

AFFIDAVIT OF JOHN F. ROSEN, M.D.

STATE OF NEW YORK)
) SS.
COUNTY OF BRONX)

JOHN F. ROSEN, M.D., being duly sworn, states as follows
under oath:

1. I am Professor of Pediatrics and Head of the Division of Pediatric Metabolism at Albert Einstein College of Medicine; Attending Pediatrician at Montefiore Medical Center, Bronx, New York; and Chairperson of the Childhood Lead Poisoning Prevention Advisory Committee of the Centers of Disease Control (CDC), United States Department of Health and Human Services. I have also been appointed to a committee formed by the National Academy of Sciences that is writing an extensive report on measuring lead exposure in sensitive populations. My statements in this Affidavit are based on my own personal knowledge.

PROFESSIONAL BACKGROUND

2. My entire professional career has been dedicated to research and writing on mineral and heavy metal metabolism. In particular, my research focuses on the pathophysiology, metabolism, and consequences of lead toxicity in children.

3. Since 1984, I have served as Chairperson of CDC's Childhood Lead Poisoning Prevention Advisory Committee. The Centers' Statement, Preventing Lead Poisoning in Young Children, issued in 1985 and again in 1991, sets forth the standards of the medical profession and the United States Public Health Service on the medical testing for and management of lead poisoning and is

recognized as the definitive federal statement on prevention and treatment of lead poisoning in children. The Public Health Service issues this statement every 6-7 years.

4. In October 1991 our CDC Committee issued a new statement on Preventing Lead Poisoning in Young Children, attached as Exhibit 1 to the Declaration of Sue Binder, M.D. As stated in the Preface to that statement, "it reflects the vision expressed in the Department of Health and Human Services' Strategic Plan for the Elimination of Childhood Lead Poisoning, which calls for a concerted, coordinated societywide effort to eliminate this disease." U.S. Dep't of Health & Human Services, Pub. Health Serv., Centers for Disease Control, Preventing Lead Poisoning in Young Children iii (Oct. 1991) (CDC Statement). The Strategic Plan (Feb. 1991) is attached as Exhibit B. I served as a Peer Reviewer of that federal report.

5. I also served as a Peer Reviewer for the Agency for Toxic Substances and Disease Registry (ATSDR) of the Public Health Service. Pursuant to § 118(f) of the Superfund Amendments and Reauthorization Act (SARA) of 1986, 42 U.S.C. § 9618(f), ATSDR was required to prepare a study of lead poisoning in children. This report, titled The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress (ATSDR Report), was completed and submitted in July 1988. The Report's most salient conclusions are that childhood lead poisoning is a pervasive disease; shockingly, it is the most common preventable childhood disease. Over 5,000,000 children in

the United States were found at high risk of lead poisoning. The report further emphasizes that lead at a level once considered safe does impair neurobehavioral and cognitive development in children. (The ATSDR Report is attached as an Exhibit to the accompanying Declaration of Paul Mushak, Ph.D.)

6. Also at the federal level, I served on the Peer Review Panel for the United States Environmental Protection Agency's Lead Criteria Document and as a consultant and author for the agency's Air Lead Quality Criteria Document.

7. At the Division of Pediatric Metabolism of the Albert Einstein College of Medicine and Montefiore Medical Center, I am the Director of the Lead Poisoning Prevention Project, which tests and treats approximately 10,000 children annually for lead toxicity. The vast majority of these patients are Medicaid recipients. The Division assesses the sources of this disease in our patients, and we provide medical, environmental, and nutritional intervention. Twelve years ago we also set up our own laboratory capacity and reporting system to handle the blood lead samples from our Lead Poisoning Prevention Project Clinic.

8. In addition to my contributions to the recent federal reports mentioned above, my most recent studies and publications include the following. CDC used one of these studies in the Strategic Plan's cost-benefit analysis. Sequential Measurements of Bone Lead Content by X-Ray Fluorescence in CaNa₂EDTA-Treated Lead-Toxic Children, 93 *Envtl. Health Perspectives* 271 (1991). This study showed that when children were detected with high

blood lead levels and then removed from leaded environments or returned to lead-abated environments, significant accumulation of lead in the children's bodies ended. Id. at 274. The children in this study were representative of the majority of children attending lead-toxicity programs nationally. Id. at 273. An extension of that study is now in press, to be published in the medical journal Neurotoxicology. Trends in the Management of Childhood Lead Poisoning. This further demonstrates the effectiveness of environmental intervention alone, without costly medical intervention, if lead poisoning is detected early.

9. CDC restricted its cost-benefit calculation to children whose blood lead levels are held to 25 $\mu\text{g.}/\text{dl.}$, even though additional benefits are now conclusively known at lower blood lead levels. Another of my recent publications draws together and analyzes all the recent, rigorously performed studies that all converge on the strong, unequivocal conclusion: No blood lead threshold is too low to be disassociated with the adverse effects on IQ and cognitive and central nervous system functioning. Health Effects of Lead at Low Exposure Levels: Expert Consensus and Rationale for Lowering the Definition of Childhood Lead Poisoning, 146 Am. J. Dis. Child. 1278 (1992). Lead exposure well below 10 $\mu\text{g.}/\text{dl.}$ is directly related to neurobehavioral and cognitive deficits. The impairments in intellectual performance are currently considered irreversible. These consistent findings result in the following societal effects: at least 50% more children falling into the borderline

IQ range and an absence of children in the superior range.

10. I have authored numerous other articles on the etiology and treatment of lead toxicity in children. These articles include: Rosen, Metabolic and Cellular Effects of Lead: A Guide to Low-Level Lead Toxicity in Children, Dietary and Environmental Lead Exposure 157-85 (K. Mahaffey ed. 1985); Piomelli, Rosen & Chisolm, Treatment Guidelines for the Management of Childhood Lead Poisoning, 105 J. Pediatrics 523 (1984); and Rosen, The Metabolism and Subclinical Effects of Lead in Children, Biogeochemistry of Lead in the Environment 151-72 (J. Nriagu ed. 1978). Articles reporting my original research on the effects of lead toxicity in children include Markowitz & Rosen, Assessment of Lead Stores in Children: Validation of an 8-Hour CaNa₂-EDTA Provocation Test, 104 J. Pediatrics 337 (1984), and Saenger, Rosen & Markowitz, Diagnostic Significance of Edetate Disodium Calcium Testing in Children with Increased Lead Absorption, 136 Am. J. Dis. Child. 312 (1983). Several other articles are listed in my curriculum vitae relating to the pathophysiology of lead toxicity.

11. I am a Fellow of the American Academy of Pediatrics and a member of the American Pediatric Society, the New York Academy of Sciences, the Society for Pediatric Research, the Society of Toxicology, and the American Institute of Nutrition. I graduated from Harvard College in 1957 and the Columbia University College of Physicians and Surgeons in 1961. I attach a copy of my curriculum vitae to this affidavit as Exhibit A.

THE RISK OF LEAD POISONING

12. As can be gleaned from all the publications and studies cited above, lead particles ingested by the human body cause severe damage to the brain and central nervous system. Children from birth through age six years are by definition at risk of lead poisoning. Their normal hand-to-mouth activity causes more frequent ingestion of such particles. More significantly, children's brains and nervous systems are particularly vulnerable in their developmental stages. Environmental factors may cause older children to be at risk also. See ATSDR Report 1, 10, III-12, IV-7; CDC Statement 7-12.

13. Ingestion of lead particles by pregnant women also causes damage to the developing fetus. The 1988 ATSDR Report estimated that nationwide, 400,000 fetuses per year are at risk of being born lead poisoned. ATSDR Report 16. It should be noted that all the statistics on frequency of lead poisoning before 1992 are based on a lead poisoning threshold of 25 $\mu\text{g.}/\text{dl.}$ In 1991, CDC lowered the threshold to 10 $\mu\text{g.}/\text{dl.}$ CDC Statement 1. Thus many more children are considered at risk today.

14. Lead paint is the greatest source of lead exposure to young children. Id. at 12. The problem stems primarily from peeling or chalking lead paint in aging or damaged housing. Id. at 18. For much of this century lead paint was used in homes on both exterior and interior surfaces. Although the federal government virtually prohibited the manufacture of household lead paint in 1977 (allowing only a narrow class of exemptions), lead

paint remains in more than 57,000,000 households nationwide; 74% of all homes constructed before 1980 contain lead paint. 16 C.F.R. pt. 1303; CDC Statement 18.

15. Because of these risks, and because lead poisoning's effects are so severe--impacting all the skills critical for educational success--children age 0-6 years, the age range most at risk, should be tested for lead poisoning a minimum of once per year. Environmental and other risk factors may require more frequent testing.

16. In the majority of cases, lead poisoning is asymptomatic. In the rare instances when symptoms are present, they are the same as many other childhood diseases and impairments. Severe cases cause coma, convulsions, kidney damage, and even death. According to currently published data, all cases cause some irreversible brain damage, reduced IQ, delayed cognitive development, and other physical and mental impairments. Such effects occur at blood lead levels above and even below 10 $\mu\text{g.}/\text{dl.}$ In fact there is no apparent threshold. Lead causes adverse effects even below 2 $\mu\text{g.}/\text{dl.}$

17. Lead toxicity produces altered behavior such as attention disorders, learning disabilities, and cognitive disturbances. Symptoms and signs of severe toxicity are fatigue, pallor, malaise, loss of appetite, irritability, sleep disturbance, sudden behavioral change, and developmental regression. Even more serious symptoms include clumsiness, ataxia, weakness, abdominal pain, constipation, vomiting, and

changes in consciousness (coma) due to early encephalopathy, which may lead to death. Mental retardation is a frequent result. See CDC Statement 7-15.

DETECTING LEAD POISONING

18. The only way to determine the presence of lead poisoning is to test for the level of lead in the blood. According to the 1991 CDC Statement, only direct measurement of the blood lead concentration has the clinical and diagnostic capability to determine whether a child has lead poisoning. See id. at 41.

19. Verbal questioning about a child's possible exposure to lead cannot elicit complete, definitive, or accurate data to indicate a child's blood lead level. Many patients, especially Medicaid recipients, simply may not know all the answers to the verbal assessment questions. Furthermore, medical histories are not substitutes for clinical laboratory measurements, when such measurements by definition have the capability to establish a medical diagnosis.

20. A blood test that does not test for the level of lead in the blood does not elicit complete, definitive, or accurate enough data either. The erythrocyte protoporphyrin (EP) test is such a test. The federal Health Care Financing Administration (HCFA) concedes the EP test is not sensitive to blood lead levels under 25 $\mu\text{g.}/\text{dl.}$, and therefore the EP test will miss all children at risk for elevated blood lead levels between 10 and 25 $\mu\text{g.}/\text{dl.}$

21. The EP test not only fails to detect levels of lead in blood below 25 $\mu\text{g.}/\text{dl.}$; the EP test also fails to detect blood lead concentrations above 25 $\mu\text{g.}/\text{dl.}$ This is because the EP test does not test for the level of lead in blood at all. In fact, it is only an indirect reflection of iron or lead status. For lead status, this test is an insensitive index of any blood lead values below 40 $\mu\text{g.}/\text{dl.}$ At blood lead values between 10 and 30 $\mu\text{g.}/\text{dl.}$, EP measurements will totally fail as a screening method to identify the overwhelming majority (over 95%) of children whose blood lead levels fall in this danger zone. As a result, the EP test only identifies a child with an elevated blood lead in about one out of seven or eight cases.

22. In our clinic alone, where we perform blood lead tests and EP tests together, we have seen a significant number of cases where the EP test result was normal, and the blood lead level was above 25 $\mu\text{g.}/\text{dl.}$ This is not only a known occurrence in our own Lead Poisoning Prevention Program; such occurrences were demonstrated nationally by the Second National Health and Nutrition Examination Survey (NHANES II), in 1976-80. See, e.g., ATSDR Report V-31.

23. For a pediatrician pursuing a diagnosis, the only responsible course of action is to carry out a test that provides safe and definitive results for all children, according to current standards of pediatric practice. A blood lead test provides definitive information concerning to what extent a child has been recently exposed to lead. An EP test provides, at best,

marginal information only for the one child out of seven or eight indicating that a child may be lead poisoned. For a pediatrician pursuing a conclusive diagnosis, to perform such an unreliable test for lead poisoning is irresponsible pediatric practice.

24. Unfortunately average American pediatricians must be encouraged to engage in responsible practice regarding lead poisoning, because many are unfamiliar with this area of practice. That being so, everything must be done to safeguard children's health, rather than to give license to do otherwise. The 1991 CDC Statement explicitly recognizes this point. The Statement was prepared with primary medical care providers foremost in mind, so they could glean the current, essential standards of pediatric practice in a readily digestible form. See especially CDC Statement ch. 4 & app. II.

THE RESOURCES FOR BLOOD LEAD TESTING

25. I cannot fathom why, more than a year after the CDC Statement dictated blood lead tests as the reasonable standard of medical practice, a state's medical care providers' capacity for performing the tests, particularly if reimbursable under the Medicaid program, would be limited.

26. The laboratory capacity certainly exists among the large commercial laboratories, such as Roche, MetPath, or Smith Kline. Similar capacity must exist at state and local public laboratories. The large commercial laboratories are located around the country and routinely assess blood samples of Medicaid patients in-state or out-of-state. These laboratories can

process the blood lead results of any children in the Medicaid program and be reimbursed by the Medicaid program. See Declaration of William Hiscock ¶¶ 15, 18.

27. CDC's 1991 Statement did allow a "grace period" for transition from EP to blood lead testing. As Chairperson of CDC's Advisory Committee, I can state that our intent was to provide a transition of up to a year for state and local agencies to order proper equipment and implement blood lead measurements as the diagnostic test for childhood lead poisoning. One year is more than twice the time required to mount such a laboratory capability de novo, including the time required to order an instrument, train a technician, and set up a reporting system. The transition period was not intended to be a bureaucratic vacation to extend until the next CDC Statement is issued. The next CDC Statement, like the last, may not be issued for another six years or more.

28. If CDC envisioned a further transition period to universal blood lead testing anywhere, it would be outside the Medicaid program, where reimbursement may not be available.

29. Although HCFA's policy to reimburse any blood lead tests that are performed eliminates cost as a factor, blood lead testing is not expensive anyway. Throughout the Montefiore Medical Center-Albert Einstein College of Medicine complex, including all their satellite clinics, we have established that the total cost per blood lead test does not exceed \$12-13. Colleagues' findings in Pennsylvania, West Virginia, and North

Carolina confirm these results. One need only check the rates from the commercial mass laboratories to find their charges in the same range.

30. Even if these nationwide resources were somehow not available in a particular area, it is not overly difficult or costly to develop independent laboratory capacity, as we did years ago at Montefiore Medical Center. The laboratory can be staffed with a laboratory technician and a clerk-typist with a computer. The required instrumentation currently runs approximately \$60,000-75,000, plus \$10,000 in consumables per year. This country certainly has the technology to perform universal blood lead tests for all Medicaid eligible children.

THE RISKS FOR MEDICAID ELIGIBLE CHILDREN IN PARTICULAR DICTATE BLOOD LEAD TESTING.

31. Medicaid eligible children are virtually by definition at high risk for lead poisoning. Their low income by itself is an indicator that they live in older, deteriorated or recently rehabilitated housing that has exposed them to lead paint. The ATSDR Report shows the incidence of lead poisoning is 2-3 times higher among Medicaid eligible children than among the same aged children in general. HCFA concedes this point. Hiscock Declaration ¶ 20.

32. Lead poisoning is the most common, preventable childhood disease. For Medicaid children, even in an urban setting, visits to the doctor may be limited. These factors make it all the more nonsensical not to require the proper blood lead test for all Medicaid children, when they do visit a doctor, and

when we know how to detect the onset of this most common preventable disease in the pediatric age group.

33. Too much attention is being paid to the possibility of risk when among this population there is only likelihood, not possibility, of exposure to lead paint or other sources of lead. Under these circumstances anything other than a blood lead test, like the EP test, for lead testing, is a waste of time and money for the Medicaid program. Even more importantly, the alternatives present unacceptable clinical and medical risks of not arriving at the correct diagnosis of lead poisoning in Medicaid eligible children.

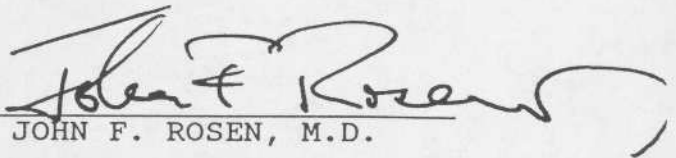
34. Furthermore, as Chairperson of CDC's Advisory Committee, I can state that it was not our intent to suggest to Medicaid providers that the use of a questionnaire should determine whether children, particularly in the most vulnerable age group of 6-36 months, should receive a blood lead test. The CDC Statement is unequivocal that for this population, a blood lead test must be conducted at least once by the time a child reaches 12 months and again by 24 months of age, even if the responses to the assessment questions indicate the child is at "low risk" of lead poisoning. CDC Statement 43. The CDC screening schedule is based on the fact that children's blood lead levels increase most rapidly at 6-12 months and peak at 18-24 months. Id. at 42. The incidence of high blood lead levels among this population is so high, it makes little sense to try to ascertain with a questionnaire who within this group is at "high

risk." See id. at Figure 6-1. The CDC Statement explicitly states that for this group the questionnaire is not a substitute for a blood lead test. Id. at 42.

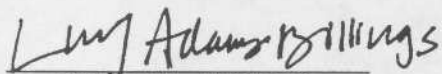
35. HCFA states that its revised guidelines incorporate CDC's methodology of using verbal assessments to determine risk categories and in this regard its methodology is consistent with the 1991 CDC Statement. See Hiscock Declaration ¶ 14. Contrary to Mr. Hiscock's statement, and as stated above, the use of questionnaires to determine risk for children age 6-36 months is indisputably inconsistent with the CDC Statement.

36. Clearly the current state of medical knowledge and standards of pediatric practice dictate measurements of blood lead levels on a frequent periodic basis for all Medicaid eligible children. Moreover, if the Medicaid Early Periodic Screening, Diagnosis and Treatment program is supposed to be preventive, then it is only proper pediatric medical practice to err on the side of considering all Medicaid children at high risk. If Congress' 1989 amendment was intended to remedy HCFA's past failures to require blood lead tests, then clearly the blood lead test should be given to all Medicaid children who fall within the age range at risk, birth through age six. Older

children should also be tested if they have not been previously tested or are otherwise at risk.


JOHN F. ROSEN, M.D.

Sworn to before me
on November 18, 1992


NOTARY PUBLIC

My Comm. Expires
24, 1993

ROSEN
EXHIBIT A

10/1/82

CURRICULUM VITAE

JOHN FRIESNER ROSEN, M.D.

BORN: JUNE 3, 1935, NEW YORK CITY

EDUCATION:

Harvard College, 1953-1957, B.A.

Columbia University College of Physicians and Surgeons
1957-1961, M.D.

POST GRADUATE TRAINING:

Montefiore Hospital and Medical Center
1961-1962, Internship

Columbia-Presbyterian Medical Center
1962-1965, Resident in Pediatrics (Babies Hospital)

Rockefeller University, 1965-1967, Guest Investigator (Post
Doctoral Fellow) (Mineral Metabolism and Peptide Chemistry)

Intern - Montefiore Hospital and Medical Center, 1961-1962

Junior Resident - Babies Hospital, New York City, 1962-1964

Senior Resident - Babies Hospital, New York City, 1964-1965

PROFESSIONAL EMPLOYMENT AND HOSPITAL APPOINTMENTS:

Assistant Physician - The Rockefeller University, 1965-1969

Guest Investigator - The Rockefeller University, 1965-1967

Research Associate - The Rockefeller University, 1967-1969

Research Collaborator - Brookhaven National Laboratory
(Departments of Medicine and Physics), 1975-Present

Chairman, Research Advisory Committee - Tandem - Van de
Graaff Facility, Brookhaven National Laboratory
Department of Physics), 1979-Present

Director, Metabolism Services
Montefiore Medical Center, 1969-Present

Head, Division of Pediatric Metabolism, Albert Einstein
College of Medicine, 1980-Present

Adjunct Attending Physician - Montefiore Hospital and Medical
Center, 1969-1974

Associate Attending Pediatrician - Montefiore Hospital and
Medical Center, 1974-1978

Attending Pediatrician - Montefiore Hospital and Medical Center, 1978-Present

Assistant Professor of Pediatrics - Albert Einstein College of Medicine, 1969-1975

Associate Professor of Pediatrics - Albert Einstein College of Medicine, 1975-1980

Professor of Pediatrics - Albert Einstein College of Medicine, 1980-Present

BOARD CERTIFICATION: Diplomate, American Board of Pediatrics, 1966

PROFESSIONAL SOCIETY MEMBERSHIPS:

American Chemical Society, 1967-Present

Sigma XI, 1967-Present

American Association for the Advancement of Science, 1967-Present

American Federation for Clinical Research, 1969-Present

Fellow of the American Academy of Pediatrics, 1966-Present

Harvey Society, 1966-Present

New York Academy of Sciences, 1971-Present

Society for Pediatric Research, 1972-Present

Lawson Wilkins Pediatric Endocrine Society, 1975-Present

American Pediatric Society, 1979-Present

American Institute of Nutrition, 1979-Present

American Society for Bone and Mineral Research, 1979-Present

Society of Toxicology, 1984-Present

OTHER PROFESSIONAL ACTIVITIES:

Research Committee - Montefiore Hospital and Medical Center, 1980-Present

Committee on Appointments and Promotions to Rank of Full Professor - Albert Einstein College of Medicine, 1982-1984

Peer Review Panel, Health Effects Chapters, Lead Criteria Document, EPA - 1982-1984

Consultant and Author, E.P.A. (Washington). Writing of Air Lead Quality Criteria Document - 1981, 1985.

Ad Hoc Member, Toxicology Study Section, Division of Research Grants, N.I.H. - 1982-1984

Chairman, Centers for Disease Control Advisory Committee on Childhood Lead Poisoning Prevention. CDC, 1984

Member, Toxicology Study Section, Division of Research Grants, N.I.H., 1985-1989

Member, National Academy of Science, National Research Council
Committee on Low Level Exposure in Susceptible Populations.
1989-

Chairman, Centers for Disease Control Advisory Committee on
Childhood Lead Poisoning Prevention. CDC, 1990-

Peer Reviewer, Centers for Disease Control's Strategic Plan
For The Elimination Of Childhood Lead Poisoning, 1991.

CURRENT GRANT SUPPORT:

1. The metabolism of lead in bone.
NIH #ES 01060-12-16
Dr. J.F. Rosen - Principal Investigator
12/01/86-11/30/96 (MERIT AWARD)
2. Treatment outcomes in moderately lead toxic children.
NIH #ES 04039-02-06
Dr. J.F. Rosen - Principal Investigator
3/1/86-4/30/92 (Renewed to 1997)
3. A Nutritional Survey in Homeless Children.
Diamond Foundation
Dr. J.F. Rosen - Principal Investigator
1988-1992 (Renewed to 1994)
4. Lead Poisoning Prevention Project.
Aron/JC Penney and Robert Wood Johnson Foundations
Dr. John F. Rosen - Principal Investigator
1987-1992 (Renewed to 1994)
5. MERIT AWARDEE of the National Institute of Environmental
Health Sciences - 1986-1996 (ES 01060)
6. SAFE House (Transition Housing) For Successfully Treated
Lead Poisoned Children and Their Families.
Robert Wood Johnson Foundation
Dr. John F. Rosen - Principal Investigator - 1990-1993.

REVIEWER FOR:

American Journal of Physiology
Annals of Internal Medicine
Journal of Clinical Endocrinology and Metabolism
Journal of Laboratory and Clinical Medicine
Journal of Neurochemistry
Journal of Pediatrics
Life Sciences
New England Journal of Medicine
Pediatric Research
Pediatrics
Science
Toxicology and Applied Pharmacology

ARTICLES: (Selected)

1. Haymovits, A.H. and Rosen, J.F.: Human thyrocalcitonin. Endocrinology 81:993-1000, 1967.
2. Rosen, J.F., and Haymovits, A.H.: Liver lysosomes in congenital osteopetrosis: A study of lysosomal function, calcitonin, parathyroid hormone, and 3',5' AMP. J. Peds. 81:518-527, 1972.
3. Rosen, J.F. and Finberg, L.: Vitamin D dependent rickets: Actions of parathyroid hormone and 25-hydroxycholecalciferol. Ped. Res. 6:552-562, 1972.
4. Rosen, J.F.: The microdetermination of blood lead in children by flameless atomic absorption. The carbon rod atomizer. J. Lab. and Clin. Med. 80:567-576, 1972.
5. Rosen, J.F. and Finberg, L.: Vitamin D dependent rickets: Actions of parathyroid hormone and 25-hydroxycholecalciferol. In, Clinical Aspects of Metabolic Bone Disease. Frame, Parfitt and Duncan (Eds)., Excerpta Medica Foundation, 1973, pp. 388-393.
6. Rosen, J.F.: The microdetermination of blood lead in children by nonflame atomic absorption spectroscopy. In, Proceedings of the Institutional Consortium on Endemic Lead Poisoning. Clinical Toxicology Bulletin 3:111-118, 1973.
7. Daum, F., Rosen, J.F. and Boley, S.J.: Parathyroid adenoma, parathyroid crisis, and acute pancreatitis in an adolescent. J. Peds. 83:275-277, 1973.
8. Rosen, J.F., Zarate-Salvador, C. and Trinidad, E.E.: Plasma lead levels in normal and lead-intoxicated children. J. Peds. 84:45-48, 1974.
9. Lamm, S. and Rosen, J.F.: Lead contamination in milks fed to infants: 1972-1973. Pediatrics 53:137-141, 1974.
10. Rosen, J.F., Roginsky, M., Nathenson, G. and Finberg, L.: 25-hydroxyvitamin D: Plasma levels in mothers and their premature infants with neonatal hypocalcemia. Amer. J. Dis. Child. 127:220-223, 1974.
11. Rosen, J.F. and Trinidad, E.E.: The significance of plasma lead levels in normal and lead-intoxicated children. Environ. Health Perspect. 7:139-144, 1974.
12. Rosen, J.F. and Lamm, S.H.: Further comments on the lead content of milks fed to infants. Pediatrics 53:144-145, 1974.

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14. Daum, F., Rosen, J.F., Roginsky, M., Cohen, M. and Finberg, L.: 25-hydroxycholecalciferol in the management of rickets associated with extrahepatic biliary atresia. J. Peds. 88:1041-1043, 1976.
15. Rosen, J.F. and Wexler, E.E.: Studies of lead transport in bone organ culture. Biochem. Pharm. 26:650-652, 1977.
16. Rosen, J.F. and Sorell, M.: Interactions of lead, calcium, vitamin D, and nutrition in lead-burdened children. In, Clinical Chemistry and Chemical Toxicology of Metals. Brown, S.S. (Ed.), Elsevier, 1977, pp. 27-31.
17. Rosen, J.F., Fleischman, A.R., Finberg, L., Eisman, J. and DeLuca, H.F.: 1,25-dihydroxycholecalciferol: Oral administration and sterol levels in the long-term management of idiopathic hypoparathyroidism in children. In, Vitamin D: Biochemical, Chemical and Clinical Aspects Related to Calcium Metabolism. Norman, A.W. et al (Eds.) Walter de Gruyter, Berlin, 1977, pp. 827-830.
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22. Fleischman, A.R., Rosen, J.F., and Nathenson, G.: Oral 25-hydroxycholecalciferol for the prevention of early neonatal hypocalcemia in premature neonates. Amer. J. Dis. Child. 132:973-977, 1978.
23. Rosen, J.F., Fleischman, A.R., Finberg, L., Hamstra, A., and DeLuca, H.F.: Rickets with alopecia: An inborn error of vitamin D metabolism. J. Peds. 94:729-735, 1979.
24. Fleischman, A.R., Rosen, J.F., Nathenson, G. and Finberg, L.: Oral 25-OHD in preventing neonatal hypocalcemia. In, Pediatric Diseases Related to Calcium. Anast, DeLuca (Eds.), Elsevier, 1980, pp. 345-354.

25. Chesney, R.W., Rosen, J.F., Hamstra, A. and DeLuca, H.F.: Serum 1,25-dihydroxyvitamin D levels in normal children and in vitamin D disorders. *Amer. J. Dis. Child.* 134:135-139, 1980.
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27. Rosen, J.F., and Markowitz, M.: D-Penicillamine: Its actions on lead transport in bone organ culture. *Ped. Res.* 14:330-335, 1980.
28. Fleischman, A.R., Rosen, J.F., Smith, C.M. and DeLuca, H.F.: Maternal and fetal levels of 1,25-dihydroxyvitamin D levels at term. *J. Peds.* 97:640-642, 1980.
29. Sorell, M., Rosen, J.F., Kapoor, N., Kirkpatrick, D., Raju S.K., Chaganti, Good, R.A., and O'Reilly, R.J.: Marrow transplantation for juvenile osteopetrosis. *Amer. J. Med.* 70:1280-1287, 1981.
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EXHIBIT B

STRATEGIC PLAN FOR THE ELIMINATION OF CHILDHOOD LEAD POISONING

Developed for the Risk Management Subcommittee, Committee
to Coordinate Environmental Health and Related Programs,
U.S. Department of Health and Human Services.

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U.S. DEPARTMENT OF HEALTH
AND HUMAN SERVICES
Public Health Service
Centers for Disease Control

*STRATEGIC PLAN FOR THE
ELIMINATION OF CHILDHOOD
LEAD POISONING*

February 1991

STRATEGIC PLAN FOR THE
ELIMINATION OF CHILDHOOD LEAD POISONING

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APPENDICES

- I. Lead exposure and its effects on children and fetuses
- II. Benefits of preventing lead exposure in the United States and costs and benefits of lead-based paint abatement
- III. History of childhood lead poisoning prevention programs
- IV. Organizations and agencies that could help promote awareness of childhood lead poisoning
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PREFACE

Three striking conclusions about childhood lead poisoning have emerged in the past several years: 1) the effects of exposure to even moderate amounts of lead are more pervasive and long-lasting than previously thought, 2) significant impairment of intelligence and neurobehavioral function is being reported at increasingly lower levels of lead in blood, and 3) millions of children in the United States have blood lead levels in this new range of concern. These findings have been reviewed in great detail elsewhere, and they are summarized here. They are not, however, the main subject of this report. The main subject is the public health response to our new understanding of childhood lead poisoning.

