed attention in June of 1996 when the Consumer Product Safety

Commission (CPSC) announced that certain imported vinyl miniblinds could present a "lead poisoning" hazard to children.[74] Following two months of testing, the CPSC reported that inexpensive, nonglossy vinyl miniblinds from China, Taiwan, Mexico, and Indonesia would eventually deteriorate, forming lead dust.

Public awareness was heightened when state health officials in Arizona and North Carolina identified vinyl blinds as the cause of "lead poisoning" among children living in mobile homes where no lead paint was present. But no information was provided about factors influencing exposure, including time spent outdoors by the children, sources of drinking water, and the degree of contact the children had with window blinds.[75]

In response to the CPSC report, a trade association group, the Window Covering Safety Council, agreed to reformulate the imported miniblinds by removing the lead intentionally added to some vinyl to enhance color, prevent deterioration, and make the slats rigid. (Vinyl miniblinds manufactured in the U.S. have not used lead as a stabilizer for the past 20 years.)

Lead in miniblinds appears to represent a low health hazard, as the exposure potential is negligible under foreseeable conditions of use. Unless lead is released through excessive wear or deterioration, there is no exposure pathway (i.e., ingestion, inhalation) that would result in significant lead exposure to humans.

Notably, there are physical safety hazards associated with windows and window treatments in general—entanglement with cords, cuts from glass, falls from open windows—that probably serve to limit the time children spend near windows, thus minimizing children's exposure potential.

If deterioration of those miniblinds that contain lead resulted in the generation of dust, potential hand-to-mouth activity of children playing with the blinds could result in ingestion of lead-containing dust. But no estimates of the amount of lead contained in miniblinds nor estimates of potential human exposure have been provided to support the contention that miniblinds represent a known health hazard.

5.2. Lead in Hair Dyes

Hair dyes that contain small amounts of lead have raised concern among some consumers and interested parties. The fear is that children will ingest lead by putting their hands in their mouths after touching their parent's hair or contacting household surfaces (e.g., sinks, countertops) containing hair-dye residues. One research group analyzed the lead content of several hair dyes containing lead acetate and suggested that "significant" exposure potential (on the order of hundreds of micrograms) exists as a result of hand-to-mouth activity by children.[76] One controversy of the study, however, is that the authors based some of their risk assessment on a reported "Total Tolerable Daily Intake" (TTDI) for children

of 6 micrograms of lead per day (6 µg/d). The derivation of this TTDI (from a study of calcium supplements) is questionable, as there currently are no federally established acceptable daily intake levels for lead. In 1977 the World Health Organization recommended that children limit their exposure to lead to less than 428 µg/d, a level provisionally deemed acceptable for adults.[77]

Other investigators have conducted a safety assessment of lead acetate as a component of hair dyes and have concluded that "the tiny contribution of lead acetate exposure from hair-coloring use can be regarded unequivocally as being toxicologically insignificant."[78] This research involved an extensive review of the lead acetate toxicology literature, although the researchers' focus was primarily on the risk to the dye user rather than on potential risk to children inadvertently exposed. Nevertheless, lead in hair dye represents a minor exposure pathway for children as a group, particularly in comparison with the potentially greater exposures received from peeling paint, dust, and soil.

The U.S. Food and Drug Administration (FDA) concluded that lead acetate was safe for use in hair dyes and approved its use subject to a maximum content of 0.6 percent lead in the product.[79,80] The FDA is not known to have received any reports of children with elevated BLLs in any way attributable to lead from hair dyes. Thus, as in the case of miniblinds, lead in hair dyes appears to pose no appreciable health risk, either to children or to adults.

5.3. Lead in Calcium Supplements

The issue of lead in over-the-counter calcium supplements has recently became a topic of debate and, predictably, has caused concern among the general public. Over the years, while the FDA has analyzed various foods for their lead content, the Council for Responsible Nutrition (CRN) has conducted similar tests on calcium-containing products and supplements (see Table 2, page 18).[81]

The FDA studies have found trace amounts of lead to be present in virtually all foods analyzed, attesting to the fact that humans are exposed daily to trace amounts of lead in concentrations that are not considered toxicologically relevant. For its part, the CRN has concluded that "calcium products contain naturally occurring trace levels of lead similar to the lead levels found in common foods and beverages, such as fruits, vegetables and milk."[81]

The trace amounts of lead detected in calcium supplements are generally lower—often much lower—than the Federal standard of 3 ppm (parts per million).[82] The Natural Resources Defense Council (NRDC) has stated, however, that pregnant women absorb more lead than do nonpregnant women; and the council has called for new manufacturing standards for products such as calcium supplements to lower their lead content.[82]

Although lead-related variables were not specifically considered, a study

recently published in the *Journal of the American Medical Association* followed nearly 2,500 pregnant women who took between 1,500 and 2,000 mg of supplemental calcium daily.[83] The researchers identified no significant adverse health effects associated with increased calcium supplementation.

The available studies on calcium supplementation unequivocally show that the health benefits of calcium, an essential mineral, clearly outweigh any hypo-

Table 2. Analysis of Food Products for Lead Content

Product	Lead (µg per 1,000 mg of elemental calcium)
Whole milk A	6.7
Whole milk B	5.0
Whole milk C	1.7
Milk, 2% fat, A	9.0
Milk, 2% fat, B	9.0
Milk, 2% fat, C	0.8
Calcium Supplement A	6.3
Calcium Supplement B	3.1
Calcium Supplement C	4.3
Calcium Supplement D	6.9
Calcium Supplement E	3.4

Product	Lead (µg per serving)
Applesauce, canned	8.5
Fruit cocktail, canned	7.1
Spinach, fresh	2.4
Peaches, canned	6.0
Pears, canned	4.9
Strawberries, fresh	1.1
Apple Juice, bottled	2.6
Wine	7.7

Source: Council for Responsible Nutrition (CRN) Analysis of FDA Total Diet Study, 1991–1993.

