LEGACY OF LEAD: AMERICA'S CONTINUING EPIDEMIC OF CHILDHOOD LEAD POISONING

A Report
and Proposal
for Legislative Action

Environmental Defense Fund
Washington, DC

March 1990

René Boux

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Written by

Karen L. Florini
Senior Attorney
Environmental Defense Fund

George D. Krumbhaar, Jr.
Consulting Author

Ellen K. Silbergeld
Senior Toxicologist
Environmental Defense Fund

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All errors are entirely the responsibility of the authors.

Environmental Defense Fund
1616 P st. NW
Washington, DC 20036
(202) 387-3500

$8.00

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EXECUTIVE SUMMARY AND OVERVIEW

Introduction

As a tragic legacy of the decades-long use of leaded products on a vast scale, lead today pervades America's environment. The result is a nationwide epidemic of low-level lead poisoning, an epidemic that is causing permanent neurologic damage to millions of American children. Recent studies demonstrate that the long-term consequences of this disease are profound: children who had moderately elevated lead levels in early childhood later exhibited seven-fold increases in school dropout rates, six-fold increases in reading disabilities, and lower final high school class standing.[1] These effects occurred even though the initial exposures caused no overt symptoms.

Although no precise national measurements have been collected, the federal government estimates that well over three million pre-school children -- more than 1 in every 6 -- have dangerously elevated lead levels. Poor and minority children are disproportionally affected, but the problem cuts across all socioeconomic lines.

The federal government estimates that well over three million pre-school children -- more than 1 in every 6 -- have dangerously elevated lead levels.

The consequences of low-level lead poisoning are devastating not only for the affected children and their families, but also for society as a whole. As the Secretary of Education observed earlier this year, reading and writing skills of the nation's children remain "dreadfully inadequate" despite a decade of educational reform. The new data suggest that lead is partly to blame. By the same token, until children's lead exposures are substantially curtailed, the nation will continue to fall short of its educational goals.

The severity of the nation's lead-poisoning crisis has gone generally unrecognized for decades largely because the great majority of cases have never been diagnosed. The effects of low-level lead poisoning, though severe, are not unique or obvious. Unlike the readily observable signs of chicken pox, for example, the impairment of intellectual ability caused by low-level lead poisoning is hard to pinpoint in individual children. Even when identified, such symptoms overlap with those of a variety of other biological and socioeconomic factors. Only recently, with the completion of sophisticated long-term studies, was the compelling association between childhood lead poisoning and significant neurologic impairment recognized.

The Toxicity of Lead

In the human body, lead is a potent poison that can affect individuals in any age group. Children and fetuses are particularly vulnerable, because their rapidly developing nervous systems are sensitive to lead's potency as a neurotoxin. Moreover, children generally are exposed to more lead than are

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[1] See HL Needleman, A Schell, D Bellinger, A Leviton, and EN Allred (1990), "The Long-Term Effects of Exposure to Low Doses of Lead in Childhood," New England Journal of Medicine, Vol. 322, pp. 83-88.

adults, and their absorption rates are substantially higher.

Lead's specific neurotoxic effects include impairments to IQ level, short-term memory, and reaction time; it also impairs the ability to concentrate. In adults, low-level lead exposure has been associated with hypertension in men and pregnancy complications in women, including minor birth defects.

Once absorbed, lead is stored primarily in bone. To a lesser degree, storage also occurs in the kidneys and the brain, while a small portion remains in circulation in the blood. Lead's persistence in the body is unequalled by virtually any other toxin. Its "half life" in bone -- the time it takes half of a given dose to be removed -- exceeds twenty years. As a result, even small amounts of lead accumulate in the body, and can cause effects that endure long after exposure ends. Further, because stored lead can be released during pregnancy and readily transferred to the fetus, lead poisoning is, in effect, a heritable disease.

Many public health experts now believe that lead presents a "continuum of toxicity," in which the slightest exposure contributes to an adverse result somewhere in the body

In the early and middle decades of this century, lead was generally thought to be harmful only at high doses. Subsequent research, however, has uncovered a variety of effects at lower and lower levels. This trend has accelerated within the last few years, as increasingly sensitive analytic techniques allow investigators to document consequences that persist for years after initial exposure. Many public health experts now believe that lead presents a "continuum of toxicity," in which the slightest exposure contributes to an adverse result somewhere in the body.

Because lead causes neurologic damage even at doses that do not cause overt toxicity, levels of lead in blood are generally used in identifying lead exposures of concern. The federal government's Centers for Disease Control (part of the Public Health Service) is currently reviewing its definition of "lead toxicity," which is now set at 25 micrograms of lead per deciliter of blood (ug/dl); CDC is expected to adopt a new definition of between 10 and 15 ug/dl within the year. The U.S. Environmental Protection Agency, along with many public health experts, has already recognized that blood-lead levels of 10 to 15 ug/dl cause neurotoxic effects in children. This report uses the term "low-level lead poisoning" to denote these levels and the associated health effects.

Even at the 25 ug/dl level, the very limited lead-screening programs now in place uncover over 10,000 previously unreported cases of poisoning each year. Indeed, though little recognized by the general public, the scale of this insidious epidemic makes it among the most common diseases of childhood.[2] It is also nationwide in scope, as an analysis of the estimated numbers of affected children throughout the country reveals. Exposures are

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[2] Some common childhood illnesses and their reported 1988 incidence rates include:

Lead Poisoning (25+ ug/dl)	11,793
Viral meningitis	6,927
Mumps	4,730
Whooping cough	3,008
Measles	2,933

Source: Centers for Disease Control. The Centers' records include only cases that were identified through screening programs and reported by health officials; therefore, these must be considered minimum figures.

endemic in some urban regions, with over 50% of children under 6 estimated to have blood lead levels over 10 ug/dl.[3]

As a practical matter, prevention is the only realistic "cure" for lead poisoning. Available treatments are expensive and painful, do not completely remove lead from the body, and are powerless to undo neurologic damage. But little has been done to prevent childrens' exposures to lead already dispersed into existing environmental reservoirs.

Exposure Levels and the Environmental Reservoir of Lead

Most children are exposed to lead as a result of its presence in paint, plumbing, gasoline, solder, and other products. Over many decades, these uses have dispersed millions of tons of lead throughout the environment. And that reservoir continues to grow each year, as the United States uses another million-plus tons of lead in products such as automotive batteries, construction materials, gasoline, and other items. Because lead is an element, no force save a nuclear reaction can transform it into a more innocuous material; once excavated from the earth and distributed in commerce, lead can exert its inherent toxicity on the biosphere almost indefinitely.

The reservoir continues to grow each year, as the United States uses another million-plus tons of lead in products such as automotive batteries, construction materials, gasoline, and other items.

Of all the sources that make up the existing reservoir of environmental lead, one is responsible for especially intense exposures for many children: the three million tons of leaded paint remaining on the walls and woodwork of American homes. Though banned for most uses in 1977, leaded paint applied during the preceding decades continues to present a hazard. An estimated 1.2 million children under 6 absorb enough lead from deteriorated paint to elevate their blood-lead levels beyond 15 ug/dl, with a significant chance of subsequent neurologic impairment.

Although the pronounced long-term consequences of childhood lead poisoning have only recently been identified, its more obvious manifestations have been a focus of concern for decades. As early as 1904, reports of childhood lead intoxication appeared in the medical literature. But due to the limited diagnostic capabilities of the time, only the most obvious cases were identified -- those involving high doses of lead resulting in readily observable effects such as convulsions, coma, and even death. With the advent of blood-lead determinations in the 1940s and '50s, however, it became increasingly apparent that the problem was far greater in scope than had been recognized previously.