In this report, we set forth a strategy for eliminating childhood lead poisoning as a public health problem. Essential actions include increased support of programs that prevent childhood lead poisoning, increased abatement of lead-based paint and paint-contaminated dust in high-risk housing, reductions in other sources and pathways of lead exposure in children, and national surveillance for children with elevated blood lead levels. Finding and treating children with lead poisoning is critical, but not sufficient. Preventive actions must be taken to remove sources of lead in the child's environment before poisoning occurs.

Any plan to eliminate childhood lead poisoning in the United States must address the formidable problems posed by lead-based paint. Lead-based paint abatement has been neither widespread nor effective. Developing an effective, long-term lead-based paint abatement effort is probably the most critical factor in eliminating childhood lead poisoning. In this plan, approaches to developing this effort receive most attention. From a national viewpoint, the relative contribution from different sources of lead for children with high blood lead levels (that is, those with or likely to get lead poisoning) is different from that for children with low or moderate blood lead levels. For children with the highest blood lead levels, lead-based paint is a particularly important source. Strategies will need to be developed to focus abatement efforts on the highest priority groups (especially children with lead poisoning severe enough to require medical intervention, e.g., blood lead levels ≥ 25 ug/dL). Initial screening efforts will also have to be focused on areas where there are the greatest numbers of children with the highest blood lead levels (e.g., ≥ 25 ug/dL).

This plan also calls for reducing lead in other major sources and pathways of exposure. Ongoing regulatory and voluntary protective actions are important and must be strengthened. Lead is widely distributed in water, food, and air, but this lead is less likely to produce lead poisoning than lead in such concentrated sources as lead paint. Reducing the amount of lead in these environmental media, however, can have a profound effect on blood lead levels throughout the entire United States. This was demonstrated when lead was removed from gasoline. Reducing the amount of lead in

water, food, and air would help reduce the prevalence of lead poisoning and would help protect children with blood lead levels below the current definition of lead poisoning from adverse effects.

The role of exposure to soil lead, both directly and through the contribution of soil lead to lead in housedust, is still being investigated. The nature and degree of soil lead abatement that would be appropriate is unclear. The research needed to resolve the soil lead issues will take years. However, since so many children are being poisoned by lead-based paint, significant action on lead-based paint abatement should not be delayed while we await the results of research. Decisions on how to set up rational soil lead abatement programs will have to be made separately as more data become available. (However, it is critical not to further contaminate the soil during lead-based paint abatement efforts.)

We have made substantial progress in reducing exposure to lead; deaths and severe illness from lead poisoning (e.g., encephalopathy) are now rare. The results of recent studies indicate, however, that blood lead levels previously believed to be safe are adversely affecting the health of children. Millions of children in the United States are believed to have blood lead levels high enough to affect intelligence and development. The need to deal with preventing exposure at these lower levels will require increased efforts. The Administration is responding to this problem with increased resources. In FY 1992, the President's budget calls for \$14.95 million for the lead poisoning prevention program at the Centers for Disease Control and \$25 million for the new HOME abatement program of the Department of Housing and Urban Development.

In many ways, the tone of this report is one of understatement. The enormity of the task of eliminating childhood lead poisoning and the extensive public health benefits to be gained are very clear. This strategic plan is at best a first step. More detailed plans for implementation must follow, and then the work itself must be done.

Childhood lead poisoning has already affected millions of children, and it could affect millions more. Its impact on children is real, however silently it damages their brains and limits their abilities. Deciding to develop a strategic plan for the elimination of childhood lead poisoning is a bold step, and achieving the goal would be a great advance.

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STRATEGIC PLAN FOR THE
ELIMINATION OF CHILDHOOD LEAD POISONING

EXECUTIVE SUMMARY

The U.S. Public Health Service Year 1990 and Year 2000 Objectives for the Nation aim for progressive declines in the numbers of lead-poisoned children in the United States, leading to the elimination of this disease. We believe that a concerted society-wide effort could virtually eliminate this disease as a public health problem in 20 years.

This plan, developed for the Committee to Coordinate Environmental Health and Related Programs of the U.S. Department of Health and Human Services, provides an agenda for the first 5 years of a comprehensive society-wide effort to eliminate childhood lead poisoning. The results and experience from this 5-year program will lead to the agenda for the following 15 years.

Lead is a poison that affects virtually every system of the body. Results of recent studies have shown that lead's adverse effects on the fetus and child occur at blood lead levels previously thought to be safe; in fact, if there is a threshold for the adverse effects of lead on the young, it may be close to zero.

Lead poisoning remains the most common and societally devastating environmental disease of young children. Enormous strides have been made in the past 5 to 10 years that have increased our understanding of the damaging, long-term effects of lead on children's intelligence and behavior. Today in the United States, millions of children from all geographic areas and socioeconomic strata have lead levels high enough to cause adverse health effects. Poor, minority children in the inner cities, who are already disadvantaged by inadequate nutrition and other factors, are particularly vulnerable to this disease.

Childhood lead exposure costs the United States billions of dollars from medical and special education costs for poisoned children, decreased future earnings, and mortality of newborns from intrauterine exposure to lead. Childhood lead poisoning continues in our society primarily because of lead exposure in the home environment, with lead-based paint being the principal high-dose source. It is the most important source for the highest-risk children (e.g., those with blood lead levels ≥ 25 ug/dL); preventive actions for such exposures should receive the highest priority.

Federal regulatory actions have significantly reduced or eliminated lead from many consumer products, including new paint and gasoline. Federal agencies continue to take actions further to reduce lead exposure from water, food, soil, air, and the workplace. Unfortunately, we are making little progress in eliminating the major source of high-dose lead poisoning, leaded paint from older housing.

In a new benefits analysis based on data from three studies, we estimate that the abatement of lead from all pre-1950 housing containing lead-based paint over the next 20 years would result in societal benefits of \$62 billion. This anticipated economic benefit is an additional incentive to society, since even if no economic benefits of abatement could be demonstrated, prevention of childhood lead poisoning would still be a worthwhile public health activity.

This plan contains recommendations for program and research activities. The four immediately essential elements of this effort are:

- 1) Increased childhood lead poisoning prevention programs and activities.
- 2) Effective abatement of leaded paint and lead paint-contaminated dust in high-risk housing.
- 3) Continued reduction of children's exposure to lead in the environment, particularly from water, food, air, soil, and the workplace.
- 4) Establishment of national surveillance for children with elevated blood lead levels.

Increased childhood lead poisoning prevention activities and national surveillance for elevated lead levels are essential parts of a national strategy to eliminate childhood lead poisoning for several reasons. Children should be screened for elevated blood lead levels so that affected children will receive appropriate medical attention and environmental follow-up. Initially, screening activities must focus on those areas with the greatest prevalence of children with the highest blood lead levels. Screening and surveillance data are also important for defining those areas in greatest need of intensive abatement programs and for evaluating the success of the national abatement program in eliminating this disease in targeted areas.

Effective lead-based paint abatement is essential for the elimination of childhood lead poisoning. Lead-based paint is the most concentrated source of lead to children and, historically, is the source most closely linked to lead poisoning in children. Many sources of lead, for example, food and soil, contribute to overall exposure of U.S. children to lead, but for children with the highest blood lead levels, that is, children with lead poisoning, lead-based paint is of particular importance.

The development of a national strategy to abate lead-based paint is critical to the success of the effort to prevent lead poisoning. At present, far too few homes are being abated. To achieve maximum impact in the shortest time, lead-based paint abatement programs need to be closely linked with public health programs.

We recommend development of a national strategy for lead-based paint abatement that includes actions by both the private and the public sectors. Since the public health benefits and cost-effectiveness of lead-based paint and dust abatement are greatest in the housing most likely to contribute to lead poisoning, in the early years the emphasis should be on abating the housing units of affected children and the units likely to poison children in the near future.

To eliminate completely this disease, however, will require that all housing with lead-based paint eventually be addressed. A prioritized program will allow the highest-risk housing to be abated first, while enhanced programs, infrastructure, and technology continue to be developed. This national lead-based paint abatement program must include an evaluation component to ensure efficacy and safety for occupants as well as workers and their families.

This strategic plan focuses heavily on lead-based paint because of its key role in lead poisoning and because of the limited nature of previous efforts to reduce this source of lead. A national plan to eliminate childhood lead poisoning, however, must also focus on other widespread sources and pathways of lead exposure to children. Lead in water, food, soil, and air, in particular, may affect large numbers of children and may contribute to overall levels of lead in the population. Continued efforts to reduce these sources and pathways of lead exposure will result in lower average blood lead levels in the United States and will thereby further diminish the likelihood of lead poisoning developing even in children exposed to a high-dose source.

Childhood lead poisoning usually does not cause distinctive clinical symptoms, but the effects of childhood lead poisoning on intellectual and neurobehavioral functioning are pronounced and may persist for life. Furthermore, lead poisoning is entirely preventable. We understand the causes of lead poisoning and, most importantly, how to eliminate them. This plan establishes priorities and identifies steps toward that end.

SUMMARY OF CHAPTERS

Chapter 1. Introduction

Lead poisoning, the most common and societally devastating environmental disease of young children, is entirely preventable. We understand the causes of childhood lead poisoning and, most importantly, how to eliminate them. A concerted societal effort could virtually eliminate this disease in 20 years.

Chapter 2. Health Effects of Lead and Lead Exposure

Lead is a dangerous and pervasive environmental poison, particularly harmful to fetuses and young children. The threshold for some of lead's health effects may be close to zero. The Agency for Toxic Substances and Disease Registry (ATSDR) estimated that between 3 and 4 million children in the U.S. (17% of all children) had blood lead levels above 15 ug/dL in 1984, levels high enough to adversely affect intelligence and behavior. Lead in the home environment, principally from lead-based paint, is the major source of lead poisoning. (See Appendix I for more details on the material in this chapter.)

Chapter 3. Benefits of Preventing Lead Exposure of Children and Fetuses

A benefits analysis was performed for this report, taking into account recent data on the effects of lead on children and fetuses. (In addition, an example of a cost-benefit analysis of a national lead-based paint abatement program, along with the detailed benefits analysis, appears in Appendix II.) For this analysis, the benefits of preventing children and fetuses from being exposed to lead are the costs that would have been associated with exposure had it occurred. On the basis of this analysis, the average benefits of preventing a child's blood lead level from exceeding 24 ug/dL (the level at which medical evaluation is necessary) are \$4,631 for avoided medical and special education costs. For all children, including those with blood lead levels below 25 ug/dL, the average increased wages to be expected from preventing each 1 ug/dL increase in a child's blood lead level are \$1,147. The average benefits of preventing a 1 ug/dL increase in the blood lead level of a pregnant woman are \$300. Based on data from three programs (see Appendix II), the benefits of abating all pre-1950 housing with lead-based paint over a 20-year period would be \$62 billion, discounted to the present.

Chapter 4. Program Agenda

The four essential program components of a strategy to eliminate childhood lead poisoning are:

- 1) Increased childhood lead poisoning prevention programs and activities.

- 2) Increased abatement of leaded paint and paint-contaminated dust in housing.
- 3) Continued reduction of children's exposure to lead in the environment, particularly from water, food, air, soil, and the workplace.
- 4) Establishment of national surveillance for children with elevated blood lead levels.

Increased childhood lead poisoning prevention activities include both funding of public lead poisoning prevention programs and increased awareness and action by private physicians. Increased abatement should also result from a combination of efforts by the private and public sectors. Before we can safely and effectively conduct as many abatements as are needed, the infrastructure for abatement must be developed. (Appendix V discusses infrastructure development in more detail.) Other environmental sources of lead should also continue to be addressed as part of the strategic plan; reductions of lead in water, food, soil, air, and the workplace are of most importance. National surveillance for elevated blood lead levels is needed to target areas requiring increased lead poisoning prevention activities and abatement, to track our progress in eliminating childhood lead poisoning, and to evaluate lead exposure in abatement workers and workers in other lead-contaminated environments.

Chapter 5. Research Agenda

Research activities to complement the four essential program components are described in this chapter.

Chapter 6. Funds Needed for Implementation of the Strategic Plan

Significant Federal, State, local, and private resources must be committed to meet the 5-year goals. Preliminary estimates indicate that as much as \$974 million in combined resources may be required to implement the first 5 years of this Strategic Plan.

Chapter 7. Summary of Recommendations

The five most urgent recommendations of this plan include increased prevention activities, increased abatement, reduced exposure to other sources of environmental lead, national surveillance, and research.

INTRODUCTION

- CHILDHOOD LEAD POISONING IS EXTREMELY WIDESPREAD.
- ALTHOUGH SUBSTANTIAL PROGRESS HAS BEEN MADE IN THE PAST 20 YEARS, KEY ENVIRONMENTAL SOURCES OF LEAD REMAIN.
- THIS DOCUMENT PRESENTS A STRATEGIC PLAN FOR THE ELIMINATION OF CHILDHOOD LEAD POISONING.

Lead poisoning remains the most common and societally devastating environmental disease of young children. Millions of U.S. children from all geographic areas and socioeconomic strata have blood lead levels high enough to be associated with adverse health effects. Poor, minority children in the inner cities, who are often already disadvantaged by inadequate nutrition and other factors, are particularly vulnerable to this disease. The pervasiveness of childhood lead poisoning was well described in The Nature and Extent of Childhood Lead Poisoning in Children in the United States: a Report to Congress, prepared by the Agency for Toxic Substances and Disease Registry (ATSDR, 1988).

Childhood lead poisoning is entirely preventable. We understand the causes of lead poisoning and, most importantly, how to eliminate them. We believe that a concerted societal effort could virtually eliminate this disease as a public health problem in 20 years.

Important progress has been made in reducing some sources of lead in the past 20 years. Federal regulatory actions have significantly reduced or eliminated lead from many consumer products, including new paint and gasoline. Voluntary programs, such as the work by the Food and Drug Administration (FDA) with can manufacturers to reduce lead in canned food, have also been highly successful in reducing exposure to lead. Federal agencies continue to take actions to further reduce lead exposure from water, food, air, and the workplace. Unfortunately, limited progress has been made in eliminating lead-based paint from older housing--the major source of high-dose lead poisoning in children. Abatement of lead-painted homes is an essential part of both the prevention of childhood lead poisoning and the treatment of poisoned children.

**LEAD-BASED PAINT ABATEMENT IS AN INTEGRAL PART
OF THE TREATMENT OF CHILDHOOD LEAD POISONING
AND THE PREVENTION OF NEW CASES. WE HAVE MADE
LITTLE PROGRESS IN ELIMINATING LEAD-BASED PAINT
IN OLDER HOMES AS A CAUSE OF CHILDHOOD LEAD
POISONING.**

The lack of progress in eliminating childhood lead poisoning is due to several factors. For example, lead poisoning has been improperly considered by many to be a disease of the poor that could be remedied by better housekeeping and childrearing; another source of confusion is that many people believe the disease was eliminated when the manufacture of lead-based paint for residential use was banned. The logistical difficulties and high costs of abating lead-based paint in homes have also been a major problem.

During the past 20 years, severe, symptomatic lead poisoning in children (e.g., encephalopathy with coma) has been markedly reduced. However, new and increased knowledge and awareness of the health effects of exposure to lead in childhood, especially at lower levels once considered safe, have dramatically increased concern about this problem in recent years. The fact that childhood lead poisoning is a societally devastating, yet totally preventable disease has focused attention on the need for a strategic plan to eliminate it.

**DEATHS AND ACUTE, SEVERE ILLNESS FROM LEAD
POISONING ARE NOW RARE. HOWEVER, WE NOW KNOW
THAT LARGE NUMBERS OF CHILDREN MAY SUFFER
ADVERSE HEALTH EFFECTS AT BLOOD LEAD LEVELS THAT
WERE ONCE CONSIDERED SAFE.**

Several recent government documents have extensively reviewed health and environmental data related to childhood lead exposure (ATSDR, 1988; Environmental Protection Agency, 1986). This strategic plan discusses these data briefly, but focuses on a detailed benefits analysis and major agenda items. The plan consists of chapters on exposure to lead and its effects on children and fetuses, a benefits analysis of reducing lead exposure, a program agenda, a research agenda, and a discussion of the funds needed for implementation. Several appendices provide the background and justification for the material in the plan.

This document has been developed at the request of Dr. James O. Mason, Assistant Secretary for Health, U.S. Department of Health and Human Services, for the Committee to Coordinate Environmental Health and Related Programs. It has been developed with the help of contributors from other Federal, State, and local agencies and the private sector. It does not, however, necessarily reflect the policies of these individuals and agencies.

EFFECTS OF EXPOSURE

- **HEALTH EFFECTS**

LEAD AFFECTS EVERY SYSTEM IN THE BODY.

**EFFECTS ON INTELLIGENCE AND BEHAVIOR
ARE MOST IMPORTANT.**

- **LEAD EXPOSURE**

**CHILDREN ARE EXPOSED TO LEAD FROM
MANY SOURCES AND PATHWAYS.**

**LEAD-BASED PAINT IS THE SOURCE OF
GREATEST CONCERN.**

Lead is an extremely dangerous and pervasive environmental poison. In 1984, at least 3 to 4 million children in the United States (17% of all children) had blood lead levels high enough to cause neurobehavioral and other adverse health effects (ATSDR, 1988). The large number of children with blood lead levels in the toxic range shows that existing environmental lead levels in the United States provide no margin of safety for the protection of children.

The risks of lead exposure are not based on theoretical calculations. They are well known from studies of children themselves and are not extrapolated from data on laboratory animals or high-dose occupational exposures. Whereas conservative approaches are used to estimate risk from low level exposures to many chemicals, especially carcinogens, this is not the case for lead.

HEALTH EFFECTS OF LEAD

ADVERSE EFFECTS OF LEAD ON CHILDREN AND THE FETUS

- Neurobehavioral
 - Decreased intelligence
 - Developmental delays
 - Behavioral disturbances
 - Seizures (at very high levels)
 - Coma (at very high levels)
- Growth
 - Decreased stature
- Endocrinologic
 - Altered vitamin D metabolism
- Hematologic
 - Elevated erythrocyte protoporphyrin levels
 - Anemia
- On the fetus
 - Decreased gestational weight
 - Decreased gestational age
 - Miscarriage and stillbirth (at very high levels)

Lead is a poison that affects virtually every system in the body. It is particularly harmful to the developing brain and nervous system; therefore, lead exposure is especially devastating to fetuses and young children.

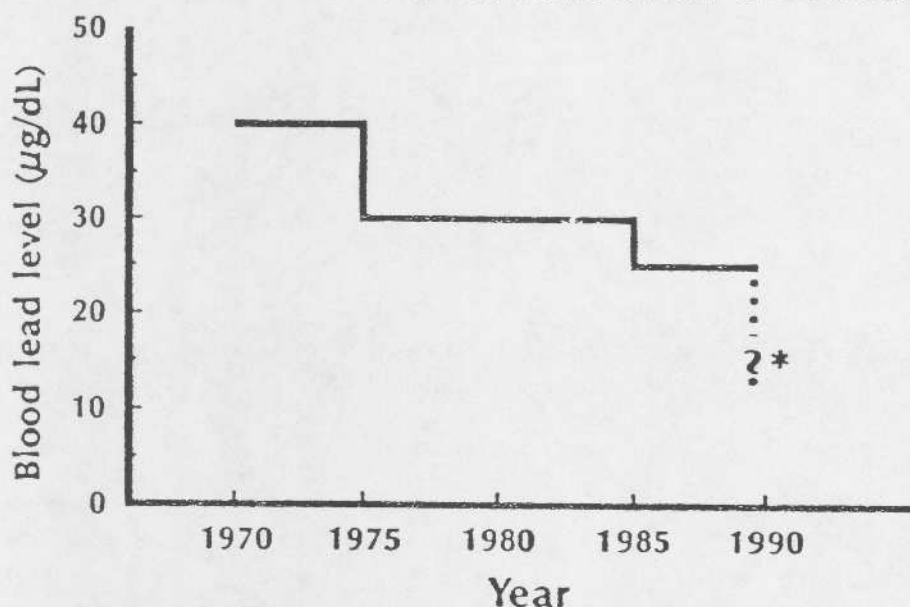
Very severe lead exposure (blood lead levels ≥ 80 ug/dL) can cause coma, convulsions, and even death. It is currently estimated that there are about 250,000 children under 6 years of age whose blood lead is 25 ug/dL and greater. The adverse effects on these children are great. They need to be identified as soon as possible to remove them from the source of lead and provide appropriate medical care. This is the highest priority. Blood lead levels as low as 10 ug/dL, which usually do not cause distinctive symptoms, are associated with decreased intelligence and slower neurobehavioral development. Other effects that begin at blood lead levels as low as 10 ug/dL include behavioral disturbances, reduced stature, and effects on vitamin D metabolism. Maternal and cord blood lead levels of 10 to 15 ug/dL appear to be associated with reduced gestational age

and reduced weight at birth (ATSDR, 1988). Blood lead levels of 10 ug/dL and above at age 2 years have been shown to result in a reduction of the General Cognitive Index at age 57 months. Most of the children studied had blood levels below 15 ug/dL (Bellinger, 1991). Although researchers have not yet completely defined the impact of blood lead levels <10 ug/dL on central nervous system function, it may be that even these levels are associated with adverse effects that will be more clear as our research instruments become better.

The neurobehavioral effects of childhood lead exposure appear to be longlasting.

In a recent long-term follow-up study (Needleman, 1990), for children who had been exposed to moderate lead levels in preschool years, the odds of those children dropping out of high school were seven times higher, and the odds of a significant reading disability were six times higher than for children exposed to lower lead levels. In addition, the children exposed to higher lead levels had lower class standing, increased absenteeism, and lower vocabulary and grammatical-reasoning scores, even after investigators controlled for other covariates. The apparent persistence or irreversibility of many of lead's neurobehavioral effects intensifies concern over exposure of fetuses and children to lead.

Blood lead levels considered elevated by CDC



*Currently undergoing revision

Studies on the health effects of lead over the past 20 years have produced a consistent trend: the more that is learned about lead's effects on children and fetuses, the lower the blood lead level at which adverse effects can be documented. In the first half of the 20th Century, medical care providers were concerned about blood lead levels ≥ 80 ug/dL; by the 1960s, they were concerned about levels ≥ 60 ug/dL; in the 1970s, as studies began showing effects at lower and lower levels, the level of concern was at 40 ug/dL; and by the middle 1980s, it was lowered to 25 ug/dL. A current reassessment will likely place the level at which interventions are recommended at 10-15 ug/dL. Blood lead levels formerly considered safe have now been clearly associated with adverse effects. If there is a threshold for lead's effects on health, it is probably near zero.

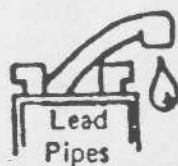
The definition of childhood lead poisoning requires a blood lead level of ≥ 25 $\mu\text{g/dL}$. This definition is being reconsidered and the blood lead level is being revised downward.

The current definition of childhood lead poisoning requires a blood lead level ≥ 25 ug/dL (CDC, 1985). This definition is being reevaluated and, as a result of recent research on the effects of low-level lead exposure in children, it will undoubtedly be lowered to 10-15 ug/dL. A Federal advisory committee is currently meeting and working on these changes.

OTHER SOURCES AND PATHWAYS TO BE ADDRESSED



- Paint
- Gasoline
- Air
- Solder
- Dust
- Stationary sources, like smelters
- Soil
- Water
- Food



Lead has some unusual characteristics that cause special concern about exposure. First, lead deposited in the environment does not biodegrade; it remains there and accumulates. Second, lead exposure is pervasive, sparing no segment of the U.S. populace. Third, lead accumulates over months and years in the bodies of children. Therefore, chronic exposure to small sources of lead can result in a large long-term accumulation in a child, increasing that child's risk of adverse health effects. During pregnancy, a woman's bone lead stores may be mobilized, exposing the fetus to lead. Thus, childhood lead exposures in one generation may result in prenatal exposure in the next generation.

Children are exposed to lead from many sources (for example, paint, gasoline, solder, and stationary sources like smelters) via multiple pathways (for example, air, dust, soil, water, and food). A child's particular environment determines the relative importance of each source and pathway.

Today, lead-based paint is the source of greatest public health concern. It is the most common cause of high-dose lead exposure. Exposure occurs not only when children ingest chips and flakes of paint (which often contain as much as 50 percent lead by weight), but also, and probably more commonly, when children ingest lead-based paint-contaminated dust and soil during normal mouthing activities.

In the mid-1980s, about 13.6 million children under 7 years of age lived in homes with lead-based paint. An estimated 1.8 to 2.0 million children lived in deteriorated lead-painted housing with unsound paint (for example, peeling paint and other damage to walls), which placed them at high risk of excessive lead exposure from this source; about 1.2 million of these children were estimated to have blood lead levels above 15 ug/dL, mainly because of exposure to lead paint (ATSDR, 1988). ATSDR has assessed existing lead paint in U.S. housing and public buildings to be an "untouched and enormously serious problem."

LEAD-BASED PAINT IS THE SOURCE OF GREATEST PUBLIC HEALTH CONCERN. OTHER SOURCES OF LEAD ALSO CAN BE IMPORTANT CONTRIBUTORS TO CHILDREN'S BLOOD LEAD LEVELS.

Estimates of numbers of children exposed to other sources and pathways of lead appear in Appendix I. The removal of lead from gasoline during the last decade, as well as reductions in other widespread sources and pathways such as lead in food, has contributed to a major drop in the mean blood lead levels of children. By lowering the average, or baseline, level of lead in children, the risk of lead poisoning is reduced, even from exposure to concentrated sources such as lead paint, because higher doses are necessary to produce lead poisoning. It is, therefore, important to continue to reduce children's exposure to lead from air, water, food, soil, and the workplace; there will also be occasions where these sources and pathways result in lead poisoning. Efforts to reduce these exposures are not a substitute for lead-based paint abatement, however, because in the geographic areas where lead-based paint and dust are a prominent hazard, they alone can, as noted above, produce childhood lead poisoning.

CHAPTER 3. BENEFITS OF PREVENTING LEAD EXPOSURE OF CHILDREN AND FETUSES (The methods and assumptions on which this benefits analysis are based are detailed in Appendix II. Numerical estimates are included only for those benefits which we believe are defensible by good, quantitative data. Not factored in the benefits analysis are those which are not able to be quantified. Appendix II also contains a detailed cost-benefit analysis, in which the benefits of reducing lead exposure are compared to the costs of lead-based paint abatement, based on the three currently available studies for which we had data both on the costs of abatement and the resultant changes in blood lead levels.)

BENEFITS OF PREVENTING LEAD EXPOSURE

THE BENEFITS WE QUANTIFIED ARE:

- **REDUCED MEDICAL COSTS**
- **REDUCED SPECIAL EDUCATION COSTS**
- **INCREASED FUTURE PRODUCTIVITY**
- **REDUCED INFANT MORTALITY**

Lead exposure in U.S. children is estimated to cost society billions of dollars a year (for example, Levin, 1986). These estimates have included costs of medical care, special education and institutionalization, and decreases in productivity and lifetime earnings resulting from impaired cognition.

For this strategic plan, we have developed a new benefits analysis. The analysis is detailed in Appendix II and is focused on the benefits of preventing exposure to lead in children and fetuses. The benefits of reducing lead exposure in persons already being exposed are likely to be substantial, but they are difficult to quantitate. For example, we do not know how long lead levels must be elevated before a child develops cognitive deficits or before these deficits become irreversible. We, therefore, did not include already exposed individuals in the main benefits analysis.

For this analysis, the benefits of preventing children and fetuses from being exposed to lead are the avoided costs that would have been associated with exposure. The four benefits for which we provide monetary values for prevention are 1) reduction in medical care costs of poisoned children, 2) reduction in special education costs for poisoned children, 3) reduction in future lost productivity from cognitive deficits in children, and 4) reduction in neonatal mortality from prenatal lead exposure. These are but a few of the benefits of preventing lead exposure. We did not evaluate the benefits related to children's stature, hearing, vitamin D metabolism, and blood production; the benefits of preventing the effects of lead on adults; or nonhealth-related benefits such as reduced personal injury court cases and improved property values.

The benefits we evaluated fall into two categories: 1) The first category consists of benefits achieved only for children whose blood lead levels are prevented from rising above a certain threshold; avoided medical and special education costs are estimated only for those children prevented from developing blood lead levels ≥ 25 $\mu\text{g/dL}$. 2) The second category consists of the benefits of preventing increased blood lead levels in children no matter what their initial levels are. For example, intellectual deficits result over a broad range of blood lead levels. Estimates of costs saved by reducing the effects of lead on intellectual functioning were made for preventing increases of 1 $\mu\text{g/dL}$ in blood lead level, regardless of the starting blood lead levels. The benefits of reducing maternal blood lead levels, which results in decreased infant mortality, are included in the second category.

Average benefits of preventing

Blood lead levels from rising above 24 $\mu\text{g/dL}$:

Avoided medical costs	\$1,300 per child
Avoided special education costs	\$3,331 per child

A 1 $\mu\text{g/dL}$ increase in blood lead level, regardless of starting blood lead level:

Increased lifetime earnings	\$1,147 per $\mu\text{g/dL}$ per child
Reduced infant mortality	\$ 300 per $\mu\text{g/dL}$ per newborn

The average total medical cost avoided by preventing a child's blood lead level from rising above 24 ug/dL is \$1,300 per child. (This amount is lower than the cost per episode for chelation, because not all children with elevated blood lead levels will be chelated.) On the average, \$3,331 per child is saved in special education costs. By preventing an increase of 1 ug/dL in a child's blood lead level, a net present value benefit of \$1,147 per child from increased future income is saved. Clearly, the greater the prevented increase in blood lead level, the greater the benefits; for the individual child, preventing the blood lead level from exceeding 24 ug/dL results in maximum benefits. Preventing a 1 ug/dL increase in the blood lead level of a pregnant woman saves an average of \$300 from reduced infant mortality. (Assumptions used in quantifying these benefits, including the monetary benefits of preventing infant mortality, are in Appendix II.)

