LEAD AND HUMAN HEALTH:
An Update

Prepared for the American Council on Science and Health (ACSH)

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Despite years of intensive research, educational efforts, and remedial measures, lead continues to receive as much attention as any modern environmental health risk. Some would still characterize lead as America's leading environmental health concern. Based on a review of the scientific literature, and assessing lead from the perspective of public health, American Council on Science and Health (ACSH) has come to the conclusions stated below.

- Lead is an important toxicant that can exert adverse effects in humans, given sufficient exposure and accumulation in the body. Systems known to be susceptible to adverse effects of high exposure include: neurological, reproductive, renal, and hematological. Children are more sensitive than adults to the effects of lead, and precautions should be taken to limit childhood exposure and keep blood lead levels (BLL) below the CDC-recommended level of 10 µg/dL.

- Federal and state regulatory standards and programs have helped to minimize or eliminate the amount of lead in consumer products, occupational settings, and the environment; this decreased presence has contributed to remarkable reductions in BLL in the U.S. population, particularly in children.

- Symptomatic childhood lead poisoning seen years ago in children with markedly elevated blood lead levels (i.e., > 40 µg/dL), has almost disappeared as a clinical finding in the U.S. Such lead poisoning no longer constitutes a widespread public health threat in the U.S., although specific sectors of the population may remain at risk as a result of elevated exposures.

- The most recent published data show that the U.S. average for blood lead is 2.9 µg/dL; the CDC action level (i.e., education and followup testing) is 10 µg/dL, and the intervention level (clinical case management) is 20–44 µg/dL. For children 1–2 years of age, the most recent data show that the mean level is 3.1 µg/dL. Blood lead levels are continuing to decline, and given the reductions in major source exposures, these levels should continue to fall until equilibrium with background exposure is reached.

- The continued focus on trace amounts of lead in such consumer products as cosmetics and dietary supplements does not adequately take into account the relative exposures these sources represent; lead in these products does not appear to be toxicologically significant and should not pose a health risk to humans.

- Claims of subtle neurobehavioral effects in children due to elevated BLL are not based on firm evidence; many studies that attempt to link low-level lead exposure with learning disabilities, behavioral problems, attention deficit disorders, and lowered IQ are complicated by multiple confounding socioeconomic and familial factors.

- There is a significant degree of public confusion around the CDC action level of 10 μg/dL. This is the lowest level at which the CDC recommends initial action, limited to education and followup testing. Specific clinical intervention measures are not recommended until BLL values exceed 20 μg/dL.

- Targeted rather than universal screening for elevated lead levels is preferred in order to identify children and other individuals with an increased risk of elevated BLL cost-efficiently.

- Lead abatement of homes should not be universally mandated, but should be considered on a case-by-case basis; proper remediation techniques and attention to resident exposure during such remediation are critical.

- Elimination or minimization of exposure to lead can be successfully achieved through alterations in personal habits, increased public education, and improvements in living conditions, particularly among population groups known to have higher likelihood of exposure.

I. INTRODUCTION

"If we were to judge of the interest excited by any medical subject by the number of writings to which it has given birth, we could not but regard the poisoning by lead as the most important to be known of all those that have been treated of, up to the present time."—Orfila, 1817

Although this was written almost two centuries ago, concern over lead and human health has not waned, and today, lead remains one of the most often discussed and well-studied public health topics of all time. Both the interest and concern over lead are related to its toxicity at certain dose levels and lack of known biological benefit. The level at which lead exerts various effects on biological systems remains the focus of much research, investigation, and debate. It has become evident that the general public is not sufficiently knowledgeable about the health risks associated with lead, in part due to the conflicting and often biased information disseminated by government agencies, industries, attorneys, and public interest groups, among others. In addition to those regulatory and public health agencies charged with providing recommendations concerning lead exposure, the public receives ample "spin" from the news media and popular press—information that is frequently inaccurate and often highly subjective regarding lead and its real risk.

The purpose of this updated monograph is to convey the current information on lead pertinent to a public health perspective, as well as to provide the basis for increased understanding of the risks that may be associated with lead depending on the different levels of exposure. A key principle of the review is the differentiation of hazard and risk, and the concept that hazard alone is not associated with a health risk. A pack of cigarettes (hazard) in your pocket won't cause lung cancer (health risk) if they are never lit and inhaled (exposure). A hazard must be combined with exposure to create a potential risk, and as exposure decreases so does the risk of adverse effects. As our analytical ability to detect smaller and smaller amounts of chemicals, microbiological agents, or other substances in the environment has increased, a concomitant impression has developed that a presumed health risk also exists simply because the mere presence of the substance can be proven or detected. Accordingly, this review will give some perspective about trace levels of lead exposure and what this means for human health.

Because of both regulatory initiatives and industrial awareness, there have been continuing declines in environmental lead, and thus

human exposure. We are on the cusp of a change in focus from lead as a widespread, major environmental health problem, to one in which small, selected subpopulations remain at risk from lead exposure. It is these more highly exposed individuals who require the attention of public health officials and for whom screening, surveillance, and abatement programs and initiatives should receive the highest priority. Billions of dollars are spent annually in the United States alone on lead-related health and abatement programs. In an age of increased demand for shrinking public financial resources, it is imperative that lead, as a health risk, be placed in perspective relative to other risk factors so that resources are assured to be available for those at greatest risk.

Lead is given the symbol "Pb," which comes from the Latin word "plumbus" or "plumbic," meaning lead.

II. WHAT IS LEAD?

Lead is a stable, silver-gray, ubiquitous heavy metal and is detectable in all phases of the inert environment (e.g., air, water, and soil) as well as in most biological systems. It is one of the more commonly used metals in the world, and like many other metals, is rarely found in its elemental form; rather, it is found in a variety of compounds, complexes, and alloys. Metallic lead is used in products such as electric storage batteries, lead solder, radiation shields, pipes, and sheaths for electric cable. It may be combined with other metals to make brass alloys for plumbing fixtures. Organic lead compounds contain a lead atom bonded to carbon to form an organic lead molecule; examples include tetraethyl and tetramethyl lead (the more toxic form of the metal) that were once widely used as gasoline additives to prevent engine knock. Inorganic lead salts are compounds containing lead combined with elements other than carbon. Examples include lead oxides, lead chromate, and lead nitrate. These compounds have been used in a variety of products such as insecticides, pigments, paints, glassware, plastics, and rubber compounds.

In the environment, lead is most commonly found as various lead salts in mineral ores. Lead in the environment occurs naturally and as a by-product of human activity, and its concentration and presence in environmental media are highly variable. Generally, lead tends to accumulate near discharge points (WHO, 1977, 1989), owing to its physical and chemical properties that minimize the potential for volatilization and airborne transport and enhance the tendency for rapid local deposition. In surface water, lead is likely to form insoluble complexes with

other substances. In soil and sediment, lead binds with other particles (e.g., complexation), thereby reducing its bioavailability (the amount of lead capable of being absorbed by the body) to organisms living in those environments. Plants and food crops may contain small amounts of lead either as a result of direct atmospheric deposition or root absorption from soil. Lead is not as pervasive in the environment as it once was due to its removal from various industrial products (e.g., gasoline, paint) and processes and the widespread efforts aimed at reducing human exposure through source reduction measures.

III. LEAD AND HUMAN HEALTH

A. Pharmacokinetics of Lead: Absorption, Distribution, and Excretion

Lead may be absorbed by the body through inhalation, ingestion, or dermal (skin) contact; it can be transferred to the fetus through the placenta (Goyer, 1990). Inhalation and dermal contact are routes of exposure more typical of occupational settings, whereas the primary route of exposure for the general population is ingestion from minor amounts in food and hand-to-mouth activity, particularly in children. Adults absorb approximately 5–15 percent of ingested lead into the circulation; of this amount, less than 5 percent is retained in the body (Goyer, 1996). Young children can absorb considerably more (30–40 percent) of ingested lead; this explains their enhanced susceptibility to the potential effects of lead (Goyer, 1996).

Blood lead level (BLL), typically measured and reported as micrograms of lead per deciliter (one deciliter is one tenth of a liter, or 100 milliliters) of blood (µg/dL), is a common biomarker (an indicator of exposure) for lead. The test to determine BLL is reliable and widely used, reasonably easy to perform, of low cost, and more reproducible and sensitive than other indirect measures of lead exposure.

A blood lead level conveys the results of a screening and diagnostic test used to assess lead exposure, particularly recent exposure. It reflects the equilibrium between absorption, excretion, and deposition in tissues. Blood lead levels respond relatively rapidly to changes in lead intake and are generally considered to show a linear relationship with exposure. Blood lead levels associated with chronic exposure may underestimate total body burden because the majority of body lead may be stored in teeth or bone and these are more difficult to sample.

Typically, measurements and estimates of lead absorption are derived from controlled animal studies, as there are few empirical human data on the absorption of lead from environmental sources, due in part to methodological and logistical difficulties. This is particularly true of child exposure from ingestion of lead-contaminated soil. When assessing potential exposure of children to lead in soil, the U.S. Environmental Protection Agency (EPA) has historically used a computer model (IEUBK[*]) to predict blood lead levels (BLL), which has default assumptions for the amount (i.e., 200 mg/kg) and bioavailability (i.e., 60 percent) of lead in soil. Studies of soil ingestion in humans as well as in laboratory animals reveal that actual BLL values may be above or less than estimates derived from models, which underscores the importance of using site-specific information and validating models when possible (Freeman et al., 1996; Casteel et al., 1997). A possible contributor to discrepancies between modeled and actual absorption is that lead absorption is dependent on the amount and form of lead ingested and on the matrix (e.g., soil or dust) in which it is consumed (Freeman et al., 1996). In a study aimed at better understanding the relationship between lead content of soil or dust and blood lead, the EPA found that, by itself, removal of soil lead as a source of exposure has minimal impact on blood lead levels, and concluded that lead in soil is not very bioavailable to humans (USEPA, 1996). The implications of these findings are significant from a risk perspective, particularly in situations in which remedial cleanup levels are directly linked to estimates of human lead exposure from soil.

Nutritional status and eating behavior appear to influence the absorption and toxicity potential of lead in several ways. Lead ingested from water and other drinks tends to be absorbed to a greater extent than lead ingested from solid food, while lead ingested under fasting conditions is absorbed to a greater extent than lead ingested during food consumption (Mahaffey, 1990; ATSDR, 1992). Lead also interacts with several essential elements—notably calcium, iron, and zinc—and dietary deficiencies of both calcium and iron are known to enhance the absorption of lead (Goyer, 1996). Thus, the Centers for Disease Control and Prevention (CDC) has made an educational effort to foster proper nutrition among children, particularly urging parents to assure an adequate daily supply of iron and calcium to help reduce lead absorption (CDC, 1991).