Citing the "epidemic proportions" of childhood lead poisoning, Congress first took action in 1970 to eliminate a primary source of children's exposures. The Lead-Based Paint Poisoning Prevention Act of 1971[4] authorized a wide range of actions designed to identify and treat those already harmed, to remove lead-based paint from federally-assisted homes, and to prohibit its use in areas thought to be accessible to children. Unfortunately, implementation of key provisions faltered badly almost from the start. As a result, two decades later the epidemic persists.

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[3] Geographical distributions of lead-affected children are described in Appendix 1.

[4] 42 U.S.C. sections 4801-4846.

The role of paint is even clearer today than it was twenty years ago, for many other major sources of lead have been at least partially controlled in the interim. For the American populace as a whole, the most significant reductions in lead exposure have resulted from the phase-down in use of leaded gasoline over the last fifteen years. But while this step has provided important benefits in reducing lead exposures for many people, it has done little to aid those children whose primary source of lead is from paint. And these children -- many of them poor and/or minorities -- are precisely the same individuals who are most disadvantaged by a myriad of other social and economic factors.

Toward a Solution: A Proposal for Legislative Action

"Ideally, in keeping with the precepts of primary prevention, lead should have been prohibited from ever having been dispersed in the modern environment."

American Academy of Pediatrics

The massive amounts of information on lead's toxicity -- bolstered by recent findings on low exposure level effects -- as well as indications of children's current exposure levels reveal an urgent need for an aggressive federal program to control America's continuing epidemic of lead poisoning. To be effective, such a program must provide a mechanism not only to ***stop adding*** lead to children's environments, but also to ***remove*** it from the areas where they are most heavily exposed: their homes. And, to be politically feasible, it must respond to current budgetary realities the nation now faces.

The Environmental Defense Fund proposes creation of a National Lead Paint Abatement Trust Fund, to be financed by placement of a substantial excise fee on the production and importation of lead. Proceeds from the fund initially would be devoted to the removal of deteriorating lead-based paint from the group of highest-risk homes. In addition, a portion of the monies could be made available for research to develop more effective lead-removal methods.

The program would be implemented jointly by the Environmental Protection Agency and the Department of Health and Human Services. It would contain provisions to enable it to reflect market conditions and, where possible, accomplish secondary goals of improving housing and creating employment opportunities by hiring and training workers for abatement programs. In addition, by avoiding the slow and resource-intensive process of developing a regulatory approach to control continuing uses of lead in products, it would yield results far more quickly than would more traditional approaches.

While the proposed program would not alleviate every aspect of the nation's current lead poisoning epidemic, it would constitute a pragmatic and timely next step. Lead poisoning already burdens America with millions of dollars of costs each year -- both the direct costs of medical treatments, and the indirect social costs of special education, lost income, and a less productive citizenry. It also imposes grave handicaps on individual children, their families, and their communities. For them and for the nation as a whole, these handicaps will only intensify as the transition to the twenty-first century's "information age" continues. By creating a nationwide paint-abatement program funded by a lead excise tax, America can permanently reduce lead exposures and bring about a significant improvement in the health and abilities of the nation's children -- now and for generations to come.

PART I: UNDERSTANDING THE PROBLEM

1. THE TOXICITY OF LEAD[1]

Lead's primary effect of concern is neurotoxic damage to fetuses and preschool children, for this effect occurs at levels of exposure that are commonplace in contemporary society. Low levels of lead exposure can also cause kidney damage and high blood pressure in adults.

Lead makes its way into the blood stream; into soft body tissue, including the brain and kidneys; and into the "hard tissues," such as bone and teeth.

Upon entering the body, lead makes its way into the blood stream; into soft body tissue, including the brain and kidneys; and into the "hard tissues," such as bone and teeth.[2] Blood-lead content is generally considered to be the most accurate measure of short-term lead exposure. The estimated half life of blood lead (i.e., the time required for one half of the lead to disappear) is 35 days.[3] While about 50 to 60 percent of the lead entering a person's body is eliminated fairly rapidly,[4] most of the remainder is stored in bone, where it stays for far longer periods. In fact, lead in bone has an estimated half life of about 20 years.[5]

Long thought to be inert, bone-based lead is now looked on as a double threat to the body. Bone is a living tissue that is itself sensitive to toxic assaults.[6] Many conditions, moreover, can rapidly release bone-based lead back into the blood stream. For example, pregnancy and osteoporosis, both

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[1] The documentation of lead's toxic effects is immense. Key sources include: Agency for Toxic Substances and Disease Registry (1988), The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress (Atlanta: U.S. Dep't of Health and Human Services/Public Health Service), Doc. No. 99-2966, especially Chapters III and IV; U.S. Environmental Protection Agency (1986a), Air Quality Criteria Document for Lead, Vols. I through IV; Centers for Disease Control (1985), Preventing Lead Poisoning in Young Children, (Atlanta: Dept of Health and Human Services/ U.S. Public Health Service). Excellent review articles include HL Needleman (1988a), "Why We Should Worry About Lead Poisoning," Contemporary Pediatrics, pp. 34 - 56; JM Davis and DJ Svensgaard (1987), "Lead and Child Development," Nature, Vol. 329, pp. 299-300; HL Needleman (1988b), "The Persistent Threat of Lead: Medical and Sociological Issues," Current Problems in Pediatrics, Vol. XVIII, pp. 703-76; EK Silbergeld (1985), "Neurotoxicology of Lead," in K Blum and L Manzo (eds.), Neurotoxicology (Amsterdam: Dekker).

[2] MB Rabinowitz, GW Wetherill, and JD Kopple (1976), "Kinetic Analysis of Lead Metabolism in Healthy Humans," Journal of Clinical Investigation, Vol. 58, p. 260.

[3] Ibid.

[4] Agency for Toxic Substances and Disease Registry (1988), p. III-7.

[5] Rabinowitz et al. (1976).

[6] Agency for Toxic Substances and Disease Registry (1988), p. III-7.

of which cause demineralization of bone, have been associated with sharp rises in blood lead levels.[7] Indeed, lead moves from bone to other parts of the body readily enough that it may well be an "insidious source" of long term lead poisoning.[8]

An important aspect of lead's menace, therefore, is its *cumulative effect.* Even seemingly trivial exposures, if often repeated, can add up to doses that exert toxic effects.[9]

"At a sufficient level of lead exposure, virtually all body systems will be injured or have a high risk of injury."

And virtually no part of the body is immune from lead. As one recent analysis put it, "At a sufficient level of lead exposure, virtually all body systems will be injured or have a high risk of injury."[10] While researchers have not yet discerned the exact biological mechanisms of lead toxicity, they have extensively documented its effects on a number of organ systems at the cellular level.

The most important effects of lead involve disruption of energy metabolism at the cellular level and interference with neural cell function in the brain. Specifically, lead interferes with the formation of heme, the molecule that carries oxygen in all cells.[11] In the nervous system, lead has a unique ability to inhibit communication and slow motor nerve conduction velocity[12] -- the speed at which nerves process signals.[13]

Neurotoxic Effects: Lead's neurotoxic effects at relatively low exposure levels include decreased intelligence, short-term memory loss, reading and spelling under-achievement, impairment of visual-motor functioning, poor perceptual integration, poor classroom behavior, and impaired reaction time. Children and fetuses are especially susceptible to these effects, because their neurologic systems are rapidly developing.[14] Growing

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[7] EK Silbergeld, J Schwartz, and KR Mahaffey (1988), "Lead and Osteoporosis: Mobilization of Lead from Bone in Menopausal Women," Environmental Research, Vol. 47, p. 79.

[8] Environmental Protection Agency (1986a), Vol. IV, p. 13-16.

[9] Centers for Disease Control (1985), p. 3.

[10] Agency for Toxic Substances and Disease Registry (1988), p. IV-3.

[11] Silbergeld (1985).

[12] Ibid.; see also, PJ Landrigan (1989), "Toxicity of Lead at Low Dose," British Journal of Industrial Medicine, Vol. 46, pp. 593-4.