EXAMPLE:

The benefits of preventing a child's blood lead level from rising from 24 $\mu\text{g/dL}$ to 34 $\mu\text{g/dL}$ are:

Avoided medical costs	\$ 1,300
Avoided special education costs	\$ 3,331
Increased lifetime earnings (\$1,147 per $\mu\text{g/dL}$ * 10 $\mu\text{g/dL}$)	\$11,470
<u>Total</u>	<u>\$16,101</u>

When these figures for the individual (average) child are applied nationally, the benefits of eliminating childhood lead poisoning are striking. For example, based on data from 3 programs (See Appendix II), the benefits of abating all pre-1950 housing with lead-based paint over a 20-year period would be \$62 billion, discounted to the present.

SEE APPENDIX II FOR

- Detailed benefits analysis
 - An illustration of the cost-benefit analysis for abatement of lead-based paint in all pre-1950 housing
 - Sensitivity analyses
-

CHAPTER 4. PROGRAM AGENDA

THE PROGRAM AGENDA FOR THE NEXT 5 YEARS CONTAINS FOUR MAIN ITEMS

- INCREASED CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES
- INCREASED ABATEMENT OF LEADED PAINT AND PAINT-CONTAMINATED DUST IN HOUSING
- REDUCTIONS IN OTHER SOURCES AND PATHWAYS OF LEAD EXPOSURE
- NATIONAL SURVEILLANCE

The program agenda for the first 5 years of the effort to eliminate childhood lead poisoning has four essential components: 1) increased childhood lead poisoning prevention activities, 2) increased abatement of leaded paint and paint-contaminated dust in housing, 3) continued efforts to reduce other widespread sources and pathways of lead exposure, and 4) national surveillance for elevated blood lead levels. Education and public awareness are essential to success in implementing all of these components.

**PROGRAM AGENDA ITEM 1. INCREASED CHILDHOOD LEAD POISONING
PREVENTION ACTIVITIES**

**INCREASED CHILDHOOD LEAD POISONING
PREVENTION ACTIVITIES MEANS**

- **INCREASED FUNDING FOR FEDERALLY-SUPPORTED PROGRAMS**
- **OTHER EFFORTS TO INCREASE SCREENING AND EDUCATION**
- **DEVELOPMENT OF INFRASTRUCTURE TO SUPPORT INCREASED PROGRAMS**

For this document, childhood lead poisoning prevention activities are defined as the screening of children for elevated blood lead levels, referral of poisoned children for medical and environmental interventions, and education about childhood lead poisoning. Such education is not limited to increasing public and medical provider awareness of lead poisoning. It also includes the education of children with elevated blood lead levels and their families about nutritional and other interventions. Expansion of childhood lead poisoning prevention activities should first focus on those children with the highest blood lead levels (e.g., blood lead levels $\geq$ 25 ug/dL).

Most children with lead poisoning are never identified.

An estimated 250,000 children had blood lead levels >25 $\mu\text{g/dL}$ in 1984 (ATSDR, 1988). (More up-to-date estimates will be available in the next couple of years from the Third National Health and Nutrition Examination Survey.) Available data indicate that the majority of such lead-poisoned children are never identified. The screening of children for elevated blood lead levels must be increased so that poisoned children can receive appropriate medical attention and environmental follow-up. (Environmental follow-up varies widely among programs and includes the measurement of lead in paint and often other potential media and interventions to prevent further exposure.) Screening data are also important for defining those areas in greatest need of intensive abatement programs and for evaluating the success of abatement programs in eliminating this disease in targeted areas.

Federally-Supported Childhood Lead Poisoning Prevention Programs

FEDERALLY-SUPPORTED PROGRAMS

- Federal programs began in 1972.
- Programs are administered by several agencies.
- Programs are directed at children at highest risk for lead poisoning.
- Programs screen only a small percentage of children at risk.

The history of childhood lead poisoning prevention programs in the United States is summarized in Appendix III. State and local childhood lead poisoning prevention programs perform many functions. They screen large numbers of children for lead poisoning and accept referrals of poisoned children from other practitioners for follow-up. They ensure that appropriate investigations are conducted of the homes and other environments of poisoned children. They may issue orders for abatement and may work with other government agencies to have abatements done. They also make sure that children receive appropriate medical treatment and that any other young children in the family or household are screened for lead poisoning. They educate parents and health care providers about lead poisoning and ways of preventing it.

Door-to-door screening in high-risk neighborhoods generally is the most productive method of identifying children with lead poisoning. Early in the 1970s, community outreach and door-to-door screening efforts were an essential component of programs. However, these activities are labor-intensive and costly. Consequently, most programs now screen children in fixed-site facilities.

The national effort to identify children with lead poisoning and abate the sources of lead in their environments began with the passage of the Lead-Based Paint Poisoning Prevention Act of 1971. Federally-funded screening began in Fiscal Year 1972 with blood lead testing, but in 1975 the Centers for Disease Control (CDC) recommended screening with erythrocyte protoporphyrin (EP) instead. (EP levels are elevated in the presence of elevated blood lead levels. Although useful for identifying children with blood lead levels above about 30 ug/dL and for detecting iron deficiency, EP is not a sensitive test for identifying children with blood lead levels below 25 ug/dL.) For most of the early years of this program, Federal funds appropriated under this Act were administered by the CDC. More than \$89 million were distributed, and over a quarter of a million children were identified with lead poisoning and received referrals for environmental and medical intervention. The improvement in the health status of children identified with lead poisoning in this program was documented in an evaluation by F.D. Kennedy (1978).

Current major sources of Federal funding for screening programs are the Maternal and Child Health (MCH) Block Grant Program, administered by the Health Resources and Services Administration (HRSA), and the Categorical Grant Program, administered by the CDC.

SOURCES OF FEDERAL FUNDING

- Maternal and Child Health Block Grants
 - Centers for Disease Control Categorical Grant Program
 - Early and Periodic Screening, Diagnostic, and Treatment Program (EPSDT)
 - Supplemental Food Program for Women, Infants, and Children (WIC)
 - Head Start
-

The MCH Block Grants serve as the principal means of Federal support to States to maintain and improve the health of mothers and children, including children with special needs. These grants are made to State health agencies to assure access to MCH services, especially for those with low income who live in areas with limited health services, and to reduce the incidence of preventable diseases and handicapping conditions in children. After assuming administrative responsibility for the Lead-Based Paint Poisoning Prevention Act in Fiscal Year 1982, HRSA issued a policy statement to all State MCH and Crippled Children's Services recommending routine periodic EP screening for all preschool children. Although not all States use MCH block grant funds for childhood lead screening, a 1984 survey indicated that 40 States, the District of Columbia, and Puerto Rico had screening activities.

The CDC Categorical Grant Program was authorized by the Lead Contamination Control Act of 1988. This program provides for childhood lead screening by State and local agencies, referral of children with elevated blood lead levels for treatment and environmental interventions, and education about childhood lead poisoning prevention. Money for this program was first appropriated in FY 1990. The President's budget for FY 1992 contains \$14.95 million for this program, an increase of \$7.16 million from FY 1991.

Other government-funded child health programs also conduct some childhood lead screening. These programs include Medicaid's Early and Periodic Screening, Diagnostic, and Treatment Program (EPSDT); the Supplemental Food Program for Women, Infants, and Children (WIC); and Head Start.

EPSDT is a comprehensive prevention and treatment program available to Medicaid-eligible persons under 21 years of age. In 1989, of the 10 million eligible persons, more than 4 million received initial or periodic screening health examinations. These are provided at a variety of sites (for example, physician offices, public health clinics, and community health centers) by private or public sector providers. Screening services, defined by statute, must include a blood lead assessment "where age and risk factors indicate it is medically appropriate." (The requirements for a blood lead assessment are not further defined.) In addition, the EP test is recommended for children ages 1 to 5 years to screen for iron deficiency. Because this test is also useful in identifying children with blood lead levels ≥ 25 ug/dL, many children being screened for iron deficiency are screened for lead poisoning at the same time. The guidelines for States indicate that environmental investigations for lead-poisoned children are covered under EPSDT, although abatement is not. However, specific criteria for screening and the determination of what Medicaid will cover are decided on a State-by-State basis. Thus, many States do not conduct much screening or do not pay for environmental investigations for poisoned children. National data are not available on the numbers of children screened for lead poisoning through EPSDT, since State-reported Medicaid performance and fiscal data are not broken down to such specific elements.

The U.S. Department of Agriculture's WIC program serves pregnant and postpartum women and children under 5 years of age in low-income households. Program benefits include supplemental food, nutrition education, and encouragement and coordination for the use of other existing health services. As of March 1988, an estimated 1.63 million children ages 1 to 4 years were participating in WIC. Although children must undergo a medical or nutritional assessment or both to be certified to receive benefits, Federal WIC regulations permit States to establish their own requirements for WIC certification examinations. These regulations permit the use of an EP test for certification and define lead poisoning as a nutritionally-related medical condition that can be the basis of certifying a child to receive WIC benefits. Most WIC programs that perform EP tests use them to screen for iron deficiency, although hematocrit or hemoglobin measurements are most commonly used for this purpose. The nutritional education and supplemental food provided by WIC are undoubtedly important in reducing lead absorption in many children and pregnant women.

Limited data on EP screening of children being seen for WIC certification or follow-up are available from CDC's Pediatric Nutrition Surveillance System (PedNSS). For calendar year 1989, 2,231,939 WIC visits for children 6 months through 4 years of age were reported to PedNSS (provisional data). Six States reported performing EP tests on 44,852 children; of these children, 10.8% had EP levels ≥ 35 ug/dL. Data are not available on how many of the children with elevated EP levels had blood lead levels measured.

Head Start provides a comprehensive developmental program for low-income children between the ages of 3 and 5 years. About 24 percent of U.S. 3- and 4-year-olds living in poverty are served by 229 Head Start programs. Although Head Start is mainly known as an education program, 99 percent of the enrolled children receive medical screening (54 percent through EPSDT). This screening can include screening for lead poisoning, if lead poisoning is prevalent in the community. National data on how much lead screening is conducted through Head Start are not available.

In 1985-86, about 785,000 children were screened through childhood lead poisoning prevention programs (ATSDR, 1988). In Fiscal Year 1988, according to data collected by the Public Health Foundation, State and local health agencies screened 970,768 children and identified 18,912 that had positive screening tests requiring diagnostic confirmation (Jane Lin-Fu, personal communication). (These latter numbers include some children screened through EPSDT, WIC, and Head Start, but they may underestimate the numbers of children screened under the MCH Block Grant Program.) Given that an estimated 250,000 children had blood lead levels above 25 ug/dL in 1984 (ATSDR, 1988), it is apparent that most lead-poisoned children are never identified.

REASONS TO INCREASE ACTIVITIES

- Increase the number of children screened
 - Increase the use of intensive screening methods
 - Ensure prompt investigations of the environments of poisoned children
 - Assure proper follow-up of poisoned children
-

More childhood lead poisoning prevention activities are needed to 1) increase the number of children screened, particularly in communities with the highest levels of blood lead in children and rates of childhood lead poisoning, 2) increase the use of intensive screening methods, such as community outreach and door-to-door screening, 3) ensure prompt investigation of the environments of poisoned children, and 4) assure proper follow-up of poisoned children. Increasing the number of States that require or encourage EP or blood lead testing through MCH Block Grant activities, EPSDT, WIC, and Head Start would probably be an efficient way of increasing screening in high-risk populations. Outreach and educational activities from the Federal level to regional and State offices and local agencies and programs could increase recognition of the importance of such screening. Better information about the amount and efficacy of screening children in EPSDT, WIC, and Head Start would be helpful in developing strategies for increasing testing through these programs where appropriate.

Other Efforts to Increase Screening and Education

OTHER EFFORTS NEEDED

- Increased outreach to children without a usual source of care
- Increased screening by health care providers
- Increased public awareness

Outreach to Children Without a Usual Source of Medical Care

On the basis of 1988 data from the National Health Interview Survey, it has been estimated that 8 percent of children less than 5 years of age do not have a regular source of medical care. Intensified childhood lead poisoning prevention activities must be directed at these children, many of whom are at high risk for lead poisoning. Some of these children could be reached by increasing enrollment in EPSDT and other programs. Others could be identified through intensified (for example, door-to-door) screening by childhood lead poisoning prevention programs. Additional strategies, such as screening children using emergency rooms in high-risk neighborhoods for primary or semiemergent care, should be evaluated for cost-effectiveness.

Screening by Health Care Providers

Education is of vital importance in increasing the amount of screening conducted by health-care providers. The American Academy of Pediatrics issued its most recent statement on lead poisoning prevention, diagnosis, and treatment in 1987. Nevertheless, many providers do not consider screening for childhood lead poisoning to be a part of routine pediatric care.

Several strategies are available for increasing health-care provider awareness. The first is to disseminate educational materials and do outreach through existing professional organizations and medical schools. (A partial list of relevant professional organizations is in Appendix IV, Table 1). A second strategy is to develop and disseminate training modules that can be completed for Continuing Medical Education credits, such as the Case Study in Environmental Medicine developed by ATSDR. A third is to provide conferences for medical care providers on childhood lead poisoning, either through the private sector (such as those held in 1989 at the University of Maryland and the University of Virginia) or through federally funded centers (such as the Health Education Centers of the Health Resources and Services Administration, the Education Resource Centers of the National Institute for Occupational Safety and Health, and the occupational and environmental clinics with activities funded by ATSDR).

Increased Public Awareness

Campaigns to increase public awareness of childhood lead poisoning and its prevention are likely to increase the amount of screening conducted. Such campaigns will not only educate medical care providers, they will also increase the public's demand for lead screening of children. Some lead poisoning cases may be prevented, for example, by informing homeowners of the potential dangers in renovating older homes. The National Maternal and Child Health Clearinghouse is a source of publications about childhood lead poisoning. This Clearinghouse and other resource centers could expand their activities, including operating a toll-free hotline and developing and disseminating simple materials about lead poisoning prevention in different languages. Information centers could also supply information on Federal, State, and local resources for dealing with childhood lead issues.

State and local health departments and childhood lead poisoning prevention programs should also be encouraged to increase public awareness of childhood lead poisoning. The categorical grants program authorized by the Lead Contamination Control Act of 1988 specifically allows funds to be used for educational activities conducted by State and local childhood lead poisoning prevention programs.

Several private sector organizations have sponsored educational activities about childhood lead poisoning. Other organizations should be encouraged to follow suit. Because childhood lead poisoning is associated with decreased intelligence and ability to learn, coalitions between organizations promoting lead poisoning prevention and organizations promoting education and the prevention of mental retardation should also be encouraged. Organizations that might be interested in such activities are listed in Appendix IV, Table 2.

Development of Infrastructure to Support Increased Childhood Lead Poisoning Prevention Programs

DEVELOPMENT OF INFRASTRUCTURE TO SUPPORT PROGRAMS MEANS INCREASED

- Training programs
- Laboratory services
- Laboratory proficiency testing programs

The expansion of screening programs will result in a demand for training programs on childhood lead screening and the investigation of environmental sources. The Louisville, Kentucky, training program can serve as a model for other such programs. This program provides methods for assessing lead poisoning in high-risk populations and demonstrates the integration of lead screening with basic child health services and the technical and management skills needed for an effective and efficient childhood lead poisoning prevention program.

In addition, increased screening will lead to a demand for increased laboratory services. In 1991 CDC will likely issue new recommendations suggesting that screening programs attempt to identify children with blood lead levels below 25 ug/dL. This change will mean that blood lead measurements must be used for childhood lead screening instead of EP measurements. When this happens, the demand for increased blood lead testing will far exceed current capacity. In addition, cheaper, easier to use, and portable instrumentation for blood lead testing will need to be developed. Furthermore, existing programs for proficiency testing and certification of laboratories will have to be expanded. With concern about health effects at low blood lead levels, laboratories will be called upon to do better measurements in the 4 to 5 ug/dL range. As a result, major efforts will be needed to improve laboratory quality assurance and control at these lower levels. Reference materials for laboratories performing blood lead measurements and technical assistance will be required to improve laboratory quality.

PROGRAM AGENDA ITEM 2. INCREASED ABATEMENT OF LEADED PAINT
AND PAINT-CONTAMINATED DUST IN HOUSING

INCREASED ABATEMENT REQUIRES

- Setting priorities for which homes are to be abated first
- Strategies for increasing the number of abatements conducted
- Assuring the safety and effectiveness of abatement
- Development of infrastructure for abatement
- Development of a national implementation plan

Lead-based paint abatement is an integral part of the treatment of childhood lead poisoning and a crucial step in the prevention of new cases. Many sources besides lead-based paint are contributors to the exposure of children to lead, but we have four reasons for focusing on abatement of lead paint in this plan. First, lead-based paint and paint-contaminated house dust are still the major cause of high-dose lead poisoning in U.S. children. Second, we have known of the dangers of lead paint since the beginning of the century. The greatest concentrations of lead in paint occur in housing built before 1950. Although the Consumer Product Safety Commission has required paint manufactured for residential use to be almost lead-free since 1977, we have made little progress in eliminating paint previously applied as a cause of childhood lead poisoning. This problem may get worse with time, as houses painted with lead-based paint deteriorate further. Third, abatement of paint is expensive, and a successful effort to eliminate poisoning from leaded paint will require a coordinated effort from the

government and private sectors. Fourth, leaded paint abatement is difficult and potentially dangerous. Poorly performed abatements have poisoned workers and their families and people living in the homes being abated. In recent years, numerous families have been poisoned while renovating homes that were not tested for lead. Until this environmental source of lead is eliminated, the United States will continue to have a significant childhood lead poisoning problem.

Setting Priorities for Lead-Based Paint Abatement

PRIORITIES FOR ABATEMENT

- Homes of children identified with lead poisoning
- Homes at high risk of housing children with lead poisoning
- Homes with lead-based paint that are being renovated or remodelled for other reasons

An estimated 30 to 40 million residences in the United States contain leaded paint (ATSDR, 1988), although not all of them pose an imminent hazard. Priorities for abatement should be based largely on public health concerns; therefore, abatement programs must work in tandem with childhood lead poisoning prevention programs to ensure the most efficient use of resources.

Three priority groups of housing for abatement can be identified: homes of children identified with lead poisoning, homes at high risk of housing children with lead poisoning (but in which poisoned children have not yet been identified), and homes with lead-based paint that are being renovated or remodelled for other reasons. Although not specifically discussed in the following, day care centers and other buildings frequented by young children are also a high priority.

The first priority for abatement is the homes of children identified with lead poisoning. This is important not only to protect these children from continued exposure, but also to prevent children who will live in these dwellings in the future from being poisoned. In particular, children with lead poisoning severe enough to require medical intervention (i.e., ≥ 25 ug/dL) should be the utmost priority.

The second priority for abatement is the homes with a large potential for poisoning children. These are homes that are likely to be causing unrecognized lead poisoning or to poison children in the near future. This category includes housing in areas with a high prevalence of lead poisoning, but could include older housing in areas where there is little or no childhood lead screening. Screening, housing, socioeconomic, environmental, and other data should be used to identify those areas where housing is most likely to poison children. Abatement of housing in this category is a crucial part of the lead poisoning prevention strategy. Within this second priority group, decisions will have to be made about which specific homes and areas should be abated first. These decisions should be based on a combination of environmental and demographic data. A "hazard ranking scheme" should be developed and validated. The more efficient the identification of homes likely to contain poisoned children and to poison children in the future, the more cost-effective the abatement will be.

Opportunistic abatements, the third priority, involve those homes that can be efficiently abated because they are being worked on anyway or have other special characteristics. An example of opportunistic abatement is the removal of leaded paint from public housing during comprehensive modernization. The comprehensive modernization program is effective because 1) the Federal government has authority over the housing to be abated and 2) lead abatement adds only a relatively small amount to the cost of ongoing modernization activities.

Data from several evaluations show that abatement of lead-based paint decreases children's blood lead levels (Kennedy, 1978; Rosen, 1990; Copley, unpublished data; Amitai et al., unpublished data). The data from these studies indicate that even less than complete abatements reduce children's blood lead levels. In general, the most thorough abatements are believed to be the most effective in reducing blood lead levels and residual lead in the environment. Given the limited resources for abatement, however, a balance must be struck between doing the best possible abatements in fewer units and using reasonably good, less expensive methods in more units. The cost-effectiveness of alternative paint abatement methods should be evaluated, and the cost of abatement should be reduced through the development of new methods and materials and the establishment of a larger infrastructure for abatement.

An important issue is that some of the housing stock, particularly in the inner cities, is deteriorated past the point of rehabilitation or may be in neighborhoods that are so economically depressed that buildings rapidly deteriorate and are abandoned. Extremely deteriorated buildings in declining neighborhoods with large numbers of abandoned units are very likely to be abandoned or razed in the next 5 years. Requiring complete abatement in such situations would be futile and could lead to families being dislocated. In such circumstances, the efficacy of preventive maintenance—cleaning and partial abatement with frequent environmental and blood lead testing—should be determined, and its role should be defined. In addition, when low-income units are abated, safeguards will be required to ensure that they remain available as low-income housing.

Strategies for Increasing the Number of Abatements

STRATEGIES FOR INCREASING ABATEMENTS

- Incentives
- Demonstration programs
- Testing and disclosure requirements
- Education and public awareness

Increasing the number of abatements performed will require a mixture of public and private sector efforts. Housing can be divided into several different sectors—for example, public housing, public-assisted rental units, privately owned rental units, and owner-occupied homes. Different strategies will be required to increase abatements for different kinds of housing. These strategies include positive and negative incentives, demonstration programs, and the use of test and disclosure requirements.

Positive and negative incentive strategies confer a financial benefit or other advantage, or withdraw a financial benefit or advantage, to promote or discourage certain behaviors. Incentive programs can be used to encourage testing for lead-based paint or abatement of identified hazards. Demonstration area programs would set aside entire neighborhoods that would be abated to serve as a model to encourage abatement elsewhere.

Another possible strategy would require testing for lead levels in housing and the disclosure of the test results. (These results would be recorded, so that units undergoing multiple transactions would not be repeatedly tested.) Requiring the abatement of units with high lead levels could be an additional option. Testing and disclosure could be required for all housing units or it could center around transactional "trigger events," such as renovation or remodeling, renting, sale, or transfer.

Education and public awareness strategies are critical to the success of abatement programs. They are designed to inform the general public, the housing industry, and other relevant parties about preventing childhood lead poisoning and the role of lead-based paint. These strategies are designed to mobilize the community to act voluntarily to address the problem of leaded paint in housing. Public education and awareness will prompt the market to encourage abatement by placing a higher value on an abated house or rental unit than on a nonabated dwelling. Without increased awareness of the dangers of lead-painted housing, incentive strategies will be ignored, and regulatory approaches will be less acceptable to the public.

The President's budget for FY 1992 includes \$25 million for the HOME program which will be administered by the Department of Housing and Urban Development (HUD). This program will assist low- and moderate-income private residential property owners, abate lead-based paint, and will be directed to homeowners with young children in high-risk housing. This program could provide a knowledge base for evaluating the effects of abatement.

Federal, State, and local governments and the private sector have roles in many of these strategies; different groups are appropriate for implementing different strategies. How these strategies should be used to ensure the abatement of homes in the three priority groups and the roles of different levels and agencies of government and the private sector should be dealt with in an implementation plan.

Development of Infrastructure for Abatement (See Appendix V for More Details on the Material in this Section.)

INFRASTRUCTURE DEVELOPMENT MEANS

- Developing testing and abatement guidelines
- Developing worker training and certification programs
- Evaluating emerging abatement technology
- Developing laboratory accreditation programs
- Ensuring the availability of insurance for contractors
- Arranging relocations for residents during abatement
- Developing guidelines for disposal of abatement debris

Although enough is known to start an effective national abatement program, the capacity to undertake large-scale abatement does not currently exist. Regulations to ensure the safety of workers and occupants and the quality of the abatement work are limited. Very few inspectors, abatement contractors, or workers have been trained to perform the needed work properly. Both contractors and property owners can have difficulty getting insurance. These deficiencies in the infrastructure for abatement must be corrected as quickly as possible so that a national abatement program can be developed. This section briefly describes the steps that must be taken to increase the national capacity to do safe and effective abatements. More details on infrastructure development appear in Appendix V.

Guidelines for testing for lead-based paint and performing safe and effective abatements are essential. In April 1990, HUD issued the first national set of comprehensive technical guidelines (the HUD Interim Guidelines) for lead paint testing and abatement. These guidelines were developed by a committee of government and nongovernment experts for public and Indian housing authorities. Since the guidelines were developed for housing that is to be extensively modified during modernization by the Federal Government, they should be modified for use by States, localities, and individuals in situations where funds are not as available, time is a critical factor, and the unit is not being gutted for other reasons.

The development of guidelines should be followed by the development of government-sanctioned model training programs for assuring the quality and consistency of worker training. As the amount of leaded paint abatement increases, market forces will meet the growing demand for training programs. Government involvement may be necessary, however, to control the quality of instruction and to assure the competence of trainees. In addition, mandatory requirements for the certification of contractors and their workers, testers, and inspectors should be established either by government or trade organizations.

Lead-based paint abatement will probably not evolve exclusively as a separate industry and skill specialty. It is an integral and inevitable part of a variety of existing building trades: painting, plastering, masonry, flooring, cabinetry, carpentry, electrical, plumbing, insulation, and door and window replacement. Some home renovation contractors will probably specialize in lead paint abatement. Thus, lead-based paint abatement should be integrated into the various building trades. Because abatement is a potentially hazardous activity, all workers involved in home renovation and repair should be familiar with the special safeguards and techniques required.

Another potential benefit of a national abatement program is increased employment. As persons with little training develop the skills needed for lead-based paint abatement, they will be likely to vacate jobs that do not require training. Because this abatement work will require a large work force, often in neighborhoods with high rates of unemployment, the training and employment of local persons will have local economic and social benefits.

Lead exposures of persons performing abatement and other workers, especially of pregnant women and of women and men who have or are planning to have children, should be reduced. At present, abatement workers are not covered by the Occupational Safety and Health Administration (OSHA) general industry standard regulating worker exposure to lead. Instead, they are covered under the safety and health standards for the construction industry, which regulate lead exposure far less strictly. A standard is needed that takes into account new data showing adverse effects of lead on adults at lead levels below the current OSHA general industry standard. Abatement workers and their families should be protected by medical monitoring and medical removal provisions, as are potentially lead-exposed workers in general industry.

During the past few years, private firms have developed a variety of new products to reduce the costs of lead-based paint abatement. Standards and performance criteria must be established to assure the effectiveness of new products. Standards for laboratories evaluating environmental samples should also be developed.

Other constraints to rapidly expanding lead-based paint abatement programs are the unavailability of liability insurance for contractors and building owners performing abatement, the lack of programs for quality assurance of lead-based paint and dust laboratory analysis, and the lack of suitable temporary housing for families whose homes are being abated. Another constraint is uncertainty about the proper disposal of abatement debris. When lead is removed from buildings, it is, in effect, being concentrated; if lead is to be kept from being dispersed in the environment, there must be rules and regulations for its safe disposal.

Development of a National Implementation Plan for Abatement

AN IMPLEMENTATION PLAN FOR ABATEMENT SHOULD FOCUS ON

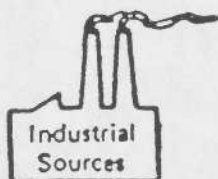
- Increased abatements by the private and public sectors
- Increased safety and efficacy and decreased cost of abatement
- Targeting of high-risk housing
- Best use of available funds

A well-designed national implementation plan for increasing the number of abatements performed should be developed immediately. Although there is an immediate need for increased resources for abatement, a phased approach to increasing abatement should be designed. The implementation plan should focus on three main issues: 1) how to increase private and public sector abatements; 2) how to increase the safety and efficacy and decrease the costs of abatements through technology development and evaluation and worker training and certification; and 3) how best to use available funds to quickly reduce the number of children poisoned by lead-contaminated housing.

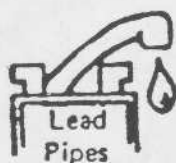
During the early years of the national abatement strategy, an evaluation component will be essential. This evaluation should include measurements of efficacy and safety through postabatement environmental and human testing, and the inspection and collection of data on numbers of abatements being funded by the private and public sectors.

PROGRAM AGENDA ITEM 3. REDUCTIONS IN OTHER SOURCES AND PATHWAYS OF LEAD EXPOSURE

OTHER SOURCES AND PATHWAYS
TO BE ADDRESSED



- Soil
- Water
- Air
- Housewares
- Food
- Workplace and hobbies



Lead-based paint and paint-contaminated dust account for most cases of lead poisoning in the United States. Other sources of lead will also have to be addressed, however, to eliminate this disease. For example, lead-contaminated soil is probably an important source for a large number of children. However, adequate information is not yet available on which to base recommendations for a national soil abatement strategy. Federal agencies are proceeding with or are evaluating further regulation of environmental lead in water, air, and housewares. In this section, some current and needed activities are summarized.

Although lead-based paint and paint-contaminated dust account for most cases of childhood lead poisoning in the United States, other sources of lead will also have to be addressed.

The Environmental Protection Agency (EPA) is evaluating the need for more stringent standards for lead in drinking water and air. EPA is also conducting a demonstration project in three cities to evaluate the benefits of removing lead-contaminated soil from yards of homes where children live.

The Food and Drug Administration (FDA) has proposed new regulatory standards for lead in ceramic pitchers and other types of ceramic foodware. FDA is also attempting to identify sources of lead in the diet other than those that have already been identified, such as lead in wine bottle cap wrappers and in calcium supplements. Mechanisms should be established so that potters and other crafts people either clearly indicate that their wares are not for food service or have their wares tested to ensure that they do not contain lead.