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[*] The IEUBK model used for predicting human blood lead levels is a multi-component, pharmacokinetic model baed on lead metabolism studies in infant and juvenile baboons.

Once absorbed in the bloodstream, lead is primarily distributed among two compartments—the more rapid turnover pool with distribution to the soft tissues such as the liver, lung, spleen, and kidney, and the slower turnover pool with distribution to the skeleton (Rabinowitz, 1991). Lead can accumulate over time, particularly in bone, and 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. Thus, lead in bone may contribute a significant amount to blood lead and serves as a key storage site and source of internal lead exposure, particularly in situations involving chronic exposure. Mobilization of lead from maternal bone is particularly relevant during pregnancy and lactation and may also occur in persons with osteoporosis (Silbergeld, 1988). The significance of internal organs as a source of lead is reflected by their respective biological half-life times (the time it takes for one half of an amount to be eliminated or removed from the body). In bone, the half-life is quite long, approximately 17–20 years (Heard and Chamberlain, 1984; Goyer, 1996), while in blood, the half-life is about 2–3 weeks (Chamberlain et al., 1978; Rosen, 1985).

Finally, the predominant mode of elimination of absorbed lead is renal (urinary) excretion, while excretion through the gastrointestinal tract (fecal) also occurs either directly (unabsorbed ingested lead) or in the bile.

B. Toxicological Effects

Lead has long been one of the most intensely studied and researched toxicants, with thousands of studies that have been conducted on lead and its effects in both animals and humans. Not surprisingly, there are numerous effects that have been reported in the literature, including neurotoxicity, carcinogenicity, reproductive toxicity, and neurobehavioral/developmental effects. It is not the intent of this monograph to review in great detail the toxicological characteristics of lead; for this, other general reviews on lead toxicity should be consulted (ATSDR, 1993; Goyer, 1993; Beck, 1992). While much of our knowledge on the effects of lead comes from experience involving excessive industrial or occupational exposure, the present challenge is to discern what effects occur, if any, at low, environmentally relevant levels.

Specific health effects associated with lead and the lowest BLL or BLL range at which those effects occur are shown in Table 1. Both the USEPA (EPA, 1986) and the Agency for Toxic Substances and Disease

Registry (ATSDR, 1988) have also reported on various lead-induced effects and the ranges at which they occur. The list of effects shown in Table 1 forms a continuum from clinical or overt effects to subtle or biochemical effects. The latter effects may not be adverse, but may merely represent a marker of exposure (Goyer, 1990a).

Neurological Effects

Neurotoxicity resulting from lead overexposure is perhaps the most well documented effect, particularly in settings involving occupational exposures. Manifestations of lead toxicity in adults consist of ataxia, memory loss, and at the highest levels, coma and death. Nerve conduction is reversibly slowed in peripheral nerves at BLL of approximately 40 μg/dL (Goyer, 1996). Overt effects on the nervous system,

Table 1. **Lowest Observed Effect Levels for Lead-Related Health Effects**		
Blood Lead Concentration (μg/dL)		
Effect	Children	Adults
Neurological		
Encephalopathy (overt)	80–100	100–12
Hearing deficit	20	—
IQ deficits	10–15	—
In-utero effects	10–15	—
Peripheral neuropathy	40	40
Hematological		
Anemia	80–100	80–100
U-ALA	40	40
B-EPP	15	15
ALA inhibition	10	10
Py-5-N inhibition	10	—
Renal		
Nephropathy	40	
Vitamin D metabolism	< 30	
Blood Pressure (males)	—	30
Reproduction		40

Source: Adapted from Casarett & Doull's Toxicology, 5th ed.,1996

such as wrist drop (weakness of the wrist and fingers caused by nerve compression), require BLL values of 60 µg/dL or greater (Goyer, 1996). Prospective and cross-sectional studies on the neurological effects of low- to moderately exposed lead workers have reported effects primarily related to slower motor nerve conduction coupled with difficulties in remembering incidental information, although these effects are not typically observed below mean BLL values of 40 µg/dL (Goyer, 1996).

As with adults, a primary target for lead toxicity in young children is the nervous system. It is generally recognized that at BLL values of 80 µg/dL or greater, lead encephalopathy occurs, characterized clinically by ataxia, coma, and convulsions, which are often fatal. Effects associated with BLL values below 80 µg/dL form a continuum from clinical, overt effects to subtle or biochemical effects (Goyer, 1990a). A number of cross-sectional and prospective epidemiologic studies (Grant and Davis, 1989; NRC, 1993) show that even without overt toxicity, children with mildly elevated (i.e., > 10–15 µg/dL) BLL values may show increased incidence of subsequent neurological or behavioral impairments. However, it has been particularly challenging and difficult to determine whether specific neurobehavioral deficits are causally linked with increased exposure to lead.

Neurobehavioral/Developmental Effects

Perhaps the greatest concern related to neurotoxicity has been that of lead's potential influence on behavior and development, particularly in children (EPA, 1989; Needleman et al., 1990; NRC, 1993). Generally, the studies have associated lead exposure with decreased intelligence, reduced short-term memory, reading disabilities, and deficits in vocabulary, fine motor skills, reaction time, and hand–eye coordination. During the past two decades, there have also been a number of epidemiological studies that have attempted to relate BLL values at the time of birth, during infancy, and through early childhood with measures involving psychomotor, cognitive, and behavioral outcomes. This monograph does not purport to review exhaustively all studies that have assessed lead's potential neurobehavioral effects on children, but rather provides a context for evaluating lead's potential role in influencing a complex trait such as intelligence.

In addressing this concern, a 1994 report in the *British Medical Journal* reviewed the epidemiological evidence concerning environmental lead and its effects on child intelligence. The investigators sought to quantify the magnitude of the relationship between IQ in children aged 5 years or older and their body burden of lead (Pocock et al., 1994). To

assess this potential relationship, 26 epidemiological studies published since 1979 were reviewed. This report concluded that while low-level lead exposure may cause a small IQ deficit, other explanations or influences (confounders) need to be considered, including: (a) Have the studies adequately controlled for confounding variables? (b) Are there selection biases in recruiting? (c) Do children of lower IQ adopt behaviors that make them more prone to lead exposure and uptake? Because of the uncertainties involved in assigning causality, health professionals and scientists have recommended that public health attention be prioritized towards reducing moderate increases in children's BLL while reviewing other social and biological determinants that may influence intellect and behavior in children (Ruff et al., 1993).

It has become a matter of some controversy as to whether slightly to moderately elevated BLL exert detectable short- and long-term adverse effects on neurobehavior and development and whether such effects are reversible. One primary reason for the controversy is the extreme difficulty in studying this suggested effect given the complexity of evaluating intelligence and the hundreds of variables that impact intelligence and behavior (and which must be controlled for in studies). Modern neuropsychological testing methods are different from more mainstream clinical psychological test methods (which primarily detect gross abnormalities) and as such, test method is important when evaluating subtle effects on IQ and behavior. There is, in addition, the challenge of investigating effects at the low end of the toxicological spectrum. Although the debate and discussion continue, meta-analyses of human epidemiological studies have generally observed a statistical association between blood lead and IQ that is small, most likely a 1–3 IQ-point deficit for a change in mean BLL from 10 to 20 μg/dL (Goyer, 1996). Not surprisingly, the association between an effect on IQ and blood lead is more apparent at moderate to high BLL values, typically in excess of 30 to 40 μg/dL (Ruff et al., 1993). This is important from two perspectives. First, the vast majority of children have BLL values well below this level (mean for children ages 1–5 was 2.7 μg/dL, while only 0.4 percent had BLL values $\geq$ 20 μg/dL; MMWR, 1997) and thus, significant changes in intelligence/behavior are not expected in the U.S. population as a whole. Second, identification of those small sectors of the population with elevated BLL remains a priority for identification and possible intervention. In reviewing the available prospective epidemiological neurobehavioral studies, Volpe et al. (1992) concluded that while individual studies may indicate an effect of some type, the weight of evidence of the collected studies does not lend support to the

concept that low-level lead exposure (i.e., BLL values below 25 µg/dL) is associated with clear neurobehavioral deficits in children. If a subtle decline in IQ is linked to lead exposure to some degree, perhaps we will see the converse—increase in IQ level—if such effects are reversible, as environmental lead levels continue to decline.

Difficulties in Studying Lead's Effect on Neurobehavior and Development in Children

While there are many challenges when studying the central nervous system effects of low-level lead, three stand out as particularly problematic:

First, interpretation of the independent variable, the lead level, can be problematic. For example, does a single point BLL indicate when a person was exposed, whether the exposure was acute or chronic, and what "developmental" period the person or child was in at the time of greatest body burden? Investigators usually must assume that the current BLL adequately measures the entire exposure history of the child until the time he or she was tested, as they generally have very little information regarding the true exposure history of the children being studied. There is no way of knowing, in most cases, whether the one or two BLL measurements represent an exposure history long enough to affect the child. In addition, there is usually no information regarding the age at which the child was exposed, which has profound consequences in assessing the degree to which lead might affect the developing brain. A "high" lead level at one point may actually reflect very recent short-term exposure, which would be unlikely to have caused the abnormalities found on testing.

Second, controlling for confounders, covariates, and biases presents substantial and even irreconcilable difficulties. Even if behavioral or cognitive alterations exist, it is extremely difficult to differentiate the neurobehavioral effects of lead from effects due to the many social, emotional and medical factors that are known to have important impacts upon neurobehavioral development (Schroeder et al., 1985; Dietrich et al., 1990; Ernhart et al., 1987). Despite the attempt to control for all confounding variables within a study, there are usually many uncontrolled variables. These include socioeconomic status, childhood diseases, parenting skills, genetic predisposition, styles of child rearing, parental time spent with the child, and skills and styles of key caretakers other than parents. One specific example of an uncontrolled variable is frequency of ear infections in young children, a variable that is often ignored in studies. Infants or toddlers prone to repeated infections of the

ear are likely to have verbal IQs that are lowered by several points when they reach school age (Kaufman, 1996). Additional factors that influence intelligence and performance include: maternal and paternal intelligence, family education and motivation, maternal cigarette smoking and drug use, home stability, child abuse, nutrition and prenatal care, labor and delivery, and personality characteristics.