[13] In addition to these effects of low-level lead exposure, effects of high-level exposures are also varied, and include anemia, brain damage, muscle palsy, kidney failure, headache and vomiting, convulsions, and death. These high-dose effects have been known for centuries. The first known clinical account of lead poisoning comes from the first century B.C., while Hippocrates offered unconfirmed descriptions two centuries earlier. See HA Waldron (1973), "Lead Poisoning in the Ancient World," Medical History, Vol. 17, pp. 391-99. In eighteenth century Massachusetts, lawmakers enacted one of the country's first public health statutes after recognizing the health effects of drinking "rum and other strong liquors" from leaded containers. See CP McCord (1953), "Lead and Lead Poisoning in Early America: Benjamin Franklin and Lead Poisoning," Industrial Medicine and Surgery, Vol. 22, p. 397.

[14] Centers for Disease Control (1985), p.1.

evidence indicates that the effects of lead poisoning occur before any overt symptoms appear and often constitute a serious health problem even in the absence of obvious symptoms.

Several key epidemiologic studies in recent years have compellingly demonstrated the range of lead's effects on a variety of populations.[16] Most of these studies are retrospective, meaning that researchers identify a group of children, determine their lead levels, and evaluate their current health status in an attempt to ascertain the effects of prior lead exposure. Although lead poisoning is often viewed as primarily a disease of the poor, wealth and social status confer no immunity. Indeed, a recent government study concluded that children living *above* the poverty level comprise the largest category of people in danger of undue exposure.[15]

A series of landmark studies on lead neurotoxicity have been conducted by Dr. Herbert Needleman of the University of Pittsburgh and his colleagues. The researchers collected baby teeth -- which, like bone, serve as long-term storage sites for lead -- from over 2300 first and second graders in two suburban Boston school districts. They then categorized the children according to dentine (tooth) lead levels [17], and identified two groups for further study: a low-lead group of 100 children who had extremely low levels and a high-lead group 58 children who had relatively high levels but who had no symptoms of overt lead poisoning.[18] Those 158 children were then evaluated using an array of standardized and some nonstandardized neuropsychological tests.

When the results were controlled for 39 other factors (such as socioeconomic status, family size, and mother's IQ), children in the high-lead group had a median IQ deficit of six points compared to their low-lead classmates, as well as shorter attention spans and imparied language skills. Even more striking was the effect on the overall distribution on IQ scores: the children in the higher-lead group were almost four times as likely to have an IQ below 80, while none of them scored above 125.[19]

Also striking were the results of evaluations by the children's teachers (who did not know their pupils' lead status). Using an 11-item scale that examined classroom behavior, attention, and overall functioning, teachers concluded that children with elevated lead levels scored significantly worse than the low-lead group.

Five years later, the researchers re-examined these two groups of

Although lead poisoning is often viewed as primarily a disease of the poor, wealth and social status confer no immunity. Indeed, a recent government study concluded that children living *above* the poverty level comprise the largest category of people in danger of undue exposure.

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[15] Agency for Toxic Substances and Disease Registry (1988), p. I-48.

[16] For an outline of epidemiological considerations, see sidebar below and Needleman (1990b), p. 677.

[17] The high lead group had dentine levels above 24 parts per million (ppm); the low-lead group had dentine levels below 6 ppm.

[18] These 158 children were a subset of the 270 children with levels above 24 or below 6. Others were excluded to avoid possible confounding factors such as head injuries, acute lead poisoning, and variable lead levels in different teeth.

[19] Needleman (1988a).

[20] Bellinger, D, HL Needleman, R Bromfield, and M Montz (1984), "A Follow-up Study of the Academic Attainment and Classroom Behavior of

children.[20] The high-lead group had lower IQ scores, needed more special academic services, and had a higher rate of school failure. Eleven years after the initial study, a second follow-up was conducted, to determine whether lead's effects persist into young adulthood.[22] The findings were dramatic: compared to the lower-lead classmates, the higher-lead group showed a 7.4 increase in school dropout rates, and a 5.8 increase in reading disabilities (defined by scoring two or more grade levels below that expected for the highest grade completed). The higher-lead group also exhibited lower class rank and higher absenteeism.

Other researchers have also found effects in epidemiological studies on lead-exposed children, though some have conducted similar studies and reported no effects. All studies published since 1972 were recently evaluated using meta-analysis, a technique that allows investigators to pool data across studies and to draw conclusions as to the statistical reliability of the data taken collectively.[23]

After eliminating studies that failed to meet key criteria such as adequate sample size, exclusion of acutely poisoned children, and controls for socioeconomic factors, data from the remaining twelve studies were pooled. The outcome strongly supports a linkage between low-dose lead exposure and intellectual deficits in children.

Further evidence of lead's neurotoxicity comes from a series of prospective studies, in which investigators measure variables over an extended period of time into the future. Recent studies have found notable effects from prenatal lead exposures at very low levels.[24] In fact, one study found effects from prenatal exposures as low as 6 to 7 ug/dl.[25]

For example, in a study of several hundred children whose prenatal lead exposure had been determined from umbilical cord blood samples at the time of birth, investigators found that even moderate lead levels affected the

THE IMPORTANCE OF SMALL NUMBERS

Figure 1 shows the frequency distribution of IQ scores between the "low lead" and "high lead" children, and indicates that high blood lead levels are associated with a left-ward shift in the overall IQ distribution curve. In addition to showing that the median IQ deficit is 6 points, these data also illustrate two other key points: (1) High lead children in this case were almost ***four times as likely to have IQs of less than 80*; and (2) *five percent of the low lead group had IQs of more than 125, while none of the high lead group did.*[21]**

In other words, lead's effect on a population *as a whole* is more dramatic than its effects on individuals, by affecting the frequency of high and low scores. The disadvantaged are further harmed, while the truly gifted are deprived of their potential.

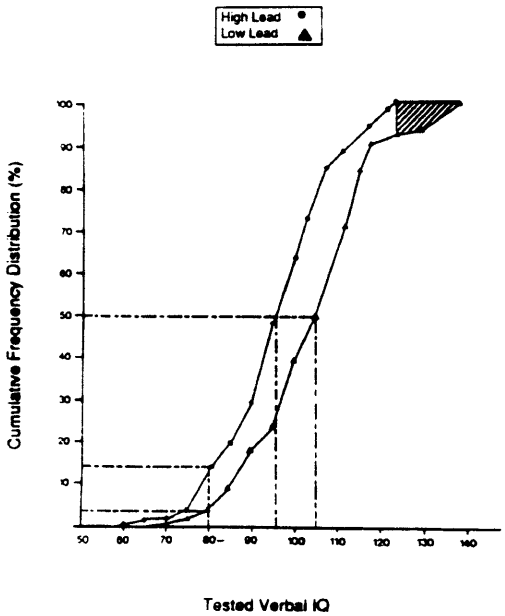

FIGURE 1
DISTRIBUTION OF IQ SCORES

Children with Elevated Dentine Lead Levels," Biological Trace Elements Research, Vol. 6, pp. 207-223.

[21] Diagram adapted from HL Needleman (1988a).

[22] HL Needleman, et al. (1990a), "The Long-Term Effects of Exposure to Low Doses of Lead in Childhood," New England Journal of Medicine Vol. 322, pp. 83-88. Researchers were able to trace and evaluate about half of the original participants. The others could not be located or refused to participate. The group that was retested tended to have lower dentine levels, higher IQs, and better school behavior reports. As a result, it seems likely that the 11-year follow-up may underestimate lead's long-term effects, since a higher percentage of the most severely affected individuals did not participate.

[23] HL Needleman, CA Gatsonis (1990b), "Low Level Lead Exposure and the IQ of Children," Journal of the American Medical Association, Vol. 263, pp. 673-678.

[24] For detailed discussion of these studies, see Agency for Toxic Substances and Disease Registry (1988), pp. IV-8 to IV-13.