thetical risk that could result from the presence of trace amounts of lead. Moreover, calcium dramatically reduces the body's absorption of lead. Thus, an adequate intake of calcium may be among the best dietary means to counteract the body's uptake of lead. A decrease in the limitation for lead in calcium supplements would result in a biologically insignificant change in the amount of lead absorbed—a change that would have no net health benefit.[84]

6. Regulatory Initiatives for Limiting Exposure to Lead

Over the years, standards and regulations have been established to limit human exposure to lead-containing materials. Included among the initiatives are the Occupational Safety and Health Administration (OSHA) Lead Standard,[85] the U.S. Department of Housing and Urban Development (HUD) Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing,[86] the U.S. Environmental Protection Agency Guidance on Residential Lead-Based Paint, Lead-Contaminated Dust, and Lead-Contaminated Soil,[87] OSHA's lead standard for construction,[88] and Title X, the Residential Lead-Based Paint Hazard Reduction Act of 1992,[89] aimed at addressing the lead paint problem in private housing. In addition, because quantification of exposure and abatement/remediation activities require accurate lead measurements, the standards-setting organization, the American Society for Testing and Materials, continues to develop standards related to the identification, monitoring, and remediation of lead hazards.[90]

Two federal laws now form the basis for the regulation of lead-based paint: the Lead-Based Poisoning Prevention Act of 1971, with subsequent amendments in 1973, 1976, 1987, and 1988; and the 1992 Residential Lead-Based Paint Hazard Reduction Act. During the discussions that led to the 1971 Act, two approaches were considered to control residential lead exposure: a health-based approach and a housing-oriented approach.

The health-based approach involved screening children to determine BLLs, treating those children found to have elevated BLLs, and deleading their homes. This approach had the distinct advantage of early detection of high-risk children. Poorly nourished children concentrated in inner cities are particularly vulnerable to lead exposure and could be more easily identified through such a targeted health-based screening approach.

The housing-oriented approach involved the removal of lead-based paint from public housing, regardless of the paint condition and exposure potential. This housing-based approach took center stage and eventually supplanted the health-based approach.

In 1992 the U.S. Congress enacted the Residential Lead-Based Paint Hazard Reduction Act. Title X of this act establishes 0.5 percent lead by weight as the lead level in *existing* paint that triggers lead hazard control measures. This measure was designed to control the most significant lead paint hazards. Rather

Table 3. Federal Efforts Related to Lead Exposure and Control

Agency	Responsibility
Food and Drug Administration (FDA)	Regulates lead content in bottled water, ceramic and other foodware, decorated glassware, lead crystal, calcium supplements, coffee urns, food, soldered cans
Environmental Protection Agency (EPA)	Monitors lead content in air, water, and soil and has some involvement in regulating lead-based paint
National Institute for Occupational Safety and Health (NIOSH)	Conducts research and surveillance on occupational lead exposure; offers health hazard evaluation programs and industrial hygiene training
Occupational Safety and Health Administration (OSHA)	Regulates lead exposure at the work site
National Institute of Environmental Health Sciences (NIEHS)	Conducts basic biomedical research on human health effects of lead
Department of Housing and Urban Development (HUD)	Funds and directs public housing authorities to contain or remove lead-based paint in public housing units
Consumer Product Safety Commission (CPSC)	Requires warning labels on lead-containing products; regulates lead paint in children's toys; issues warnings about the hazards of lead-based paint in the home
Agency for Toxic Substances and Disease Registry (ATSDR)	Makes health assessments of lead-containing areas near Superfund sites

than requiring the removal of lead-based paint from all exterior and interior surfaces, the statute draws a distinction between an *imminent* hazard, such as lead-contaminated dust and soil or flaking and accessible paint, and a *latent* hazard, meaning intact lead paint on surfaces not accessible to a child. Perhaps most significantly, under this statute lead-containing paint removal was no longer mandated under all circumstances.

In June 1995 HUD released a report, Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing, which devotes particular attention to testing for lead in housing.[86] The HUD guidelines also place emphasis on worker health and safety and on the role of the Occupational Safety and Health Administration (OSHA) in lead-related activities. This is a logical development, given the growth of the lead testing and abatement industry and the number of workers potentially exposed to lead during remediation activities. These guidelines mandate the use of personal protective equipment, decontamination procedures, and medical surveillance techniques, although largely without consideration of the cost of these measures.

The FDA, the agency responsible for establishing permissible lead levels in foods and related consumer products, has acted to eliminate lead solder in can manufacture, to reduce the use of lead-based pesticides on fruits and vegetables, and to promote the packaging of baby foods and juices in glass containers. Limits have been placed on permissible amounts of lead leachable from domestic and imported ceramic products and from silver-plated hollowware. Lead glaze on most ceramic foodware sold in the U.S. is now formulated, applied, and fired in such a way that lead will not leach into food and beverages. Table 3 (opposite) lists a selection of federal agencies along with their specific areas of responsibility, either for regulating human exposure to lead or for researching the health effects of lead.