Nutrition is particularly important in development and represents a key confounding variable that must be carefully assessed in interpreting studies on lead and IQ. Over the years, we have come to learn that poor nutrition is an important factor in the behavior and intelligence of children (Benton and Roberts, 1988; Schoenthaler et al., 1991). Numerous studies have investigated the positive correlation between diet supplementation and nonverbal IQ scores (Schoenthaler et al., 1991; Benton and Buts, 1990; Benton and Cook, 1991). Several investigations have demonstrated the particular importance of iron in influencing cognitive development and performance (Oski, 1993; Lozoff et al., 1991), and one group of investigators concluded that "the most important systemic abnormality produced by iron deficiency in infancy is the alteration in cognitive performance" (Oski, 1993). The results of these studies are significant because nutritional deficiencies thought to affect intellectual development are also expected to increase the efficiency of lead uptake from the gastrointestinal tract.

The third challenge to analysis of such effects, and perhaps the most important, is the difficulties inherent in the objective and accurate measurement of the dependent variable—subtle neurobehavioral effects, such as classroom behavior and IQ, which are "soft" endpoints with limited sensitivity and specificity.

The point of this discussion is to bring to light the many potential confounding factors and complexities, including the influence of essential trace elements such as iron, that must be addressed when investigating low-level effects of lead on human intelligence and behavior. Given that the much higher historical BLL of past decades, compared to today's levels, have not produced a discernible effect on intelligence, it is likely that until we develop more sensitive or reliable indicators of intelligence and behavior, this debate will continue.

Reproductive Effects
The effect of high lead exposure on reproductive health in both adult males and females has been recognized for some time. Severe lead toxicity in women has been associated with sterility, miscarriage, stillbirth, and neonatal morbidity and mortality from exposure in utero

(Oliver, 1911; Taussig, 1936; Rom, 1976). In men, heavy occupational exposure has been shown to have an adverse effect on semen quality (Lerda, 1992; Hu et al., 1992; Assennato et al., 1987).

In both men and women, the evidence for low-level exposure effects is weaker. It has been difficult to demonstrate effects on neurodevelopment in infants and children (Ernhart, 1992). For example, there is limited evidence that prenatal exposures resulting in maternal BLL values above 15 µg/dL are associated with reduced birth weight (Dietrich et al., 1987) or increased risk of preterm delivery (Dietrich et al., 1986; Fahim et al., 1976). Spontaneous abortion has not been reported to be associated with maternal BLL values below 30 µg/dL, and the weight of evidence suggests that lead does not cause congenital anomalies (Ernhart, 1992). It appears that the lowest maternal BLL value (i.e., approximately 15 µg/dL) that may be associated with effects in the fetus (e.g., reduced birth weight) are above those (2.1 µg/dL) found in the vast majority of all adults (MMWR, 1997). However, because pregnancy can cause mobilization of lead from bones, women either with a history of elevated BLL values or with known elevated exposures should be evaluated and advised if pregnancy is considered.

In males, many studies have been conducted pertaining to the possible reproductive effects of lead, and most have focused on the highly variable yet important parameters of semen quality and sperm production. Aberrant sperm morphology, decreased sperm count and decreased sperm density have all been demonstrated in heavily exposed individuals (Lerda, 1992; Hu et al., 1992; Assennato et al., 1987), although it is difficult to determine the precise levels of exposure associated with such effects. In general, however, BLL values less than approximately 50 µg/dL appear to have little if any impact on semen quality (Goyer, 1996; Tuohima and Wickmann, 1985; Wildt et al., 1983). The collective data suggest that attributable effects on semen quality are most pronounced when BLL values are consistently elevated to 50–60 µg/dL or higher (Tuohimaa and Wickmann, 1985), a level rarely seen today in the general population. Aside from possible effects on sperm quality at high exposures, there are no known teratogenic effects (contributed by paternal exposure) or clinical effects on male fertility except at very high exposure levels (Lerda, 1992; Coste et al., 1991; Wildt et al., 1983).

Cancer

The human epidemiological data on the carcinogenicity of lead is considered inconclusive. The evaluation of the carcinogenic risk from lead exposure has been based primarily on observations from epidemio-

logical studies, experimental animal studies, and short-term tests. The International Agency for Research on Cancer (IARC) has classified lead and inorganic lead compounds as possibly carcinogenic to humans, citing inadequate evidence for carcinogenicity in humans but sufficient evidence for carcinogenicity in experimental animals. The U.S. Environmental Protection Agency (EPA) classifies lead similarly, based on the same interpretation of the weight of experimental evidence.

Experimental studies on laboratory rodents have investigated the carcinogenic potential of several lead compounds following long-term (typically lifetime) administration of high dose levels (EPA, 1989). In these studies, the kidney of the male rat has been the prominent target organ, giving rise to both benign and malignant tumors; the development of renal adenocarcinoma (cancer) is dependent on both the length and severity of lead exposure. Prevailing hypotheses for kidney-related carcinogenicity have thus tended to focus upon the susceptibility of male rats to nephropathy (kidney damage) (Goyer, 1992).

Epidemiological studies of lead-exposed individuals have found little, if any, relationship between occupational exposure and the onset of cancer. Studies have been inconclusive and confounded by coexposures to other carcinogenic metals (e.g., arsenic, chromium) and have often failed to control for smoking among those exposed (Goyer, 1992; Cooper et al., 1985; McMichael and Johnson, 1982; Selevan et al., 1985; Gerhardsson et al., 1986; Dingwall-Fordyce and Lane, 1963). Additionally, sufficient data on BLL values and actual human exposure in such studies is frequently lacking. The collective weight of evidence does not support an association between low-level lead exposure and carcinogenesis in humans.

Other Effects

The Kidney
The adverse effects of lead overexposure on the kidney have been well documented (Goyer, 1971). These changes may progress to generalized kidney disease, which is characterized by disruption of function of the tubular structures. Chronic and excessive lead exposure may result in end-stage renal disease (Weeden, 1982), though it is important to note that kidney effects require relatively high and persistent exposure to lead (EPA, 1986). Studies have shown that BLL values in the range of 40–80 µg/dL are associated with biological changes in the kidney that are largely reversible (Goyer and Rhyne, 1973). For reference purposes, the vast majority of the population have BLLs well below

these levels (mean BLL of 3.4 μg/dL; MMWR, 1997). Some studies involving lead-exposed workers suggest a threshold value of 60 μg/dL for the prevention of kidney effects in adult males (Buchet et al., 1980; Goyer et al., 1989). Other studies (Cardenas et al., 1993; Gehardsson et al., 1992) have reported subtle effects on renal biomarkers below this threshold, although the physiological and toxicological significance of these changes, if any, has not been demonstrated.

Hearing

While less attention has focused on lead and potential effects on sensory functions, there have been some indications of auditory system processing deficits in lead-exposed children (Otto and Fox, 1993). Otto et al. (1985) reported an increased latency of brainstem auditory evoked potential (BAEP—a measure of nerve conduction) in school children with a history of high lead exposure (BLL values ranging from 60 to 90 μg/dL) in early childhood. Other studies have associated deficits in hearing with a relatively low (i.e., 10 μg/dL) BLL, with no apparent lower threshold (Schwarz and Otto, 1987, 1991). Osman et al. (1999) reported hearing test results in children indicating that auditory function is impaired at BLL values ranging from 1.9 to 28.1 μg/dL. Given the presence of some lead, albeit at trace levels, in the blood of most humans, it will be difficult to discern if a true threshold exists, and whether confounding variables contributing to hearing loss can be controlled for adequately. In addition, interindividual differences in hearing ability must be considered when assessing potential effects of lead on hearing.

Hematological

The effects of lead overexposure on heme (an iron-containing compound involved in oxygen transport by hemoglobin) synthesis have been thoroughly investigated, and there is a consensus that adverse effects on hemoglobin are associated with BLL values of 50 μg/dL in adults and 80 μg/dL in children (Goyer, 1996). Among the earliest toxicological effects of increased lead accumulation on the heme synthesis pathway is a reduction in hemoglobin production, a phenomenon that has not been demonstrated conclusively below approximately 40 μg/dL. Frank anemia can occur as BLL values approach 80 μg/dL, which results from both shortened red cell life span and impairment of heme synthesis. Lead affects several hematological enzymes, as shown in Table 1, and changes in some of these enzymes correlate closely with BLL values and may serve as early biomarkers of lead exposure (Goyer, 1996).

Cardiovascular

For more than two decades, the relationship between lead and blood pressure has been investigated in the general population as well as in occupational and experimental animal studies. Several reviews have concluded that there is only a weak association between BLL and elevated blood pressure for those with BLL values below 45 µg/dL (Hertz-Picciotto and Croft, 1993; Staessen et al., 1995; Nowack et al., 1992; IPCS/WHO, 1995). This association has been inconsistent across studies because of potential confounders and the inability to establish a clear dose-response relationship (the demonstration that a change in exposure is directly related to a change in response).

Meta-analyses of the literature on this potential effect of lead indicate that there may be a weak positive association. Although plausible mechanisms have been suggested for lead-related blood pressure effects based on animal studies, it is difficult to determine whether extrapolation of the animal data to humans is appropriate. Given the weak association, it is improbable that significant excesses of cardiovascular morbidity and mortality occur in the general population as a result of low-level lead exposure. 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" (IPCS/WHO, 1995).

Vitamin D Metabolism

Bone is a major organ for lead deposition and accumulation, and skeletal lead has been used as a measure of cumulative lead exposure (Pounds et al., 1991). It has been suggested that lead might affect bone integrity and function by altering growth and stature, and by perturbing vitamin D metabolism. Various investigators have reported associations between blood lead and decreasing levels of vitamin D metabolites at BLL values ranging from 12 to 120 µg/dL (Rosen et al., 1980; Mahaffey et al., 1982). No threshold for this effect has been conclusively demonstrated. Another study reported 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 (Koo et al., 1991).

IV. INTERPRETATION OF BLOOD LEAD LEVELS: WHAT IS SAFE? WHAT IS HARMFUL?

There remains a healthy debate as to whether a threshold or no-effect level exists for lead-induced effects, particularly those associated with effects on intelligence and neurobehavioral and developmental endpoints. Whether a threshold exists for any particular endpoint depends on the sensitivity of the test measure and on an accurate distinction between what constitutes a biologically relevant toxicologic effect and what constitutes an effect that signifies only exposure (i.e., an effect that is not discernibly adverse).