[25] Bellinger, D, A Levitan, C Waternaux, HL Needleman, and M Rabinowitz (1989),"Low-level Lead Exposure, Social Class, and Infant Development," Neurotoxicology and Teratology, Vol. 10, pp 497-503.

children's performance on mental-development tests up to two years later.[26] Similar outcomes have been found in studies in Port Pirie, Australia and Cincinnati, Ohio.[27] In the words of the American Academy of Pediatrics, the available data have "shown conclusively" that reduction in intelligence and alteration in behavior occur in children with elevated blood lead levels.[28]

In addition to these extremely disturbing findings on the consequences of lead exposure in children and fetuses, a growing body of research is showing that low levels of lead also exert toxic effects on adults, including cancer, reproductive effects, and high blood pressure.

Cancer: The U.S. Environmental Protection Agency has classified lead as a "probable human carcinogen," based on data from animal studies.[29] Recently, EPA's Science Advisory Board, which is comprised of outside experts from industry and academia, formally reviewed EPA's classification and endorsed it.[30] Researchers are currently comparing lead's potency as a carcinogen to its potency as a neurotoxin.[31]

Reproductive Effects: Experiments on laboratory animals give ample evidence of lead's toxic effects on the reproductive system (e.g., failure of ovulation, delayed sexual maturity, impotence, sterility, spontaneous abortions).[32] While there are fewer data on the reproductive effects in humans, there are numerous reports of an increase in spontaneous abortions, structurally abnormal sperm, and decreased fertility in lead-poisoned adults.[33]

Effects on Blood Pressure: An additional threat to adult males is indicated by evidence showing a link between low-level lead exposure and

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[26] D Bellinger, A Leviton, C Watermaux, HL Needleman, and M Rabinowitz (1987), "Longitudinal Analyses of Prenatal and Postnatal Lead Exposure and Early Cognitive Development, New England Journal of Medicine, Vol. 316, pp. 1037, 1039.

[27] AJ McMichael, PA Baghurst, NR Wigg, GV Vimpai, EF Robertson, RJ Roberts (1988), "Port Pirie Cohort Study: Environmental Exposure to Lead and Children's Abilities at the Age of Four Years," New England Journal of Medicine, Vol. 319, pp. 468-75; KN Dietrich, KM Krafft, RL Bornschein (1987), "Low Level Fetal Exposure Effect on Neurobehavioral Development in Early Infancy," Pediatrics, Vol. 5, pp. 721-30.

[28] American Academy of Pediatrics (1987), "Statement on Childhood Lead Poisoning," Pediatrics, Vol. 79, pp. 457.

[29] See 50 Fed. Reg. 46936 (Nov. 13, 1985).

[30] Environmental Protection Agency, Scientific Advisory Board (December 1989), "Report of the Joint Study Group on Lead: Review of LEad Carcinogenicity and EPA Scientific Policy on Lead," (Doc. No. EPA-SAB-EHC-90-001), p. 1.

[31] Research underway at University of Maryland, Program in Toxicology.

[32] HL Needleman and PJ Landrigan (1981), "The Health Effects of Low Level Exposure to Lead," Annual Review of Public Health, Vol. 1981, pp. 277-98.

[33] Ibid.

DETERMINING CAUSE AND EFFECT THROUGH EPIDEMIOLOGICAL STUDIES

In epidemiological studies, groups of people are studied in order to determine patterns of disease. Those patterns are then analyzed statistically in order to reveal links between a particular substance and certain health effects.

Scientists attempting to show cause and effect through epidemiological studies use five rules of thumb that, taken together, serve as a rigorous test of causality. There are:

1. Order of precedence. The "cause" must precede the "effect."

2. Consistency. There must be broad consistency among data both internally, and among different studies.

3. Dose-response. Causality can be more strongly inferred when variations in the "cause" are associated with variations in the "effect."

4. Specificity. If the same effect can be produced by other means, the cause-effect relationship under scrutiny is weakened. If the effect can be produced only by the cause, the relationship is strengthened.

5. Biological plausibility. Put simply, this test asks whether, in light of current knowledge of human biology, the cause/effect relationship seem likely.

All five criteria are met for studies on the neurotoxicity of lead.

high blood pressure.[34] Although differences between blood-pressure values were relatively small, the effect nonetheless is of concern from a public health perspective. Like lead's effects on IQ distribution, the consequences of even a small shift in the distribution curve for blood pressures can be severe on a population-wide basis. Given the role of cardiovascular disease as the number one cause of death in America, even "small" increases in average blood pressure are of significant concern.

Given the role of cardiovascular disease as the number one cause of death in America, even "small" increases in average blood pressure are of significant concern.

[34] In a statistical analysis based on a national health survey of 9,932 persons of all ages, one researcher found a "robust relationship between low-level lead exposure and blood pressure" in adult males. J Schwartz (1988), "The Relationship Between Blood Lead and Blood Pressure in the NHANES II Survey," Environmental Health Perspectives, Vol. 78, pp. 15-22. A reanalysis of the same data for males between the ages of 12 and 74, using a different and rather conservative statistical technique, also found a significant linear association between blood lead levels and blood pressures. JR Landis and KM Flegal (1988), "A Generalized Mantel-Haenszel Analysis of the Regression of Blood Pressure on Blood Lead Using NHANES II Data," Environmental Health Perspectives, Vol. 78, pp. 35-42. While the actual differences in blood pressure in these and other studies are small, the consistency across studies is strong. W Victery, HA Tyroler, R Volpe, and LD Grant (1988), "Summary of Discussion Sessions: Symposium on Lead-Blood Pressure Relationships," in U.S. Department of Health and Human Services, Environmental Health Perspectives, Vol. 78, pp. 139-155.

2. EVALUATING EXPOSURES TO LEAD

Most public health experts now agree that lead exhibits a "continuum of toxicity," where the smallest exposure can have a consequence somewhere in the body. This marks a radical departure from the approach to the problem only a few years ago.

Because most cases of lead poisoning have no overt symptoms, screening programs are critically necessary to identify children in need of treatment. Unfortunately, screening programs in many cities were curtailed or eliminated in the early 1980s after the federal government discontinued funding for such programs, and nationwide data-collection efforts were also dropped. As a result, estimates of numbers of affected children must be derived from limited sampling programs, and extrapolated using figures on other variables known to be related to lead poisoning.

In measuring the amount of lead absorbed by an individual and determining whether treatment is needed, doctors generally rely on measurements of the amount of lead in the individual's blood.[1] Though such measurements do not reveal the individual's lifetime history of lead exposure or the amount that is currently stored in bone, blood-lead levels can provide a "snapshot" of recent lead exposures.[2] Results are generally expressed as micrograms of lead per deciliter of blood, or ug/dl.

Though such measurements do not reveal the individual's lifetime history of lead exposure or the amount that is currently stored in bone, blood-lead levels can provide a "snapshot" of recent lead exposures.

One practical way to obtain some longer-term data for children involves collecting children's teeth as they are naturally shed (generally between ages five and nine). This approach, however, means that parents must be informed of the need to collect teeth and must agree to participate, and is obviously inapplicable to adults and older children.

Prior to the mid 1960s blood lead levels of 60 micrograms of lead per deciliter of blood (ug/dL) or less were generally considered as not dangerous

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[1] Because analysis of blood samples takes up to two weeks, screening tests are sometimes used to give a preliminary indication of whether further testing is warranted. The so-called "EP" test, which is a finger-prick test that gives immediate results, was used as a screening tool for many years. The test measures the presence of a naturally occurring protein that is produced at higher levels in response to lead exposure. Unfortunately, the accuracy of the EP test is limited for blood-lead levels below 40 ug/dl. State of California, Department of Health Services (1989), Childhood Lead Poisoning in California: Causes and Prevention, p. 8, 14 (interim report). As a result, it is not useful in screening for exposures at current levels of concern. Researchers at the University of Maryland Department of Toxicology are attempting to develop a substitute test that will serve as a preliminary screening test for blood-lead levels around 10 ug/dl.