7. Lead: What Is a Safe Level?

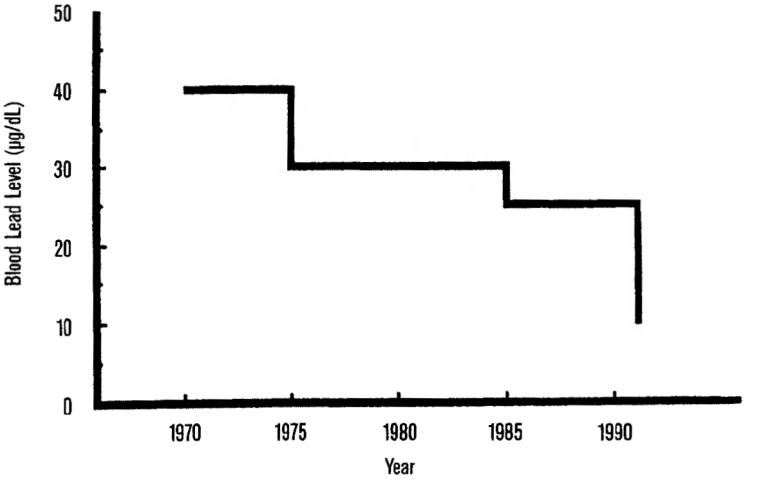
Over the years the CDC has lowered its recommended-action BLL, the level at which some intervention or monitoring is advised (see Figure 2,page 22). In 1970 the CDC lowered its intervention level from 55 µg/dL to 40 µg/dL. It lowered it further, to 30 µg/dL, in 1975 and then to 25 µg/dL in 1985. The intervention level—the point at which public health intervention begins—was set at the current 10 µg/dL in 1990.[15] During the decades of the 1970s and 1980s nearly 9 out of every 10 American children under age 5 had BLLs exceeding 10 µg/dL. By today's definition those children would have been considered "lead poisoned." Today, however, fewer than 5 percent of American children in the 1-to-5 age group have BLLs in excess of 10 µg/dL.[9]

One of the more unfortunate outcomes of the lead controversy is the wide-

spread and scientifically inaccurate use of the term "lead poisoning." This term is often used incorrectly to define asymptomatic children with BLLs that exceed 10 µg/dL. A "poison," in toxicological terms, has a definite dose level at which the agent in question exerts highly toxic—often fatal—effects. Over the years the term "lead poisoning" has been systematically applied to lower BLLs, a practice which is inaccurate from a toxicological standpoint. The term "lead poisoned" is justifiably used at high BLLs, but poisoning in the literal sense does not occur at BLLs as low as 10 µg/dL. A 1984 report in the *Journal of Pediatrics* found that symptomatic lead poisoning in children was associated with BLLs greater than 70 µg/dL. At levels less than 50 µg/dL, the report suggested that other diagnoses and descriptors be sought.[91]

The misuse of the word "poison" is unfortunate. One consequence of such misuse is that many states or health agencies now interpret 10 µg/dL to be a frank effect level and have passed legislation based on that interpretation. Use of alternative descriptors such as "elevated blood lead" or "minimally or modestly increased blood lead" would be a more accurate depiction of BLLs in the 10 to

Figure 2. Blood Lead Levels Considered Elevated by the Centers for Disease Control and the Public Health Service



Source: CDC. (1991). *Preventing Lead Poisoning in Young Children.* U.S. Department of Health and Human Services.

20 µg/dL range. It is clear that the CDC makes a distinction between a BLL of 10 µg/dL and one of 70 µg/dL; it seems reasonable that descriptors of severity should also be differentiated.

The CDC's justification for lowering the blood lead guideline level is related to studies and information about *potential* neurologic, reproductive, and hypertensive effects resulting from low-level lead exposure.[21] Of particular note, the CDC has asserted that "as more data become available, the definition of lead toxicity level will likely continue to be lowered." Blood lead action levels and definitions of toxicity should be based on clear indications of adverse effects to the most sensitive individual (in this case, a child). Such action levels should not be periodically lowered because studies have associated low BLLs with measurable effects that may not be adverse or toxic in nature. If future studies and scientific evidence demonstrate the adversity of effect(s) resulting from low-level lead exposure, then recommended BLLs and exposure limits should be adjusted accordingly. Currently, the weight of evidence does not clearly indicate a need to change the blood lead guideline; and to lower the "lead poisoning" level—particularly in the absence of clear, unequivocal data—is questionable from a scientific perspective.

An unfortunate ramification of the continual reduction of the lead action level is that attention may mistakenly be focused on children with BLLs that are not associated with clinical effects, making it harder to help those subpopulations truly in need of intervention (i.e., socioeconomically disadvantaged children or young adults with BLLs that are clearly associated with adverse effects). Using the label "poisoning" when referring to BLLs that are without defined health consequences may also serve to confuse people and may cause anxiety for parents who are unfamiliar with the significance of blood lead concentrations. Increased use of the CDC classification (see Table 4, page 24) would be instrumental and educational in alleviating parental fear.

The continuing decline of the lead action level mirrors a trend in environmental health assessment today: the trend of associating increasingly smaller levels of contaminants detected in the environment with some measurable effect in humans. According to the principles of toxicology, for virtually all substances there is some level at which there is no adverse effect. The critical step in public health protection is to establish the lowest observed effect level resulting from exposure and then to determine whether that effect represents an adverse effect or is simply an indicator of exposure.

Table 4. CDC Recommendations for Comprehensive Follow-up Services, According to Diagnostic Blood-Lead Level (BLL)*

BLL (µg/dL)	Action
<10	Reassess or rescreen in 1 year. No additional action necessary unless exposure sources change.
10–14	Provide family lead education. Provide follow-up testing. Refer for social services, if necessary.
15–19	Provide family lead education. Provide follow-up testing. Refer for social services, if necessary. If BLLs persist (ie., 2 venous BLLs in this range at least 3 months apart) or worsen, proceed according to actions for BLLs 20–44.
20–24	Provide coordination of care (case management). Provide clinical management. Provide environmental investigation. Provide lead-hazard control.
45–69	Within 48 hours, begin coordination of care (case management), clinical management, environmental investigation, and lead hazard control.
≥70	Hospitalize child and begin medical treatment immediately. Begin coordination of care (case management), clinical management, environmental investigation, and lead-hazard control immediately.

*A diagnostic BLL is the first venous BLL obtained within 6 months of an elevated screening BLL.

Source: CDC (1997). *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials.* U.S. Department of Health and Human Services.

8. Lead Abatement

8.1. When Is It Needed?

One of the principal ways to decrease the risk of toxicity from any substance is to reduce the source of exposure. Over the past decade remediation of lead-containing paint has become popular; such remediation is now mandated by many state programs. While the intent and motivation for remediating lead-containing paint are laudable, there are certain conditions under which abatement is not advised.