A. CDC Recommendations for Intervention and Interpretation of BLL Values

Over the years, the CDC, which has taken a particularly active role in lead education and elevated blood lead level prevention, has lowered its recommended action level, the level at which some intervention is advised (Figure 1). Despite widespread public perception that lead poisoning "occurs" at BLLs as low as 10 μg/dL, the CDC recommends no clinical management for individuals until BLL values reach the 20–44 μg/dL level (Table 2). This recommendation is generally consistent with the lowest observed effect levels for various toxicological effects in humans (Table 1). Since 1970, this level has been decreased from 55 μg/dL to the current 10 μg/dL (Figure 1), a stepwise decrease that has mirrored both our increased ability to detect various changes and effects (not necessarily adverse or toxicologically important) at smaller and smaller concentrations. It is striking to note that during the decades of the 1970s and 1980s, nearly 9 out of every 10 American children under the age of 5 had BLL values exceeding 10 μg/dL (Pirkle et al., 1994); by today's standard, they would be considered "lead-poisoned." Presently, however, less than 5 percent of American children aged 1–5 have BLL values above 10 μg/dL (CDC, 1997). It will be an interesting as well as necessary sociobehavioral observation to note the sequelae of those "lead-poisoned" children of the '70s and '80s as to whether any overall health impact will become manifest. Presently, there is no widespread, discernible adverse effect at the population level. Some of the discrepancy concerning ever decreasing recommended action levels despite an absence of population-wide discernible effects from low-level lead exposure can be attributed to the

Figure 1. **Blood Lead Levels Considered Elevated by the Centers for Disease Control and Prevention (CDC) and Public Health Service**
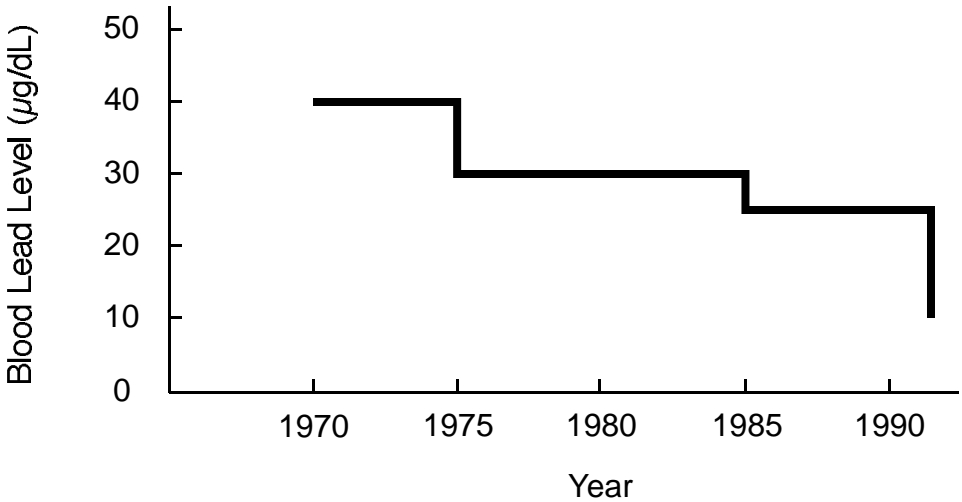
Source: CDC (1991) Preventing Lead Poisoning in Young Children. U.S. Department of Health and Human Services.

inherent differences between personal and public health risk (see below).

B. A Distinction Between Personal and Public Health Risk

A critical issue in all environmental regulatory matters, including lead issues, is the distinction between personal and public risk. When a parent is told by her pediatrician that her child's BLL is 12 µg/dL, she is understandably frightened, as the popular impression is that the CDC and other regulatory bodies have declared 10 µg/dL the bright line that denotes "lead poisoning." This impression is not consistent with either CDC guidance or toxicological interpretation. Thirty years ago, most of us who lived in cities had lead levels well above 10 µg/dL, and as noted earlier, most average childhood lead levels were higher still (Pirkle et al., 1994; Cosgrove et al., 1989). The extensive epidemiological literature finds few lead effects at BLL values less than 30 µg/dL and even at that level the effects are considered minor. Why then is there such a pervasive public perception that a level of 10 µg/dL constitutes a known effect level, one associated with lead poisoning?

Public health agencies are concerned about optimal protection for

hundreds of millions of people. They therefore set levels low enough to minimize the possibility of harm to populations at risk. This is what we might call a "public risk." A personal risk, by contrast, asks: "What does this level mean to me or my child?" It is the question posed by the anxious mother to her child's physician. Physicians, who are on the front line dealing with patients' worries, often find themselves troubled by this question. They may know that a BLL of 12 µg/dL poses little or no risk to a specific child, but they rarely have sufficient knowledge of the regulatory process to understand the basis for the 10 µg/dL criterion or

Table 2. **Comprehensive Follow-up Services According to Diagnostic* 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 followup 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 (i.e., 2 venous BLLs in this range at least 3 months apart) or worsen, proceed according to actions for BLLs 20–44.
20–44	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, clinical management, environmental investigation, and lead hazard control.
> 70	Hospitalize child and begin medical treatment immediately. Begin coordination of care, 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, Screening Young Children for Lead Poisoning, 1997a.

that this represents the lowest level at which some initial followup is recommended. They too may believe that the government has determined that this represents the bright line between safe and dangerous. It is important, therefore, that the public understand the meaning of various BLL values. They are regulatory (public risk) numbers, designed for optimal protection, and they have little direct meaning for an individual child's personal risk.

Although unrelated to lead, a well-known example of the importance of proper interpretation and understanding of such analytical numbers occurred when 15 parts per billion of benzene was found in Perrier water, while the regulatory limit is 5 parts per billion. Worried Perrier drinkers, exposed to three times the "acceptable" amount, naturally assumed they were at risk. They were not. The public risk level is set thousands of times lower than the personal risk level. The EPA attempted, through television and other media, to reassure those exposed and to explain this distinction between a public health risk and a personal risk. But it proved a difficult task for them. The public wants regulatory standards to reflect safety versus danger, and is troubled to learn that this is not necessarily the case.

C. "Lead Poisoning"

One of the more unfortunate developments concerning lead as a public health issue is the widespread and scientifically inaccurate use of the term "lead poisoning." This term is often indiscriminately used to describe any asymptomatic child (or adult) with a BLL that exceeds 10 μg/dL. The term "poison," in toxicology, is typically reserved for highly toxic substances that exert acute and potentially fatal effects at very low dose levels. Over the years, the expression "lead poisoning" has been systematically applied to lower and lower BLLs, a practice that is inconsistent with the justifiably termed toxic effects at high BLLs. From a toxicological perspective, poisoning does not occur at low BLLs (e.g., 10–20 μg/dL), as has been suggested from the public interpretation of CDC's recommended action at this level (Table 2).

The misuse of the term "lead poisoning" when referring to BLL values that are without defined health consequences serves to confuse people, and it may cause anxiety for parents who are unfamiliar with the significance of various blood lead concentrations. Increased parental and consumer understanding of what various BLL values signify (Table 1) and at what levels CDC recommends direct clinical intervention

(Table 2) would be helpful in alleviating fear and undue concern.

Another consequence is that many states or health agencies now consider 10 µg/dL to be a frank effect level, which it is not, and have passed legislation based on that interpretation. Avoidance of the term "lead poisoning" when referring to BLL values in the range of 10–20 µg/dL is essential, as effects in that range clearly do not indicate clinical poisoning. There are distinct differences in the effects associated with BLL values of 10 vs. 80 µg/dL and in the future it would be appropriate for federal and state agencies to use more accurate descriptors when discussing various BLL values.

From the public health perspective, an important ramification of the continual reduction of the lead action level is that attention is mistakenly focused on those children with BLL that are not clearly associated with clinically or toxicologically relevant effects. This misplaced focus comes at the expense of those individuals within the population truly in need of intervention—socioeconomically disadvantaged children or young adults with BLL values clearly associated with adverse effects. If we continue to place the greatest scrutiny on those children with BLL in the range of 5–15 µg/dL, we will be devoting research dollars and energies to a population for whom there are no obvious clinical effects and potentially ignoring those easily identified individuals with much higher BLL levels. It is doubtful there is useful intervention for the former, while clearly we can help the latter.

V. TRENDS IN POPULATION BLOOD LEAD LEVELS

A. Current Population Blood Lead levels

Screening of children in major U.S. cities in the early 1960s revealed that 20–45 percent of children evaluated had elevated BLLs of about 40 µg/dL (Cosgrove et al., 1989). In the 1970s regulatory and public health efforts at the national level were undertaken to reduce lead exposure. These included actions to limit the use of lead in paint, gasoline, and soldered cans (ATSDR, 1988). The second National Health and Nutrition Examination Survey (NHANES II, 1976 to 1980) conducted by the CDC established the first set of baseline BLL for the U.S. population and demonstrated the pervasiveness of lead exposure across race, urban and rural residence, and income level (Mahaffey et al., 1982). A 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 per-

sons evaluated) and the Hispanic Health and Nutrition Examination Survey (HHANES), (1982 to 1984; 5682 persons evaluated) found that the mean BLL of persons aged 1–74 had dropped 78 percent, from 12.8 to 2.8 μg/dL, over four years (CDC, 1991; Figures 2a and 2b). Importantly, the mean BLL of children aged 1–5 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 over this