[2] While promising, methods of directly measuring the total amount of lead stored in an individual's body are still undergoing development and are not yet widely available. See, for example, JF Rosen et al. (1988), "L-Line X-ray fluorescence of Cortical Bone Lead Compared with the CaNa2EDTA Test in Lead-Toxic Children: Public Health Implications," Proceedings of the National Academy of Sciences (USA), Vol. 86, pp. 685-689.

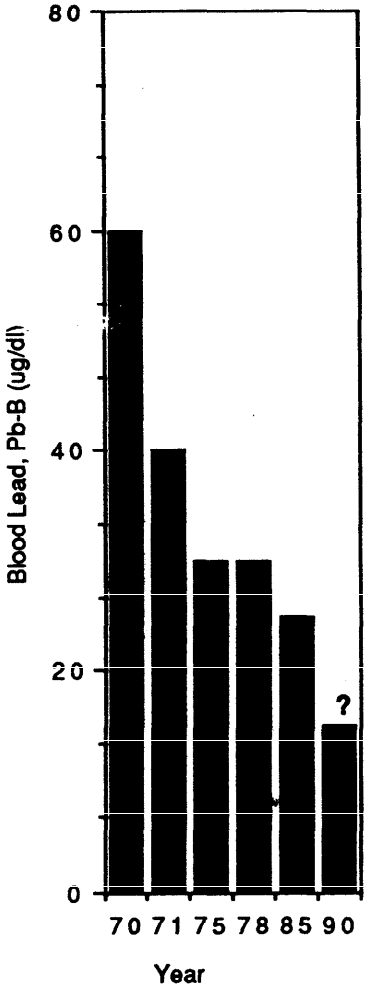

FIGURE II: RECOMMENDED BLOOD LEAD LEVELS FOR MEDICAL INTERVENTION

Figure II shows the erosion of the recognized threshold for lead toxicity as new evidence has emerged through increasingly sophisticated scientific studies.

enough to require monitoring or treatment.[3] This became an official standard in October 1970 when the U.S. Surgeon General issued a report defining 60 ug/dL as a level of "undue lead absorption." But even within the year, further analysis prompted the Public Health Service to circulate a draft lowering that threshold for undue absorption by a third, to 40 ug/dL.[4] Within five years the threshold fell again, to 30 ug/dL, while the threshold for outright lead poisoning was set at 80.[5]

In 1978, the Public Health Service revised its finding, with 30 ug/dL as the threshold for undue lead absorption and 70 ug/dL as the threshold for poisoning.[6] In 1985 the Service's Centers for Disease Control (CDC) issued a statement lowering its thresholds for both excessive lead absorption and lead toxicity to 25 ug/dL.[7] And in late 1989, CDC announced that it was convening an Advisory Committee to update its statement on preventing lead poisoning in young children, to reflect research findings since 1985. Public health experts interpret this action as portending another downward revision of the standard, probably to 15 ug/dl or lower.[8]

The Environmental Protection Agency has also evaluated lead's toxicity in developing regulations under a variety of environmental statutes, including the Clean Air Act, the Clean Water Act, and the Safe Drinking Water Act. A 1986 report prepared as a background document on air regulations cited 10-15 ug/dL as the range associated with "neurological

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[3] Centers for Disease Control/Public Health Service (1985), p. 1.

[4] Public Health Service, Bureau of Community Environmental Management, Control of Lead Poisoning in Children - pre publication draft (Washington, U.S. Department of Health, Education, and Welfare, December 1970), p. 2.

[5] Public Health Service, Increased Lead Absorption and Lead Poisoning in Young Children: A Statement by the Center for Disease Control (Washington, U.S. Department of Health, Education, and Welfare, March 1975), p. 1. That document used lead "poisoning" or "toxicity" to mean a condition showing acute or obvious symptoms; "undue lead absorption" or "elevated blood lead level" means a level warranting medical intervention but where obvious symptoms may not be present.

[6] Public Health Service, Preventing Lead Poisoning in Young Children: A Statement by the Center for Disease Control (Washington, U.S. Department of Health, Education, and Welfare, April 1978), p. 1.

[7] Centers for Disease Control/Public Health Service, (1985), pp. 1-2. Lead toxicity is defined by two factors: a blood lead level of 25 ug/dl together with an erythrocyte protoporphyrin ("EP") of 35 ug/dl. EP is a naturally occurring protein that plays a key role in the manufacture of hemoglobin; an elevated EP level is one of the earliest and most reliable signs of impaired function due to lead. Ibid., p. 3.

[8] 54 Fed. Reg. 48026 (November 20, 1989). The CDC convenes such committees at irregular intervals, when it considers the evidence strong enough to warrant re-examination of the standard.

deficits."[9] In its proposed regulations on lead in drinking water, the EPA again cited 10-15 ug/dL as an "appropriate range of concern for health effects that warrant avoidance."[10] A December 1989 statement of EPA's Science Advisory Board concludes that "...there is likely to be no threshold for lead neurotoxicity, at least within the contemporary range of blood lead levels (i.e., 1-10 ug/dl)."[11] And as another EPA advisory group pointed out even more recently, "[t]he value of 10 ug/dl refers to the maximum blood-lead level permissible for all members of these groups, and not mean or median values."[12]

The steady erosion of the accepted threshold for lead's toxic effects, coupled with lead's known biochemical properties, has convinced many public health experts that lead has no threshold. Rather, the emerging view is that lead presents a "continuum of toxicity" in which traditional symptoms associated with high dosage, such as kidney failure and anemia, have their low dosage counterparts, such as IQ deficits and decreased nerve conduction.[13]

Public health experts have also found that the same level of lead exposure may affect different people unequally. Among the chief variables appear to be dietary intake of essential trace minerals. Lead's effects are aggravated in people who lack adequate dietary calcium, iron, zinc, or phosphorous.[14] This places poor families, where malnutrition may be more common, at greater risk of adverse effects from lead.

"Because the 'baseline' level of lead in blood in the U.S. population is apparently about 10 - 15 ug/dl, it is virtually impossible to demonstrate effects of lead at lower blood levels... the physiological states now defined as "normal" might actually be "abnormal" conditions associated with typical levels of lead in the body. The hypothesis that people would be healthier in subtle ways if the average blood lead level were 1 - 2 ug/dl (or less) deserves sober consideration..."

National Research Council (1980), p.137

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[9] Environmental Protection Agency (1986a), Addendum to Vol. 4, p. A-48.

[10] 53 Fed. Reg. 31524 (August 18, 1988).

[11] Environmental Protection Agency, Science Advisory Board (1989), p. 11.

[12] Environmental Protection Agency, Science Advisory Board (1990), p. 1.

[13] Ibid. See also PJ Landrigan (198_), p. 593.

[14] Centers for Disease Control (1985), p. 3.

3. SOURCES AND PATHWAYS OF LEAD EXPOSURE

Lead can enter the environment directly, as from industrial emissions, or indirectly, as when automotive batteries are incinerated or when dust from lead paint forms part of household dust. The most critical source of lead exposure for most children is deteriorating lead paint in dwellings; lead from gasoline and from drinking water are also significant. Additional exposures occur through continuing uses of lead. Each year industry produces, and consumers use and discard, products containing well over a million tons of lead. The lead in each of those products is indestructible.

Because lead is an element, it cannot break down or decompose into something less toxic. Both pure lead and its compounds are harmful to humans. Once introduced into the biosphere -- that part of the earth's surface and atmosphere where living organisms exist -- lead remains toxic indefinitely.