Intact and well-maintained lead-based paint should not, in most cases, be abated. Lead-based–painted surfaces become a hazard only when they have been allowed to deteriorate. Lead-contaminated dust may be generated as lead-based paint deteriorates over time; is damaged by moisture; is abraded on friction and impact surfaces; or is disturbed in the course of renovation, repair, or abatement projects.

We have learned from past experience with other environmental health concerns, such as asbestos, that in many situations (intact asbestos in pipe wrappings) remediation or abatement activities are either ineffective or may actually increase health risk by dispersing the substance and increasing the potential for human exposure. There is often no need to renovate, strip, or raze a home merely because it contains lead paint. If the paint is intact and not peeling, and there is no evidence of lead dusting, then costly and disruptive remedial activities may not be advisable. On the other hand, abatement should be considered if (a) lead exposure has been confirmed, (b) a health risk to children is probable, and (c) the risk can be reduced through effective remedial techniques.

During 1993–1994 the New York State Department of Health assessed lead exposure among children resulting from renovation and remodeling of homes containing lead-based paint.[92] The study identified 320 children in New York State (excluding New York City) with BLLs greater than 20 μg/dL, levels considered to be attributable to residential renovation and remodeling. In this study, in most cases (86 percent) the paint removal was not performed by a professional contractor (who might be expected to be more aware than a layperson of lead hazards and protective measures). The study determined that home renovation and remodeling in which lead-based paint is altered or disturbed constitutes an important source of lead exposure among children.

We should be promoting the philosophy of "lead-safe" rather than "lead-free," particularly in terms of remediation efforts in private homes and public housing. Maintaining intact lead paint in a safe condition is prudent until future renovation or abatement activities necessitate the complete removal or encapsulation of lead-containing paint.

The Baltimore, Maryland, Jobs and Energy Project is a successful program that incorporates both lead abatement and public education.[93] Because the majority of lead paint hazards are found in single-family units, the Baltimore Project was designed to provide affordable lead paint and dust hazard identification, remediation, and prevention programs for single-family homes, duplexes, and small apartment buildings.

The basic components of the Baltimore program include identification and evaluation of the extent of the lead problem on a community-, neighborhood-, or apartment-complex–wide basis and the assignment of an abatement schedule based on a needs assessment. The needs assessment includes, but is not limited to, the number of vulnerable children present, the levels of lead dust on surfaces, the degree of lead paint deterioration, the size of the surfaces to be treated, the rate of lead dust generation, and the BLLs of the resident children.

The actual abatement work is conducted by trained local contractors and appropriately trained volunteers. Abatement, if conducted, is followed by education for residents on the proper maintenance of their abated or partially abated home. The Baltimore program has been successful because (1) it has been affordable; (2) it has selectively addressed areas in need of lead-based paint and dust abatement rather than arbitrarily removing all lead-based painted surfaces; (3) it meets HUD clearance standards; and (4) the local contractors and volunteers have performed the work in a safe manner.[93]

8.2. Abatement Techniques: Which Are the Best?

Under the federal Residential Lead-Based Paint Hazard Reduction Act of 1992, lead-based paint hazard controls are categorized into three types: interim controls, abatement of lead-based paint hazards, and complete abatement of all lead-based paint.

Interim controls are a set of measures designed to temporarily reduce the likelihood of human exposure to lead-based paint hazards. Such controls may include dust removal, paint film stabilization, and treatment of surfaces (e.g., window wells and sills) that are subject to friction and impact. Education, ongoing maintenance, monitoring, and periodic reevaluations by certified professionals are also a part of interim controls. Interim controls for lead-contaminated soils include covering the area with grass or gravel and adding fences, bushes, or decks.

Abatement of lead-based paint hazards may include the removal of deteriorated lead-based paint and lead-contaminated dust; the permanent containment or encapsulation of lead-based paint (encapsulation involves the bonding of coatings and rigid coverings to the existing paint film); the replacement of lead-painted surfaces or fixtures; and, in the environment, the removal or covering of lead-contaminated soil.

Finally, complete abatement involves the permanent elimination of all interior and exterior lead-based paint, regardless of paint condition. Abatement of lead-contaminated soils would include complete removal of at least the top six inches of soil, soil cultivation, soil treatment and replacement, or paving with concrete or asphalt.

The effectiveness of a given form of lead control (interim control or abatement) may be measured both by how well it eliminates or reduces an individual's exposure to lead-contaminated materials and how well its implementation reduces the BLLs of exposed individuals. In 1994 a group of researchers reviewed the available literature regarding lead-based paint, dust, and soil interim controls and abatement techniques. The researchers identified 14 studies conducted during the period from 1974 to 1993.[94] They concluded that both in-place management (interim control) and source isolation or removal (abatement) techniques for lead-based paint and lead-contaminated soil and dust were only partially effective in reducing blood-lead concentrations. There was no conclusive evidence that either of these methods was more effective than the other.

A recent study on pre- and post-abatement BLLs of children from deleaded homes suggest that current abatement techniques may be limited in terms of their effectiveness in reducing BLLs.[95] This study was a review of the effect of home lead removal on the BLLs of 132 children who had not undergone medical treatment for lead exposure and whose homes were lead-abated between 1987 and 1990.

In the majority of children with BLLs equal to or in excess of 25 μg/dL, and particularly in those with BLLs above 30 μg/dL, residential deleading was associated with an 18 percent decrease in BLLs in the year following abatement. When the child's pre-abatement BLL was below 25 μg/dL, however, and particularly when it was below 20 μg/dL, the child's BLL was more likely to increase than to decrease following the deleading.