Figure 2a. **Blood Lead Levels for Persons Aged 1 to 74 Years: United States, Second National Health and Nutrition Examination Survey (1976 to 1980, top) and Phase 1 of the Third National Health and Nutrition Examination Survey (1988 to 1991, bottom)**
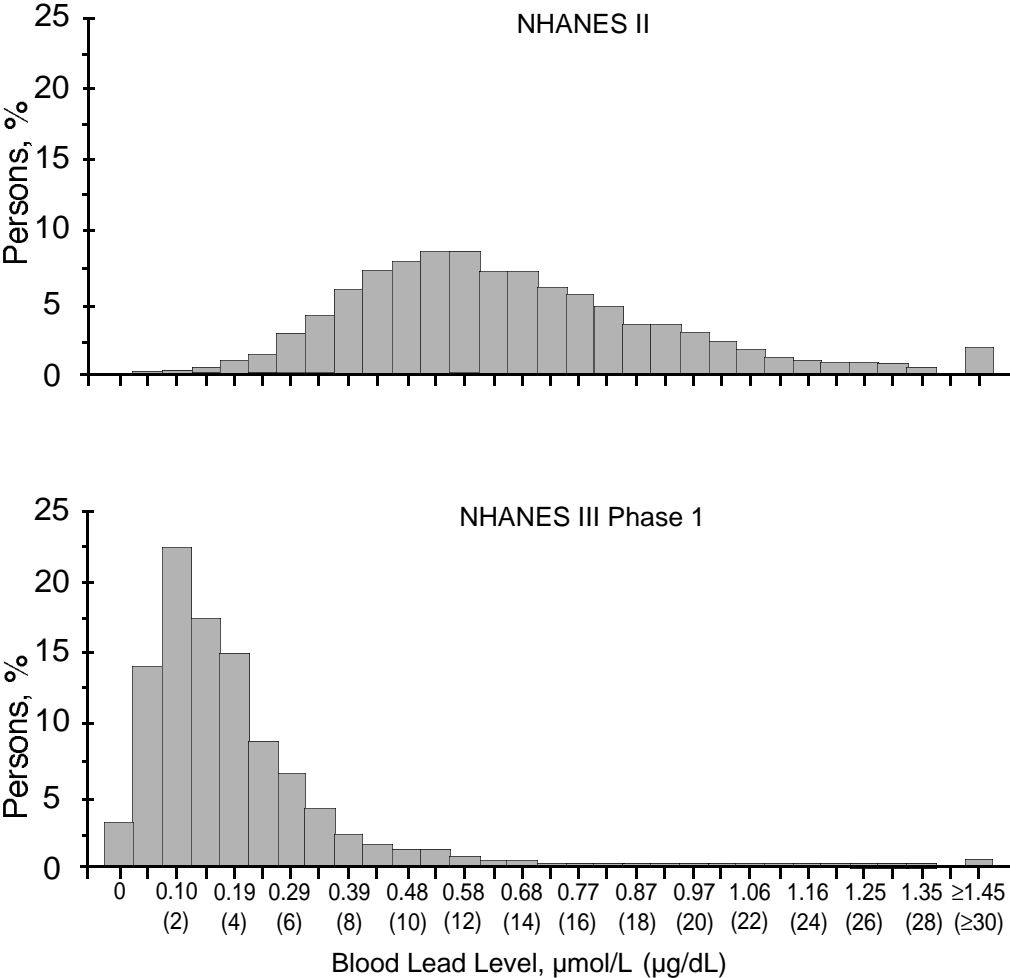
Figure 2b. **Blood Lead Levels for Persons Aged 1 to 5 Years: United States, Second National Health and Nutrition Examination Survey (1976 to 1980, top) and Phase 1 of the Third National Health and Nutrition Examination Survey (1988 to 1991, bottom)**

NHANES II

NHANES III Phase 1

Blood Lead Level, μmol/L (μg/dL)
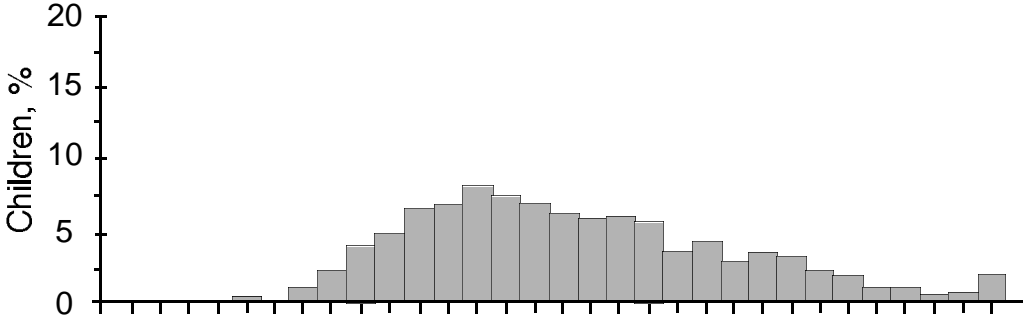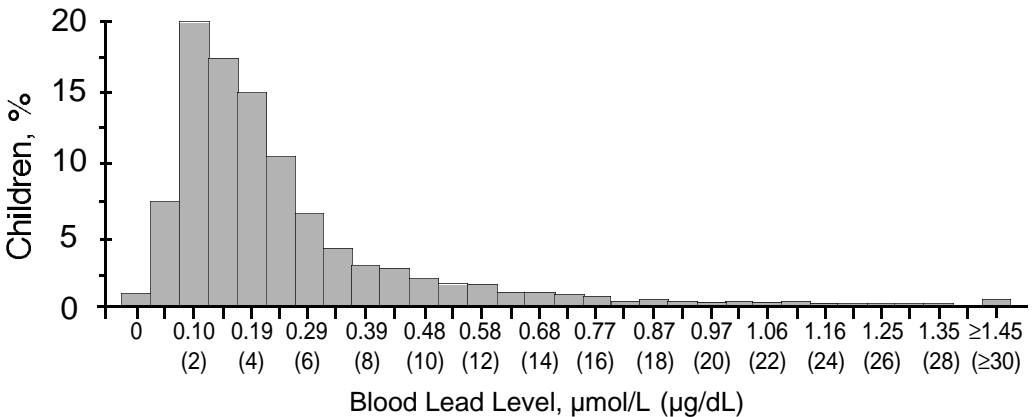
time. The prevalence of BLLs of 10 μg/dL or greater for children aged 1–5 years declined from 85 percent to 5.5 percent for non-Hispanic white children and from 97.7 percent to 20.6 percent for non-Hispanic black children living in older homes.

The third National Health and Nutrition Examination Survey (NHANES III) specifically addressed BLLs in the U.S. and their correlation with sociodemographic factors. Notably, BLL values were consis-

tently higher for younger children than for older children, for older adults than for younger adults, for males than females, for blacks than whites, and for central-city residents than for non–central-city residents. Other correlates of or risk factors for higher BLL included low income, low educational achievement, and residence in the Northeastern region of the U.S. (Brody et al., 1994). In children aged 1–5 years, the prevalence of BLL values above 10 µg/dL was higher among those who were non-Hispanic blacks or Mexican-Americans, from lower-income families living in metropolitan areas with a population > 1 million, or living in older housing (MMWR, 1997).

The most recent published national data available, phase II of NHANES III, as reported by the CDC, continue to indicate declining BLL in the U.S. population (CDC, 1997b). This update of BLL values confirmed that BLL among children aged 1–5 years were more likely to be elevated among those who were poor, of non-Hispanic black race, and living in large metropolitan areas or in older housing. During the 1991–94 survey period, the mean BLL of the U.S. population older than 1 year was 2.3 µg/dL, down from 2.8 µg/dL reported for the period 1988–1991. These data represent the latest currently available on a national level (C. French, personal communication, CDC, 2000). Although national data beyond 1994 are not available, a recent study (Bowers et al., 2000) evaluated BLL levels for children under six years of age using 12 longitudinal data sets from 11 states and one city. Geometric mean BLL values of these children declined between 4 and 16 percent per year (since 1994) in ten of the 12 data sets. Based on the quality of the data sets and the reproducibility of the decline rates, the investigators reported the average decline rate for BLL values since 1994 to be 4–7 percent per year. This is comparable to the decline rate observed in earlier years of 6–7 percent per year based on the NHANES II and III surveys. Using these decline rates, this analysis suggests that the mean BLL for U.S. children today is between approximately 2.0 and 2.3 µg/dL (Bowers et al., 2000).

It remains encouraging that the average BLL of the most susceptible group of individuals, i.e., children, continue to decline, with the most recent national data reporting that among those aged 1–5 years, approximately 4.4 percent (890,000) had BLL in excess of 10 µg/dL, down from 8.9 percent (1.7 million) of those surveyed during the 1988–91 survey period. This is quite dramatic in that in only a 6-year time span, trend data such as these show a 50-percent decrease. For children with BLL greater than 20 µg/dL (levels at which children are at greater risk of effects than at 10 µg/dL), the percentage has declined

from 24.7 percent in 1976–1980 to 1.1 percent in 1988–1991, to 0.4 percent in 1991–1994 (Table 3; CDC, 1997b).

Collectively, the NHANES survey data clearly demonstrate a very encouraging decline in BLL for the U.S. population as a whole. The data provide convincing evidence that BLL for the vast majority of those assessed are below levels considered important toxicologically, and even below the CDC benchmark for educational intervention (10 µg/dL). The CDC (CDC, 1997) and others (Arnetz and Nichlolich, 1990) have shown that reductions in the major sources of lead exposure, including lead content of food, the removal of lead from paint, soldered cans, and plumbing systems, and the removal of more than 99 percent of the lead from gasoline, have played a significant role in reducing human lead exposure and hence BLLs. Given the source reductions and continuing declines in dietary intake of lead, decrements in BLL values across the U.S. population should continue to levels that essentially constitute background exposure.

B. Subpopulations and Areas of Concern

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, housing environment, parental occupation, low income level, or suboptimal nutrition) continue to be associated with higher BLLs. Because of the overall decline in BLL among the U.S. population, the CDC has shifted emphasis in screening, from universal to targeted screening of higher risk individuals, particularly young children. This 1997 recommendation differs substantially from the 1991 pronouncement that recommended virtual universal screening of children aged 12–72 months. The CDC notes that "many children, especially those living in older housing or who are poor, need screening and, if necessary, appropriate interventions to lower their BLL. 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" (CDC, 1997a). Specifically, the CDC's current recommendation is for statewide targeted screening (except where a state plan does not exist, in which case statewide screening is advised) based on an assessment of local data and an inclusive planning process.

It will become increasingly important, both from a public health perspective and from a resource management viewpoint, to continue to identify accurately those individuals and population sectors that have higher exposures to lead, which places them at an elevated risk of lead

Table 3. 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 I 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

Source: Adapted from Brody et al., 1994; Pirkle et al., 1994

toxicity. In this regard, individuals with excessive exposure to lead, regardless of the source (e.g., dietary intake, soils or dusts, or lead-based paint), should be targeted for screening and should be the focus of lead education and intervention activities in the years ahead.

VI. HUMAN EXPOSURE

Human exposure to lead may occur through various environmental pathways (air, water, soil, dust), from ingestion of food and water containing trace amounts of lead, and/or from the use of lead-containing consumer products. Airborne lead is an important source of contamination; when this lead is deposited onto soil and dusts, it may be ingested by young children (Charney et al., 1983; Bellinger et al., 1986; Bornschein et al., 1986). Background exposures can result from indirect sources (e.g., airborne lead deposited on soil can be taken up by food plants and then ingested). Lead used in manufacturing and occupational settings is an important source for some exposed individuals. Lead smelters and other industrial plants utilizing lead-containing coal may

also be important point sources for those residing nearby.

One way to investigate sources of lead exposure is to use isotopic ratio analysis (IRA), a method utilizing the fact that lead has varying amounts of radioactive isotopes, the precise amount depending upon the ore's geological age. Thus, by determining the ratio of radioactive isotopes to each other and to nonradioactive lead in a particular sample, investigators can determine its likely source (Manton et al., 2000; Jaeger et al., 1998).