In coordination with FDA, domestic manufacturers of food cans have markedly reduced their use of solder with a high lead content. This change has resulted in large reductions in the lead levels in canned foods in the United States. Nevertheless, a total ban on the use of solder with high lead content in domestically produced canned goods should be seriously considered. The frequency of use of solder with high lead content in imported food cans is unknown; a ban on the use of solder with high lead content in imported food cans should also be considered.

Childhood exposures from parental occupations and hobbies involving lead should be reduced. This can be done through a combination of good work practices and education. The use of folk remedies containing lead continues to be a problem in certain ethnic populations. Educational activities, intensified lead screening, and intervention strategies could reduce exposure to this source of lead.

PROGRAM AGENDA ITEM 4. NATIONAL SURVEILLANCE FOR ELEVATED LEAD LEVELS

USES OF SURVEILLANCE DATA

- To target interventions
- To track progress
- To evaluate worker exposures

The only national data available for estimating the number of children who may have elevated blood lead levels are derived from national surveys of nutritional and health status that, in the past, have been conducted about once a decade. These data are extremely valuable for providing unbiased estimates of the blood lead levels of children and workers in the United States. In the future they will be conducted more often, and this will make it possible to evaluate national and regional blood lead levels more frequently. As these data are now collected, however, they cannot be used to monitor short-term trends over several months or a few years. They cannot be used to characterize geographic distributions of poisoning in the community or to target interventions where they are most needed. A national surveillance program for elevated blood lead levels in children and workers is essential for the development of a "lead priority list" for targeting interventions, for tracking our progress in eliminating childhood

lead poisoning, and for evaluating lead exposure in abatement workers and workers in other lead-contaminated environments.

Several sources of data could be used for surveillance. These include childhood lead poisoning prevention programs, other government programs that conduct or reimburse for screening for lead poisoning, and laboratories that perform blood lead testing.

The development of better systems for managing data in childhood lead poisoning prevention programs should be a high priority. Data from childhood lead poisoning prevention programs could be extremely important for evaluating the yield of screening in specific areas, the yield of alternative screening strategies, and the efficacy of interventions. Since screening takes place in only limited geographic areas, however, data from screening programs cannot provide national information. Furthermore, although many areas that need targeted abatement programs could be identified through screening data, areas that have no screening programs could not be evaluated. In addition, many large programs have not yet computerized their data, and those computer systems that exist are often cumbersome or cannot link data on screening and medical follow-up with data on environmental investigations and interventions.

Data from other government programs conducting or reimbursing for screening, like EPSDT, could also be useful, but these data have serious limitations. They would provide information on only a small segment of the population being tested for lead poisoning, and they would not include follow-up data.

The optimal model for national surveillance is the notifiable disease system that CDC has used since 1961. Through this system, cases of illnesses are reported electronically to CDC by State epidemiologists. Since lead poisoning is diagnosed on the basis of laboratory tests, reporting for lead would depend upon laboratories sending their data on persons with elevated blood lead levels to State health departments for transmission to CDC. The State health department would also be responsible for ensuring that multiple tests on the same individual are identified as such and that persons needing follow-up are referred appropriately. An evaluation component is essential for determining that the data collected are complete and representative. The American Academy of Pediatrics, the American Medical Association, and the Council of State and Territorial Epidemiologists have endorsed the development of such surveillance.

KEY ORGANIZATIONS ENDORSING NATIONAL SURVEILLANCE

- American Academy of Pediatrics
 - American Medical Association
 - Council of State and Territorial Epidemiologists
-

The feasibility of developing national surveillance for elevated lead levels is illustrated by the National Institute for Occupational Safety and Health (NIOSH) efforts to develop a system for reporting elevated blood lead levels in workers. NIOSH receives reports from eight State health departments that provide data about numbers of workers with elevated blood lead levels and industries in which lead poisoning is occurring. The States with surveillance systems also ensure follow-up of the affected workers. In 1988, 4,804 workers in seven States were reported to have blood lead levels ≥ 25 ug/dL.

CHAPTER 5. RESEARCH AGENDA

RESEARCH IS NEEDED FOR

- INCREASED PREVENTION ACTIVITIES
- INCREASED ABATEMENTS
- REDUCTIONS IN OTHER SOURCES

Enough is already known to start an effective campaign to eliminate childhood lead poisoning, and intensified efforts to prevent this disease should get under way immediately. There are, however, several questions that must be answered if this disease is to be successfully eradicated in the most cost-effective manner. The following are key elements of a research agenda designed to provide essential information for future years of a program to eliminate childhood lead poisoning. Many of these elements appeared in the Committee to Coordinate Environmental Health and Related Programs ad hoc committee report on the implementation of the ATSDR report to Congress.

The results of basic research have shown the need for a strategic plan for the elimination of childhood lead poisoning.

This research agenda does not include a discussion of or a budget for many basic research activities. Such activities include evaluating the amount of lead absorbed by children and adults, identifying new biomarkers for lead exposure, and determining the impact of pharmacological treatment of lead poisoning on children's cognitive functioning. Although these activities are not essential for the first 5 years of the Strategic Plan, they are important. The findings of basic research have made a plan such as this necessary, and they make it possible to develop a program agenda at this time. These research activities should receive financial support.

RESEARCH AGENDA ITEM 1. RESEARCH FOR CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES

RESEARCH FOR INCREASED CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES

- Cost-effectiveness of screening strategies
- Better instruments for blood lead testing
- Evaluation of capillary blood collection devices
- Evaluation of educational and nutritional interventions

Studies should be conducted on the cost-effectiveness of different strategies for childhood lead screening. These strategies include screening in inner-city emergency rooms to reach children who have no ongoing source of care and "cluster testing" of all children in multiple dwelling units where cases of childhood lead poisoning have been identified. The usefulness of screening in day care centers and nursery schools should also be evaluated. In addition, Federal programs now funding childhood lead screening should be evaluated to see how they can work together for a most efficient use of resources.

At present it is much cheaper and easier to perform an EP test than a blood lead measurement; however, the EP test is not a useful screening test for blood lead levels below 25 ug/dL. Both because of the expected increase in screening and because of the concern about the health effects of lower blood lead levels, the demand for blood lead testing is likely to increase. The development of portable, easy-to-use, cheaper instrumentation for blood lead measurement is extremely important.

Because capillary (or fingerstick) blood samples may be easily contaminated with lead on the skin, venous blood must be used to confirm lead poisoning in children. Several capillary blood collection devices now on the market purport to collect blood free of surface finger contamination from lead. These devices should be evaluated for ease of use and ability to collect an uncontaminated sample.

The education of families about lead poisoning by childhood lead poisoning prevention programs often includes information about the importance of nutrition. Because of our growing concern about the adverse effects of low blood lead levels, nutritional interventions are likely to be recommended for more children. A number of nutritional factors have been shown experimentally to influence the absorption of lead and its concentrations in tissues. Intervention studies or clinical trials should be conducted to establish that increasing the regularity of meals and ensuring adequate dietary intake of iron and calcium can reduce blood lead levels.

Educational strategies for increasing medical care provider and public awareness of lead poisoning should also be evaluated for their efficacy in reducing children's blood lead levels and preventing lead poisoning.

RESEARCH AGENDA ITEM 2. RESEARCH ON LEAD-BASED PAINT AND
PAINT-CONTAMINATED DUST ABATEMENT

RESEARCH ON ABATEMENT

- Long-term follow-up postabatement
- Efficacy of abatement methods
- Better methods for measuring lead in paint and dust
- Evaluation of worker exposures
- Abatement of forced air ducts, rugs, furniture, etc.
- Determination of safe environmental levels
- Efficacy of preventive maintenance

The techniques recommended in the HUD Interim Guidelines have been shown to be effective in abating lead-based paint and reducing dust levels. However, no long term evaluations have been conducted to ensure that dust lead levels and children's blood lead levels remain low once abated units have been reoccupied. Long-term follow-up of units abated under these guidelines and their occupants should be conducted.

Few childhood lead poisoning prevention programs perform as rigorous an abatement as that recommended in the HUD Interim Guidelines. Better data on the long-term efficacy of less stringent abatement methods should also be collected, and the cost-effectiveness of alternative methods of lead-based paint abatement should be evaluated. These analyses should be used to determine how best to spend resources,

given that more complete and expensive abatements probably result in greater reductions of blood lead levels but may result in fewer units being abated.

Current methods for measuring lead in paint and dust are sometimes inaccurate, expensive, or both. Accurate, inexpensive methods for such measurements would decrease the cost and increase the reliability of preabatement and postabatement testing. These methods include improved X-ray fluorescence (XRF) devices and chemical spot tests. Preferred methods are those that can be used onsite, instead of requiring offsite laboratory analysis, and those that do not destroy surfaces.

All abatement methods should be evaluated to determine worker exposures to lead and other hazards. Laboratory and field studies should be conducted, when appropriate, before new methods are recommended for widespread use, and they should include evaluations of worker safety. HUD, EPA, and other agencies have already started some of these evaluations.

Methods for abating such items as forced air ducts, rugs, and furniture have not been evaluated adequately. Furthermore, there is no consensus on whether such abatement is appropriate. For example, discarding lead-contaminated rugs and upholstered furniture has been advocated.

Environmental lead levels used for determining whether a home needs abatement or if an abated unit can be reoccupied are based on limited scientific data. These levels should be evaluated to ensure that they are both adequate to protect health and do not result in unnecessary abatements. Included in this work would be the paint lead concentration at which paint abatement is recommended; the dust lead concentration at which dust abatement is recommended, even in the absence of lead-based paint; and the soil lead concentration at which abatement should occur. In addition, a system for estimating the total lead hazard in a building or housing unit, combining information on household demographics, paint lead concentration, quantity, and condition, and dust and soil lead levels should be developed. The lead levels of paint, dust, and soil that are to be considered safe after abatement should be evaluated. One important outcome of this work would be algorithms for identifying which housing units are most likely to poison children in the future.

Because of the limited money available for abatement, inexpensive interim methods must be developed and evaluated for preventing children from being exposed to high environmental lead levels in units awaiting abatement. Such interventions may include regular professional cleaning with high-efficiency vacuum cleaners and scraping and repainting small areas of peeling paint. Outcome measurements should include measurement of lead in house dust and children's blood. Such preventive maintenance strategies should be evaluated over several years.

RESEARCH AGENDA ITEM 3. RESEARCH ON REDUCTIONS IN OTHER
SOURCES AND PATHWAYS OF LEAD EXPOSURE

RESEARCH ON REDUCTIONS IN OTHER
SOURCES AND PATHWAYS

- Relative contributions of different sources
- Cost-effectiveness of soil abatement
- Drinking water lead levels and treatments
- Sources of dietary lead
- Improved food lead measurement
- Bioavailability
- Mobilization of lead during pregnancy



Studies should be conducted to determine the relative contributions of various sources and pathways of lead to children's blood lead levels. These studies should also investigate the relationship between lead in the various environmental compartments to which children are exposed. Sources and pathways to be investigated should include paint, dust, soil, air, food, water, and exposure from parental occupations and hobbies.

Current methods for remediating soil are expensive, and their efficacy under varying conditions has not been proven, particularly in urban areas. Studies should be conducted to examine the efficacy and cost-effectiveness, in terms of blood lead level reductions, of various methods of remediating soil (such as removing soil and planting ground cover). These efforts should complement EPA's ongoing efforts.

Lead levels in drinking water in the United States, including levels in water fountains, should be assessed more completely. Alternative treatment approaches aimed at reducing lead in drinking water should be evaluated.

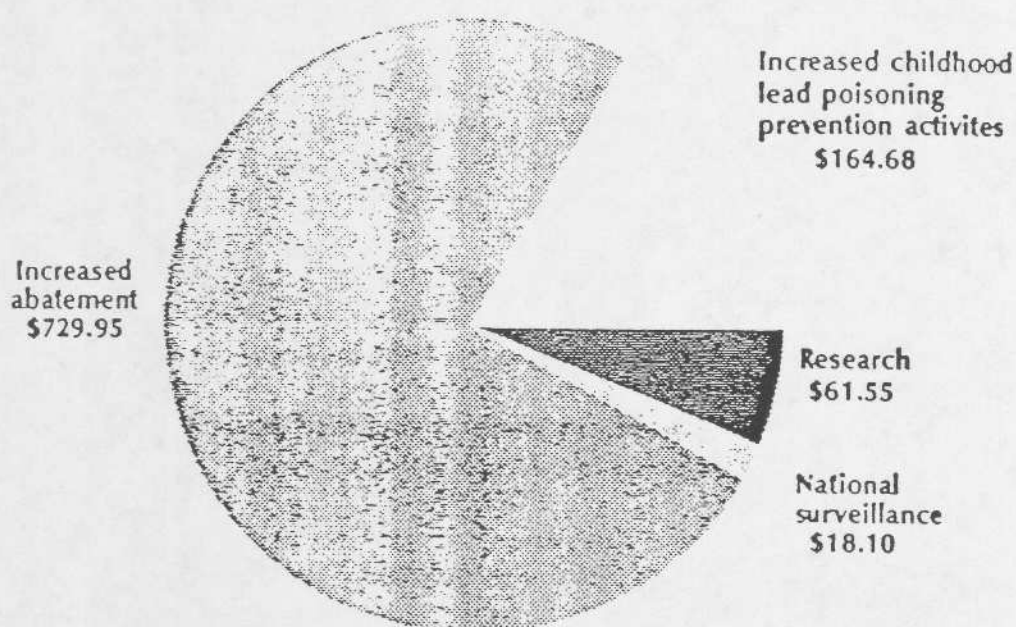
While a great deal is known about many dietary sources of lead, others have not been identified or evaluated. Lead in calcium supplements is of particular concern because of the many pregnant women taking these preparations. Other inadequately studied sources of lead include wine (from lead in the wine itself or in caps or seals), coffee produced in institutional coffee urns, infant foods, and bottled waters. Surveys of lead ingested by special populations should also be conducted. These surveys should focus on canned foods, housewares, and folk remedies used by special populations, such as ethnic groups. For these evaluations, analytical procedures will have to be improved.

The bioavailability of lead probably varies according to the substrate (for example, paint, dust, soil, food) and the chemical form and particle size of the lead. Criteria for cleanup may need to vary according to the probable bioavailability of lead at a given site. Animal feeding studies and collection of data on human populations are needed to provide information on how bioavailability issues should be considered when decisions on remediation and clearance are made.

Studies should be conducted on the mobilization of bone stores of lead during pregnancy and on the biokinetics of fetal lead exposure. If bone stores prove to be an important determinant of blood lead levels during pregnancy, interventions to reduce lead mobilization in pregnancy should be developed and studied.

CHAPTER 6. FUNDS NEEDED FOR IMPLEMENTATION OF THE STRATEGIC PLAN

SUMMARY OF FUNDS PROJECTED TO BE NEEDED FOR THE FIRST 5 YEARS OF THE STRATEGIC PLAN* (millions of dollars)



Total = \$974.28 (million)

*Costs reflect the amount of money needed to implement the program agenda and a shared commitment of the public and private sectors.

Our estimate of the cost of implementation for the first 5 years of the Strategic Plan is expected to be \$974 million. Ninety-four percent of this money is for program activities; six percent is for research. The source of funds is not discussed in this report; these costs reflect a shared commitment of the public and private sectors.

Funds Needed for Implementation of the Program Agenda

Implementation of the program agenda will require the efforts and cooperation of many Federal, State, and local agencies. The first five years of this agenda will cost \$913 million. This budget does not include funds for program activities needed to reduce sources and pathways of exposure other than lead-based paint and paint-contaminated dust. Many of these are already being addressed through Federal and other actions.

The estimate of the additional costs for increased abatement requires further discussion. Because of the lack of baseline data, it is difficult to project how many more housing units should be abated as part of a strategic plan to eliminate childhood lead poisoning. Furthermore, development of cheaper abatement methods and of an infrastructure for abatement is an essential part of the first years of any national abatement strategy. Therefore, a phased increase in the number of abatements performed is proposed, with an emphasis on research and development and the testing of strategies and materials in the first 2 years of the program. Within 3 years, resources should be made available to perform 20,000 to 30,000 more abatements annually than are currently being performed. These resources would be enough to abate the homes of all lead-poisoned children currently being identified by childhood lead poisoning prevention programs who have no other source of funding for abatement. (As the amount of screening increases, the estimate of additional units to be abated annually will also need to be increased.) These resources would also make it possible to have demonstration projects and to abate units in the second priority group, homes that have a large potential for poisoning children. At this rate, eliminating all lead paint from housing stock in the United States will take a long time, but it is important to make a start--to eliminate lead-based paint from those units that have the greatest potential to adversely affect health.

The costs of abatement vary greatly according to the size and kind of housing unit, the region of the country, and other factors. For this plan, we assumed that an average abatement costs around \$6,500. This estimate was developed by Anne Elixhauser, Battelle, under a contract with CDC through interviews with screening programs. (The abatement methods used in the three studies whose data form the basis of the benefits analysis and the cost-benefits analysis in Appendix II were much cheaper and less comprehensive; however, data are not available on how much blood lead levels might be reduced by more expensive methods. We assume that the reduction would be correspondingly greater. Information on the costs and benefits of abatement will need to be continually updated as new information becomes available.) Thus, the abatement of 20,000 to 30,000 units a year could be expected to cost around \$130 to \$195 million a year. Since it will take a couple of years to build up the infrastructure for abatement and increase the number of abatements performed, we estimate that the increased abatements needed to complete the first 5 years of this Strategic Plan would cost a total of \$710 million. The unit cost of abatement is likely to decrease over the next several years as new abatement methods are developed and the infrastructure for abatement increases.

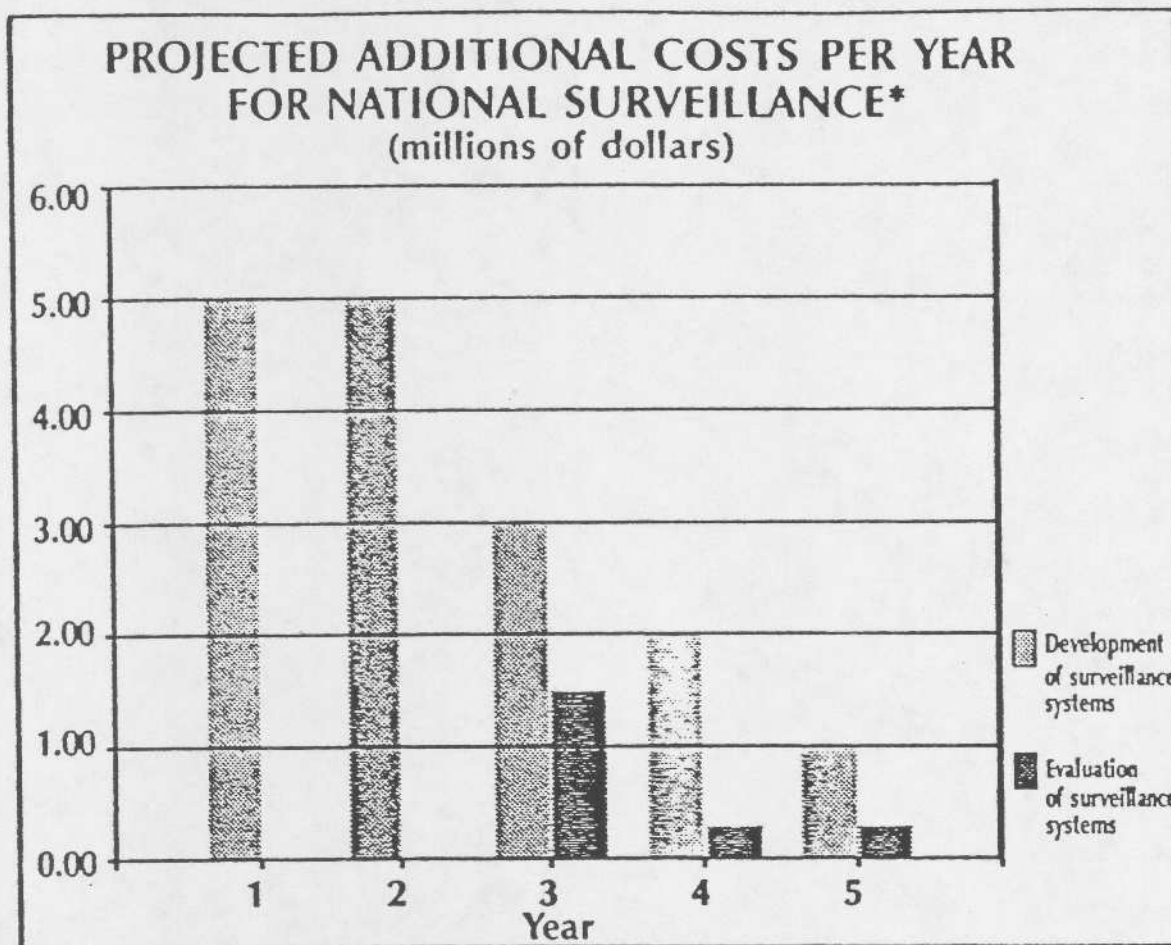
Increasing the amount of abatement conducted will also require development of a national abatement plan and infrastructure development, as described in Chapter 4. We estimate that the costs of such development work will be between \$3 and 6 million a year for the first 5 years of the Strategic Plan, and will total \$19.95 million over 5 years.

Following are detailed budgets for increased childhood lead poisoning prevention activities and national surveillance.

**PROJECTED ADDITIONAL COSTS PER YEAR FOR INCREASED
CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES***

	Cost per Year (millions of dollars)					Total Cost
	Year 1	2	3	4	5	
Increased funding for programs	25	25	25	35	45	155
Increased screening through EPSDT, WIC, and Head Start	0.25	0.25	0.25	0.25	0.25	1.25
Educational materials and outreach	0.5	0.3	0.01	0.01	0.01	0.83
Federal campaign to increase awareness	0.5	0.5	0.5	0.5	0.5	2.5
Clearinghouse	0.75	0.5	0.2	0.1	0.05	1.6
Infrastructure development for prevention activities	1.5	0.8	0.8	0.2	0.2	3.5
Total	28.50	27.35	26.76	36.06	46.01	164.68

*Costs reflect the amount of money needed to implement the program agenda and a shared commitment of the public and private sectors.



*Costs reflect the amount of money needed to implement the program agenda and a shared commitment of the public and private sectors.

Funds Needed for Implementation of the Research Agenda

Implementation of the research agenda will cost \$62 million. The next three tables summarize the budgets for the three main categories of research needed to support the program agenda.

PROJECTED ADDITIONAL COSTS PER YEAR FOR RESEARCH FOR CHILDHOOD LEAD POISONING PREVENTION ACTIVITIES*

	Cost per Year (millions of dollars)					Total Cost
	Year 1	2	3	4	5	
Cost-effectiveness of alternative screening strategies	0.5	0.5	0.5	-	-	1.5
Capillary collection devices	0.3	0.1	-	-	-	0.4
New instrumentation for measuring blood lead levels	0.2	0	-	-	-	0.2
Nutritional interventions	2	1	1	0.25	-	4.25
Educational strategies	2	1	0.5	0	0	3.5
Total	5.0	2.6	2.0	0.25	0	9.85

*Costs reflect the amount of money needed to implement the research agenda and a shared commitment of the public and private sectors.

**PROJECTED ADDITIONAL COSTS PER YEAR FOR
RESEARCH ON ABATEMENT***

	Cost per Year (millions of dollars)					Total Cost
	Year 1	2	3	4	5	
Efficacy of abatement	1.3	0.5	0.5	0.5	0.5	3.3
Alternative abatement methods	2	2	2	1	0.5	7.5
Measurement of lead in paint and dust	0.6	0.1	--	--	--	0.7
Worker exposure studies	0.5	0.5	0.2	0.2	0.2	1.6
Abatement of air ducts, etc.	1	--	--	--	--	1
Safe levels of lead in paint, dust, and soil	1	2	2	1	1	7
Preventive maintenance	0.8	0.5	0.2	0.2	0.2	1.9
Total	7.2	5.6	4.9	2.9	2.4	23.0

*Costs reflect the amount of money needed to implement the research agenda and a shared commitment of the public and private sectors.

PROJECTED ADDITIONAL COSTS PER YEAR FOR RESEARCH
ON REDUCTIONS IN OTHER SOURCES

	Year	Cost per Year (millions of dollars)					Total Cost
		1	2	3	4	5	
Sources of children's exposure	1	1.5	1.5	0.5	0.5	5	
Cost-effectiveness of soil abatement	2	2	2	2	1	9	
Drinking water lead levels	1	0.5	--	--	--	1.5	
Treatment for lead in water	0.3	0.3	--	--	--	0.6	
Sources of dietary lead	2	1	1	1	0.6	5.6	
Food lead measurement	0.4	0.15	0.15	0.15	0.15	1	
Bioavailability studies	1	1	--	--	--	2	
Lead biokinetics in pregnancy	1	1	1	0.5	0.5	4	
Total		8.7	7.45	5.65	4.15	2.75	28.7

*Costs reflect the amount of money needed to implement the research agenda and a shared commitment of the public and private sectors.

CHAPTER 7. SUMMARY OF RECOMMENDATIONS

In summary, childhood lead poisoning is a preventable disease with a huge societal cost. This plan outlines several steps that must be taken to eliminate sources of lead exposure for children. These steps will require a combination of government financial assistance and strategies to maximize the role played by the private sector.

The most urgent elements of the plan are the following:

- o Increased childhood lead poisoning prevention activities--These activities are essential to identify poisoned children and assure appropriate interventions are conducted. They are also important for targeting neighborhoods that need more intensive, communitywide interventions for preventing lead poisoning.
- o Increased abatement--A nationwide lead-based paint abatement program must be designed that will maximize the number of children benefited, given the fixed resources for abatement, using safe and effective methods.
- o Reductions in other sources and pathways--Ongoing efforts to limit children's exposure to lead from water, food, air, soil, and the workplace require continued attention.
- o Surveillance--A national surveillance system for elevated blood lead levels should be developed for tracking progress in eliminating childhood lead poisoning, identifying areas in need of further evaluation or interventions, and evaluating exposures of persons performing abatement and other workers.
- o Research - Research on lead should focus on developing and evaluating cost-effective methods for screening children, testing paint and dust for lead, and reducing the sources of lead to which children can be exposed as much as possible.

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APPENDIX I

LEAD EXPOSURE AND ITS EFFECTS ON CHILDREN AND FETUSES

The problem of human exposure to lead has been extensively studied, probably more than exposure to any other toxic substance. For public health policy, the following summary findings are especially important:

- o Lead is an extremely dangerous and pervasive environmental poison.
- o Today, far too many children are still exposed to excessive levels of lead: most recent national estimates indicate that in 1984, between 3 and 4 million children had lead in their bodies at levels which justify significant public health concern and which have been associated with neurobehavioral and other adverse health effects.
- o Our children have not been effectively protected from the major sources of lead exposure—especially leaded paint and lead-contaminated dust and soil.

LEAD EXPOSURE

Lead has some unusual characteristics that cause special concern. First, lead deposited in the environment remains there and accumulates. Therefore, lead distributed in the areas where we live from paint, gasoline, and stationary sources remains there. As long as lead continues to be added to our environment, more lead will accumulate.

Second, lead exposure is pervasive, sparing no socioeconomic segment of the United States. Since lead is dispersed into air, food, soil, dust, and water, children of all socioeconomic backgrounds in all geographic areas experience unacceptably high lead exposures. Overall, children living in or around old, dilapidated inner city housing are at highest risk for lead poisoning.

Third, lead accumulates over months and years in the bodies of children. Therefore, chronic exposure to small amounts of lead can lead to a large long-term accumulation in a child, increasing that child's risk of adverse health effects. In addition, it is believed that, during pregnancy, women's body lead stores may be mobilized, exposing the fetus to lead. Therefore, childhood exposures in one generation may result in prenatal exposure for the next generation.

Measurement of Lead Exposure

All Americans are exposed to some amount of lead. The amount of exposure has most often been quantified by measuring lead in blood (common units are ug/dL). Lead in blood reflects exposure during the previous weeks or months, whereas bone (or tooth) lead is a measure of cumulative lead exposure over months and years.

In most studies of the health effects of lead, measurements of outcome (such as IQ or behavioral changes) have been compared with blood lead measurements, and most public health decisions have been based on blood lead levels. Developing more practical methods to measure bone lead may substantially increase the use of such methods in assessing lead exposure; but at present, blood lead measurements remain the most generally used method of assessing human exposure to lead. The current definition of childhood lead poisoning is a blood lead level ≥ 25 ug/dL with an erythrocyte protoporphyrin (EP) level ≥ 35 ug/dL (Centers for Disease Control, 1985). This definition is currently being reevaluated, and the blood lead level will be revised downward to the level of 10-15 ug/dL.

Sources and Pathways of Lead Exposure

Children are exposed to lead from multiple sources such as paint, gasoline, solder, batteries, and stationary sources via multiple pathways such as air, dust, dirt, water, and food. The distinction between sources and pathways is not always clear. For example, dust and dirt are pathways for lead exposure. Because so much lead has been deposited in dust and dirt, they are sometimes also considered sources of lead exposure. In addition, in some discussions of lead exposure, water, food, and air are classified as sources of lead, although lead in these media comes almost totally from other sources.

The important public health point is that lead comes from known sources and moves through and is deposited in identified pathways to enter children. Although accurately tracing lead through all the complex pathways once it has left the source (e.g. leaded paint on a wall) may be difficult, it is not difficult to establish that reducing the amount of lead coming from the source will reduce the amount of lead going into children. For example, it is difficult to accurately trace all the pathways by which lead from gasoline enters children. Nonetheless, children's blood lead levels are well-correlated with gasoline usage patterns, and these levels have fallen dramatically in response to the reduction of lead in gasoline.

Children are exposed, therefore, when lead moves from its source through environmental pathways to be ingested or inhaled by a child. Reducing the amount of lead coming from these primary sources of lead (e.g. leaded paint on a wall) will reduce children's exposure to lead.

For the fetus, exposure comes from the mother's blood lead burden. The placental barrier is not effective in stopping lead from crossing over to the fetus (ATSDR, 1988). Generally, prenatal exposure is assessed by measuring the mother's blood lead level. The role of mobilization of maternal bone lead stores in prenatal exposure is yet to be determined.