The researchers concluded that if home lead abatement is to be effective for children with BLLs below 30 μg/dL, and particularly for those below 20 μg/dL, caution must be exercised in order to minimize exposure to lead-containing dust during the removal.[95]

A review of the results of abatement studies generally supports the notion that intact and well-maintained lead-based paint to which there is limited, if any, human exposure should not be removed. This conclusion is in agreement with the HUD lead-based paint guidelines, which call for greater focus on correcting lead-based paint hazards rather than removing all lead-based paint.[86] Regardless of the residential lead-based paint remediation methodology employed, all methods should be followed up with education for adults regarding identification and management of lead-based paint hazards, periodic and proper house

cleaning and maintenance procedures, and proper nutrition and hygiene for children living in the home.

9. Conclusions and Recommendations

Lead is one of the most pervasive and persistent heavy metals in the environment. It can be toxic to humans if sufficient exposure and absorption occur. Because lead has no known beneficial or necessary function within living systems, there is a need to protect individuals from excessive lead exposure and to educate the general population in personal habits that will help in this effort.

In recent years much has been written about the role of lead in causing behavioral and neurodevelopmental effects in children. While lead is clearly capable of adversely affecting the central nervous system at high BLLs, it is difficult to attribute toxicologically significant behavioral or neurological effects to low BLLs, because of the numerous confounding factors that influence intellect and neurobehavior in children.

Major federal programs introduced to reduce lead in the environment have been successful, as demonstrated by dramatically lower BLLs in the U.S. population. Approximately 95 percent of all young children in the U.S. currently have BLLs under 10 μg/dL, supporting the CDC statement that childhood lead poisoning is not "a major environmental health problem in the United States but remains primarily a disease of the poor and underprivileged."[14]

The current (1991–1994 time period) mean BLL in the U.S. population is 2.3 μg/dL, down from 2.8 μg/dL during the 1988–1991 time period.[9] Recent data indicate, however, that 93,000 U.S. children are estimated to have BLLs above 25 μg/dL, a level at which significant effects are more likely to be clinically evident. Of the U.S. children estimated to have BLLs in excess of 25 μg/dL, at least 61 percent are African-American or Mexican-American. Among those of the remaining 39 percent who are Caucasian, the majority are estimated to be among the urban poor. Individuals who fall into these higher risk categories or who are known to have elevated BLLs should be among the first groups targeted for surveillance and intervention programs.

For the majority of children not considered high risk, an approach based on education and lead-exposure reduction and prevention is recommended. The following simple strategies will help to minimize risk of exposure and adverse health effects for children and adults alike.

9.1. General Personal Strategies

- For homes built before approximately 1960 that contain peeling or flaking paint, determine analytically if the paint contains lead and seek expert advice on whether paint removal is warranted. (Note: Loose paint is especially likely to be found on window sills and window wells, since the opening and closing of windows tends to damage paint.) Residents of the home (particularly young children and pregnant women) should not remain in the house during lead abatement activities.

- Avoid storing acidic foods (e.g., tomatoes, vinegar, and orange juice) in older or imported ceramic products and do not store food or beverages in lead crystal.

- Because exposure to dirt by children is in most cases unavoidable, monitor the play activities of children to prevent intentional ingestion of dirt.

- Educate children as to the importance of good hygiene practices, particularly the washing of hands before eating.

- Emphasize the importance of good nutrition, particularly since individuals with iron or calcium deficiency tend to have higher blood lead levels and nutritionally deficient individuals may be more vulnerable to the toxic effects of lead.

- If living in an older house with outdated plumbing, let tap water run for 30 seconds or until it runs cool before using. Do not use hot water for drinking or cooking purposes since lead leaches more easily into hot water.

- Request venous (as opposed to fingerstick) blood lead testing for your child if there is evidence (presence of known or suspected lead sources in addition to clinical signs such as fatigue, behavioral changes, or gastrointestinal disturbances) to suggest that excessive exposure may have occurred.

9.2. General Public Strategies

- Recycle or properly dispose of lead-containing consumer products, particularly lead-containing batteries, following federal, state, or local guidelines, if applicable.

- Store, handle, and dispose of lead-containing or lead-contaminated materials (i.e., paint dust and chips) carefully and appropriately.

- If you are an employer, and lead exposure is a potential concern, review, understand, and implement the OSHA lead standard for workers occupationally exposed to lead.

- Avoid excessive exposure to lead-containing materials used in hobby activities (e.g., production of bullets and fishing sinkers, lead soldering, preparation of leaded stained-glass windows).

- Support those lead-control programs that seek to identify high-risk individuals through continued research, educational efforts, and community awareness.

While currently we may not fully understand the implications and ramifications of low BLLs on human health, as environmental levels and subsequent human exposures are reduced, BLLs and the risk of adversity will also decrease. Lead levels will continue to decline in the ambient environment. Over the ensuing years BLLs should continue to decline as well—an observation which has been recently confirmed for the U.S. population.[9] In the years ahead we must strive to identify those children who remain at heightened risk of lead exposure and must intervene when appropriate to reduce BLLs.

Finally, from a broader public health viewpoint, we must continue to mitigate lead exposures for high-risk sectors of the population while continuing to identify and place into perspective other known environmental health hazards that deserve equal or greater attention.