A. Primary Routes of Human Exposure

While humans of any age can be exposed to lead by inhalation or dermal contact, ingestion of dietary lead remains a large contributor to low-level daily exposure. For most exposed children, ingestion—of food, contaminated soil or dust, flaking paint, and/or water—is the principal route of exposure. Inhalation is generally a key pathway only in occupational settings; similarly dermal exposure and absorption of inorganic lead compounds are not appreciable in terms of overall intake. Lead that is deposited from the atmosphere onto soils may then be ingested directly, or become entrained in dusts, particularly house dust, which then may become an important source of oral exposure, particularly for children (Paustenbach et al., 1997; EPA, 1986; Bornschein et al., 1986). Young children are more prone to ingestion of lead-containing soil and dust because of frequent hand-to-mouth activity and/or exhibition of pica (the craving for and ingestion of nonfood substances) behavior.

There is a scientific and medical consensus that for children with BLL > 20 µg/dL (e.g., those residing in homes, particularly in the eastern U.S., with flaking or peeling lead-based paint), ingestion of lead-based paint remains an important direct route of exposure. Paint containing up to 50 percent lead was in widespread use in the U.S. through the 1940s, although in subsequent years (up to the 1970s), binder paints containing approximately 5 percent lead were more common. In 1978, the Consumer Product Safety Commission (CPSC) banned the manufacture of paint containing more than 0.06 percent lead by weight for use on interior and exterior residential surfaces, toys, and furniture (EPA, 1990). 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 (EPA, 1990).

For adults, key pathways of exposure largely remain inhalation of lead-containing dusts and fumes in occupational settings, particularly

during mining, smelting, and refining operations or during battery manufacturing and reclamation operations (Gittleman et al., 1994). Exposure to lead may also occur through eating or smoking in a lead-contaminated environment (Sittig, 1991; Baxter et al., 1985). Drinking water can also serve as a source of lead exposure because of leaching from lead-containing pipes and fixtures, although with the eventual replacement of older lead-soldered residential plumbing, this source will become less important.

Regarding dietary intake, lead occurs in and on food, both naturally and as a result of human activity (e.g., exposure from glazes on low-fired pottery). Lead may also be introduced into food inadvertently during harvesting, transportation, processing, packaging, or preparation. Sources of 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 discontinuing the soldered cans formerly used in infant formula packaging. Since then, can manufacturers have stopped producing soldered cans for the food industry.

Dietary ingestion of lead continues to decline due to (a) reduction in ambient particulate fallout to crops as a result of the elimination of leaded gasoline, (b) phaseout of lead-soldered cans by manufacturers, and (c) decline in lead levels in water used in food processing and preparation (Figure 3). Historical estimates of dietary lead exposure for adults in the U.S. have included 95 µg/day (Podrebarac, 1984) and 82 µg/day (Gartrell et al., 1986), among others. Data from other studies (Total Diet Study—USFDA) indicated 35–37 percent reductions in dietary lead intake for young children between 1982 and 1986 compared with previous levels (ATSDR, 1988). Similarly, estimates made using the USEPA Multiple Source Food Model suggested a decline in dietary lead intake for a 2-year-old from approximately 45–50 µg/day in 1978 to 13 µg/day in 1985 (Flegel et al., 1988). More recent estimates from the USFDA Total Diet Study (FDA, 2000) indicated that since 1982–84, daily intake of lead from food has dropped 96 percent in 2–5-year-olds (from 30 to 1.3 µg/day) and almost 93 percent in adults (from 38 to 2.5 µg/day).

B. Other Potential Sources of Exposure

There are several minor sources of lead exposure, relevant only to small sectors of the population, from some rather unique and specialized types of products and activities. Consumers can be

exposed to lead through a variety of goods, including unglazed ceramic dishes and pottery, pewter dishes or cups, lead crystal glassware and decanters, and materials used in hobbies, crafts, or leisure activities. These materials contain lead that may be leachable and capable of entering the human body through some exposure pathway. These products include lead-based ammunition and fishing weights, lead-acid batteries, cigarettes, certain printing pigments and inks. Some hobbies with potentially elevated risk of lead exposure include artwork in which lead-containing paints or colored pencils are used, glazed pottery making, stained glass production, and lead soldering (e.g., home electronics).

VII. INCONSEQUENTIAL TRACE SOURCES OF LEAD EXPOSURE

Advances in our analytical ability to detect smaller and smaller levels of trace constituents or pollutants in our environment have directed increasing attention at lead in consumer products and applications. This ability to detect ever smaller amounts of contaminants analytically has spawned a belief, among some, that any detectable concentration of

Figure 3. **U.S. Food Lead Trends, 1935–1990**
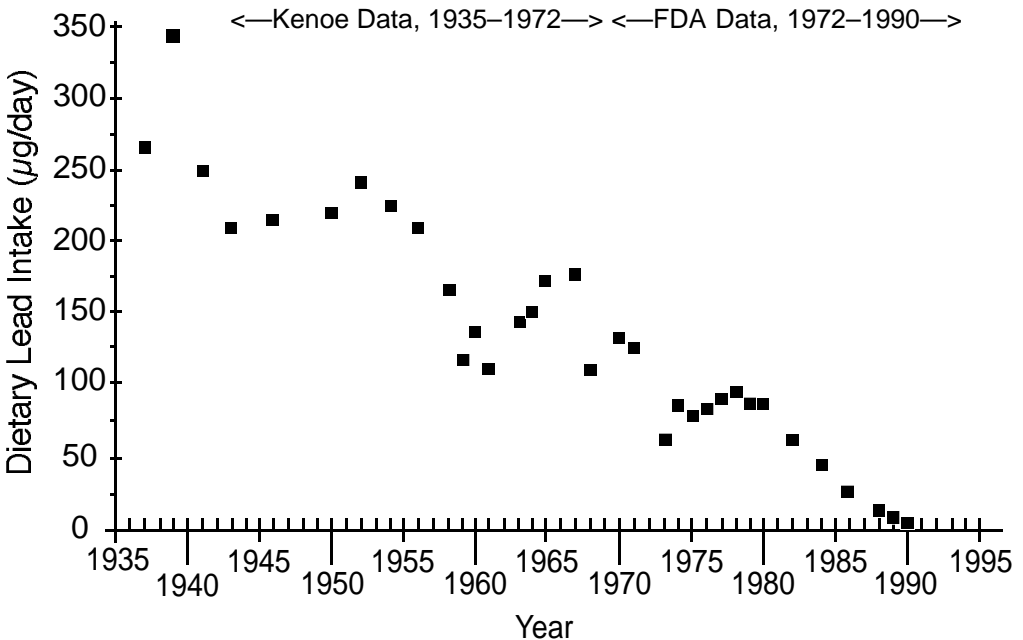
Source: Kenoe Data—Gross, 1981, FDAData—Bolger et al., 1991

a chemical found in the environment constitutes a health risk. Clearly lead can be toxic, but we must not forget that, as with all compounds, the level of exposure and the bioavailability of lead in the body are critical determinants in predicting adverse effects. Several product-related issues involving lead—in miniblinds, hair dyes, calcium supplements, and candles—illustrate the situation we encounter with increasing frequency, one in which scientific reason and principles of toxicology have been summarily ignored or discounted. These "health scares" undoubtedly have left many consumers confused and questioning their potential health risk from using such products.

A. Miniblinds

Windows became the object of renewed attention in June 1996 when the CPSC announced that certain imported vinyl miniblinds could present a "lead poisoning" hazard to children (CPSC, 1996). Following additional 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 officials in Arizona and North Carolina identified vinyl blinds as the cause of "lead poisoning" among children living in mobile homes where no lead-based paint was present. But no information was provided about other factors that might have influenced exposure, including the time spent outdoors by the children, sources of drinking water, and the degree of contact the children had with window blinds (Brown, 1996).

In response to the CPSC report, a trade association group (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 risk as the exposure potential is negligible under foreseeable conditions of use. If deterioration of lead-containing miniblinds 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 present a realistic health risk. Moreover, since the report and "scare" in 1996, there have been no known documented case

reports of lead in miniblinds causing health effects in humans and this issue has remained dormant ever since.

B. Cosmetics

Hair dyes that contain small amounts of lead have also raised concern among some consumers and public interest groups. The fear is that children will ingest lead by putting their hands in their mouths after touching a parent's hair or contacting household surfaces containing hair-dye residues. Some 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" (Cohen and Roe, 1991). This viewpoint has a sound toxicological basis, as the dermal route of exposure is generally considered insignificant because of the low dermal bioavailability of lead acetate in humans. Cohen and Roe (1991) reported that human exposure to lead acetate from a hair coloring agent only accounted for about 0.5 percent of the total absorption of lead from the environment. Even if such residual lead were ingested by children, this source represents a very minor exposure pathway for children as a group, particularly in comparison with the potentially greater exposures from peeling paint, dust, and soil.

Some 20 years ago, the U.S. 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 (FDA, 1980, 1981). 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 with lead in miniblinds, lead in hair dyes appears to have resulted in no apparent adverse effects in children or adults.

C. Dietary Supplements

The issue of lead in over-the-counter calcium supplements is another recent example of public concern over trace amounts of lead that might be ingested. Over the years, the FDA has analyzed various foods for their lead content, while the Council for Responsible Nutrition (CRN) has conducted similar tests on calcium-containing products and supplements (Table 4). The FDA studies have found trace amounts of lead to be present in virtually all foods analyzed; this is not unexpected given its ubiquity as a natural element of this earth. What is more important, the overall daily dietary exposure to lead is not toxicological-

ly 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 (CRN, 1997a). And the nutritional value of these foods outweighs any detrimental effect associated with exposure to trace levels of lead in them. A similar conclusion (in terms of risk/benefit analysis) has been reached by government agencies when concerns over trace amounts of pesticide residues on plants and trace dioxin levels detected in breast milk were issues of concern to the public. A study published in *The Journal of the American Medical Association* followed nearly 2,500 pregnant women who took 1,500–2,000 mg of supplemental calcium daily (Bucher et al., 1996). The researchers did not report any significant adverse health effects associated with increased calcium supplementation.

A risk/benefit analysis related to calcium supplements helps to put the issue of lead exposure into proper context. The available studies on calcium supplementation unequivocally show that the health benefits of calcium, an essential mineral, clearly outweigh any hypothetical 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 or other nutritional supplements, while satisfying the fearful concerns regarding trace-level exposures, would not result in improvements to individual or general public health (CRN, 1997b).