For an individual child, the particular environment in which the child lives determines the relative importance of each lead source. For example, for a child living in a home with deteriorating lead paint, the paint will almost certainly account for a significant portion of exposure.

Although the immediate environment determines the importance of various lead sources for an individual child, estimates can be made of the overall relative importance of lead sources to U.S. children as a group. The Agency for Toxic Substances and Disease Registry (ATSDR) recently reviewed the available information on childhood lead exposure by source. The following are ATSDR's estimates of the number of children exposed by lead source (ATSDR, 1988). As noted in that report, these estimates are based on the best available information, and the estimation errors are difficult to quantify. The assumptions involved in the calculations differ for each source. Some numbers are for children potentially exposed and some for children actually exposed.

Lead in paint: Currently, leaded paint is the source of greatest public health concern. It is the most common cause of high-dose lead exposure. Exposure occurs not only when children ingest chips and flakes of paint (which often contain as much as 50% lead by weight) but also when children ingest lead paint-contaminated dust and soil, usually during normal mouthing activities. ATSDR has assessed that existing leaded paint in U.S. housing and public buildings is "an untouched and enormously serious problem."

About 13.6 million children under 7 years of age are potentially exposed in their homes to paint that contains lead at concentrations of 0.7 mg/cm^2 or higher. About 1.8 to 2.0 million children live in housing with unsound lead-based paint (e.g., holes in walls, peeling paint), which places them at high risk of excessive lead exposure; about 1.2 million of these children are estimated to have blood lead levels above 15 ug/dL , mainly due to exposure to leaded paint.

Lead-based paint abatement has been an essential part of all lead poisoning prevention programs in high-risk areas, despite cost constraints which limit the extent of such abatements. Historically, many studies have shown that the risk of lead poisoning is related to the presence of lead-based paint, and also to deteriorated or dilapidated housing (Gilbert et al., 1979); lead in dust is undoubtedly an important pathway for such exposures. Bornschein et al. and Chisholm have shown that children living in or returning to rehabilitated lead-free or lead-reduced housing after medical treatment for lead poisoning have significantly lower lead levels than children living in similar, non-rehabilitated housing (Bornschein et al., 1986; Chisholm, 1988). Three studies cited

in Appendix II demonstrate decreased blood lead levels in children with lead poisoning after the abatement of lead-based paint in their homes. (There may also be additional input to dust lead from lead in outdoor soil. Exposure to soil lead may occur from direct exposure to soil or indirectly as a result of its contribution to dust lead indoors. Lead in soil may arise from past use of exterior lead-based paint or from other external sources (see below). The value and role of soil abatement in addition to lead-based paint and dust abatement are currently being investigated in an Environmental Protection Agency (EPA) demonstration project; this issue will probably not be clarified for at least several years.)

Lead in gasoline: Since the introduction of lead as a gasoline additive in the mid-1920s, millions of tons have been used for this purpose (EPA, 1986). The recent reduction in the amount of lead in gasoline in the United States has been of major benefit to children. In the 13 years between 1976 and 1989, the amount of lead used in gasoline was reduced by more than 99% (EPA, 1990). Because of this, the blood lead levels in the U.S. population have decreased substantially.

Lead from primary and secondary smelters: About 230,000 children live near enough to a primary or secondary smelter to be exposed to lead from that source. Up to 13,000 of these children are estimated to have blood lead levels above 20 ug/dL from exposure to smelting by-products.

Lead in drinking water: In the United States, lead in water comes predominantly from lead in plumbing such as lead-soldered joints in copper pipes. The EPA maximum contaminant level (MCL) for lead is currently 50 ug/L. EPA estimated that in 1988, about 3.8 million children were exposed to water with a lead concentration higher than 20 ug/L. In 1988, EPA proposed that the allowable level for lead in drinking water be reduced. A revised lead standard is currently under consideration.

Lead in food: EPA estimated that about 42% of lead in food comes from lead-soldered cans or other metal sources, about 45% is deposited from the atmosphere, and the remainder comes from unidentified sources (EPA, 1986). Thus, almost 90% of lead in food comes from sources external to the food. Because of major decreases in the production of lead-soldered food and beverage cans and decreases in air lead levels due to decreases in gasoline lead, food lead levels are declining. The most obvious means of reducing lead in food is to reduce further lead in soldered cans and reduce lead emissions into the air (this has essentially been accomplished for mobile sources, i.e. automobiles, but not yet for stationary sources, such as smelters, incinerators, and other industrial sources).

Lead in dust and soil: Dust and soil act as a pathway to children for lead deposited by primary lead sources such as leaded paint, leaded gasoline, and stationary lead emitters. Since lead does not dissipate, biodegrade, or decay, the lead deposited into dust and soil becomes a long-term source of lead exposure for children. For example, although lead

emissions from gasoline are much reduced, gasoline lead deposited in years past remains in the dust and soil, and children continue to be exposed to it. The same is true for lead-based paint used in previous years. ATSDR (1988) has concluded that the "actual number of children exposed to lead in dust and soil at concentrations adequate to elevate blood lead levels cannot be estimated with the data now available."

Other sources and pathways of lead exposure: Several other sources and pathways are also important causes of elevated lead levels in many populations. These include lead in ceramic ware, folk remedies, hobbies or craftware, and childhood exposure to lead brought home by parents from their workplaces. As battery recycling increases, exposure to lead from this activity should be limited by control of emissions and lead levels in the workplace.

ADVERSE EFFECTS OF LEAD EXPOSURE

The adverse health effects on children from exposure to lead are a major public health concern. The risks associated with many chemicals, especially carcinogens, are extremely uncertain; for such chemicals, conservative approaches are used to extrapolate risks from animal or human occupational studies to estimate the upper limit of the risk posed to children and other populations. The adverse effects and risks of lead are well-known from studies of children themselves, and risk assessment calculations, with their inherent uncertainties, are not needed. Moreover, environmental lead levels in the United States provide no margin of safety to protect children; this is well-illustrated by the large number of children with lead levels in the toxic range. These effects of lead have been reviewed elsewhere in detail (ATSDR, 1988; EPA, 1986; EPA, 1989) and are only briefly summarized in this discussion.

High levels of lead in the body cause encephalopathy manifested by convulsions, mania, confusion, somnolence, or coma; if untreated, lead poisoning often results in death. Encephalopathy has been reported in persons with blood lead levels as low as 80 ug/dL (EPA, 1986). Since blood lead levels of 80 ug/dL can cause frank encephalopathy, it is not surprising that lower levels cause adverse effects on the central nervous system. In addition, lead affects the kidney, reproductive system, hematopoietic system, and virtually all other systems of the body.

Particularly disturbing are the following effects of lead exposure: 1) neurobehavioral effects of lead (including electrophysiologic changes) that occur at blood lead levels at least as low as 10 to 15 ug/dL; 2) reduced gestational age and reduced weight at birth that occur at levels at least as low as 10 to 15 ug/dL; 3) reduced growth rates up to 7 to 8 years of age that occur at levels at least as low as 10 to 15 ug/dL; 4) effects on heme metabolism starting at levels of about 15 to 20 ug/dL; and 5) effects on vitamin D

metabolism starting at levels of about 15 to 20 ug/dL (ATSDR, 1988; EPA, 1986; EPA, 1989). Some studies have even indicated effects at levels below 10 ug/dL; some effects appear to have no threshold. Millions of children have blood lead levels above or near these values.

In addition, studies on health effects of lead exposure during the past 20 years have produced a consistent trend: the more that is learned about lead's effects on children and the fetus, the more concern is generated by lower and lower blood lead levels. The lowest observed adverse effect level (LOAEL) continues to drop. Blood lead levels formerly considered safe, or without adverse effect, have now been clearly associated with adverse effects.

Although other health effects are of significant concern, a dominant focus of recent studies of lead is the effect of lead on central nervous-system cognitive function (e.g., intelligence). When the results are viewed collectively, a series of both prospective and cross-sectional studies provide persuasive evidence of lead's effects on children's cognitive function at blood lead levels as low as 10 ug/dL (ATSDR, 1988; EPA, 1986; EPA, 1989). Blood lead levels of 10 ug/dL and above at age 2 years have been shown to result in a reduction of the General Cognitive Index at age 57 months. Most of the children studied had blood lead levels below 15 ug/dL (Bellinger, 1991). Although researchers have not yet fully defined the impact of blood lead levels <10 ug/dL on central nervous system function, it may be that even these levels are associated with adverse effects that will be more clear as our research instruments become better. If there is a threshold for lead's effects, it is near zero.

In a recent long term follow-up study (Needleman, 1990), for children exposed to moderate lead levels during preschool years, the odds of dropping out of high school were seven times higher and the odds of a significant reading disability were six times higher than for children exposed to lower lead levels. In addition, these children had lower class standing, increased absenteeism, and lower vocabulary and grammatical-reasoning scores, even after controlling for other covariates. The magnitude and persistence of these impacts on ability to learn and perform well in school suggest that lead exposure may have a significant deleterious effect on how well a child will function in society.

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APPENDIX II

BENEFITS OF PREVENTING LEAD EXPOSURE IN THE UNITED STATES AND COSTS AND BENEFITS OF LEAD-BASED PAINT ABATEMENT

Lead exposure among U.S. children has been estimated to cost society billions of dollars annually (e.g., Levin, 1986). For this Strategic Plan, we have developed a new benefits analysis, taking into account recent data on the effects of lead on children and fetuses. In addition, we have developed an example of a cost-benefit analysis for the abatement of lead-based paint in pre 1950 housing. We based this analysis on data from three studies conducted between 1983 and 1988; information on the costs and benefits of abatement will have to be continually updated as newer information becomes available. The Department of Housing and Urban Development and others are attempting to develop new and more effective abatement practices.

THE BENEFITS OF PREVENTING LEAD EXPOSURE AMONG CHILDREN AND FETUSES

This analysis will focus on the benefits of preventing exposure to lead among children and fetuses. The benefits of reducing lead exposure of persons already being exposed are likely to be substantial, but they are difficult to quantify. For example, we do not know how long a child needs to have an elevated blood lead level to develop cognitive deficits, although presumably longer durations of exposure have greater and possibly more longlasting effects. Therefore, the benefits of reducing exposure in already-exposed persons will not be included in the main portion of this analysis, although they will be included in the sensitivity analysis. For purposes of this analysis, the benefits of preventing exposure to lead in children and fetuses are the avoided costs that would have been incurred had exposure occurred. The benefits for which we provide monetary values are 1) reduction in medical care costs incurred by poisoned children, 2) reduction in special education costs for poisoned children, 3) reduction in future lost productivity due to cognitive deficits in children, and 4) reduction in neonatal mortality due to prenatal lead exposure.

The above benefits are only a few of the benefits of preventing lead exposure. Many benefits cannot be described in monetary terms, (e.g., avoiding the emotional costs to families of having a lead-poisoned child). Other benefits, such as preventing lead's effects on children's stature, hearing, vitamin D metabolism, and blood production, will not be explored in this analysis. The reason is not that they are unimportant, particularly when summed over millions of children; rather, it reflects the absence of methods for estimating appropriate monetary values for these effects. We also have not evaluated the potential contribution of lead to juvenile delinquency (Needleman, 1989), the administrative costs of personal injury lawsuits, the improvement in property values from

improved housing conditions resulting from abatement, or the effects of lead on adults, such as increased rates of hypertension, stroke, and cardiovascular disease. By not including these effects, we grossly underestimate the costs of lead exposure to society.

The benefits evaluated in this analysis fall into two categories. The first category consists of only the benefits that will be achieved for children whose blood lead levels are prevented from rising above a certain threshold; avoided medical and special education costs are estimated only for those children who would have had blood lead levels ≥ 25 ug/dL. These costs are presented as the average cost for each child in this category; the figure derived takes into account that not all children with blood lead levels ≥ 25 ug/dL will need chelation therapy or special education. The second category consists of benefits of preventing increased blood lead levels in children no matter what their initial levels are. For example, intellectual deficits result over a broad range of blood lead levels. We estimated the avoided costs due to the effects of lead on intellectual functioning for preventing increases of 1 ug/dL in blood lead level, regardless of the child's starting blood lead level. The benefits of reducing maternal blood lead levels (i.e., decreased infant mortality) are also included in this latter category.

The Benefits of Preventing Children from Developing Blood Lead Levels ≥ 25 ug/dL

Medical costs: We assume, per the 1985 statement by the Centers for Disease Control, Preventing Lead Poisoning in Young Children, that children identified with blood lead levels ≥ 25 ug/dL will receive medical attention. Estimates of the medical care these children would need are based on data from Piomelli et al. (1984). We updated cost data from the regulatory impact analysis prepared by the Environmental Protection Agency (EPA) for reducing lead in gasoline (Schwartz et al., 1985) to 1989 using data from the medical care component of the Consumer Price Index.

Follow-up tests and administrative expenditures for all children whose blood lead levels are ≥ 25 ug/dL will total \$148 per child. Previous benefit analyses have used data from Piomelli et al. indicating that 70% of children with blood lead levels ≥ 25 ug/dL will have erythrocyte protoporphyrin levels ≥ 35 ug/dL and will receive provocative disodium calcium-edetate (EDTA) testing and follow-up. Provocative chelation requires a one-day hospitalization and one physician visit and is assumed to cost \$740. These same children will require a further series of follow-up tests and physician visits totaling \$444.

Five percent of children with blood lead levels ≥ 25 ug/dL will receive chelation therapy (Schwartz et al., 1985), requiring five days of hospitalization, several physician visits, laboratory testing and a neuropsychological evaluation. Half of these (2.5%) will require a second chelation therapy because their blood lead levels will rebound to ≥ 25 ug/dL. Half of these (1.25%) will require a third round of chelation therapy. (Therefore, an average of .0875 chelation therapies will be required for every child with a blood lead level ≥ 25 ug/dL.) The cost of each chelation therapy is estimated to be \$3,700.

To estimate the average medical cost per child with a blood lead level ≥ 25 ug/dL, the costs are multiplied by the associated probabilities by using the following equation:

$$\begin{aligned} \text{AMC} &= \text{PFU}(\$FU) + \text{PEDTA}(\$EDTA) + \text{PCHEL}(\$CHEL) \\ &= 1.0(\$148) + 0.70(\$740 + \$444) + 0.0875(\$3,700) = \$1,300 \end{aligned}$$

where

AMC	=	Average medical costs for children ≥ 25 ug/dL
PFU	=	Probability of follow-up testing for children ≥ 25 ug/dL
\$FU	=	Cost of follow-up testing
PEDTA	=	Probability of receiving provocative EDTA testing and follow-up
\$EDTA	=	Cost of EDTA testing and follow-up
PCHEL	=	Probability of receiving chelation therapy
\$CHEL	=	Cost of chelation therapy

Therefore, the total medical cost that can be avoided by preventing a child from developing a blood lead level above 24 ug/dL is \$1,300.

Costs of special education: Children with high blood lead levels are more likely to have decreased school performance and require reading or speech therapy or psychological assistance. The costs of such treatment can be substantial. In a 3-year follow-up of children with high and low blood lead levels, de la Burde and Choate (1975) reported a relative risk of 7 for poor academic progress and a relative risk of 4 for repeating a grade. In addition, they reported that cognitive effects persisted for at least 3 years. Bellinger et al. (1984) reported that an excess of 17% of children with high blood lead levels were receiving daily assistance outside the classroom. Needleman et al. (1990) recently reported an odds ratio of 5.8 for reading disability among the children in their high lead group. Lyngbye et al. (1990) reported an odds ratio for learning disability of 4.3 for children with tooth lead levels above 16 parts per million (ppm).

On the basis of these reports and previous benefits analyses (Schwartz et al., 1985), we assume that 20% of children with blood lead levels ≥ 25 ug/dL will require special education (defined as assistance from a reading teacher, school psychologist, or other specialist) for an average of 3 years. Costs for part-time special education have been estimated by Kakalik et al. (1981) to be \$5,827 per year (updated to 1989 by using the Consumer Price Index). Because costs would be incurred over 3 years, costs in years 2

and 3 are discounted at 5%* to the year special education begins. The average special education costs for children with blood lead levels ≥ 25 ug/dL in year 1 are computed by using the following equation:

$$\begin{aligned} \text{ASEC} &= (\text{PSE})(\$SE) \\ &= (0.20)(\$5,827) = \$1,165 \end{aligned}$$

where ASEC = Average special education costs
 PSE = Probability of requiring special education
 \$SE = Cost of special education

Discounting years 2 and 3 by 5% results in total special education costs of \$3,331 per child with a blood lead level ≥ 25 ug/dL.

The Benefits of Preventing a 1 ug/dL Increase in the Blood Lead Levels of Children

Most children with lead-related cognitive deficits do not require special education or other assistance; however, their losses can still be substantial in monetary terms. Impaired cognitive functioning and IQ decrements can reduce a person's productivity in society. In this benefits analysis, we use this loss in productivity as a proxy for the cost to society of cognitive impairment. This cost is clearly an underestimate because it puts no value on the losses sustained by the individual that are not reflected by decreased economic productivity. In addition, this analysis does not consider unearned income (e.g., interest, dividends), which would presumably be affected as the wage rate. We assume for this analysis that the benefits of reducing lead exposure on the cognitive functioning of children exhibit no threshold.

Figure 1 depicts the relationship between lead exposure and earnings. The first way lead exposure affects earnings is through its effect on IQ.

Lead has a direct effect on cognitive functioning, as measured by changes in IQ (pathway a). This reduction in IQ then has a direct effect on wage rate (pathway b), which affects

*When costs or benefits occur in the future, they should be adjusted by discounting. The principle behind discounting is that there is a social as well as a personal preference for postponing costs and obtaining benefits as soon as possible. Therefore, dollars available in the future are less valuable than those available today. Mathematically, discounting future dollars can be thought of as the opposite of computing a return on an investment. Discounting, therefore, has the effect of reducing the numerical value of benefits or costs occurring in the future. For all calculations, we use a discount rate of 5% real (i.e., 5% above the rate of inflation).

lifetime earnings. Lead also affects earnings through its effect on educational attainment by reducing IQ and by other effects, such as decreased attention span (pathway c). The effect of educational attainment on earnings is traceable through two main pathways. First, educational attainment is directly associated with wage rates and, therefore, with lifetime earnings (pathway d). Second, educational attainment is also associated with labor force participation (pathway e), which again has an effect on lifetime earnings.

The relationship between lead exposure and IQ (pathway a): Needleman and Gatsonis (1990) reported on a meta-analysis of the recent studies associating lead exposure with cognitive deficits. Although they reported only joint p values and partial r values, we used this information to perform a meta-analysis on effect size. We computed the estimated change in IQ for a 1 ug/dL change in blood lead for the six studies for which regression coefficients relating blood lead levels to IQ decrements were reported. Weighting by the inverse of the variance of each estimate, we estimate that each 1 ug/dL change in blood lead level results in a 0.25 point change in IQ.

The direct effect of IQ on wage rate (pathway b): A large body of literature exists on the relationship between IQ and wage rate. For example, in studies that examined the economic impact of increased schooling, it was important to control for differences in IQ; thus, the marginal impact of IQ on wage rate was estimated. In a review of the literature, estimates of the direct effect of IQ on wage rate (pathway b) ranged from a 0.2% to a 0.75% change in wage rate for each one IQ point change (Barth et al., 1984).

Structural equations modeling can be used to estimate the impact of multiple variables on an outcome of interest. Griliches (1977) used structural equations modeling and estimated the direct effect of IQ on wage rate to be slightly more than 0.5% per IQ point. Because this method has conceptual advantages and 0.5% is roughly the median estimate in the review by Barth et al. (1984), we used this value in these benefits estimates.

The impact of lead exposure on educational attainment (pathway c): From Needleman et al. (1990) and Needleman and Gatsonis (1990), it is possible to estimate the change in years of schooling attained per 1 IQ point change. The regression coefficients for the effect of tooth lead on achieved grade in those studies provide an estimate of current grade achieved, not of expected grade. Some of the children in those studies were, however, in college at the time of data collection and were expected to attain a higher grade. After adjusting the published results for the fact that a higher than reported percentage of the children with low tooth lead were likely to be attending college, we estimated a 0.59 year difference in expected maximum grade achieved between the high and the low exposure groups. We assumed that educational attainment scales with blood lead levels in proportion to IQ. The difference in IQ score between the high and the low exposure groups was 4.5 points. By dividing .59 by 4.5, we estimate that, the increase in blood lead level that reduces IQ by one point, reduces years of schooling achieved by 0.131 years.

Education and wage rate (pathway d): Studies that allow estimates of the relationship between educational attainment and wage rate (pathway d) are less common than those assessing the direct effects of IQ on wage rate. Chamberlain and Griliches (1977) estimated that a one year's increase in schooling would increase wages by 6.4%. In a model with similar specifications, Olneck (1977) reported a 4.8% increase. In a longitudinal study of 799 subjects for 8 years, Asbenfelter and Ham (1979) reported that an extra year of education increased the average wage rate by 8.8%. We have taken 6% as a reasonable and slightly conservative estimate of the effect of a year of schooling on wage rate.

Education, labor force participation, and earnings (pathway e): In addition to affecting wages, lead exposure is likely to affect participation in the labor force for several reasons. Labor force participation is correlated with failure to graduate from high school, principally through higher unemployment rates and earlier retirement ages. Lead exposure is also strongly correlated with attention span deficits and other effects which would also be likely to reduce labor force participation.

The differences in labor force participation between high school graduates and nongraduates were obtained from an analysis of the data in the 1978 Social Security Survey of Disability and Work by Cropper and Krupnick (1989), which controlled for age, marital status, number of children, race, region, and other socioeconomic and medical variables. We have estimated, using their regression coefficients, that average participation in the labor force is reduced by 10.5% for persons who fail to graduate from high school (pathway e). It is possible that this analysis overcontrols for other factors in estimating the effect of schooling. For example, high school drop-outs are more likely to have occupations with a higher risk of disability, which was also included as an independent variable in the regression analysis. Using the 1978 Current Population Survey, stratified by age groups between 25 and 65 years, we found that the mean number of hours worked in the previous year was 20% lower for persons with less than a high school education than the number worked by those with a high school education and those who graduated from high school. This difference results from reduced participation in the labor force and reduced hours worked by participants, and suggests that lead exposure sufficient to cause a 1 IQ point decrease would decrease expected earnings by about twice as much as reported by Cropper and Krupnick. To be conservative, we have used the results derived by using the regression models in Cropper and Krupnick as the estimate in this analysis. Using the study of Needleman et al. (1990), we estimate that lead exposure sufficient to cause a 1 point reduction in IQ would result in a 4.5% increase in the risk of failing to graduate (pathway c').

Lifetime earnings: Annual lifetime-earnings benefits achieved by preventing a 1 ug/dL increase in a child's blood lead level are computed as the net present value of the increased earnings expected from preventing the increase, discounted to age 6. To calculate the net present value of lifetime earnings, a number of assumptions are required. First, dollars available in the future must be discounted (see footnote, page 4).

The second assumption is that the real wage growth in the future will be 1% per annum from the 1987 distribution of incomes. (Historically, real wage rates have increased approximately 2% per annum; however, in the last decade that growth rate has fallen.) This assumption is conservative because (1) it assumes that the same percentage of the work force will have a college education in the future as in 1987, and (2) it assumes the 1987 ratio of female to male earnings will remain unchanged, whereas the ratio has increased from 0.6 to 0.7 in the past 15 years and is expected to continue to increase in the future. On the other hand, this assumption is not conservative because it assumes that women will participate in the labor force at the same rate as in 1987, whereas their participation is likely to increase. If women continue to earn less than men, the real overall wage rate may not grow as quickly as 1% per year.

A third assumption made in calculating the net present value of lifetime earnings concerns labor force participation and the value placed on the productivity of nonparticipants. Many adults do not participate in the work force at all during their potential working years. The largest group are women who remain at home doing housework and child rearing. There is no consensus on how to put a monetary value on this nonmarket productivity. Work in the home has been valued in economic studies by using either the opportunity cost (the value of foregone income) or the market value of substitute labor for this work. The opportunity cost is usually taken as the average wage earned by persons of the same age, sex, and educational level. This may be too high, as the employed members of these cohorts tend to have more work experience, more training, and more relevant education than those who remain at home. The estimate based on the market value of substitute labor is often too low, as many of the substitute workers have less education than the persons they would replace. The most appropriate value is likely to be between these two estimates.

Given an estimate of the value of this nonmarket work, an additional assumption must be made about whether the impact of lower IQ on nonmarket productivity is the same as on market wages. There appears to be an association between maternal IQ and child's IQ, which is unlikely to be entirely hereditary. Moreover, in recent lead studies the Home Observation for Measurement of the Environment (H.O.M.E.) score (a measure of the quality of the home rearing environment) has been positively correlated with the mother's and the child's IQs (Bellinger et al., 1984). These findings suggest that IQ has an impact on nonmarket work, at least on the child-rearing component. For this analysis, we have taken the value of lost productivity due to lead exposure for nonparticipants in the labor force as half the value for employed workers.

Data for calculating the values of the expected lifetime earnings of an average child in the United States, under these assumptions, were obtained from 1987 earnings profiles from the U.S. Bureau of the Census. To compute the average lifetime earnings, we assumed that the numbers of men and women in the population would be equal, and part-time workers and non-labor force participants would earn half as much as average full-time workers. The net present value of average lifetime earnings per child,

discounted to age 3, is estimated to be \$260,000. The average child born into a housing unit 1 year after abatement is 4 years younger than the child currently occupying the unit; discounting that child's lifetime earnings to today's value yields a net present value of \$223,000.

Total earnings benefits: Figure 1 shows the pathways through which lead exposure affects total earnings benefits. The lower case letters a to e correspond to the pathways on Figure 1., on page 25.

We used the following equations to calculate the total lost wages attributable to reductions in earnings because of lead exposure:

1. The estimated change in wage rate for a 1 ug/dL change in blood lead level can be expressed as follows:

$$a*b = .25*.5\% = .125\%$$

where a = estimated change in IQ for each 1 ug/dL change in blood lead level
 (.25 IQ points per 1 ug/dL change in blood lead level)

 b = estimated percentage change in wages for a 1 IQ point change (.5%
 wage change per IQ point)

2. The average change in wage rate from the decreased educational attainment resulting from lead exposure can be expressed as follows:

$$a*c*d = .25*.131*6\% = .197\%$$

where c = estimated change in grade attained for a 1 IQ point change (.131
 years schooling per 1 IQ point change resulting from lead exposure)

 d = estimated percentage change in wage rate for a 1 years change in
 grade attained (6% per year of schooling)

3. The average change in wage rate from decreased labor force participation from failure to graduate from high school can be expressed as follows:

$$a*c'*e = .25*4.5\%*10.5\% = .118\%$$

where c' = estimated change in the probability of graduating from high school
 for a 1 IQ point change resulting from lead exposure (4.5%
 increased probability of failure to graduate for each 1 point
 decrease in IQ)

c = estimated percentage change in labor force participation because of failure to graduate from high school (10.5% decrease in labor force participation because of failure to graduate)

4. Therefore, the change in the expected present value of lifetime earnings from a 1 ug/dL change in blood lead levels can be expressed as follows:

$$\begin{aligned}\Delta E &= E[(ab) + (cd) + (c'e)] \\ &= \$260,000[(.125\% + .197\% + .118\%)] \\ &= \$260,000 \cdot .441\% = \$1,147\end{aligned}$$

where ΔE = the expected change in lifetime earnings from exposure to lead
E = the net present value of lifetime earnings

We estimate, therefore, that prevention of an increase of 1 ug/dL in a child's blood lead level will produce a net present value benefit of \$1,147 per child. We again note that lost income is a clear underestimate of cognitive impairment, reduced educational attainment, and reduced labor force participation.

The Benefits of Preventing Prenatal Exposure to Lead

Prenatal lead exposure has been linked with reduced gestational age, lower birth weight, and decreased cognitive functioning, even in children exposed to low-to-moderate maternal blood lead levels (Dietrich et al., 1987). In this analysis, we assess only the impact on mortality of low gestational age due to lead exposure, since the data supporting the relationship between prenatal lead exposure and gestational age are stronger than the data supporting the relationship between lead exposure and low birth weight. In addition, to avoid the possibility of counting some infants in both prenatal and postnatal estimates, we did not assess the consequences of cognitive damage. Prenatal exposure has also been linked to stillbirths in a number of studies (Vimpani et al., 1990), but we have not computed any benefits of avoiding fetal loss because the evidence is not complete and no study provides a dose-response function. We also have not computed costs from hospitalizations for premature and low birthweight infants. These omissions result in an underestimate of the benefits of reducing prenatal exposure to lead.

The impact of prenatal exposure on mortality: We used data from the Linked Birth and Infant Death Record Project (National Center for Health Statistics) to estimate infant

*The numbers shown are based on calculations using the most precise numbers possible. Because of rounding, there may be small differences between the numbers shown and those obtained by performing the calculations described.

mortality as a function of gestational age. The impact of prenatal lead exposure on gestational age is obtained from Dietrich et al. (1987). These estimates yield a predicted reduction of 10^{-4} (or 0.0001) in risk of infant mortality for each 1 ug/dL reduction in maternal blood lead level. In this analysis, we assume that the relationship between neonatal mortality and low gestational age is the same whether it results from prenatal lead exposure or from all other causes of low gestational age.

Valuing reductions in mortality: Placing a monetary value on reductions in mortality is highly controversial. The U.S. Department of Transportation has used lifetime wages (human capital approach) as a proxy, an approach common in litigation as well. This approach has obvious faults. For example, the value of reducing early mortality among retired persons or housewives is not zero, even though they may not be expected to earn wages. Because this approach underestimates the value of human life by approximating its value with the economic productivity of an individual, most economists prefer the willingness to pay method for valuing reductions in mortality.

Numerous methods for valuing people's willingness to pay for reducing their risk of mortality have been employed. The two most common methods include surveys that present realistic scenarios of trade-offs between expenditures and mortality risks or contingent valuation studies (Jones-Lee et al., 1985; Gegax et al., 1985) and assessments of market transactions that reveal implicit trade-offs between risk and dollars (e.g., Thaler and Rosen, 1976; Smith, 1976; Viscusi, 1978; Viscusi and O'Connor, 1984). Estimates resulting from these studies range from \$500,000 to \$9 million per statistical life, with most estimates falling between \$1 million and \$5 million (Violette and Chestnut, 1989). We have taken \$3 million per statistical life as the best estimate of the willingness to pay to avoid excess mortality risk.