References

1. Sittig, M. (1991). *Handbook of Toxic and Hazardous Chemicals and Carcinogens,* 3rd ed. Noyes Publications, New Jersey.
2. Gittleman, J.L., Engelgau, M.M., Shaw, J., Wille, K.K., and Seligman, P.J. (1994). Lead poisoning among battery reclamation workers in Alabama. *J. Occup. Med.* **36**, 526–532.
3. Baxter, P.J., Samuel, A.M., and Holkham, M.P.E. (1985). Lead hazard in British stain glass workers. *Br. Med. J.* **291**, 383.
4. U.S. Environmental Protection Agency (EPA). (1990). Report on the National Survey of Lead-Based paint in Housing; base report. Report no. EPA 747-R95-003. Washington, DC: Office of Pollution Prevention and Toxics.
5. Agency for Toxic Substances and Disease Registry (ATSDR). (1988). *The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress.* Atlanta, GA: US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control.
6. Houk, V.N., Ing, R.T., and Falk, H. (1979). Assessing lead exposure from drinking water. *Am. J. Public Health* **79**, 823–824.
7. U.S. Environmental Protection Agency (EPA). (1986). *Air Quality Criteria for Lead.* Research Triangle Park, NC: Office of Air Quality Planning and Standards.
8. United Nations Environment Program (UNEP). (1991). Environmental Data Report. GEMS Monitoring and Assessment Research Center, London, UK.
9. Center for Disease Control and Prevention (CDC). (1997). Update: Blood lead levels—United States, 1991–1994. *Morbid. Mortal. Weekly Report* **46**, 141–145.
10. Cosgrove, E., Brown, M.J., Madigan, P., McNulty, P., Okonski, L., and Schmidt, J. (1989). Childhood lead poisoning, case study traces source to drinking water. *J. Environ. Health* **52**, 346–349.
11. Mahaffey, K.R., Annest, J.L., Roberts, J., and Murphy, R.S. (1982). National estimates of blood lead levels: United States 1976–1980. *New Engl. J. Med.* **307**, 573–579.
12. Annest, J.L., Pirkle, J.L., Makuc, D., Neese, J.W., Gayse, D.D., and Kovar, M.G. (1983). Chronological trend in blood lead levels between 1976 and 1980. *New Engl. J. Med.* **308**, 1373–1377.
13. Pirkle, J.L., Brody, D.J., Gunter, E.W., Kramer, R.A., Paschal, D.C.,

Flegal, K.M., and Matte, T.D. (1994). The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys (NHANES). *JAMA* **272**, 284–291.

14. Brody, D.J., Pirkle, J.L., Kramer, R.A., Flegal, K.M., Matte, T.D., Gunter, E.W., and Paschal, D.C. (1994). Blood lead levels in the US population. Phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). *JAMA* **272**, 277–283.
15. Center for Disease Control and Prevention (CDC). (1997). *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials.* U.S. Department of Health and Human Services.
16. Goyer, R.A. (1996). Toxic effects of metals. In *Casarett and Doull's Toxicology: The Basic Science of Poisons*, 5th ed. (CD Klaassen, Ed.) McGraw-Hill, New York.
17. Schoof R.A., Steele, M.J., Petito-Boyce, C., and Evans, C.G. (1993). Assessing the validity of lead bioavailability estimates from animal studies. *Toxicologist* **13**, 478.
18. Freeman, G.B., Dill, J.A., Johnson, J.D., Kurtz, P.J., Parham, F., and Matthews, H.B. (1996). Comparative absorption of lead from contaminated soil and lead salts by weaning Fischer 344 rats. *Fund. Appl. Toxicol.* **33**, 109–119.
19. U.S. Environmental Protection Agency. (1996). Urban Soil Lead Abatement Demonstration Project. Volume I: EPA Integrated Report. EPA/600/P-93/001aF (April).
20. Mahaffey, K.R. (1990). Environmental lead toxicity: nutrition as a component of intervention. *Env. Hlth. Perspec.* **89**, 75–78.
21. Agency for Toxic Substances and Disease Registry (ATSDR). (1992). *Case Studies in Environmental Medicine: Lead Toxicity.* U.S. Department of Health and Human Services.
22. Silbergeld, E.K. (1991). Lead in bone: implications for toxicology during pregnancy and lactation. *Env. Hlth. Perspec.* **91**, 63–70.
23. Centers for Disease Control and Prevention (CDC). (1991). *Preventing Lead Poisoning in Young Children.* U.S. Department of Health and Human Services.
24. Goyer, R.A. (1993). Lead toxicity: current concerns. *Env. Hlth. Perspec.* **100**, 177–187.
25. Goyer, R.A. (1971). Lead and the kidney. *Current Topics in Pathology.* **55**, 147–176.

26. Wedeen, R.P. (1982). The role of lead in renal failure. *Clin. Exp. Dialysis Asphereris.* **6**, 113–146.

27. Goyer, R.A. and Rhyne, B. (1973). Pathological effects of lead. *Int. Rev. Exp. Path.* **12**, 1–77.

28. Pounds, J.G., Long, G.J., and Rosen, J.F. (1991). Cellular and molecular toxicity of lead in bone. *Environ. Health Perspect.* **91**, 17–32.

29. Rosen, J.F., Chaney, R.W., Hamstra, A., DeLuca, H.F., and Mahaffey, K.R. (1980). Reduction in 1,25-dihydroxy vitamin D in children with increased lead absorption. *N. Eng. J. Med.* **302**, 1128–1131.

30. Mahaffey, K.R., Rosen, J.F., Chesney, R.W., Peeler, J.T., Smith, C.M., and DeLuca, H.F. (1982). Association between age, blood lead concentrations and serum 1,25-dihydroxycholecaldiferol levels in children. *Amer. J. Clin. Nutr.* **35**, 1327–1331.

31. Koo, W.W.K., Succop, P.A., Bornschein, R.L., Krug-Wispe, S.K., Steichen, J.J., Tsamg, R.C., Hammond, P.B., and Berger, O.G. (1991). Serum vitamin D metabolites and bone mineralization in young children with chronic low to moderate lead exposure. *Pediatrics* **87**, 680–687.

32. Oliver, T. (1911). Lead poisoning and race. *Brit. Med. J.* **1**, 1096.

33. Taussig, F.J. (1936). *Abortion: spontaneous and induced.* Kimpton, London.

34. Rom, W.N. (1976). Effects of lead on female reproduction: A review. *Mt. Sinai J. Med.* **43**, 542–552.

35. Ernhart, C.B. (1992). A critical review of low-level prenatal lead exposure in the human: Effects on the fetus and newborn. *Reprod. Toxicol.* **6**, 9–40.

36. Dietrich, K.N., Krafft, K.M., and Bornschein, R.L. (1987). Low-level fetal lead exposure effect on neurobehavioral development in early infancy. *Pediatrics.* **80**, 721–730.