D. Candles

Most recently, certain candles that reportedly contain trace amounts of lead in the metal-core wicks have come under attack from the Public Citizen Health Research Group, a consumer organization, based on speculation that lead-wicked candles could produce airborne lead levels that are higher than permitted by the U.S. EPA. Again, this is an example of focusing on the hazard because of its low-level presence at the expense of conducting a risk assessment to determine if any health risk is present. To determine that, one would need: (a) to assess the amount of lead in a metal-core wick; (b) to determine if lead is released through candle burning and, if so, what the ambient concentrations are; and (c) to determine through personal monitoring what the actual exposure and absorption are. Additionally, the relative contribution of lead-containing candles should be assessed rela-

Table 4. **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.

tive to all other daily trace sources to put this source into perspective. Given the rare occurrence of low-level lead-containing candles in commerce (a random survey conducted by the group determined that about three percent of candles [9 of 285] contained some lead), and the other factors associated with assessment of health risk determined that above, there appears to be no scientific or clinical basis for a public health concern related to lead in candles.

A. What Is Lead Abatement and When Is It Needed?

One of the principal ways to decrease the risk from any hazard is to limit or reduce one's exposure to that substance. While there are certain means and strategies by which individuals can limit their exposure to certain sources of lead, there are also natural background levels that will remain in our environment and about which we can do little. However, for some people living in older housing that contains lead-based paint in need of repair, there are means by which human exposure can be minimized.

Removal of such lead in homes, known more commonly as lead abatement, has become a significant industry over the years, even to the point where some states have mandated lead removal at all costs. While removing lead-based paint is well-intentioned, there are many situations under which abatement is not advised. As with asbestos, intact and well maintained lead-based paint should not, in most cases, be abated. Painted surfaces only become a hazard when they have been allowed to deteriorate and when flaking, peeling, or dusting is evident. 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 during renovation, repair, or abatement projects.

We have learned from experience with other well-known potential environmental (e.g., PCBs in sediments) and human health (e.g., asbestos) concerns, that in many situations (intact asbestos in pipe wrappings) remediation or abatement activities are ineffective and may even increase health risk by dispersing the substance and increasing the potential for exposure. There is often no need to renovate, strip, or raze a home merely because it contains lead-based paint. If the paint is intact and not peeling and if children residing in the home do not have behaviors involving ingestion of wall paint, costly and disruptive remedial activities may not only be contraindicated—they may increase the health risk to residents. Abatement should be considered if (a) lead exposure from existing paint has been confirmed, (b) there is clear evidence of cracking or peeling paint, (c) a health risk to children is probable, and (d) the risk can be reduced through appropriate remedial techniques.

During the period 1992–1994, the New York State Department of Health assessed lead exposure among children resulting from renova-

tion and remodeling of homes containing lead-based paint (CDC, 1997c). The study identified 320 children in New York State (excluding New York City) with BLL values greater than 20 µg/dL, levels consid-ered attributable to residential renovation and remodeling. In most cases (86 percent), the paint removal was not performed by a professional contractor, who would presumably be more aware of the proper tech-niques and protective measures employed during lead-paint removal. The study concluded that home renovation and remodeling in which lead-based paint is altered or disturbed, especially when done without proper expertise, actually continues to be an important source of lead exposure in children.

Instead of lead-free homes, a more prudent and cost-effective approach is that of lead-safe homes, particularly in terms of remediation efforts in homes and public housing. Just because lead is present does not mean that it poses a health risk. Exposure is the operative word. Maintaining intact lead-based paint in a safe condition is prudent until deteriorating conditions or future renovation plans necessitate either removal or other intensive abatement measures.

B. A Successful State Prototype Program

The Baltimore, Maryland Jobs and Energy Project is a successful program that incorporates both lead abatement and public education (Livingston, 1994). Because the majority of lead-based paint haz-ards are found in single-family units, the Baltimore Project was designed to provide affordable lead-based paint and dust hazard identifi-cation, 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-com-plex–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-based paint deterioration, the size of the surfaces to be treated, the rate of lead dust generation, and the BLL of the resident children.

Appropriately trained local volunteers and contractors conduct the actual abatement work. Abatement is followed by education for resi-dents on the proper maintenance of their abated or partially abated homes. The Baltimore program has been successful because: (1) it has been affordable (as opposed to complete and indiscriminate abatement

of every painted surface in lead-containing homes, as in some states); (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 safely (Livingston, 1994). This successful approach considers and evaluates all information relevant to an assessment of health risk, instead of blindly remediating simply because lead is known to be present.

C. Appropriate and Useful Abatement Techniques

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 reduce temporarily 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. The primary intent is to reduce or remove access to exposure.

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 the removal or covering of lead-contaminated soil.

Complete abatement involves the permanent elimination of all interior and exterior lead-based paint, regardless of the paint condition. This approach has all too often been selected based on fear, not consideration of the relative exposure and risk, has been costly, and has likely increased the health risk in some situations. Complete abatement of lead-contaminated soil would include removal of at least the top six inches of soil, soil cultivation, soil treatment and replacement or paving with concrete or asphalt. Regardless of the approach taken, it is imperative that the approach be tailored to site-specific conditions.

D. Cost-Effectiveness and Health Protection

The effectiveness of a given abatement approach (i.e., interim control or abatement) may be assessed primarily by (a) how well it eliminates or reduces exposure to lead-containing materials (in this discussion, paint), or (b) how well its implementation reduces the BLL of an exposed individual or population, a particular measure that involves some time to determine. In 1994 a group of researchers reviewed 14 studies conducted during the period 1974–1993 (Burgoon et al., 1994) regarding lead-based paint, soil, and dust interim control and abatement techniques. 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.

Another study on pre- and post-abatement BLL of children from deleaded homes also suggested that current abatement techniques may be limited in terms of their effectiveness in reducing BLL (Swindell et al., 1994). This study was a review of the effect of home lead removal on the BLL 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 of or above 25 μg/dL, and particularly in those with BLLs above 30 μg/dL, residential deleading was associated with an 18-percent decrease in BLL 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 conclusions reached were that if home lead abatement is to be effective for children with BLLs below 30 μg/dL, and particularly for those with BLLs below 20 μg/dL, caution must be exercised in order to minimize exposure to lead-containing dust during the removal (Swindell et al., 1994). These results also suggest that lead abatement is most effective in those homes where lead exposure is greater, while lead abatement activity itself temporarily causes some increase in BLL of children with lower initial BLL.

A review of published studies on lead abatement, focusing on BLL, supports the notion that intact and well-maintained lead-based paint to which there is minimal human exposure should not be removed. This empirical evidence 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 (HUD, 1995). Regardless of the technique employed, education for adults regarding identification and management of lead-based paint hazards should follow, along with periodic and proper cleaning and maintenance procedures. Finally, proper nutrition and hygiene for children living in a lead-abated home are essential.

IX. FEDERAL AND STATE PROGRAMS RELATED TO LEAD

Over the past 30 years, various initiatives, standards, and regulations have been established to limit human exposure to lead-containing products and environmental media (air, water, soil). Included among these are the Occupational Safety and Health Administration (OSHA) Lead Standard (OSHA, 1990), the U.S. Department of Housing and Urban Development (HUD) Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing (HUD, 1995), the U.S. Environmental Protection Agency Guidance on Residential Lead-Based Paint, Lead-Contaminated Dust, and Lead-Contaminated Soil (EPA, 1994), OSHA's lead standard for construction (OSHA, 1993) and Title X, the Residential Lead-Based Paint Hazard Reduction Act of 1992 (Title X, 1992), aimed at addressing lead-containing paint in private housing. In addition to these broad-based initiatives, because quantification of exposure and abatement/remediation activities require accurate lead measurements, the American Society for Testing and Materials developed standards related to the identification, monitoring, and remediation of lead hazards (Ashley and McKnight, 1993). Table 5 lists various federal agencies along with their specific areas of responsibility, either for controlling human exposure to lead or educating the public as to the hazards and potential risks associated with lead.

Because lead in homes presented a readily detectable and potentially broad-based target for lead mitigation efforts, federal efforts were implemented to control and regulate lead-based paint in homes—the Lead-Based Poisoning Prevention Act of 1971 and the 1992 Residential Lead-Based Paint Hazard Reduction Act. During the discussion 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 BLL, treating those with elevated BLL, and implementing abate-

ment procedures for lead-based paint removal. This approach had the distinct advantage of early detection of higher-risk children. For example, children concentrated in older inner city housing containing lead-based paint, often in a deteriorated state, are high-risk individuals for lead exposure and could more easily be identified through a targeted health-based screening approach.

The alternative "housing-oriented" approach involved the removal of lead-based paint from public housing, regardless of the paint condition and exposure potential or associated BLL of residents. This housing-based approach eventually replaced the health-based approach in 1992, when the U.S. Congress enacted the Residential Lead-Based Paint Hazard Reduction Act. Title X of this Act established 0.5 percent 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-based paint hazards. Rather than requiring the removal of lead-based paint from all exterior and interior surfaces, the statute drew 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-based paint on inaccessible surfaces). Perhaps most significantly, under this statute lead-containing paint removal was no longer mandated under all circumstances. This represented an important progression in thinking about lead as a hazard and reflected the understanding that lead-based paint does not need to be removed in every situation, particularly if exposure potential is minimal or nonexistent. Similar to the case with asbestos, we have learned (and will discuss further below) how to avoid situations requiring full abatement and removal, without regard to the actual risk that can result in increased BLL from such measures.

A. Federal Programs and Initiatives

Department of Housing and Urban Development (HUD)
In other federal actions, HUD released a report in 1995 entitled "Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing," which devoted considerable attention to the testing recommendations for lead in housing (HUD, 1995). The HUD guidelines also emphasize worker health and safety and the role of OSHA in controlling human exposure to lead in occupational and work settings. 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.

Table 5. **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

Food and Drug Administration (FDA)

The FDA, responsible for establishing permissible lead levels in foods and other consumer products, has controlled human exposure to lead by eliminating lead solder in can manufacture; reducing the use of lead-containing pesticides on fruits and vegetables; and promoting the packaging of baby food 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. By law, lead glazes on most ceramic foodware sold in the U.S. are now formulated, applied, and fired in a manner that prevents lead from leaching from the glaze into food and beverages.

Centers for Disease Control and Prevention (CDC)

The CDC, in its most recent publication on childhood lead screening (CDC, 1997a), stated that "childhood lead poisoning is a major, preventable environmental health problem." Furthermore, the CDC "intends to eliminate childhood lead poisoning and to employ blood lead screening as an important element of this mission." Given our current knowledge about the dramatic declines in BLL values and the statistics on lead in the environment, the contention that childhood lead poisoning is a major environmental health problem does not reflect the current situation of the U.S. population as a whole. Certain sectors of the population or various demographic regions may have higher exposures to lead, but this is the exception, and this situation is no longer widespread in the U.S.