Under these assumptions, the monetary benefit associated with reducing infant mortality is (0.0001) (\$3,000,000) or \$300 per ug/dL increase in blood lead level prevented for each pregnant woman.

Total Benefits of Preventing Lead Exposure

On the basis of the above analyses, the benefits of preventing a child's blood lead level from reaching 25 ug/dL are \$4,631 for avoided medical and special education costs. The increased productivity to be expected from preventing a 1 ug/dL increase in a child's blood lead level is \$1,147. Clearly, the greater the prevented increase in blood lead level, the greater the benefits; for the individual child, preventing the blood lead level from exceeding 24 ug/dL results in maximum benefits. The average benefits of preventing a 1 ug/dL increase in the blood lead level of a pregnant woman are \$300.

ABATEMENT OF HOMES

In this section we describe the costs and effectiveness of lead-based paint abatement.

Effectiveness of Lead-Based Paint Abatement in Reducing Lead Exposure

Studies have shown that abatement of lead-based paint in housing is effective in reducing children's blood lead levels (Kennedy, 1978), but quantitative data on these reductions are limited. We obtained both cost of abatement and effectiveness data for three evaluations of the efficacy of abatement--a study by Rosen et al. in New York City (in press), a study from St. Louis (G. Copley, unpublished data), and a study from Massachusetts (Y. Amitai et al., unpublished data). These data are not necessarily representative of abatements as they are currently performed. However, because of the lack of other data, we used them for our cost-benefit analysis.

In a study on the use of bone lead measurements in New York City children, Rosen et al. (in press) reported on children who did not receive chelation therapy but who did have their homes abated. At 24 weeks, the children's blood lead levels had declined from an initial mean level of 29 ug/dL to 21 ug/dL. Abatement methods used in this study included scraping, spackling and repainting surfaces with deteriorating lead paint.

An unpublished study from the City of St. Louis Division of Health (G. Copley, unpublished data) reported that children who did not receive chelation and whose homes were abated experienced a mean reduction of 9.3 ug/dL in blood lead levels (from 43.9 to 34.2 ug/dL) measured 6 to 12 months after the abatement. Abatement consisted of scraping or encapsulating deteriorated surfaces.

An evaluation of data collected in 1984 and 1985 by the Massachusetts Childhood Lead Poisoning Prevention Program (Y. Amitai et al., unpublished data) examined the intraabatement and postabatement blood lead levels of children who received no chelation therapy. The purpose of the study was to examine the impact of abatement method on intraabatement blood lead levels when children were not relocated during abatement. Several abatement methods were employed, including dry scraping, sanding, and encapsulation. Mean blood lead levels 8 months postabatement decreased by 10.2 ug/dL (from 35.7 ug/dL to 25.5 ug/dL).

In these three studies, the approximate mean decrease in blood lead levels after abatement was 9 ug/dL for children in lead-contaminated housing and with initial blood lead level ≥ 25 ug/dL.

These studies only included children with blood lead levels ≥ 25 ug/dL. No data are available on the effects of abating homes of children with lower blood lead levels. In the studies, the mean decrease in a child's blood lead level with abatement was 25%. For the cost-benefit analysis presented here, we assume that the reduction in blood lead levels from abatement of homes of children with initial levels < 25 ug/dL will be proportional to the reduction for children with higher blood lead levels. Thus, children with blood lead levels < 25 ug/dL will experience a 25% decrease in blood lead levels

from lead-based paint abatement. Using estimates from models developed by EPA and others, we estimate that the mean blood lead level for children whose preabatement levels are between 10 and 24 ug/dL is 15 ug/dL (J. Schwartz, personal communication). For children whose blood lead levels are between 10 and 24 ug/dL, the mean decrease in blood lead levels expected from abatement is 3.75 ug/dL.

Costs of Lead-Based Paint Abatement

We contacted individuals associated with the abatement programs in New York City, St. Louis, and Massachusetts to ascertain the nature and approximate costs of the abatement methods used at the time that data for these studies were compiled. All three programs relied extensively on scraping, spackling, and repainting areas with deteriorated lead-containing paint. Encapsulation was less frequently used (only in Boston and St. Louis), and there was no replacement of doors, windows, or woodwork. Only deteriorated or damaged lead-containing surfaces were abated routinely in New York City and St. Louis, while Boston abatements included stripping of all chewable, accessible surfaces below 5 feet (e.g., window sills, baseboards, door frames), regardless of condition, if they contained lead. Costs for abating an average unit of 5 to 6 rooms for each of the cities were as follows: St. Louis - \$2,000, New York City - \$2,500, and Boston - \$1800 (inflated when necessary to 1989 prices by using the Consumer Price Index). These prices include abatement of common areas and exteriors when necessary and costs of materials, labor, insurance, overhead, whatever worker protection was employed, preparation of the unit before abatement, and cleanup. An average cost of \$2,100 will be assumed for these studies.

It should be noted that some currently recommended abatement methods and procedures are much more expensive than those discussed above. A cost-benefit analysis was not conducted for these more rigorous abatements because data on associated changes in blood lead levels are not available.

The investigators in New York City, St. Louis, and Massachusetts were questioned about the longevity of the effectiveness of these abatement methods--that is, about how long a relatively "lead-free" environment would be maintained in the home. In all three cases, the investigators reported that repointing after abatement was very infrequent (considerably less than 1% within a year). This does not address the problem of long-term effectiveness of the abatement, for example, 5 to 15 years after the original abatement is completed.

The average charge for home inspections in several lead poisoning prevention programs is \$97 per unit (K. O'Connor, C. Torres, H. Billingsly, personal communications), which we round to \$100. If we assume that 80% of pre-1950's housing contains leaded paint (Shier and Hall, 1977), the cost for an investigation per positive home is \$125. Therefore, the total cost of abatement is \$2,225 per unit. Several costs are not included in these estimates because they are more difficult to quantify or are extremely variable.

One source of costs is court proceedings, for example, when notices to landlords are challenged; another is for the dislocation of families from their homes and the effects on neighborhoods when landlords refuse to abate marginally viable housing. An additional cost results if families are relocated to alternative housing at program expense.

COST-BENEFIT ANALYSIS

In the following section, we present a cost-benefit analysis for abatement of an average house with lead-based paint built before 1950. In the analysis, we will use the data presented earlier on the costs and effectiveness of abatements performed in St. Louis, New York City, and Boston.

The benefits used in this analysis are likely to be substantial underestimates of the true benefits of abatement. In addition to the reasons for underestimation already discussed, a very important component of underestimation in this analysis is that we will not assign monetary value to the benefits of abating homes of children and pregnant women who already are currently being exposed to lead. This assumption may be unjustified for several reasons. First, children remaining in a lead-contaminated environment may need repeated courses of medical treatment for continued elevations in blood lead levels. Second, the blood lead levels of some of these children will increase further as a result of living in lead-contaminated homes, thereby increasing the probability that they will need medical care and special education and further reducing their future earnings. Third, decreasing the amount of time children and pregnant women have elevated blood lead levels will probably decrease the adverse effects from lead. Data are not available, however, to allow these benefits to be quantified. Therefore, this cost-benefit analysis is conducted under the assumption that we target homes for abatement in a high-risk area based on the home's containing lead-based paint and having been built before 1950. All benefits are accrued by children who will enter a high-risk age group in the house in the future and by fetuses potentially exposed to lead in the future. No attempt is made to target abatement to the homes of currently lead-poisoned children.

For this cost-benefit analysis, we use the following assumptions:

Assumptions:

Assumption 1: In general, children's exposure to lead-based paint and paint-contaminated dust and soil begins to increase when they become mobile and decreases as they practice less mouthing behavior. We will assume that children less than 10 months and greater than 6 years of age are unlikely to be poisoned by lead-based paint, regardless of their housing. Therefore, quantitative benefits can be assessed for children who are less than 10 months of age and are now living in the unit, for children who are likely to be born into or move into the abated unit, and women who will become pregnant while living in the abated unit.

Assumption 2: On average, there are 0.287 children per house built before 1950 (Pope, 1986). The average number of children less than 10 months old per pre-1950 housing unit is $(0.287 \text{ children} \times (9/72 \text{ months})) = 0.036 \text{ children}$.

Assumption 3: This analysis is performed for the average home built before 1950 which is painted with leaded paint.

Assumption 4: The average overall loss rate of housing, both rental and owner-occupied, built before 1950 is 1% per year (D. McGough, Department of Housing and Urban Development, personal communication). On the basis of this assumption, the median remaining life of existing housing stock built before 1950 is 68 years.

Assumption 5: A targeting strategy is employed that abates homes built before 1950 that contain lead-based paint, whether or not these homes currently contain children.

Assumption 6: Were the targeted homes not abated, some of the children who occupied them would have become lead poisoned. For these children the increase in blood lead level prevented by abatement is 9 ug/dL. For children who would not have become poisoned, the average prevented increase in blood lead level is 3.75 ug/dL.

Assumption 7: On the basis of data from Cincinnati, the difference in blood lead levels among pregnant women in lead-contaminated 19th century housing and those in lead-free public housing is 2.13 ug/dL (R. Bornschein, personal communication). On the basis of these data, we assume that abating a unit results in the prevention of a 2.13 ug/dL increase in the blood lead levels of pregnant women.

Assumption 8: Almost 6 million children under 7 years of age live in pre-1950 housing with high levels of lead in paint (ATSDR, 1988). Of these, 0.2 million, or 3.4%, have blood lead levels above 25 ug/dL. Thus, for this analysis we will assume that abatement will prevent blood lead levels ≥ 25 ug/dL in the 3.4% of children who would be expected to develop them otherwise and will prevent levels between 10 and 24 ug/dL in the rest. Thus, the average prevented increase in blood lead level for a child living in a house contaminated with lead-based paint is:

$$(0.034) (9 \text{ ug/dL}) + (0.966) (3.75 \text{ ug/dL}) = 3.93 \text{ ug/dL}$$

Assumption 9: An average of 0.045 infants below age 1 year are present in housing units built before 1950 (Pope, 1986). We will therefore assume that 0.045 children are born into each unit each year after abatement and that 0.045 represents the proportion of pregnant women in such houses each year. Abatement prevents an increase of 2.13 ug/dL in the blood lead level of a pregnant woman. Thus, the average prevented increase in blood lead levels in pregnant women in abated housing is:

$$(0.045) (2.13) = .096 \text{ ug/dL}$$

Assumption 10: The medical costs avoided by preventing a child born into a unit the year after abatement from developing a blood lead level ≥ 25 ug/dL is \$1,069 (discounted to 4 years into the future, since we assume costs are incurred at age 3 years). The avoided special education costs are \$2,365 (discounted to 7 years into the future). For each 1 ug/dL blood lead level increase prevented in a child, \$1,085 in lost earnings is avoided.

Assumption 11: Each avoided increase of 1 ug/dL blood lead in a pregnant woman by abating the unit the year before she becomes pregnant, when discounted to 1 year in the future, results in an average savings of \$286 from the prevention of infant mortality.

Assumption 12: A discount rate of 5% is used.

Assumption 13: Analysis is done for a set time. Both benefits and costs are discounted to that year.

Assumption 14: Benefits can be assessed for each cohort of children entering the home. Because we assume that the average remaining lifespan of housing units built before 1950 is approximately 68 years, benefits are calculated for 68 cohorts of children, with benefits being discounted appropriately.

Assumption 15: The discounted total value of benefits for children is equal to the sum of the benefits accruing for current resident children less than 10 months of age and the benefits accruing to children who will move into or be born into the residence in the future. These are the benefits of avoiding medical and special education costs and increasing earnings.

Assumption 16: Benefits are also accrued by reducing blood lead levels in pregnant women who will live in the house in the future.

The following equations summarize this information. In these calculations, figures are only presented up to 4 decimal places. As a result, an attempt to duplicate the calculations performed will result in rounding errors; final values are based on the most precise figures possible.

1. The proportion of children now living in the home who will accrue benefits from the avoidance of medical and special education costs can be expressed as follows:

$$\begin{aligned} f &= g \cdot h \\ &= .034 \cdot .036 = .0012 \end{aligned}$$

where f = average number of children per house less than 10 months of age living in pre-1950 housing whose blood lead levels would be expected to rise above 24 ug/dL

g = average proportion of children with blood lead levels above 24 ug/dL (.034; see Assumption 8, page 14)

h = average number of children less than 10 months of age per housing unit built before 1950 (0.036; see Assumption 2, page 14)

2. The proportion of future children who will accrue benefits from avoidance of medical and special education costs can be expressed as follows:

$$\begin{aligned} i &= g*j \\ &= .034*.045 = .0015 \end{aligned}$$

where i = average number of children per house who will be born into the average pre-1950 house each year of the house's remaining lifespan and whose blood lead levels would be expected to rise above 24 ug/dL without abatement

j = average number of pregnant women per house per year (.045; see Assumption 9, page 14)

3. The net present value of medical costs avoided through abatement is the sum of the avoided costs for children currently living in the unit plus the avoided costs for 67 cohorts of future children.

$$\begin{aligned} MED &= f*AMC + i*[AMC_1 + AMC_2 + \dots + AMC_{67}] \\ &= .0012*\$1,300 + .0015*\$21,605.23 \\ &= \$1.59 + \$33.06 = \$34.65 \end{aligned}$$

where AMC = average medical costs for children with blood lead levels above 24 ug/dL the present year (\$1,300; see page 3)

AMC_n = average medical costs for children with blood lead levels above 24 ug/dL in year n , discounted to the present year

4. The net present value of special education costs avoided through abatement is the sum of the avoided costs for children currently living in the unit plus the avoided costs for 67 cohorts of future children.

$$\begin{aligned} SEC &= f*ASEC + i*[ASEC_1 + ASEC_2 + \dots + ASEC_{67}] \\ &= .0012*\$3,331 + .0015*\$47,783.47 \\ &= \$4.06 + \$73.11 = \$77.17 \end{aligned}$$

where $ASEC$ = average special education costs for children with blood lead levels above 24 ug/dL in the present year (\$3,331; see page 4)

$ASEC_n =$ average special education costs for children with blood lead levels above 24 ug/dL in year n , discounted to the present year

5. The average net present value of lost earnings prevented by abatement for children less than 10 months of age currently living in the home is:

$$INC_c = h * k * \Delta E$$

$$= .036 * 3.93 * \$1,147 = \$161.65$$

where $INC_c =$ lost earnings prevented for current children less than 10 months of age living in lead-painted homes

$k =$ average decline in blood lead levels of children from abatement (3.93 ug/dL; see Assumption 8, page 14)

$\Delta E =$ change in earnings that can be attributed to a 1 ug/dL change in blood lead level (\$1,147; see page 9)

6. The net present value of lost earnings prevented through abatement for future children is the avoided costs for 67 cohorts of future children.

$$INC_f = j * k * [E_1 + E_2 + \dots + E_{67}]$$

$$= .045 * 3.93 * \$19,781 = \$3,497$$

where $INC_f =$ net present value of lost earnings prevented for future children who would live in pre-1950 homes

$E_n =$ average present value decrease in earnings for each cohort, discounted to the present year

7. The net present value of lives saved from avoided mortality from reducing prenatal lead exposure can be expressed as follows:

$$LIFE = i * [M_1 + M_2 + \dots + M_{67}]$$

$$= .096 * \$5,571.72 = \$553.22$$

where $i =$ average prevented increase in a pregnant woman's blood lead level from abatement (.096 ug/dL; see Assumption 9, page 14)

$M_n =$ average benefits from reduced fetal mortality of preventing a 1 ug/dL increase in blood lead level in year n , discounted to the present year

8. The total benefits of abatement are:

$$\text{MED} + \text{SEC} + \text{INC}_c + \text{INC}_f + \text{LIFE} \\ = \$34.65 + \$77.17 + \$161.65 + \$3,497.00 + \$553.22 = \$4,323.70$$

Costs Versus Benefits of Abatement

We estimate that abating an average pre-1950 lead-painted home using the methods employed for the three studies described above earlier costs \$2,225, and the benefits over the lifetime of the home are \$4,323. Thus, abatement of a home results in a net benefit of \$2,098. This net benefit does not take into account any benefits sustained by a child who is already poisoned in the unit or the numerous benefits to which we could not assign monetary values.

This cost-benefit analysis provides an economic justification for a national program of abating lead-contaminated housing to prevent childhood lead poisoning. This analysis is conservative because a number of important benefits remain unquantified. Moreover, prevention of lead poisoning would be an important public health activity, even if no economic benefits could be demonstrated.

This analysis indicates what is needed for a rational national abatement program. Obviously, the better a plan for setting priorities for abatement can be targeted to homes likely to house children in the future, the greater the net benefits. Furthermore, if strategies can be developed for determining which homes are most likely to poison children, the efficiency and benefits of any abatement program will be markedly increased.

Sensitivity Analysis

Changing certain values may have an impact on the conclusions that are drawn from an analysis; consequently, we perform sensitivity analysis to test the impact of changing our assumptions. In this section, we report the results of sensitivity analyses aimed at testing whether the values of key variables significantly alter the conclusions that can be drawn from the study. Table 1 displays the results of the base case analysis along with the sensitivity analyses. Benefits are expressed in terms of net benefits--that is, the difference between the total benefits and the cost of abatement.

Changing the number of children per home: In the base case analysis, we assumed that abatement would not be targeted to homes with children; therefore, the average number of children per pre-1950 home was used in estimating the benefits of abatement. In this variation, we assume that abatement is conducted in communities with more children than the average. For this analysis, we assume that the average unit to be abated houses three times more children than the national average. Thus, we assume that, on the average, 0.108 (or $0.287 \times 3 \times (9/72)$) children less than 10 months of age now occupy the

unit and, for future cohorts, we assume that an average of 0.135 (or 0.045×3) children will be born into each unit each year. Under these assumptions, the net benefits are \$10,747 per unit. Alternatively, if we assume that five times the average number of children occupy these units than is the average for the nation, the net benefits are \$19,395 per unit.

Changing the discount rate: In the base case analysis we assumed a discount rate of 5%. If we discount all future benefits and costs by 3% the net benefits are approximately \$6,357. When all future benefits and costs are discounted by 7%, net benefits become \$404.

Changing the lifespan of houses: Assuming a 50-year lifespan rather than the median of 68 years reduces net benefits to \$1,866, and decreasing the lifespan of houses to 30 years reduces net benefits to \$1,212 per abated unit.

Changing the effectiveness of the abatement (ug/dL reduction): In the base case analysis, we assumed that children with blood lead levels ≥ 25 ug/dL experience a 9 ug/dL blood lead decline after abatement and that children with lower levels will have a 3.75 ug/dL decline. In this variation, we will assume that abatement is more effective than in the three studies from which we obtained data. We will assume that children with blood lead levels ≥ 25 ug/dL experience a 21.6 ug/dL decline (60% of the average baseline blood lead level) and that children with lower blood lead levels experience a 9 ug/dL decline. We also assume a proportionately greater decrease in the blood lead levels of pregnant women (5.11 ug/dL). Under this scenario, net benefits increase to \$7,992. This analysis implies that a more effective abatement method that results in an approximate average decline in the blood lead level of 9.4 ug/dL could cost as much as \$10,000 per unit and we could still expect to see net benefits from abatement.

Changing assumptions about the impact of abatement on children 9 months of age or older currently in lead-painted homes: In the base case analysis we estimated benefits only for those children currently living in the home who were less than 10 months of age. No benefits were assumed in the analyses for children above that age. In this variation, we will assume that all children 6 years of age and under living in an abated home will experience full benefits. In this case, the net benefits are \$3,290. If we assume that children between 10 months and 6 years of age receive only half the benefits of children less than 10 months, the net benefits are \$2,693.

These analyses show that targeting abatement to homes with children and improving the efficacy of abatement will result in greater net benefits. Furthermore, if strategies can be developed for determining which homes are most likely to poison children, the efficiency and benefits of any abatement program will be markedly increased.

THE BENEFITS OF A NATIONAL EFFORT TO ABATE ALL PRE-1950 HOUSING UNITS WITH LEAD-BASED PAINT

In this analysis, we estimate the benefits from abating all homes in the United States.

1. Results of a study by Shier and Hall (1977) show that 80% of pre-1950 housing contains lead-based paint. Since there are 28,971,000 occupied pre-1950 housing units in the United States (U.S. Bureau of the Census, 1989), we estimate that there are 23,176,800 occupied pre-1950 housing units containing lead.
2. For this analysis, we use a cost estimate of \$2,225 per abatement. Therefore, the cost of abating all 23,176,800 units today would be \$51,568,380,000. Were the abatement conducted over the next 20 years (performing an equal number of abatements each year), the total present cost of abatement would be \$33,739,550,000.
3. We have estimated that the total benefits of abatement are \$4,323 per housing unit. If all abatements were performed now, the total benefits would be \$100,193,306,000. If abatements were conducted over the next 20 years, the total present value of the benefits would be \$61,742,270,000. (This number takes into account the fact that a house abated in the future has a shorter lifespan as a lead-free dwelling than a unit abated today; therefore, fewer cohorts of children would benefit.)
4. We have estimated that the net benefits of abatement (total benefits of abatement - costs of abatement) are \$2,098. If all pre-1950 lead-painted housing units in the United States were abated today, the net benefits of abatement would be \$48,624,926,000. If units were abated over the next 20 years, the present value of the net benefits would be \$28,002,830,000.

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Table 1. Results of the base case cost-benefit analysis and sensitivity analysis, expressed as net benefits for abatement of the average pre-1950 home with lead-based paint, discounted to the present. (Net benefits = total benefits - cost of abatement.)

<u>Description of Analysis</u>	<u>Net Benefits</u>
Base case analysis (see text for assumptions)	\$ 2,098
Sensitivity analyses	
Number of children per home	
If increased 3-fold	10,747
If increased 5-fold	19,395
Discount rate of 5%	
If decreased to 3%	6,357
If increased to 7%	404
Life span of houses is 68 years	
If decreased to 50 years	1,866
If decreased to 30 years	1,212
Effectiveness of the abatement	
With 60% decrease in blood lead levels	7,992
Benefits accrue to children 10-72 months	
With 100% benefits	3,290
With 50% benefits	2,693

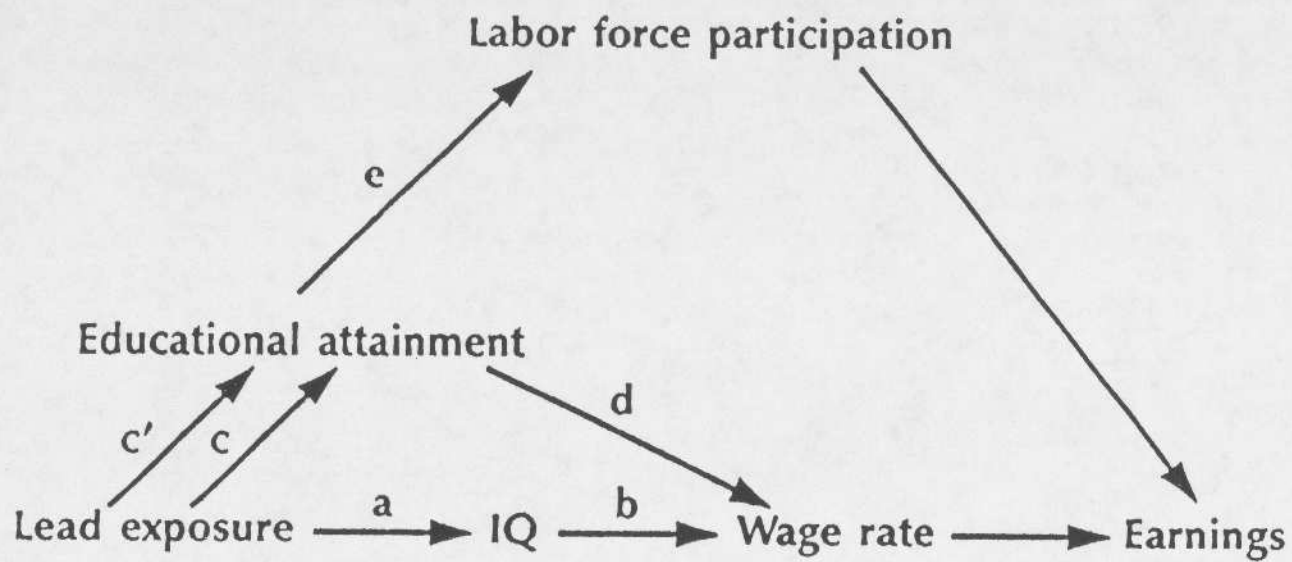


Figure 1. Effect of lead exposure on earnings.

APPENDIX III

HISTORY OF CHILDHOOD LEAD POISONING PREVENTION PROGRAMS

The first cases of childhood lead poisoning from lead paint in housing were reported in 1892 in Australia. Although many severe cases of the disease were reported in subsequent decades in the United States, little effort was made to find additional cases until the 1950s, when caseworkers in a few large cities attempted to find lead-poisoned children. In 1966, Chicago began the first mass screening program, followed shortly by New York and other cities (Lin-Fu, 1980).

The Lead-Based Paint Poisoning Prevention Act, passed in 1971, initiated a national effort to identify children with lead poisoning and abate the sources of lead in their environments. For most years of this program, Federal funds appropriated under this Act were administered by the Centers for Disease Control (CDC). More than \$89 million were distributed, and over a quarter of a million children were identified with lead poisoning and received referrals for environmental and medical intervention.

In 1981 the Omnibus Budget Reconciliation Act amended Title V of the Social Security Act, which had authorized the Maternal and Child Health (MCH) Services Program since 1935. The amendment created the MCH Services Block Grant Program and consolidated many categorical programs, including that for childhood lead poisoning prevention, into the Block Grant. In 1982, the administrative responsibility for the Lead-Based Paint Poisoning Prevention Act was transferred to the Office of Maternal and Child Health (now the Maternal and Child Health Bureau) of the Health Resources and Services Administration.

Under the provisions of the MCH Services Block Grant Act, each State decides how to use these Federal funds. Data on whether these funds are used to support childhood lead poisoning prevention activities has not been reported to the Federal government. The 1989 Omnibus Reconciliation Act includes a requirement for State MCH Block Grant Programs to be consistent with the Public Health Service Year 2000 Objectives for the Nation and to submit an annual report with specified content in a standardized format. Since reduction of the numbers of children with lead poisoning is likely to be included as a Year 2000 Objective, more information on childhood lead poisoning prevention activities funded by the MCH block grant is anticipated.

The Lead Contamination Control Act of 1988 authorized \$20 million for Fiscal Year 1989, \$22 million for Fiscal Year 1990, and \$24 million for Fiscal Year 1991 for CDC to administer a childhood lead poisoning prevention grant program. Under this law, \$4

million were appropriated in Fiscal Year 1990, and \$8 million were appropriated in 1991. The President's budget for 1992 includes \$14.95 million for this program. The majority of this money will be provided as grants for State and local agencies to perform childhood lead screening, referral for medical and environmental follow-up, and education about lead poisoning in those communities with children with the highest blood lead levels. This money is directed at communities with large numbers of children with higher blood lead levels (e.g., ≥ 25 ug/dL). Although clearly many more States and communities need comprehensive programs to address childhood lead poisoning, CDC's current grant program is an important step in our effort to eliminate childhood lead poisoning.

The President's budget for FY 1992 also includes \$25 million for the HOME program, which will be administered by the Department of Housing and Urban Development (HUD). This program will assist low- and moderate-income private residential property owners to abate lead-based paint, and will be directed to homeowners with young children in high-risk housing. This program could provide a knowledge base for evaluating the effects of abatement.