37. Dietrich, K.N., Krafft, K.M., Bier, M., Succop, P.A., Berger, O., and Bornschein, R.L. (1986). Early effects of fetal lead exposure: Neurobehavioral findings at 6 months. *Int. J. Biosocial Res.* **8**, 151–168.

38. Fahim, M.S., Fahim, Z., and Hill, D.G. (1976). Effects of subtoxic lead levels on pregnant women in the State of Missouri. *Res. Commun. Chem. Pathol. Pharmacol.* **13**, 309–331.

39. Lerda, D. (1992). Study of sperm characteristics in persons occupationally exposed to lead. *Amer. J. Ind. Med.* **22**, 567–571.

40. Hu, W.Y., Wu, S.H., Wang, L.L., Wang, G.I., Fan, H., and Liu, Z. (1992). A toxicological and epidemiological study on reproductive functions of male workers exposed to lead. *J. Hyg. Epidem. Microbiol. Immunol.* **36**, 25–30.

41. Assennato, G., Paci, C., Baser, M.E., Molinini, R., Candela, R.G., Altamura, B.M., and Georgino, R. (1987). Sperm count suppression without endocrine dysfunction in lead exposed men. *Arch. Environ. Health* **42**, 124–127.

42. Lancranjan, I., Popescu, H.I., Gavanesca, O., Klepsch, I., and Serbanescu, M. (1975). Reproductive ability of workmen occupationally exposed to lead. *Arch. Environ. Health* **30**, 396–401.

43. Tuohimaa, P., and Wickmann, L. (1985). Sperm Production of Men Working Under Heavy-Metal or Organic Solvent Exposure. In *Occupational Hazards and Reproduction.* (K. Hemminiki, M. Sorsa, H. Vanio, Eds.), Hemisphere, New York.

44. Coste, J., Mandereau, L., Pessione, F., Bregu, M., Faye, C., Hemond, D., and Spira, A. (1991). Lead-exposed workmen and fertility: A cohort study on 354 subjects. *Eur. J. Epidemiol.* 7(2), 154–158.

45. Wildt, K., Eliasson, R., and Berlin, M. (1983). Effects of Occupational Exposure to Lead on Sperm and Semen. In *Reproductive and Developmental Toxicity of Metals.* (J.W. Clarkson, G.F. Nordberg, P.R. Sager, Eds.), Plenum Press.

46. U.S. Environmental Protection Agency (EPA). (1989). Supplement to the 1986 EPA Air Quality Criteria for Lead. Addendum. Vol. 1. EPA/600/8-89/045A. Washington, DC: U.S. EPA Office of Health and Environmental Assessment, Pp. A1–A67.

47. Goyer, R.A. (1992). Nephrotoxicity and carcinogenicity of lead. *Fund. Appl. Toxicol.* **18**, 4–7.

48. Cooper, W.C., Wong, O., and Kheifets, L. (1985). Mortality among employees of lead battery plants and lead producing plants, 1947–1980. *Scand. J. Work Environ. Hlth.* **11**, 331–345.

49. McMichael, A.J., and Johnson, H.M. (1982). Long term mortality profile of heavily exposed lead smelter workers. *J. Occup. Med.* **24**, 375–378.

50. Selevan, S.G., Landrigan, P.J., Stern, F.B., and Jones, J.H. (1985). Mortality of lead smelter workers. *Amer. J. Epidemiol.* **122**, 673–683.

51. Gerhardson, L., Lundstrom, N.G., Nordberg, G., and Wall, S. (1986). Mortality and lead exposures: A retrospective cohort study of Swedish smelter workers. *Brit. J. Ind. Med.* **43**, 707–712.

52. Dingwall-Fordyce I., and Lane, R.E. (1963). A follow-up study of lead workers. *Br. J. Ind. Med.* **20**, 313–315.

53. Hertz-Picciotto, I., and Croft, J. (1993). Review of the relation between blood lead and blood pressure. *Epidemiol. Rev.* **15**, 352–373.

54. Staessen, A., Roels, H., and Amery, A. (1995). Low-level lead exposure and blood pressure. *J. Hum. Hypert.* **9**, 303–328.

55. Nowack, R., Wiecek, A., and Ritz, E. (1992). Lead and hypertension. *Contrib. Nephrol.* **100**, 25–34.

56. IPCS/WHO. (1995). Environmental Health Criteria 165. Inorganic Lead. World Health Organization, Geneva.

57. Staessen, J.A., Roels, H., and Fagard, R. (1996). Lead exposure and conventional and ambulatory blood pressure. *JAMA* **275**, 1563–1570.

58. Needleman, H.L., Schell, A., and Bellinger, D. (1990). Long-term effects of childhood exposure to lead at low doses: an eleven-year follow-up report. *New Engl. J. Med.* **322**, 83–88.

59. National Research Council (NRC). (1993). *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations.* Washington, DC: National Academy Press.

60. Pocock, S.J., Smith, M., and Baghurst, P. (1994). Environmental lead and children's intelligence. A systematic review of the epidemiological evidence. *Brit. Med. J.* **309**, 1189–1196.

61. Ruff, H.A., Bijur, P.E., Markowitz, M., Ma, Y-C, and Rosen, J.F. (1993). Declining blood lead levels and cognitive changes in moderately lead-poisoned children. *JAMA* **269**, 1641–1646.

62. Shannon, M., and Graef, J. (1996). Lead intoxication in children with pervasive developmental disorders. *Clin. Toxicol.* **34**, 177–181.

63. Bergomi, M., Borella, P., Fantuzzi, G., Vivoli, G., Sturloni, N., and Cavazzuti, G. (1989). Relationship between lead exposure indicators and neuropsychological performance in children. *Dev. Med. Child Neurol.* **31**, 181–190.