The CDC, as part of its core function through the U.S. Department of Health and Human Services (DHHS) and the Public Health Service (PHS), acts as a central agency for assisting states in the development of their individual lead screening and educational programs. In addition, the CDC has conveyed its own perspective (CDC, 1991) on lead as a health risk in the U.S., and within that perspective has offered its views on (a) the role of pediatric providers, (b) the role of state and local public agencies, (c) blood lead screening, (d) diagnostic evaluation and medical management of children with elevated blood lead levels, and (e) management of lead hazards both for the individual and the community at large.

Almost 10 years ago, the CDC lowered its intervention level from 25 µg/dL to 10 µg/dL based on the view that some adverse health effects occur at this lower level. In 1997, the CDC revised its intervention recommendations and again these are shown in Table 2. The CDC

Table 6. **Child Lead Poisoning Prevention Activities and Associated Policies**	
Activity	Examples of Associated Policies
Primary Prevention	
Evaluation and control of residential lead-based paint hazards	Protective housing codes or statutes
Public lead education	State or area-wide plan calling for community-wide lead education
Professional lead education and training	State certification for lead abatement workers
Anticipatory guidance by child health care providers	State Medicaid policies requiring anticipatory guidance
Identification and control of sources of lead exposure other than lead-based paint	State or area-wide plan to reduce exposures from industry and drinking water
Secondary Prevention	
Childhood blood lead screening	State or area-wide screening plan; state Medicaid policies and contracts calling for screening; protocols and policies for providers and managed-care organizations
Followup care for children with elevated BLLs	Local policies to establish a followup care team; protocols for care coordination and for medical and environmental management; Medicaid policies and contracts calling for followup care
Monitoring (Surveillance)	
Monitoring of children's BLLs	State policy requiring laboratories to report all BLL test results of resident children
Monitoring of targeted (older deteriorating) housing stock, hazard-reduction activities, and lead-safe housing	State certification and licensing procedures for monitoring safety of lead-hazard reduction activities and occurrence of such activities in areas with targeted housing; procedures for tracking lead-safe housing

stressed that primary prevention (e.g., elimination of lead hazards) is an important facet of any overall program. Other prevention activities and associated policies are noted in Table 6.

An important distinction in any surveillance program is the difference between medical intervention and educational intervention. Medical intervention is not recommended until BLLs are well above 10 µg/dL (e.g., at least 20 µg/dL). It is appropriate to determine the source of exposure and to eliminate or reduce such exposures so that BLLs can return to a level considered acceptable.

It is important to note that in 1991, the Public Health Service called for a societywide effort to eliminate childhood lead "poisoning" in 20 years, and thus we are at midpoint, time-wise, in this concerted effort. It will become increasingly necessary for the PHS to define what is meant by lead poisoning for further gains to be made. If the goal is to reduce all childhood BLLs below 10 µg/dL, that is probably achievable and not far from attainment. However, the complete absence or removal of all detectable lead from human blood is impossible, given its trace level natural presence in the environment and in virtually all living systems.

The Environmental Protection Agency (EPA) and the Occupational Safety and Health Administration (OSHA)

The EPA has general oversight and responsibility for monitoring and regulating lead in the environment, specifically its presence in air, water, and soil. EPA has established regulations that restrict the amount of lead in air, in water, and in soil. OSHA is responsible for regulating lead in occupational settings and currently has regulations restricting lead concentrations in air. In addition, if a worker has a BLL that exceeds 40 µg/dL, he or she is to be removed from the worksite. OSHA also maintains a lead-specific standard that covers air monitoring, respiratory protection, medical surveillance and intervention and other aspects related to worker protection from potential effects of lead.

B. State Programs and Initiatives

The CDC published guidance in 1997 with the intent of providing information and direction to those state and local public health officials during their development of lead screening and educational programs. Many of the state programs reflect the guidance of the CDC, in part, because some degree of funding for these programs emanates from the CDC. CDC's basic recommendation to state health officials is

the development of statewide plans for childhood lead screening programs. These should address:

1. Division of the state, if necessary, into areas with different recommendations for screening
2. Screening recommendations for each area
3. Dissemination of screening recommendations for each area
4. Evaluation

In the absence of a state-developed program, the CDC recommends that universal screening for virtually all young children be carried out (CDC, 1997a), which can be a costly and inefficient process compared to a targeted approach. Certainly selective and targeted sampling can frequently reduce the cost considerably. We have come to understand that universal screening is not a prudent use of financial or medical resources.

There is no central coordination among the states to develop similar state-by-state plans: for the most part, each has autonomy in developing its surveillance, educational, and intervention programs. This is typified by the current placement of various lead programs within the states—some are housed within environmental health programs, some within the state health departments, but there is no consistent directive as to where or by whom these programs should be administered. Based on current information from the CDC, not all states have lead programs, and while CDC funding is potentially available to individual states, at present not all receive CDC funding. While state-by-state surveillance data are not available, it is the hope of the CDC that in future years, they will be able to capture more of these data (C. French, personal communication).

Those states with CDC funding are required to report surveillance and other monitoring data to the CDC annually; however in reality not all states have been able to implement this requirement. In terms of actual monitoring programs, the CDC developed a software program designated STELLAR (systematic tracking of elevated lead levels and remediation), aimed at tracking child-specific lead data. While this software is available to individual states as well as to local programs, states also have the autonomy to develop their own surveillance systems and initiatives. While guidance on recommended BLL screening and intervention programs is provided by the CDC, individual states may opt to develop their own criteria, and given this, a bright line number does not universally trigger a common educational or medical intervention

across all 50 states.

The essential components that CDC-funded state programs are required to provide include those listed below.

1. a statewide/jurisdictionwide screening plan
2. a statewide surveillance system
3. implementing screening and followup care
4. public and professional health education and health communication/ education
5. primary prevention
6. program impact evaluation

Among those states with programs, there is wide variety in the types, goals and structures of each state program. At the national level, it will be important in the years ahead to gather, track, and coordinate data on a regular and consistent basis so that meaningful, objective, and accurate measures of impact on BLL can be realized. In an age of increasing communication, and given the public's predisposition to use technology to obtain guidance and information, accurate and reliable advice is imperative. For example, in the New York state guidance on lead-based paint removal in homes, while there is discussion of home-owners' consideration of hiring a professional to remove lead-based paint, there is no mandate for this; in fact, the guidance is written as though homeowners are the primary contractors for lead-based paint removal. Clearly, there will need to be better management and oversight of informational and educational material on lead, including at the Internet level.

X. SUMMARY

Lead is among the more pervasive and persistent heavy metals in the environment and has garnered more attention than has virtually any other toxicant. Lead can be toxic if sufficient exposure and absorption occur, and it gained its first notoriety from high occupational exposures in the past. Because lead has no known beneficial or necessary function within living systems, there is good reason 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, the dominant focus has been on effects of low-level lead exposure related to child development and behavior. While lead is clearly capable of causing neurological effects at high doses, it

remains difficult if not impossible to attribute toxicologically significant behavioral or neurological effects to increasingly lower BLL values because of the numerous confounding factors that influence intelligence and development in children. The larger picture reveals that at one time blood lead levels in Americans were significantly higher than they are today, and if lead were exerting a major and permanent effect on neurological systems, such effects should have become apparent by this time.

Many government and private programs and efforts aimed at reducing human exposure to lead through source reduction and other means have been successful as demonstrated by dramatically lower BLL values in the U.S. population, a welcome trend that continues today. Well under 5 percent of all young children in the U.S. currently have BLL over 10 μg/dL, supporting the statement that childhood lead poisoning is not "a major environmental health problem in the United States, but remains a disease of the poor and underprivileged" (Brody et al., 1994). The most recent available U.S. data indicate that while the mean BLL value was 2.3 μg/dL in the period 1991–94, there were approximately 93,000 U.S. children with BLL above 25 μg/dL. Of these, 61 percent were African American or Mexican American. Among the remaining 39 percent, the majority were believed to reside among the urban poor. These are the groups that fall into the higher risk categories and are among the first groups that should be targeted for surveillance and intervention programs. To say otherwise is not supported by scientific data and credible evidence, and shifts the emphasis away from those sectors of the population that will most likely benefit from identification and intervention.

While debates and differing views will continue to surface on the low-level effects of lead, one fact that is indisputable is the continuing decline of BLL values and of human exposure to lead from various sources. The maxim that without sufficient exposure, there is no risk will again rise to the forefront.

XI. RECOMMENDATIONS

Population BLLs continue to decline after increased efforts at minimizing environmental introduction through source control. Since lead is not an essential human element, coupled with its increasing toxicity as blood levels rise, it is prudent to limit exposure to it. The following are reasonable, cost-effective, and appropriate steps that can be taken to reduce exposure to lead:

A. Personal Family Strategies

- For older homes with peeling, flaking, or dusting paint, determine analytically if the paint contains lead and seek expert advice on whether paint removal, complete or partial, is warranted. Loose paint is especially likely to be found on windowsills and wells since the opening and closing of windows tends to cause flaking and dusting over time.

- If lead-based paint abatement is required, licensed and trained individuals should be sought for assistance. Importantly, residents of the home (particularly young children and pregnant women) should not remain in the house during lead-based paint abatement activities.

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

- While some childhood exposure to dirt is in most cases unavoidable, parents should monitor the play activities of children to prevent intentional, excessive, or chronic ingestion (e.g., pica behavior) of dirt.

- Educate children and reeducate adults 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, consider allowing 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 higher risk of lead exposure, particularly in situations when fatigue, behavioral changes, or gastrointestinal disturbances are observed.

B. 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 an employer: review, understand and implement the OSHA lead standard for workers occupationally exposed to lead.

- Avoid excessive exposure to lead-containing materials used in home maintenance and hobby activities such as bullets (firing ranges), fishing sinkers, lead soldering, and preparation of lead stained glass windows.

- Support those lead-control programs that seek to identify high-risk individuals through continued research, educational efforts, and community awareness, as well as education of those population sectors at increased risk of lead exposure resulting from lifestyle factors, living conditions, or other predisposing factors.

Although we may not fully understand the implications and ramifications, if any, of low blood lead levels for human health, a simple axiom is that as exposure decreases, so does risk. The data are quite clear on this point and levels have dropped dramatically. As lead levels continue to decline in the environment, blood lead levels will continue to decline as well, an observation that appears to be true for the U.S. population (CDC, 1997b). It is important in the years ahead that we strive to identify those children who remain at increased risk of lead exposure and to intervene when appropriate to reduce blood lead levels. Finally, from a public health viewpoint, it is important that we continue to mitigate lead exposures in high-risk sectors of the population, and to identify and place in perspective other known environmental health hazards.

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