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APPENDIX IV

ORGANIZATIONS AND AGENCIES THAT COULD HELP PROMOTE AWARENESS OF CHILDHOOD LEAD POISONING

Table 1. Professional Organizations That Could Increase Practitioner Awareness of Childhood Lead Poisoning

Primary Care Physicians (family practice, internal medicine,
pediatrics, and emergency medicine)

Ambulatory Pediatric Association
American Academy of Pediatrics
American Association of Family Physicians
American Board of Pediatrics
American College of Emergency Physicians
American College of Physicians
American Medical Association
American Medical Student Association
American Osteopathic Association
American Pediatric Society
American Society of Internal Medicine
Association of American Physicians
Association of American Indian Physicians
Association of General Practitioners/Family Physicians
Association of Medical School Pediatric Department Chairmen
Association of Program Directors in Internal Medicine
Federal Physicians Association
National Association of Residents and Interns
National Medical Association
North American Primary Care Research Group
Society of Teachers of Family Medicine
Society of General Internal Medicine

Public Health Physicians

American Association of Public Health Physicians
American College of Preventive Medicine
American Osteopathic College of Preventive Medicine
Association of Teachers of Preventive Medicine
Association of Preventive Medicine Residents

Table 1 (continued). Professional Organizations that Could Increase Practitioner Awareness of Childhood Lead Poisoning

Other Physician Speciality Organizations

American Association of Obstetrics and Gynecology
American College of Occupational Medicine

Nurses

American Society of Pediatric Hematology/Oncology
American Nursing Association
American Academy of Nursing
American Association of Neuroscience Nursing
American Association of Occupational Nursing
American College of Nurse-Midwives
American Licensed Practical Nurses Association
American Nurses' Association
American Organization of Nursing Executives
Assembly of Hospital Schools of Nursing
Association of State and Territorial Directors of Nursing
Frontier Nursing Service
National Association of Hispanic Nurses
National Association of Pediatric Nurse Associates and Practitioners
National Association of Physician Nurses
National Association of Registered Nurses (State Associations)
National Association of School Nurses
National Association of Black Nurses
National Board of Pediatric Nurse Practitioners and Associates
Nurses Association of the American College of Obstetricians and Gynecologists

Physician Assistants

American Academy of Physician Assistants
Association of Physician Assistant Programs

Pharmacists

American Pharmaceutical Association
American Society of Hospital Pharmacists
National Association of Retail Druggists
National Pharmaceutical Association
National Pharmaceutical Foundation
State Boards of Pharmacy
State Pharmaceutical Associations

Table 1 (continued). Professional Organizations that Could Increase Practitioner Awareness of Childhood Lead Poisoning

Public Health Professionals

American College of Epidemiology
American Industrial Hygiene Association
American Public Health Association
Association of Schools of Public Health
Association of State and Territorial Directors of Public Health Education
Association of State and Territorial Health Officers
Association of State and Territorial Public Health Laboratory Directors
Association of University Programs in Occupational Health and Safety
Conference of Public Health Laboratorians
Conference of State Health and Environmental Managers
Council of State and Territorial Epidemiologists
Council on Education for Public Health
National Association of County Health Officials
National Coalition of Hispanic Health and Human Services Organizations
National Conference of Local Environmental Health Administrators
National Environmental Health Association
National Foundation of Rural Medical Care
National Rural Health Association
Society for Occupational and Environmental Health
United States Conference of Local Health Officials
World Federation of Public Health Associations

Other Health Organizations

American Indian Science and Engineering Society
American Industrial Health Council
Asian American Health Forum
Association of American Medical Colleges
Association of Minority Health Professions Schools
National Association of Community Health Centers
Society for Pediatric Research

Table 2. Other Organizations that Would be Interested in Educating the Public About Childhood Lead Poisoning

Maternal and Child Health

American Association of University Affiliated Programs for Persons with
Developmental Disabilities
Association of Maternal and Child Health Programs
Be Healthy, Inc.
Healthy Mothers, Healthy Babies Coalition
March of Dimes
National Association for the Education of Young Children
National Black Women's Health Project
National Center for Education in Maternal and Child Health
National Maternal and Child Health Clearinghouse
Safe Kids Coalition

Health Education, Information, and Promotion Organizations

American Hospital Association, Health Promotion Center
American Dietetic Association
American Lung Association
American Red Cross
Association for the Advancement of Health Education
Consumer Health Information Resource Institute
Consumer Information Center
Environmental Defense Fund
Health Education Center
Health Education Foundation
Health Insurance Association of America
Health Media Education
HealthWorks Northwest
The Henry J. Kaiser Family Foundation
National Health Information Center
The National Health Network
National Information System for Health Related Services
National Public Health Information Coalition
Patient Education Resource Center
Society for Public Health Education
Women's Occupational Health Resource Center

Table 2 (continued). Other Organizations that Would be Interested in Educating the Public About Childhood Lead Poisoning

Civic Organizations

Federation of Women's Clubs
Kiwanis
Knights of Columbus
League of Women Voters
Shriners
Young Mens' Christian Association
Young Womens' Christian Association

Housing and Finance Organizations

Association of Local Housing Finance Agencies
Building Owners and Managers Association
Council of State Governments
Federal National Mortgage Association
Housing Assistance Council
Mortgage Bankers of America
National Apartment Association
National Association of Counties
National Association of Governments
National Association of Home Builders
National Association of Housing and Redevelopment Officials
National Association of Realtors
National Community Development Association
National Council of State Housing Agencies
National Council of State Legislatures
National Housing Conference
National Leased Housing Association
National Low Income Housing Coalitions

Advocacy Groups

Alliance to End Childhood Lead Poisoning
American Association on Mental Retardation
Association for Retarded Citizens of the United States
American Federation of Teachers
Child Welfare League of America
Children's Defense Fund
Citizen's Clearinghouse for Hazardous Waste
Coalition on Human Needs

**Table 2 (continued). Other Organizations that Would be Interested in
Educating the Public About Childhood Lead Poisoning**

Advocacy Groups (continued)

Foundation for Child Development
The Lead Coalition
Legal Services Corporation
National Association for Rights, Protection, and Advocacy
National Education Association
National Parent-Teacher Association

Artist Safety Organizations

Arts, Crafts and Theatre Safety (ACTS)
Center for Safety in the Arts

APPENDIX V

INFRASTRUCTURE DEVELOPMENT FOR ABATEMENT OF LEAD HAZARDS IN HOUSING

In the past two decades progress has been limited in reducing childhood lead poisoning caused by lead-based paint and dust in homes. Only a small fraction of the housing units with lead-based paint have had the lead abated. To make matters worse, improper techniques were used in many past abatement projects. The high levels of lead in dust generated during abatement resulted in poisoning of workers and their families, and children left in their homes during abatement had exacerbations of lead poisoning. Inadequate abatement and cleanup procedures also resulted in children being re-poisoned upon returning to their "deleaded" homes.

Great strides have been made in the past few years in improving abatement technology and protecting workers and their families. Although further improvements in abatement technology and practice are needed, we now have the tools to start a national abatement program. This section details the steps that must be taken to increase the national capacity to do safe and effective abatement work.

GUIDELINES DEVELOPMENT

The first set of comprehensive technical guidelines for lead-based paint testing and abatement, developed by a committee of government and nongovernment experts, were issued on an interim basis in April 1990, by the Department of Housing and Urban Development (HUD) for public and Indian housing authorities (the HUD Interim Guidelines). The HUD Interim Guidelines emphasize lead abatement of large blocks of units at the same time that other renovation work is done (comprehensive modernization). These guidelines were developed for housing that is to be extensively modified during modernization by the Federal government. These guidelines must be modified for use by States, localities, and individuals in situations where funds are scarce, time is critical, or the unit is not being gutted for other reasons.

WORKER TRAINING AND CERTIFICATION

Worker Safety

Lead-based paint abatement is a potentially hazardous occupation. Exposures among abatement and other workers, especially among pregnant women and among women and men who have or are planning to have children, should be reduced. Currently, lead abatement workers are not covered by the Occupational Safety and Health

Administration (OSHA) general-industry standard regulating worker exposure to lead. Instead, they are covered under the safety and health standards for the construction industry, which regulate lead exposure less strictly. A standard is needed that takes into account new data showing adverse effects of lead on adults at levels well below the current OSHA general industry standard. Abatement workers should be protected by medical monitoring and medical removal provisions, as are potentially lead-exposed workers in general industry. Since many companies performing abatement are likely to have only a few employees, all companies, regardless of size, must be required to conform with Federal standards.

Curriculum Development

Federally developed or sanctioned model training programs are a method for assuring the quality and consistency of worker training. Basic course curricula must be developed to meet the training needs of different groups: HUD staff, public housing authorities, individual homeowners and landlords, contractors, workers, architects, designers, testers, and inspectors. Since such courses are a prerequisite for all other training activities, developing these course curricula should be given highest priority. Some curriculum development has already begun for implementation of the HUD Interim Guidelines.

Course Delivery Mechanisms

As the amount of lead paint abatement increases, market forces will meet the growing demand for training programs. In the short term, however, government involvement may be necessary. One option, establishing government-funded pilot training centers, was used successfully to deliver training to asbestos workers quickly. This approach offers a high degree of quality control and assures that training is available in all geographic areas. Pilot training centers, however, are expensive and could discourage centers without government funding from entering the market. Alternatively, the government could establish core curricula or curriculum requirements for each course. Federal or State governments or some other group could then evaluate private instruction programs and certify their adequacy. This approach would encourage the immediate involvement of universities, labor organizations, and others and would probably provide the greatest training capacity in the long run. Although low in cost, this alternative does require government personnel or contract staff to review and approve each training program. In any event, mechanisms to control the quality of instruction and assure the competence of trainees are essential.

Certification

Institutionalizing lead paint abatement training will be difficult without mandatory requirements for certifying contractors and their workers, testers, and inspectors. At a minimum, individual training programs must be approved by a Federal or State agency or some other body, such as a trade organization. The Environmental Protection Agency

(EPA) used this approach--certifying training materials and approving course providers--in the initial phase of its asbestos program. Another alternative is for the certifying body to require that workers simply pass a standardized test. EPA is using this general approach to license radon-testing personnel. A third approach is performance-based accreditation, as in Massachusetts.

Institutionalizing Abatement Training

Lead-based paint abatement will probably not evolve exclusively as a separate industry and skill speciality. Lead-based paint abatement is an integral and inevitable part of a variety of existing building trades: painting, plastering, masonry, flooring, cabinetry, carpentry, electrical, plumbing, insulation, and door and window replacement. Therefore, lead-based paint abatement should be integrated into the various building trades, and all workers involved in home renovation and repair should be familiar with the special safeguards and techniques required.

A potential benefit of a national abatement program is increased employment. Most of the neighborhoods that will be targeted for lead abatement have high unemployment rates. As persons with little training develop the skills needed for leaded-paint abatement, they are likely to leave jobs that do not require training. Because this abatement work will require a large work force, training and employing local persons will have local economic and social benefits.

LABORATORY ACCREDITATION

Although laboratory testing protocols and quality assurance mechanisms currently exist for analysis of lead in air, water, and blood, no similar program, either mandatory or voluntary, exists for the analysis of lead in paint film or dust. Currently, EPA is distributing detailed instructions on standard test procedures for laboratories. However, within the next 18 to 24 months, some laboratory accreditation program is clearly needed to assure that consistent and reliable laboratory results are obtained. Options include a direct Federal laboratory certification program, a new independent voluntary accreditation program, or an expansion of existing accreditation programs for analyzing lead in other media to include tests of paint and dust.

EVALUATING EMERGING ABATEMENT TECHNOLOGY

During the past few years, private firms have developed a variety of new products to reduce the costs of lead-based paint abatement. Currently, more than a dozen new encapsulants and chemical strippers are being marketed across the country. Unfortunately, few independent standards have been developed or tests conducted to evaluate the effectiveness of these products or substantiate the claims made by manufacturers. Standards must be set and performance criteria established to assure the effectiveness of emerging products, either by the Federal government or by

nongovernment consensus. Such standards would allow private laboratories to test new lead abatement products at the manufacturer's or vendor's expense.

DISPOSAL OF ABATEMENT DEBRIS

At present, a significant impediment to broad scale abatement of lead-based paint in housing is uncertainty about whether the debris generated can go in regular municipal landfills as solid waste or must be disposed of as hazardous waste, at substantially greater expense. When lead is removed from buildings, it is, in effect, being concentrated; therefore, rules and regulations for its safe disposal are critical to prevent widespread dispersal throughout the environment. Although at present disposal is not an issue for the individual homeowner because of a household exemption, it is a problem for society. Certain wastes, such as stripping agents and cleanup materials with high dust concentrations, may be subject to hazardous waste classification and disposal requirements. Most abatement debris, especially bulky items, such as old window and door frames painted with lead-based paint, are likely to be considered not hazardous. Nevertheless, contractors are having difficulty finding laboratories to do toxicity testing, and insurance companies are wary of these requirements which place the responsibility and burden of proof on the unit owner and contractor. The situation is further confused by the conversion to a new toxicity test method planned for the summer or autumn of 1990. As soon as requirements for the new toxicity tests are finalized, clear and practical guidance must be given to contractors and owners of multifamily units as to how they should segregate waste debris so that as much of the debris as possible will not be classified as hazardous.

RELOCATION DURING ABATEMENT

Under most circumstances, residents and their pets should not occupy their housing during abatement. One of the most serious problems faced by local abatement programs is the lack of suitable temporary housing for families while their homes are being abated. Although relatives and friends have traditionally provided such housing, consideration should be given to special provisions in government-subsidized or other housing programs to deal with this special problem. Since most abatement projects take only a week or two, each unit provided for relocation purposes could be used for 25 to 50 families per year.

INSURANCE FOR CONTRACTORS

Another constraint to rapidly expanding lead-based paint abatement programs is the lack of insurance for contractors and building owners performing abatement work. As in the case of asbestos, improper abatement techniques used in the early years raised concerns among insurance companies about providing liability coverage. With the availability of guidelines on safe practices, the marketplace can be expected to respond with coverage at reasonable prices. Comprehensive coverage is already being provided by the

self-insurance risk retention pool established by many large public housing authorities. It is hoped that private insurers will soon recognize this market and provide coverage at competitive rates. Federal, State, and local agencies should take steps to encourage or require such coverage.

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2 DECLARATION OF PAUL MUSHAK, Ph.D.

3 I, Paul Mushak, Ph.D., declare as follows:

4 1. The matters stated herein are true of my own personal
5 knowledge. If called as a witness, I would competently and
6 truthfully testify consistent with the following.

7 2. I am a principal at PB Associates, a scientific
8 consulting firm in health and exposure assessment of toxic
9 metals and metalloids -- particularly lead. I am a senior
10 scientific and health hazards consultant to the U.S.
11 Environmental Protection Agency, the U.S. Department of
12 Justice, the U.S. Public Health Service, the World Health
13 Organization, and the Ontario, Canada, Environment Ministry.
14 I serve as an adjunct professor of environmental/chemical
15 pathology at the University of North Carolina School of
16 Medicine in Chapel Hill, North Carolina. Formerly, I was a
17 tenured medical school faculty member at the University of
18 North Carolina and was director of the Heavy Metals
19 Laboratory, an internationally recognized research and
20 consulting facility involved in various functions, including
21 blood lead research.

22 PROFESSIONAL BACKGROUND

23 3. As a chemical toxicologist and environmental health
24 scientist, I have more than 20 years' professional experience
25 researching and writing on the toxicology, biology, and
26 environmental/biological chemistry of pollutant metals and
27 other agents, especially lead. My curriculum vitae is attached
28 as Exhibit A hereto.

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4. I have published extensively in the national and international scientific and public health literature with over 130 papers, abstracts, and book chapters, plus more than 15 authored/co-authored federal and international public health risk assessment documents on metal pollutants that have served as scientific blueprints for regulatory and legislative actions. I have authored/co-authored numerous articles and papers dealing specifically with lead, including: Mushak, "U.S. Agency for Toxic Substances and Disease Registry's Report to Congress on Childhood Lead Poisoning in America Review and Update," Proceedings of the First National Conference on Laboratory Issues in Childhood Lead Poisoning Prevention 79-104 (1991); Mushak, Davis, Crocetti and Grant, "Prenatal and Postnatal Effects of Low-Level Exposure: Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning," 50 Environmental Research 11-36 (1989); Mushak and Crocetti, "Determination of Numbers of Lead-Exposed American Children as a Function of Lead Source: Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning," 50 Environmental Research 210-29 (1989); Mushak and Crocetti, "Methods for Reducing Lead Exposure in Young Children and Other Risk Groups: An Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning," 89 Environmental Health Perspectives 125-35 (1990); Crocetti, Mushak, and Schwartz, "Determination of Numbers of Lead-Exposed U.S. Children by Areas of the United States: An Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning," 89

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2 Environmental Health Perspectives 109-20 (1990); Crocetti,
3 Mushak, and Schwartz, "Determination of Numbers of Lead-Exposed
4 Women of Childbearing Age and Pregnant Women: An Integrated
5 Summary of a Report to the U.S. Congress on Childhood Lead
6 Poisoning," 89 Environmental Health Perspectives 121-24 (1990).

7 5. I have been extensively involved as a principal author
8 and evaluator of a number of public agency health risk
9 assessment documents. I was a principal co-author of the EPA's
10 4-volume 1986 criteria document for lead, generally considered
11 to be one of two federal "bibles" for lead. I was an external
12 expert reviewer for the U.S. Public Health Services/U.S. Centers
13 for Disease Control Advisory Committee on Preventing Childhood
14 Lead Poisoning's October 1991 Statement on Preventing Lead
15 Poisoning in Young Children ("Statement"). This Statement sets
16 forth the standards of the medical profession and the U.S.
17 Public Health Service on the medical testing for and management
18 of lead poisoning and is recognized as the definitive federal
19 statement on prevention and treatment of lead poisoning in
20 children.

21 6. I was a senior advisor to the committee preparing the
22 CDC's 1985 Statement on Preventing Lead Poisoning in Young
23 Children and actively assisted in its preparation.

24 7. In 1988, I served as the senior author and scientific
25 manager of the Agency for Toxic Substances and Disease
26 Registry's report, The Nature and Extent of Lead Poisoning in
27 Children in the United States: A Report to Congress (July
28 1988) ("Report to Congress"), which is the second of the two

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2 federal "bibles" for lead. The Report to Congress was prepared
3 in compliance with Section 118(f) of the 1986 Superfund
4 Amendments and Reauthorization Act, 42 U.S.C. § 9618(f). Upon
5 completion, the Report to Congress was presented to the House
6 of Representatives' Committee on Energy and Commerce. The
7 Report represents the first systematic effort to quantify the
8 extent of the U.S. child lead-poisoning problem and to place
9 such numbers in some context of distribution of the children,
10 the lead sources, the adverse health responses, and strategies
11 for lead reduction. As discussed below, two of the "take home"
12 messages of the Report to Congress are: (1) exposure to even
13 low levels of lead can have serious, persistent effects on
14 children and (2) lead toxicity must be measured using a blood
15 lead test. Excerpts from the Report to Congress are attached
16 as Exhibit B hereto.

17 8. I am a member of the American Chemical Society,
18 American Association for the Advancement of Science, American
19 Public Health Association, Association of Clinical Scientists,
20 International Society for the Study of Xenobiotics, New York
21 Academy of Sciences, Society for Environmental Geochemistry and
22 Health, Society for Occupational and Environmental Health,
23 Society for Risk Analysis (national, N.C.), and the Society of
24 Toxicology (national affiliation & N.C. chapter).

25 THE EFFECTS OF LEAD POISONING

26 9. Lead affects virtually every body system; however, in
27 its early stages, lead poisoning is often asymptomatic. Toxic
28 effects may range from subtle to profound. See Report to

1
2 Congress at Executive Summary ("Ex. Summ.") 9-11.

3 10. The primary target organ for lead toxicity is the
4 brain or central nervous system, especially during early child
5 development. Very severe lead poisoning (associated with lead
6 levels at or above 80 $\mu\text{g}/\text{dL}$) can result in coma, convulsions,
7 profound mental retardation, and even death. At lower
8 concentrations, lead mainly interferes with normal development
9 of IQ and other neurobehavioral measures in children; with the
10 body's heme-forming system, which is critical to the production
11 of heme and blood; and with the vitamin D regulatory system,
12 which involves the kidneys and plays an important role in
13 calcium metabolism.

14 11. Effects of lead on children's IQ and other neural
15 measures appear to persist. The evidence is quite compelling
16 that these persistent effects exist at even low levels of lead
17 exposure (10 $\mu\text{g}/\text{dL}$ or lower). I am familiar with at least
18 three longitudinal studies of young children in Boston,
19 Cincinnati, and Australia -- all are showing that prenatal and
20 early childhood exposure to lead has adverse effects at low
21 exposure levels on a child's IQ and other neurobehavioral
22 measures and that these effects persist into the school-aged
23 years. Thus, one of the "take home" messages of our Report to
24 Congress and later papers is that even low levels of lead
25 absorption can have persistent, adverse effects on the highly
26 vulnerable central nervous systems of young children.

27 SOURCES OF LEAD POISONING

28 12. Lead is pervasive in the environment. Children are

1
2 exposed to lead from six environmental sources, three of which
3 still persist as major sources or pathways: paint, dust/soil,
4 and water. Lead in gasoline, foods, and stationary sources is
5 declining in importance. Paint is the major source of lead
6 poisoning. Although residential use of lead-based paint is now
7 banned, lead paint in existing housing remains the most
8 widespread and dangerous high-dose source of lead exposure for
9 young children. Lead paint continues to be used on the
10 exteriors of painted steel structures, such as bridges and
11 expressways. Children living near such sites as expressways
12 have been shown to have elevated blood lead levels.

13 13. Lead deposited in soil or as dusts does not
14 biodegrade or decay since lead is a chemical element. Children
15 playing around contaminated dusts and soils will remain exposed
16 indefinitely.

17 14. The water distribution system can also pose a lead
18 risk. Contamination of drinking water occurs in or near
19 residences and schools when lead leaches into the water from
20 lead connectors, service pipes, lead-soldered joints, or lead-
21 lined water coolers.

22 15. Lead entering the body from different sources and
23 through different pathways presents a combined toxicological
24 threat. Thus, multiple, low-level inputs of lead can result in
25 significant aggregate exposure. See Report to Congress, 6-8,
26 Chapters VI and VIII.

27 THE EXTENT OF LEAD POISONING IN YOUNG CHILDREN

28 16. Lead poisoning is the major environmental threat to

1
2 the health of children. In the Report to Congress, we
3 concluded that, in 1984, 2.4 million white and African-American
4 preschool U.S. children -- 17% of all such children -- in
5 metropolitan areas had projected blood lead levels exceeding 15
6 $\mu\text{g/dL}$. See Report to Congress, at Ex. Summ. 3-4. While some
7 progress has been made to reduce some sources of lead toxicity,
8 scientific determinations of what constitutes "safe" levels of
9 lead exposure are concurrently declining even further. The
10 history of research in this field has, in fact, shown a
11 progressive decline in the lowest exposure levels at which
12 adverse health effects can be reliably detected. Since the
13 Report to Congress was published, the 1991 CDC statement on
14 Preventing Lead Poisoning in Young Children has identified a
15 blood lead level of 10 $\mu\text{g/dL}$ as the new value of concern.
16 Notably, any subsequent declines in the prevalence of elevated
17 blood lead for our 1984 base year in the Report to Congress
18 have been offset by more children having blood lead at or above
19 the new CDC action level.

20 17. For my recent presentation to the First National
21 Conference on Laboratory Issues in Childhood Lead Poisoning
22 Prevention (Exhibit C hereto), I used additional 1984
23 prevalence projection data from the Second National Health and
24 Nutrition Examination Survey (NHANES II) to calculate the
25 prevalence of elevated blood lead concentrations and associated
26 early adverse effects at the new CDC 10 $\mu\text{g/dL}$ level.
27 Prevalences for 5 $\mu\text{g/dL}$ were estimated as well. The NHANES II
28 data were used heavily in the Report to Congress. NHANES III

1
2 data will not be available until sometime in 1994.

3 18. These new calculations show that the elevated blood
4 lead levels and early poisoning risks in America's poor
5 communities are astounding. In smaller cities (population less
6 than one million), 79% of all children with family incomes
7 below \$6000 -- 91.6 percent of African-American children --
8 were projected to have lead levels exceeding the new CDC action
9 level of 10 $\mu\text{g/dL}$ in 1984. In larger cities, 88 percent of all
10 children with family incomes below \$6000 -- 96.5 percent of
11 African-Americans -- were projected to be affected. For
12 children living in smaller cities and whose families earn
13 between \$6000 and \$14,999, approximately 67% had blood lead
14 levels associated with early toxicity; 83.5 percent of African-
15 American children in this category were affected. In larger
16 cities, 78.5 percent of all children whose families earn
17 between \$6000 and \$14,999 -- 92 percent of African-American
18 children -- had lead levels at or exceeding the 10 $\mu\text{g/dL}$ level.
19 Prevalence of blood lead levels above 5 $\mu\text{g/dL}$, a level only
20 one-half that for early toxicity, in poor children was
21 virtually 100 percent. While corresponding prevalence for the
22 current year, 1992, will be lower overall (that is, over all
23 national strata), it is likely that inner-city, poor children
24 will show the smallest declines.

25 19. As noted in the CDC's 1991 Statement (page 39),
26 almost all U.S. children are at risk for lead poisoning, and
27 some children are at higher risk than others. Clearly, the
28 statistical findings quoted in this declaration show that poor

1
2 children in the U.S. are at the highest risk for lead poisoning
3 and are virtually assured of experiencing elevated lead
4 absorption. Lead poisoning in its early stages is often
5 asymptomatic, so these children must be screened for lead
6 poisoning using a test. Neither the exposure setting itself
7 nor an oral risk questionnaire is capable of assessing blood
8 lead levels.

9 THE APPROPRIATE TEST

10 20. To confirm, i.e. reliably measure, the amount of
11 toxic lead in body fluid, a blood lead test must itself be
12 used. See Report to Congress at II-8, 9. The blood lead (Pb-
13 B) test directly measures the level of lead in whole blood.
14 Blood for measuring lead content can be taken from a vein in
15 the arm (venous blood) or from puncture of a finger tip
16 (capillary blood). If the latter type of test is used,
17 contamination protocols must be followed and an elevated Pb-B
18 confirmed by venous puncture. A blood lead measurement should
19 not be confused with an "EP test."

20 21. During the early and mid 1970s, the erythrocyte
21 protoporphyrin (EP) test became a popular (low cost, high
22 volume) test for predicting whether a child was lead poisoned.
23 The EP test does not directly yield a specific blood lead
24 concentration. In children exposed to elevated concentrations
25 of lead, the lead interferes with the body's ability to make
26 the important substance, heme. Heme is used to carry oxygen
27 when in the form of hemoglobin. Lead retards formation of heme
28 from the protoporphyrin molecule and protoporphyrin builds up

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in the blood. The EP test measures this build up of protoporphyrin. The EP test does not give directly discreet blood lead concentrations; rather, protoporphyrin levels are a surrogate indicator for the likelihood of elevated blood lead levels. The test, in brief, is used to predict lead absorption.

22. The EP test is not unique for predicting lead exposure. It can also be used to determine iron deficiency anemia. Therefore, one must take account of the iron nutritional status to avoid confounding.

23. The EP test is a reasonably reliable predictor of lead absorption at blood lead levels around 40 $\mu\text{g/dL}$. As lead levels dip below this amount, the EP level begins to "uncouple" from the blood lead level. Uncoupling means that the EP level is losing its predictive value. That is, EP can occur within the normal limits while the blood lead level is actually elevated and toxic. By 25 $\mu\text{g/dL}$, the uncoupling is quite significant, and there are unacceptable numbers of false negatives. The uncoupling occurs quite steeply as the blood lead level moves farther downward, from 25 $\mu\text{g/dL}$ to 10 $\mu\text{g/dL}$. At 10 $\mu\text{g/dL}$, the new CDC action level, there is probably total uncoupling, and the EP test cannot be used as a predictive surrogate for lead poisoning.

24. Data contained in the Report to Congress illustrate this problem with false negatives. Of 118 children with Pb-B levels above 30 $\mu\text{g/dL}$ (the CDC criterion level at the time of the NHANES II study), 47% had EP levels at or below 30 $\mu\text{g/dL}$,

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and 58% had EP levels less than an EP cutoff value of 35 µg/dL. Report to Congress at II-9. Of course, the CDC has revised the criterion level to 10 µg/dL, an action which introduces even more false negatives into the EP test results.

25. With respect to lead poisoning, then, the EP test does not act as a preventive test which allows the health care provider to detect lead poisoning at its early, more manageable stages. Rather, the EP test can only accurately predict that unacceptably high lead exposure and greater risk of more severe poisoning have already occurred.

26. In July 1988, the Report to Congress formally recommended to the Energy and Commerce Committee of the United States House of Representatives that accurate screening tests replace the continued use of the EP test (see Report to Congress Recommendation 2(b), at XI-8). The Report repeatedly told Congress of the problems associated with continued use of the EP test to test for lead poisoning and noted that the rate of false negatives using the EP test is considerable. See Report to Congress at II-8-9, V-20, V-47, XI-8.

27. The health delivery system has responded often with concern over changes to lead screening and testing protocols. There had been complaints to the 1991 CDC Statement advisory group regarding increased work requirements that will result from lowering the definitions of lead poisoning, for example. Experience has shown us, however, that any predicted chaos does not occur. The system has been able to handle the change.

28. Cost is not an adequate justification for continued

1
2 use of the EP test in testing young, Medicaid-eligible children
3 for lead poisoning. Because of its unreliability for
4 accurately predicting elevated Pb-B around the new CDC action
5 level, the EP test -- which I estimate to cost somewhere under
6 \$10 (depending on economies of scale and type of testing) is
7 probably a waste of money for lead absorption prediction. EP
8 testing for iron deficiency, of course, remains appropriate.
9 I estimate the cost of the blood lead tests in public health
10 laboratories at somewhere between \$10-\$15, depending on volume
11 and other economies of scale. Private laboratories may be able
12 to provide the test even more cheaply if large volumes of
13 screening samples were contractually guaranteed.

14 29. As noted in the Report to Congress, screening
15 programs, especially blanket screening, are particularly cost-
16 effective. This is demonstrated by comparing data on the costs
17 of treating children who are poisoned because early lower
18 levels of lead intoxication were not detected by screening with
19 the costs of testing. In the example used in the Report, the
20 ratio of medical costs to child screening costs was about 50:1
21 in 1986 dollars. Given this highly favorable cost-
22 effectiveness, the issue of a somewhat more expensive blood
23 lead test over an EP test is relatively trivial in terms of
24 overall costs of lead poisoning to the health system and
25 society. See Report to Congress at IX-21-22.

26 30. Delaying blood lead testing of Medicaid-eligible
27 children is also not justified by concerns regarding laboratory
28 capacity. Recent legislation should already have moderated to

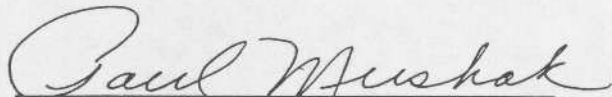
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2 some extent the effect of any stated concerns regarding quality
3 control. In 1988, the Clinical Laboratory Improvement Act was
4 amended to tighten quality assurance and quality control in all
5 laboratories and for all measurements, not just blood lead.
6 So, laboratories should already be operating with more of the
7 stringent quality control measures than before and, implicit in
8 this, be able reliably to measure Pb-B at or around the CDC 10
9 $\mu\text{g/dL}$ value. Public laboratories exist in every state and
10 territory, and there are huge private laboratories (e.g. Roche,
11 MetPath) that can be mobilized in a big way to share the test
12 volume. These private laboratories already adhere to tight
13 confidentiality and quality control standards in their use of
14 tests for employers and some public entities in, e.g., drug use
15 screening. Both public and private laboratories can react
16 quickly to an increased volume of blood lead tests -- the
17 laboratories and their administrative infrastructures already
18 exist, so the expansions needed are horizontal, i.e., lateral
19 in nature (to expand staff and equipment) rather than vertical
20 (to build new infrastructures and create new facilities).