64. Winneke, G., Brochhaus, A., Ewers, U., Kramer, U., and Neuf, M. (1990). Results from the European multicenter study on lead neurotoxicity in children: implications for risk assessment. *Neurotoxicol. Teratol.* **12**, 553–559.

65. McMichael, A., Baghurst, P.A., Vimpani, G.V., Wigg, N.R., Robertson, E.F., and Tong, S. (1994). Tooth lead levels and IQ in school-age children: the Port Pirie cohort study. *Am. J. Epidemiol.* **140**, 489–499.

66. Kaufman, A.S. (1996). IQ, Lead Levels, and Inferences from Research Studies. Submission to the Department of Housing and Urban Development. Docket No. FR-3482-P-01.

67. Harrell, R.F., Woodyard, E., and Gates, A.I. (1955). *The Effects of Mother's*

Diet on the Intelligence of Offspring. New York: Bureau of Publications, Teachers College, Columbia University.

68. Benton, D., and Roberts, G. (1988). Effects of vitamin and mineral supplementation on intelligence of a sample of school children. *Lancet.* **1**, 140–143.
69. Schoenthaler, S.J., Amos, S.P., Eysenck, H.J., Perity, E., and Yudkin, J. (1991). Controlled trial of vitamin-mineral supplementation: effects of intelligence and performance. *Personal Indiv. Diff.* **12**, 343–350.
70. Benton, D., and Buts, J.P. (1990). Vitamin/mineral supplementation and intelligence. *Lancet.* **335**, 1158–1160.
71. Benton, D., and Cook, R. (1991). Vitamin and mineral supplements improve the intelligence of six-year-old children. *Pers. Indiv. Diff.* **72**, 1151–1158.
72. Oski, F.A. (1993). Iron deficiency in infancy and childhood. *New Eng. J. Med.* **329**, 190–193.
73. Lozoff, B., Jimenez, E., and Wolf, A.W. (1991). Long-term developmental outcome of infants with iron deficiency. *N. Engl. J. Med.* **325**, 687–694.
74. Consumer Product Safety Commission (CPSC). (1996) *Consumer Product Safety Review: Lead Paint on Public Playground Equipment.* **1(2)**.
75. Brown, P.L. Imported blinds pose lead risk to children. *NY Times,* June 27, 1996.
76. Mielke, H.W., Taylor, M.D., Gonzales, C.R., Smith, M.K., Daniels, P.V., and Buckner, A.V. (1997). Lead-based hair coloring products: Too hazardous for household use. *J. Am. Pharm. Assoc.* **37**, 85–89.
77. U.S. EPA Integrated Risk Information System (IRIS). (1996).
78. Cohen, A.J., and Roe, F.J.C. (1991). Review of lead toxicology relevant to the safety assessment of lead acetate as a hair colouring. *Food Chem. Toxicol.* **29**, 485–507.
79. U.S. Food and Drug Administration (FDA). *Fed. Reg.* **45**, 213. October 31, 1980. 21 CFR parts 73 and 81. Pp. 72112–72118.
80. U.S. Food and Drug Administration (FDA). *Fed. Reg.* **46**, 44. March 6, 1981. 21 CFR parts 73 and 81. Pp. 15500–15504.
81. Council for Responsible Nutrition (CRN). *Facts about Calcium & Calcium Products.* January 27, 1997.
82. Bourgoin, B.P., Evans, D.R., and Cornett, J.R. (1993). Lead content in 70 brands of dietary calcium supplements. *Am. J. Public Health* **83**, 1155–1160.
83. Bucher, H.C., Cook, R.J., Guyatt, G.H., Lang, J.D., Cook, D.J., Hatala,

R., and Hunt, D.L. (1996). Effects of dietary calcium supplementation on blood pressure. A meta-analysis of randomized controlled trial. *JAMA* **275**, 1016–1022.

84. CRN News Release. *Calcium supplements are beneficial and safe, tighter standards would produce no medical or scientific advantage.* January, 1997.
85. Occupational Safety and Health Administration (OSHA). (1990). 29 CFR Ch. XVII. 1910.1025. Lead.
86. U.S. Department of Housing and Urban Development (HUD). (1995). Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing.
87. U.S. Environmental Protection Agency (EPA). (1994). Memorandum: Guidance on Residential Lead-Based Paint, Lead-Contaminated Dust, and Lead-Contaminated Soil.
88. Occupational Safety and Health Administration (OSHA). (1993). *Lead in Construction.* OSHA 3142.
89. Title X. (1992). Residential Lead-Based Paint Hazard Reduction Act of the Housing and Community Development Act of 1992 (Public Law 102-550).
90. Ashley, K., and McKnight, M.E. (1993). Lead Abatement in Buildings and Related Structures: ASTM Standards for Identification and Mitigation of Lead Hazards. *ASTM Standardization News.*
91. Piomelli, S., Rosen, J.F., Chisolm, J.J., Jr., and Graef, J.W. (1984). Management of Childhood Lead Poisoning. *J. Pediatrics* **105**, 523.
92. Centers for Disease Control (CDC). (1997). Children with elevated blood lead levels attributed to home renovation and remodeling activities—New York, 1993–1994. *JAMA* **277**, 1030–1031.
93. Livingston, D. (1994). Community Resources. Summary of presentation to the Michigan Environmental Sciences Board, Lead Panel. Attachment 1, 6–8.
94. Burgoon, D.A., Rust, S.W., and Schultz, B.D. (1994). A Summary of Studies Addressing the Efficacy of Lead Abatement. In *Lead in Paint, Soil, and Dust: Health Risks, Exposure Studies, Control Measures, Measurement Methods, and Quality Assurance*, ASTM STM 1226. (M.E. Beard and S.D. Allen Iske, Eds.) Philadelphia: American Society for Testing and Materials.
95. Swindell, S.L., Charney, E., Brown, M.J., and Delaney, J. (1994). Home abatement and blood lead changes in children with class III lead poisoning. *Clin. Pediatrics* **Sept.**, 536–541.