Lead's widespread usage and its resulting dispersal into the environment have been no accident. Its convenient properties have been recognized from the earliest historical times. It is malleable and easy to work. It insulates well and does not rust. It alloys readily. Lead compounds make excellent pigments in paints that also weather especially well. Egyptians in the time of the pharaohs used lead in ornaments and cosmetics.[1] Chalices made of lead-silver alloys carried wine for the ancient Greeks and lead piping still carries rainwater from the roofs of medieval cathedrals. Indeed, the word "plumbing" is itself derived from the Latin word for lead, "plumbum" (as is its chemical symbol, Pb).

The United States currently consumes well over one million tons of lead per year.[2] A substantial fraction of that amount -- approximately 60% -- comes from secondary refining (recycling). About a third comes from primary refining of lead, while imports (primarily from Canada) slightly exceed exports.[3] Almost 90% of U.S. lead mining occurs in Missouri, with some operations in Alaska, Colorado, Idaho, and Montana, as well as very limited mining in half a dozen additional states.[4] Major industrial sources alone dispose of or release 15,000 tons of lead wastes in the U.S. annually, in forms ranging from placement in landfills to fugitive emissions from facilities.[5]

Figure III. Above, the medieval chemical symbol for lead. Below, the modern equivalent.

Pb

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[1] See HA Waldron (1973), p. 392.

[2] Bureau of Mines (1989c) Mineral Commodity Summaries 1989, pp. 90 - 91 (Washington: U.S. Dept of the Interior) (1988, data converted from metric tons to short tons; one metric ton equals about 2,200 pounds, while a U.S. or "short" ton equals 2,000 pounds).

[3] Ibid.

[4] Ibid.

[5] Environmental Protection Agency, Office of Pesticides and Toxic Substances (1989), The Toxics Release Inventory: A National Perspective.

The prevalence of lead in the environment, and the public health problem it poses, is almost entirely the result of human activity. Ice layers in Greenland, far from industrial centers, reveal a record of increasing lead use by humans, with certain phases (the industrial revolution, widespread use of leaded gasoline) clearly marked in the frozen strata.[6] The increased exposure to lead in our society is so pronounced that the skeletons of modern humans contain 200 times more lead than those of their preindustrial ancestors.[7]

From the standpoint of public health and environmental quality, therefore, the threat posed by lead is inescapably cumulative. Simply curtailing additional releases of lead does not solve the problem; it is also necessary to take action to remove *existing* sources of lead exposure. Reducing current lead usage can, however, slow the rate at which the cumulative exposure problem worsens. Ongoing releases of lead -- whether through a product's manufacture, its use, or its disposal -- add to existing environmental stockpiles. In sum, controlling lead exposures requires a two-fold approach of both limiting ongoing uses of lead and attacking stockpiles created by past uses.

Simply curtailing additional releases of lead does not solve the problem; it is also necessary to take action to remove *existing* sources of lead exposure.

Because lead accumulates in the body, all sources that add lead to the environment contribute to lead poisoning. Some, however, play a far greater role than others, particularly for children's exposures. Currently, lead in house paint is the most significant of these, especially for those children with

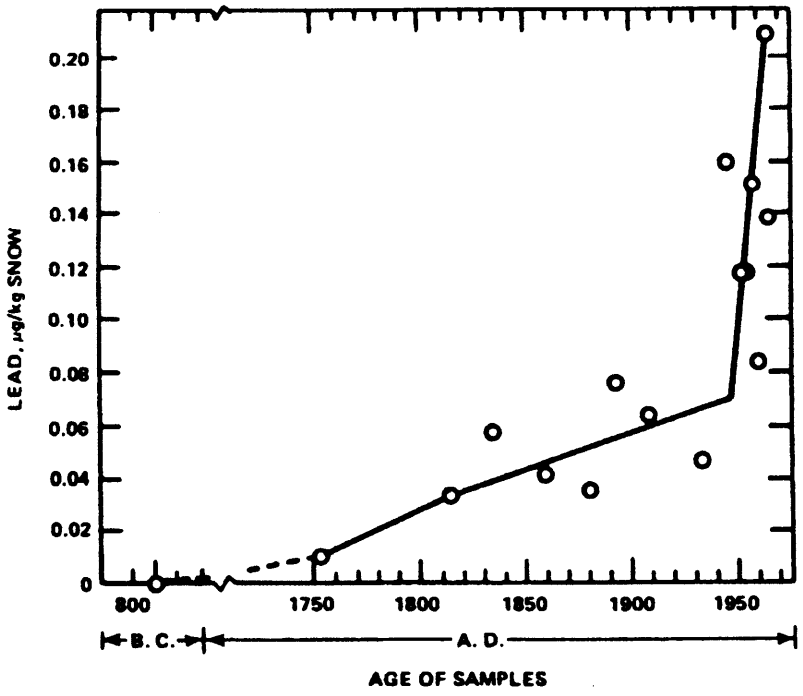

Figure IV. Lead concentration profile in snow strata of Northern Greenland. Source: Environmental Protection Agency (1986a), Vol. I, p. 1-26.

1987 (Doc. No. EPA 560/4-89-005), p. 59. The toxics release inventory program covers manufacturing in the industrial sector (as defined by Standard Industrial Codes 20 through 39) that employ more than ten individuals and use more than certain amounts of specified chemicals, including lead compounds. TRI requires reporting the amounts of substances released directly to the environment or transferred to off-site locations, including gaseous or particulate emissions to air, discharges to water, disposal of solid wastes in landfills, injection into underground wells, or transfer to off-site treatment, storage, or disposal facilities.

Worldwide, annual production of lead waste from industrial sources amounts to an estimated 1.3 million tons. JO Nriagu, and JM Pacyna (1988), "Quantitative Assessment of Worldwide Contamination of Air, Water and Soils by Trace Metals," Nature, Vol. 333, p. 139.

[6] Needleman and Landrigan (1981), p. 279.

[7] Environmental Protection Agency (1986a), Vol. I, p. I-81. This relationship was first recognized by CC Patterson (1980), "An Alternative Perspective -- Lead Pollution in the Human Environment: Origin, Extent, and Significance," pp. 265-348, in National Research Council, Lead in the Human Environment (Washington, DC: National Academy of Sciences).

moderate to heavy total lead burdens; indeed, a majority of the cases of lead poisoning that show up in clinics today are the result of paint that was applied to houses decades ago.[8]

But other sources also make a notable contribution. Some of these -- such as leaded gasoline -- are products whose use has fallen in recent years, but their legacy of contamination persists. In addition, over a million tons of lead continue to be introduced into commerce in the form of new products each year; the manufacture, use, and disposal of those products adds still more lead to the environment.

Lead-based paint

During the early and middle decades of this century, lead-based paint was the preferred medium in millions of homes.[9] The lead content of such paint varied, with some -- particularly in earlier years -- containing as much as fifty percent lead by dry weight.[10] Even as late as 1971, the New York City Health Department tested 76 different paints and found eight of them to contain lead concentrations between 3 and 11%.[11] Today an estimated three million tons of lead from paint still remains in dwellings.[12]

Simply by behaving like children, they get dust particles on their clothes and hands, and into their mouths

At one time, the general consensus was that children were exposed to lead-based paint primarily when they actually ate flakes of the sweet-tasting product or chewed on readily accessible surfaces such as window sills. More recently, however, researchers have realized that the primary exposure route starts with the transformation of lead paint into ordinary household dust. Children absorb lead by playing in the dust that is contaminated with these fine particles of paint. Simply by behaving like children, they get dust particles on their clothes and hands, and into their mouths.[13]

Even a well-maintained home is likely to have some deterioration of paint on window sills, particularly in parts of the nation exposed to freeze-thaw cycles. Many older houses, however, have been poorly maintained. Paint from these dwellings can easily be converted into dust-size particles that pose an extra menace to any active child. Over 40 million houses

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[8] Personal Communications with Dr. John Graef, Director, Boston Lead Screening Program, Boston, MA; Dr. J. Julian Chisholm, Director, John Hopkins School of Medicine Lead Program, Baltimore, MD.

[9] Hearings before the Subcommittee on Housing and Community Development of the House Committee on Banking, Finance and Urban Affairs, 100th Cong., 2d Sess. (1988) (testimony of James Keck, Deputy Commissioner, Baltimore Department of Housing and Community Development). See also R. Rabin (1989), "Warnings Unheeded: A History of Child Lead Poisoning," American Journal of Public Health, Vol. 79, pp. 1668-1674.

[10] HL Needleman and PJ Landrigan, (1981), p. 279.

[11] Rabin (1989). p. 1673.

[12] Agency for Toxic Substances and Disease Registry (1988), p. II-5.

[13] Centers for Disease Control (1985), p. 7.

Over 40 million houses containing leaded paint are the homes for each successive generation of American children, with 1.97 million of these houses particularly unsound from deteriorating paint.

containing leaded paint are the homes for each successive generation of American children, with 1.97 million of these houses particularly unsound from deteriorating paint.[14]

Lead based paint was also used extensively on exterior applications, and it too presents a hazard. Particles from exterior paint -- along with air borne lead from gasoline and other sources -- can settle on the ground. These particles then mix into the soil and accumulate with other falling particles year after year. A recent study in Oakland, California found that exterior paint had an even higher average lead content than paint on interior walls. The same study identified a strong correlation between the lead levels in a house's exterior paint and the blood lead levels of children residing in that house.[15]

Lead in soil does not come solely from paint. The same sources that have deposited lead in measurable amounts on Greenland's icecap spread dust in the nation's front and back yards each day. In some regions, total soil-lead levels are alarmingly high. For example, a majority of soil samples from Oakland exceeded 1000 parts per million -- a level that defines materials as hazardous waste under California law.[16] The same study also found that a child's blood lead level increased an average 4-5 ug/dL for every 500 ppm rise in front or back yard soil lead level.[17] In light of the emerging consensus among public health experts that blood-lead levels above 10 or 15 ug/dl are associated with significant toxicity, this exposure source is of obvious concern.

Gasoline

Leaded gasoline was once a major source of lead releases, and lead from this source undoubtedly still remains in the soils of virtually every urban and suburban area throughout the country. EPA estimates that the lead level of soil alongside roadways can reach 10,000 ppm, or more than eight times the levels associated with elevated blood lead levels in the Oakland study.[18] Areas alongside heavily traveled urban arteries -- such as sidewalks, parking lots, or street-front playgrounds -- may have even higher levels of lead in soil and dust.[19] Because farm vehicles long ran on leaded gasoline, and are still allowed to do so, lead is widely dispersed into agricultural soils as well.

Data gathered during the first phasedown of leaded gasoline provides remarkable evidence of the strong association between blood lead levels and the use of leaded gas. The Environmental Protection Agency noted in a 1986

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[14] Agency for Toxic Substances and Disease Registry (1988), pp. VI-13, VI-14.

[15] State of California (1989), p. 19.

[16] Ibid.

[17] Ibid.

[18] Environmental Protection Agency (1986a), Vol. IV, p. 13-5.

[19] Ibid., Vol. IV, p. 13-7.

study that ambient air lead levels declined markedly during the changeover to nonleaded gasoline that started in 1978.[20] While other factors were simultaneously at work (e.g., a reduction in the use of leaded solder in food cans), physicians and epidemiologists observed a strong drop in blood lead levels attributable to the changeover. Between 1976 and 1980, the period covered by a national health survey of over nine thousand persons, the average blood lead level of all respondents fell by 5.8 ug/dL, from above to below the CDC's current 25 ug/dL standard (itself soon to be revised as noted above).[21] And in a study of umbilical cord blood lead in 12,000 newborn children in Boston, doctors charted a twenty percent drop in blood lead content between 1979 and 1981.[22]

While sales of leaded gasoline are but a small fraction of what they were at their height, they are nonetheless substantial. Current law still allows the use of leaded gasoline in pre-1975 vehicles, and in farm vehicles of any age.[23] Indeed, leaded gasoline still accounts for about 9 percent of total U.S. gasoline consumption.[24] During 1989, Americans burned approximately 9.8 billion gallons of leaded gasoline containing a total of 880 million grams of lead.[25] Pending amendments to the Clean Air Act, if enacted, would ban the sale of leaded gasoline for motor vehicles by 1991 (with a two year extension available for farm vehicles).[26]

Leaded gasoline still accounts for about 9 percent of total U.S. gasoline consumption.

Drinking water

Lead can enter drinking water in a number of ways. Atmospheric lead, or lead leached from solid waste sites, can accumulate in the water supply. By far the most significant source, however, is lead pipes or lead solder in the plumbing system. Lead pipes may be present in the water main, in the connecting service line, or within the home; lead solder may likewise be found in a variety of locations. Lead is more easily leached by water that is "soft" (has a low mineral content) and/or acidic (has a low pH). However, any water can leach lead, especially water that is hot or that sits in pipes for

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[20] Ibid., Vol. IV, pp. 13-4, 13-6. See also Agency for Toxic Substances and Disease Registry (1988), p. VI-19.

[21] American Academy of Pediatrics (1987), "Statement on Childhood Lead Poisoning," Pediatrics, Vol. 79, p. 458. See also Environmental Protection Agency (1986a), Vol. I, pp. 1-87 through 1-95 for a detailed discussion of associations between blood lead levels and the reduction in use of leaded gasoline.

[22] Bellinger, et al. (1987).

[23] See 42 U.S.C. section 7550.

[24] Data from Environmental Protection Agency, "Lead in Gasoline," for the quarter 1 July 1989 to 30 September 1989.

[25] Ibid. (extrapolated from quarterly data to annual basis).

[26] S. 1630, 101st Cong., 1st Sess. section 216 (1989). Section 211 of the bill requires EPA to issue regulations prohibiting the sale of vehicle engines that require leaded gasoline as of 1992. This provision is designed to curtail production of farm machinery and small gasoline-powered engines,

extended periods such as overnight. And, by contrast to paint-associated exposures that primarily occur in pre-1960 homes, recently constructed or renovated housing may pose a problem because plumbing with freshly soldered joints can leach high levels of lead if lead solder was used.[27]

Recently constructed or renovated housing may pose a problem because plumbing with freshly soldered joints can leach high levels of lead if lead solder was used.

Food

Adults consume an estimated 32-45 micrograms of lead each day through food. This is the major route by which lead enters adult bodies.[28] Lead contaminates food through a variety of routes. Atmospheric lead can land on crops at any stage of growth, harvest or food preparation. Lead-contaminated water used in food preparation or cooking can contribute lead to the food. Finally, the same household lead dust that gets on children's hands and faces can get into food during preparation.

Lead also enters food from lead-soldered cans. In 1979, more than ninety percent of food cans had lead-soldered seams.[29] Subsequently, the Food and Drug Administration has been working with the National Food Processors Association, the Can Manufacturers Institute, and the major can manufacturers on a "voluntary phaseout" of leaded solder.[30] According to industry statistics, the number of cans with leaded solder in 1988 fell to 5.8 percent of total U.S. production.[31] However, recent tests of canned tomato products by an independent consumers group found that about *one quarter of the cans tested contained lead-soldered seams*.[32] Even using the industry data, the problem remains extensive: Americans purchase more than twenty-eight billion cans of food and beverages yearly,[33] which at the 5.8 percent rate leaves more than 1.6 billion leaded cans passing through the nation's grocery stores each year. The FDA effort, moreover, does not apply to imported canned foods.

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which are still being manufactured to run on leaded gasoline. S. Rep. No. 101-228, 101st Cong., 1st Sess. 105 (1989).

[27] Agency for Toxic Substances and Disease Registry (1988), p. VI-37.

[28] Environmental Protection Agency (1986a), Vol. IV, p. 13-7.

[29] Agency for Toxic Substances and Disease Registry (1988), p. I-30. The statistics in this paragraph refer to food cans only; since 1983, soft drink cans have been made entirely without leaded solder. [Source: Food and Drug Administration.]

[30] Interview with Mr. Pat Lombardo, Associate Director for Contaminants, Division of Contaminants Chemistry, Center for Food Safety and Applied Nutrition, Food and Drug Administration.

[31] Can Manufacturers Institute, letter dated March 28, 1989, to Mr. Jerry Burke, Acting Director, Office of Physical Sciences, Center for Food Safety and Applied Nutrition, Food and Drug Administration.

[32] Consumer Reports, July 1989, p. 473.

[33] Source: Can Manufacturers Institute.

Continuing Uses of Lead in Commerce

Currently, U.S. industries introduce approximately 1 million tons of lead into the economy each year. By far the largest use for lead today is in automobile starting/lighting/ignition batteries, with over 70% of lead devoted to that one use.[34] But a wide variety of other products also continue to be made with lead.

Many workers experience occupational exposures well above government standards, as do communities near primary and secondary smelters.[35] Individuals living in the vicinity of such plants -- or of formerly operating plants -- may receive particularly high lead exposures.[36]

These continuing uses of lead are supported by federal tax policy: domestically mined lead has long benefitted from a 22% depletion allowance, and continues to do so today.[37] Interestingly, though lead consumption in the United States has decreased over the past decade, it has increased throughout much of the rest of the world.[38]

An estimated 138,000 tons of automobile battery lead was discarded in 1989.

Auto batteries: The principal public health threat from lead in batteries stems from their manufacture and disposal, rather than from their use. While 80% of automobile batteries are recycled, the remainder forms the predominant source -- about 65% -- of the lead in municipal garbage, and an estimated 138,000 tons of automobile battery lead was discarded in 1989.[39] Although States have begun enacting mandatory recycling laws for auto batteries, there are no federal regulations affecting disposal by individual consumers; batteries continue to be dumped in the open, placed in landfills, and incinerated under conditions that allow environmental release of lead to occur.[40]

Other: Other continuing uses of lead in commerce include ammunition, brass, coverings for power and communication cables, in glass (primar-

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[34] Bureau of Mines (1989b), p. 5, Table 5 (1988 annual data). This category includes other large storage batteries as well, such as for other vehicles and computers.

[35] See Bureau of Mines (1988), Impact of Existing and Proposed Regulations on the Domestic Lead Industry, pp. 10-13.

[36] Environmental Protection Agency (1986a), Vol. I, p. 1-49 (noting residence near a lead smelter as the single highest-intensity pathway for lead exposure by children).

[37] 26 U.S.C. section 613.

[38] Bureau of Mines (1990b), "Mineral Industry Surveys: Lead Industry in November 1989," p. 1 (citing statistics from the International Lead and Zinc Study group indicating the lead metal consumption in the "western world" increased for a seventh consecutive year to a record 4.41 million tons).

[39] Environmental Protection Agency (1989b), Characterization of Products Containing Lead and Cadmium in Municipal Solid Waste in the United States, 1970 to 2000: Final Report, (Washington: U.S. Environmental Protection Agency), Doc No. EPA/530-SW-89-015A), pp. 1 & 81.

[40] Recently introduced federal legislation would bar disposal and instead mandate recycling of all lead-acid batteries. H.R. 3735, section 107, 101st Cong., 1st Sess. (1989).

ily for color TV tubes), pipes and other extruded products, radiation shielding, and sheet lead as a sound-insulation material.[41] Other uses included solders, pigments, and plastics.[42] As with batteries, when lead-containing consumer items are discarded into municipal incinerators or landfills, that lead may be released into the environment.

Finally, it is worth noting that although lead paint has been banned for household use, it still has many other applications. Lead paint's durability and its rust inhibiting qualities have made it popular for traffic paints and for outdoor installations such as bridges, and it is still used extensively for those purposes.

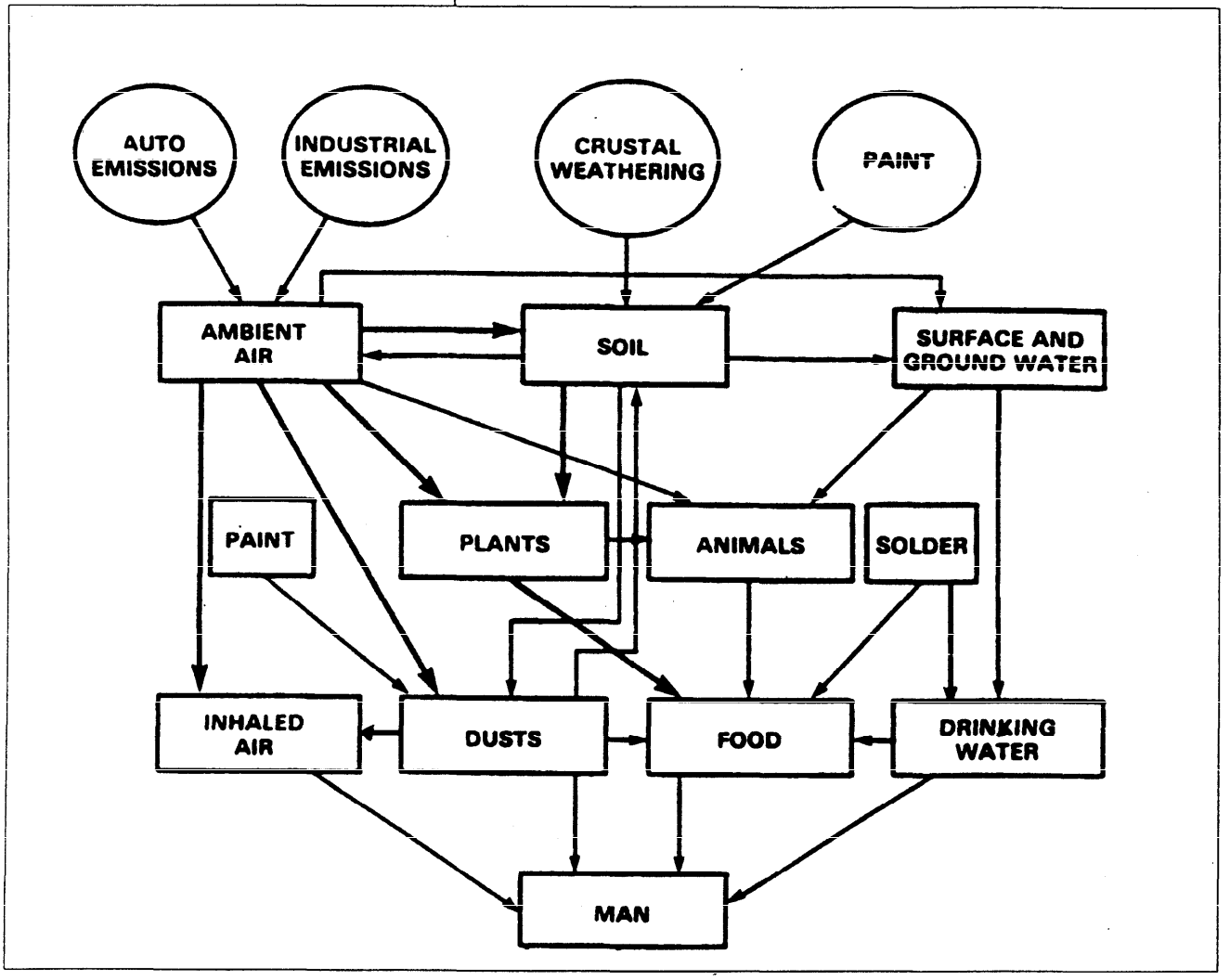

Figure V. Environmental pathways of lead. Adapted from Environmental Protection Agency (1986), Vol. I, p. I-12.

[41] Bureau of Mines (1989b), p. 4, Table 5.

[42] Environmental Protection Agency (1989b).