21 31. In summary, then, the question of an appropriate test
22 for lead exposure and lead poisoning in Medicaid children is
23 not arguable on cost or other logistical grounds but on such
24 technical merits as accuracy and freedom from under-reporting
25 the prevalence of lead poisonings. Using the example from our
26 Report, the averted medical cost would be about \$20,000 to
27 \$25,000 per child in 1992 health care dollars. The unit child
28 costs for expanding or setting up more accurate blood lead

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screening would itself be a trivial fraction of the medical costs that are averted through use of the appropriate test.

I declare under the penalty of perjury that the foregoing is true and correct, this 20th day of November 1992 in Durham, North Carolina.


Paul Mushak, Ph.D.

MUSHAK
EXHIBIT A

179

CURRICULUM VITAE

NAME: Paul Mushak SSN:208-28-8852

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DATE/PLACE OF BIRTH: Dec. 9, 1935; Dunmore, Pa.

MARITAL STATUS: m. Elizabeth W. Mushak

MILITARY SERVICE: U.S. Army (Medical Corps), 8/54- 8/57;
Honorable discharge

EDUCATION:

B.S. Chemistry (Magna Cum Laude), University of Scranton,
Scranton, Pa., 1961

Ph.D. Metalloorganic/organic chemistry and biochemistry,
University of Florida, Gainesville, Fla., 1970.

Research assistant/associate, clinical toxicology/clinical chemistry,
Pathology Department, University of Florida School of Medicine,
1/67-11/69.

Dissertation Title: Addition and Rearrangement Reactions
Involving Various Organopalladium Compounds.

Postdoctoral Study: Fellow, Department of Molecular Biophysics
and Biochemistry, School of Medicine, Yale University, New
Haven, Conn., 11/69-6/71.

EMPLOYMENT HISTORY:

Present Position: Principal, PB Associates; Consulting toxics scientist: toxicology,
risk assessment, and relevant chemical aspects of pollutant metals
and metalloids

Adjunct Professor, Department of Pathology, School of Medicine,
University of North Carolina at Chapel Hill, Chapel Hill, N.C.

Past Positions:

11/23/86-6/5/87 Special appointment as expert adviser, U.S. Public Health Service,
Agency for Toxic Substances and Disease Registry, Atlanta, Ga.

7/1/77-12/31/85 Associate Professor, Department of Pathology, School of Medicine,

University of North Carolina, Chapel Hill, N.C.

7/77-12/31/85 Research Scientist, Biological Sciences Research Center, University of North Carolina.

7/1/71-7/1/77 Assistant Professor, Department of Pathology, University of North Carolina, Chapel Hill, N.C.

8/75-6/76 Consultant, Environmental Health Hazard Project, American Public Health Association, Washington, D.C.

1/67-11/69 Research Assistant/Associate, Department of Pathology, University of Florida School of Medicine, Gainesville, Fla.

9/62-11/69 Predoctoral Research Fellow, Chemistry Department, University of Florida.

6/61-7/62 NIH Trainee, Biochemistry Department, University of Florida School of Medicine.

PROFESSIONAL SOCIETIES:

American Chemical Society
American Association for the Advancement of Science
American Public Health Association
Association of Clinical Scientists
International Society for the Study of Xenobiotics
Society for Environmental Geochemistry and Health
Society for Occupational and Environmental Health
Society of the Sigma Xi
Society for Risk Analysis (National & N.C.)
Society of Toxicology (National affiliation & N.C. Chapter)

PROFESSIONAL SERVICE:

Consultancies:

1/1/77- Senior Scientific and Health Hazards Consultant, Environmental Protection Agency, Environmental Criteria and Assessment Office, Research Triangle Park, N.C., and Cincinnati, Ohio.

1/1/81- Health Effects Consultant, Office of Carcinogen Standards, Occupational Safety and Health Administration, U.S. Department of Labor, Washington, D.C.

6/1/75- National Institute of Environmental Health Sciences, P.O. Box 12233, Research Triangle Park, N.C.

4/1/85- External Review Committee, Hazardous Substances Branch, Ontario

Ministry of the Environment, Toronto, Ontario, Canada.

Committees of the National Academy of Sciences:

Committee on Measuring Lead Exposure in Infants, Children and Other Sensitive Populations, National Academy of Sciences/National Research Council

Joint Workshop Committee: National Academy of Sciences and Councils of Academies of Sciences and Arts of Yugoslavia: Exposure to Heavy Metals in the Environment

Zinc Panel, Medical and Biological Effects of Air Pollutants, National Academy of Sciences/ National Research Council

Committees of the World Health Organization:

Advisor and Rapporteur, Air Quality Guidelines Program, Regional Office for Europe, World Health Organization, 8 Scherfigsvej, DK 2100 Copenhagen, Denmark.

Member, Interim Working Group, International Programme on Chemical Safety, Environmental Health Criteria 101 Methyl Mercury. December 1988/ June 1989.

Other Public Committees:

North Carolina Child Lead Poisoning Advisory Committee

Lead Exposure Subcommittee, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency

Corresponding Member, Subcommittee on Nickel, International Union of Pure and Applied Chemistry, 6/79-1/88

Other Professional Activities:

Testimony before the U.S. Congress:

U.S. House Subcommittee on Health and the Environment, Rep. Henry Waxman (D-Calif.), Chairman, Dec. 10, 1987, Washington, D.C., Lead in Drinking Water of Schools and Other Public Facilities;

U.S. House Subcommittee on Health and the Environment, Rep. Henry Waxman (D-Calif.), July 13, 1988, Washington, D.C., testimony in support of H.R. 4939 to regulate lead in drinking water.

Expert Witness for the U.S. Department of Justice:

A principal expert witness, United States vs. Sharon Steel Corp. et al., Midvale, UT; action under CERCLA (Superfund).

A principal expert witness, United States vs. Apache Chemical Co. et al., California Gulch, Leadville, CO; action under CERCLA (Superfund).

Service in various capacities for the U.S. Environmental Protection Agency, including Clean Air Act pollutants, health assessment documents for hazardous pollutants, toxic substances, and Superfund scientific activities. These activities include numerous workshop reviews, 1977-present.

ADMINISTRATIVE ACTIVITIES:

1975-1985: Co-Principal Investigator, Neurobiology of Environmental Pollutants, NIEHS.

1971-1985: Director, Heavy Metals Laboratory, University of North Carolina School of Medicine.

1/86-Current: Owner and Principal, Scientific consulting firm in health and exposure assessment of toxic metals, metalloids, and organic pollutants for Federal and international agencies

5/88-6/90: Owner and technical director, TRUE-TEX, laboratory services to the historical and decorative fiber arts

HONORS:

Undergraduate research scholarships, 1958-1960. Joint Program, University of Scranton, Jefferson Medical College, and Health and Welfare Fund, United Mine Workers of America.

NSF Undergraduate Research Program, 1960-1961.

Research Award, University of Southern California- Continental Oil Co., Annual National Contest in Surface and Colloid Chemistry, 1961.

NIH Trainee, University of Florida, 1961-1962.

American Cancer Society Institutional Fellowship, University of Florida, 1962-1963.

NSF and U.S. Air Force AFOR Research Assistantships, University of Florida, 1963-1967.

Yale Corporation Fellow, Yale Medical School, 1969-1971

Award for Meritorious Service, U.S. EPA, Cincinnati, Ohio- January 1981.

RESEARCH INTERESTS:

Toxicological chemistry and toxicology of metals/organometals; toxic metal/metalloid monitoring; metal toxicokinetics and metabolism; toxicokinetics of environmental metals

in large populations.

PUBLICATIONS

- P. Mushak, J. Weiss and W. Haab: Some in-vitro relationships with reference to surface activity and cholesterol. Proc. Penna. Acad. Sci. 35: 169-173 (1961).
- J. Savory, P. Mushak, N.O. Roszel and F.W. Sunderman, Jr.: Determination of chromium in serum by gas-liquid chromatography. Fed. Proc., 777 (1968).
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- P. Mushak: Fluoro-B-diketones and metal fluoro-B-diketonates. Fluorine Chemistry Reviews. Vol. 6, P. Tarrant, Ed., Marcel Dekker, Inc., New York, 1973, pp. 43-133.

- P. Mushak, F.E. Tibbetts, III, P. Zarnegar and G.B. Fisher: Perhalobenzenesulfonates as reagents in the determination of inorganic mercury in various media by gas-liquid chromatography. J. Chromatog. 87: 215-226 (1973).
- P. Zarnegar and P. Mushak: Quantitative measurements of inorganic mercury and organomercurials in water and biological media by gas-liquid chromatography. Anal. Chim. Acta 69: 389-401 (1974).
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- M.R. Krigman, D.T. Traylor, E.L. Hogan and P. Mushak: Subcellular distribution of lead in the brains of intoxicated and control rats. J. Neuropath. Exper. Neurol. 33: 176 (1974).
- B.A. Fowler, G.W. Lucier and P. Mushak: Phenobarbital protection against methylmercury nephrotoxicity. Proc. Soc. Exp. Biol. Med. 149: 74-79 (1975).
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D.D. Dietz, D.E. McMillan and P. Mushak: Effects of chronic lead administration on acquisition and performance of serial positional sequences by pigeons. Toxicol. Appl. Pharmacol. 47: 377-384 (1979).

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R.A. Goyer, J. Bachmann, T.W. Clarkson, G.F. Ferris, Jr., J. Graham, P. Mushak, D.P. Perl, D.P. Rall, R. Schlesinger, W. Sharpe and J.M. Word: Potential Human Health Effects of Acid Rain: Report of a Workshop. Environ. Health Perspect. 60: 355-368 (1985).

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S.R. Schroeder, B. Hawk, D.A. Otto, P. Mushak and R.E. Hicks. Separating the effects of lead and social factors on IQ. Environ. Res. 38: 144-154 (1985).

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M.R. Krigman, T.W. Bouldin and P. Mushak. Metal toxicity in the nervous system. In: The Pathologist and the Environment, (D.G. Scarpelli, J.E. Craighead and N. Kaufman, Eds.), International Academy of Pathology Monograph, Williams and Wilkins, Baltimore, 1985, pp. 58-100.

B.A. Hawk, S.R. Schroeder, G. Robinson, D. Otto, P. Mushak, D. Kleinbaum and E. Dawson. Relation of lead and social factors to IQ of low SES children: a partial replication. Am. J. Ment. Defic. 91: 178-183 (1986).

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D.J. Thomas, H.L. Fisher, M.R. Sumler, P. Mushak, L.L. Hall. Sexual differences in the excretion of organic and inorganic mercury by methylmercury-treated rats. Environ. Res. 43: 203-216 (1987).

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- P. Mushak. Interactive relationships as modifiers of metal toxicity with special reference to those of lead and those of selenium. In: *Selected Aspects of Exposure to Heavy Metals in the Environment*, National Academy Press, Washington, D.C., 1987, pp. 36-41.
- P. Mushak. co-editor, *Air Quality Guidelines for Europe (The Non-Carcinogenic Metals Cadmium, Lead, Manganese, Mercury and Vanadium)*. WHO Regional Publications: European Series No. 23, World Health Organization, Regional Office for Europe, Copenhagen, 1987.
- D.J. Thomas, H.L. Fisher, M.R. Sumler, L.L. Hall, P. Mushak. Distribution and retention of organic and inorganic mercury in methyl mercury-treated neonatal rats. *Environ. Res.*, 47: 59-71, 1988.
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- P. Mushak, J.M. Davis, A.F. Crocetti, and L.D. Grant. Prenatal and postnatal effects of low-level lead exposure: Integrated summary of a report to the U.S. Congress on childhood lead poisoning. *Environ. Res.* 50: 11-26, 1989.
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ABSTRACTS

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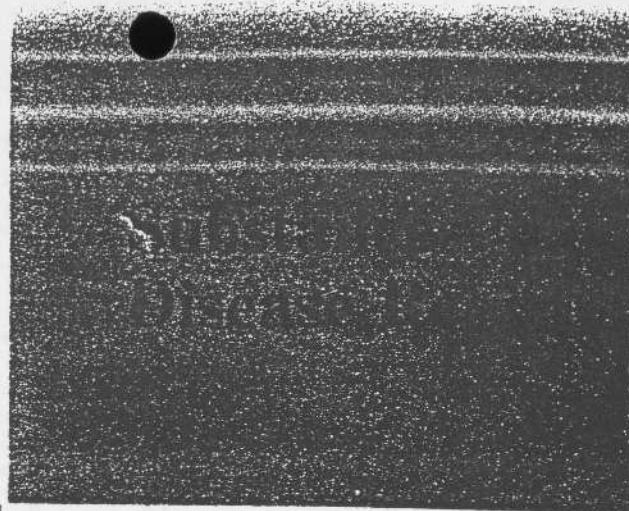
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EXHIBIT B

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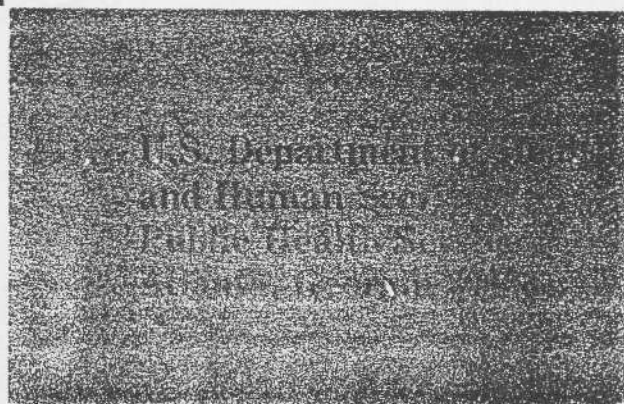


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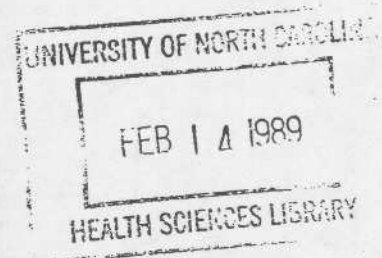
The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress

EXCERPTS



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THE NATURE AND EXTENT OF LEAD POISONING
IN CHILDREN IN THE UNITED STATES:
A REPORT TO CONGRESS



Agency for Toxic Substances and Disease Registry
Public Health Service
U.S. Department of Health and Human Services
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In 1981, Federal resources for screening were put under the program of the Maternal and Child Health Block Grants to States. Although the States' use of Federal funds for lead screening programs was estimated by one source to have been reduced initially by 25% (Farfel, 1985), a precise figure cannot be readily given since allocations of the block grant funds for particular projects are determined by the States according to their priorities, and data are not systematically collected on these State funding allocation decisions.

The evidence of the national impact of this initial reduction in Federal resources appears to be mixed. While it appears that the total number of screening program units in the nation has decreased from 60 to between 40 and 45 (Chapter V), there is also evidence in some States and localities that the number of children currently being screened has increased since 1981 (CDC, 1982; Public Health Foundation, 1986). However, a study of data from Newark, NJ for a nine-year period prior to implementation of the block grants showed that the rate of high-lead exposures in asymptomatic children increased about fourfold after funding for lead screening and public education programs was reduced (Schneider and Lavenhar, 1986). Based upon this report, it is likely that those areas that choose to decrease the efficiency of their lead screening services can expect to experience increases in the number of children with lead poisoning.

A key point in the Schneider and Lavenhar (1986) report and additional information given below is that screening programs, especially those supported at a level that allows blanket screening, are particularly cost-effective. This is demonstrated by comparing data on the costs of treating children who are poisoned because early lower levels of lead intoxication were not detected by screening with the costs of community screening programs. According to O'Hara (1982), the cost of repeat admissions to Baltimore hospitals for 19 lead-poisoned children in 1979 was \$141,750, or at least \$300,000 in 1986 dollars. In summary statistics submitted to ATSDR for the 1985-1986 program year, St. Louis listed budgetary support of \$303,453 from the city and \$100,000 from the State of Missouri. During that funding year, all agencies in the St. Louis program tested 12,308 children, of whom 1,356 or 11.02% tested positive for lead exposure using CDC classifications. Of these positives, 849 tested as Class II, 445 as Class III, and 62 as Class IV, the most severe level of toxicity. The new CDC guidelines of 1985 were implemented midway through the 1985 screening year (July 1, 1985), so these figures represent a low boundary for the number of positives.

In St. Louis, the entire screening program cost \$403,453 and identified 1,356 cases of toxicity; that is just under \$300 per poisoned child. In Baltimore, the multiple hospital admissions required for only some of the poisoned children cost about \$16,000 per child in 1986 dollars. This does not account for additional essential costs for adequate management of severe toxic cases. These additional huge costs stem from medical follow-up care and treatment, remedial education, etc. The monetized costs of the sequelae in significant toxicity cases are spelled out in U.S. EPA (1985 and 1986b). The effectiveness of screening children for lead poisoning is well demonstrated in terms of deferred or averted medical interventions, and in most settings, is quite cost-effective.

In March 1987, the Committee on Environmental Hazards, American Academy of Pediatrics, issued its "Statement on Childhood Lead Poisoning." It includes this statement:

"...to achieve early detection of lead poisoning, the Academy recommends that all children in the United States at risk of exposure to lead be screened for lead absorption at approximately 12 months of age.... Furthermore, the Academy recommends follow-up...testing of children judged to be at high risk of lead absorption."

These guidelines from America's pediatric medicine community probably cannot be effectively implemented or coordinated with the current levels or existing type of program support at local, State, and Federal levels.

b. Environmental Hazard Identification and Abatement for Severe Poisoning Cases

When cases of toxicity were found, mass screening programs for lead poisoning routinely made efforts to find the sources. A careful examination of the information on reducing lead exposure by completely or partially removing leaded paint clearly shows that, at best, the effect is debatable. At worst, the approach may not work. In a prospective study, Chisolm et al. (1985) observed that when children return to "lead abated" structures, their Pb-B levels invariably return to unacceptable levels. This is not a case of endogenous Pb-B increase from the release of bone lead, because children heavily exposed before treatment will respond better when placed in lead paint-free housing.

PART 1
EXECUTIVE SUMMARY

Exposure to lead continues to be a serious public health problem -- particularly for the young child and the fetus. The primary target organ for lead toxicity is the brain or central nervous system, especially during early child development. In children and adults, very severe exposure can cause coma, convulsions, and even death. Less severe exposure of children can produce delayed cognitive development, reduced IQ scores, and impaired hearing -- even at exposure levels once thought to cause no harmful effects. Depending on the amount of lead absorbed, exposure can also cause toxic effects on the kidney, impaired regulation of vitamin D, and diminished synthesis of heme in red blood cells. All of these effects are significant. Furthermore, toxicity can be persistent, and effects on the central nervous system (CNS) may be irreversible.

In recent years, a growing number of investigators have examined the effects of exposure to low levels of lead on young children. The history of research in this field shows a progressive decline in the lowest exposure levels at which adverse health effects can be reliably detected. Thus, despite some progress in reducing the average level of lead exposure in this country, it is increasingly apparent that the scope of the childhood lead poisoning problem has been, and continues to be, much greater than was previously realized.

The "Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress" was prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) in compliance with Section 118(f) of the 1986 Superfund Amendments and Reauthorization Act (SARA) (42 U.S.C. 9618(f)). This Executive Summary is a guide to the structure of the document and, in particular, to the organization of the responses to the specific directives of Section 118(f). It also provides an overview of issues and directions to the U.S. lead problem.

The report comprises three parts: Part 1, consisting of the Executive Summary; Part 2, consisting of Chapter I. "Report Findings, Conclusions, and Overview," which provides a more detailed overview of information and conclusions abstracted from the main body of the report; and Part 3, consisting of Chapters II through XI, which constitute the main body of the report.

Before addressing the specific directives of Section 118(f), it is important to point out that childhood lead poisoning is recognized as a major public health problem. In a 1987 statement, for example, the American Academy of Pediatrics notes that lead poisoning is still a significant toxicological hazard for young children in the United States. It is also a public health problem that is preventable.

In recognition of evolving scientific evidence of the harmful effects of lead exposure, Congress directed ATSDR to examine (1) the long-term health implications of low-level lead exposure in children; (2) the extent of low-level lead intoxication in terms of U.S. geographic areas and sources of lead exposure; and (3) methods and strategies for removing lead from the environment of U.S. children.

The childhood lead poisoning problem encompasses a wide range of exposure levels. The health effects vary at different levels of exposure. At low levels, the effects on children, as stated subsequently in this report, may not be as severe or obvious, but the number of children adversely affected is large. Moreover, as adverse health effects are detected at increasingly lower levels of exposure, the number of children at risk increases. At intermediate exposure levels, the effects are such that a sizable number of U.S. children require medical and other forms of attention, but usually they do not need to be hospitalized, nor do they need conventional medical treatment for lead poisoning. For these children, the only appropriate solution, at present, is to eliminate or reduce all significant sources of lead exposure in their environment. At high levels, the effects are such that children require immediate medical treatment and follow-up. Various clinics and hospitals, particularly in larger cities, continue to report such cases.

Lead exposure may be characterized in terms of either external or internal concentrations. External exposure levels are the concentrations of lead in environmental media such as air or water. For internal exposure, the most widely accepted and commonly used measure is the concentration of lead in blood, conventionally denoted as micrograms of lead per deciliter (100 ml) of whole blood -- abbreviated $\mu\text{g/dl}$. For example, when ATSDR estimated the number

of children considered to be at risk for adverse health effects, the Agency used blood lead (Pb-B) levels of 25, 20, and 15 µg/dl to group children by their degree of exposure.

These levels are not arbitrary. In 1985 the Centers for Disease Control (CDC) identified a Pb-B level of 25 µg/dl along with an elevated erythrocyte protoporphyrin level (EP) as evidence of early toxicity. For a number of practical considerations, CDC selected this level as a cutoff point for medical referral from screening programs, but it did not mean to imply that Pb-B levels below 25 µg/dl are without risk. More recently, the World Health Organization (WHO), in its 1986 draft report on air quality guidelines for the European Economic Community, identified a Pb-B level of 20 µg/dl as the then-current upper acceptable limit. In addition, the Clean Air Scientific Advisory Committee to the U.S. Environmental Protection Agency (EPA) has concluded that a Pb-B level of 10 to 15 µg/dl in children is associated with the onset of effects that "may be argued as becoming biomedically adverse". In this connection, the available evidence for a potential risk of developmental toxicity from lead exposure of the fetus in pregnant women also points towards a Pb-B level of 10 to 15 µg/dl, and perhaps even lower. These various levels represent an evolving understanding of low-level lead toxicity. They provide a reasonable means of quantifying aspects of the childhood lead poisoning problem as it is currently understood. With further research, however, these levels could decline even further.

A. RESPONSE TO DIRECTIVES OF SECTION 118(f) of SARA

Section 118(f) and its five directives give ATSDR the mandate to prepare this report. These directives are identified in the five subsections below.

1. Section 118(f)(1)(A)

This subsection requires an estimate of the total number of children, arrayed according to Standard Metropolitan Statistical Area (SMSA) or other appropriate geographic unit, who are exposed to environmental sources of lead at concentrations sufficient to cause adverse health effects. Chapter V, "Examination of Numbers of Lead-Exposed Children by Areas of the United States," and Chapter VII, "Examination of Numbers of Lead-Exposed Women of Childbearing Age and Pregnant Women," respond to this directive.

Valid estimates of the total number of lead-exposed children according to SMSAs or some other appropriate geographic unit smaller than the Nation as a whole cannot be made, given the available data. The only national data set for Pb-B levels in children comes from the National Health and Nutrition Examination Survey II (NHANES II) of CDC's National Center for Health Statistics. The NHANES II statistical sampling plan, however, does not permit valid estimates to be made for geographic subsets of the total data base.

In this report, the numbers of white and black children (ages 6 months to 5 years) living in all SMSAs are quantified according to selected blood lead levels and 30 socioeconomic and demographic strata. Within large SMSAs (those with over 1 million residents each) for 1984, an estimated 1.5 million children had Pb-B levels above 15 $\mu\text{g}/\text{dl}$. In smaller SMSAs (with fewer than 1 million residents), an estimated 887,000 children had Pb-B levels above 15 $\mu\text{g}/\text{dl}$.

In short, about 2.4 million white and black metropolitan children, or about 17% of such children in U.S. SMSAs, are exposed to environmental sources of lead at concentrations that place them at risk of adverse health effects. This number approaches 3 million black and white children if extended to the entire U.S. child population. If the remaining racial categories are included in these totals, between 3 and 4 million U.S. children may be affected. The numbers of children in SMSAs with blood lead levels above 20 and 25 $\mu\text{g}/\text{dl}$ are 715,000 (5.2%) and 200,000 (1.5%), respectively. These figures, however, are for all strata combined; many strata (e.g., black, inner-city, or low-income) have much higher percentages of children with elevated Pb-B levels.

Although these projected figures, based on the NHANES II survey, provide the best estimate that can now be made, they were derived from data collected in 1976-1980 (the years of NHANES II) and extrapolated to 1984. With respect to bounds to the above projections, variables in the methods used to generate these figures contribute to both overestimation and underestimation. The major source of overestimation is the unavoidable omission of declines in food lead that may have occurred in the interval 1978-1964 and that would have affected the results of the projection methodology. On the other hand, two significant factors contribute to underestimation. One is the restriction of the estimates to the SMSA fraction of the U.S. child population, some 75% to 80% of the total population. The other is the unavoidable omission of children of Hispanic, Asian, and other origins in the U.S. population. In a number of SMSAs in the West and South west, children in such segments outnumber black children. In balancing all sources of overestimates and underestimates, including variance

in the projection model itself, the projections given are probably close to the actual values.

A breakdown of the above estimates according to national socioeconomic and demographic strata shows that no economic or racial subgrouping of children is exempt from the risk of having Pb-B levels sufficiently high to cause adverse health effects. Indeed, sizable numbers of children from families with incomes above the poverty level have been reported with Pb-B levels above 15 $\mu\text{g/dl}$. Nevertheless, the prevalence of elevated Pb-B levels in inner-city, underprivileged children remains the highest among the various strata. Although the percentage of children with elevated Pb-B levels is not as high in, for example, the more affluent segment of the U.S. population living outside central cities, the total number of children with these demographic characteristics is much greater than the number of poor, inner-city children. Consequently, the absolute numbers of children with elevated Pb-B levels are roughly equivalent for some of these rather different strata of the U.S. child population.

In this report, ATSDR has also used data from lead screening programs and 1980 U.S. Census data on age of housing to estimate SMSA-specific numbers of children exposed to lead-based paint. In December 1986, ATSDR conducted a survey of lead screening programs. Of 785,285 children screened in 1985, 11,739 (1.5%) had symptoms of lead toxicity by one of two definitions. Because CDC criteria for lead toxicity changed in 1985, some programs were still using the 1978 CDC criteria (Pb-B $\geq 30 \mu\text{g/dl}$ and EP $\geq 50 \mu\text{g/dl}$) in 1985, whereas others used the new 1985 CDC criteria (Pb-B $\geq 25 \mu\text{g/dl}$ and EP $\geq 35 \mu\text{g/dl}$).

Differences in the estimates of children with lead toxicity become apparent when using the NHANES II data and the childhood lead screening program data. Estimates derived from screening program data very likely underestimate the actual magnitude of childhood lead exposure by a considerable margin. This is especially evident when the percentages of positive test results from screening programs are compared with the much higher NHANES II prevalences of elevated Pb-B levels in strata corresponding to screening program target groups, for example, poor, inner-city children in major metropolitan areas.

An analysis of 318 SMSAs, based on 1980 Census data on age of housing, showed that 35 SMSAs had 50% or more of the children living in housing built before 1950. A total of 4,374,600 children (from these 318 SMSAs alone) lived in pre-1950 housing. The percentage of these children with lead exposures sufficient to cause adverse health effects could not be estimated, but the

older housing in which they live is likely to contain paint with the highest levels of lead and is, therefore, likely to pose an elevated risk of dangerous lead exposure. A noteworthy finding concerns the distribution of children in older housing according to family income. Actual enumerations (not estimates) show that children above the poverty level constitute the largest proportion of children who reside in older housing. The implication, consistent with the conclusion based on projections from NHANES II data that was stated above, is that children above the poverty level are not exempt from lead exposure at levels sufficient to place them at risk for adverse health effects. Children above the poverty level are the most numerous group within the U.S. child population.

Although Section 118(f)(1)(A) does not explicitly request such information, an accurate description of the full childhood lead poisoning problem requires an estimate of the number of fetuses exposed to lead in utero, given the susceptibility of the fetus to low-level lead-induced disturbances in development that first become evident at birth or even some time later during early childhood. Accordingly, in a given year, an estimated 400,000 fetuses (within SMSAs alone) are exposed to maternal Pb-B levels of more than 10 µg/dl and are therefore at risk for adverse health effects. This number pertains to a single year; the cumulative number of children who have been exposed to undesirable levels of lead during their fetal development is much greater, particularly in view of the higher average levels of exposure that prevailed in past years.

2. Section 118(f)(1)(B)

This subsection requires an estimate of the total number of children exposed to environmental sources of lead arrayed according to source or source types. Chapters VI ("Examination of Numbers of Lead-Exposed Children in the United States by Lead Source") and VIII ("The Issue of Low-Level Lead Sources and Aggregate Lead Exposure of Children in the United States") respond to this directive.

The six major environmental sources of lead are paint, gasoline, stationary sources, dust/soil, food, and water. Dust/soil is more properly classified as a pathway rather than a source of lead, but since it is often referred to as a source, it is included. (Figure 11-1 in the main report shows how lead from these sources reaches children.) The complex and interrelated pathways from

these sources to children severely complicate efforts to determine source-specific exposures. Consequently, exact counts of children exposed to specific sources of lead do not exist.

The first step in approximating the number of children exposed to lead from each of the six major sources is to define what constitutes exposure. For each lead source, approximate exposure categories are defined and range from potential exposures through actual exposures known to cause lead toxicity. Because the type and availability of data for each lead source vary considerably, definitions of exposure categories also differ for each lead source. The total numbers of children estimated for each source and category are therefore not comparable and cannot be used to rank the severity of the lead problem by source of exposure in a precise, quantitative way. Furthermore, because of the nature of methods used to calculate the numbers of children in these exposure categories, it is not possible to provide estimate errors. Some numbers are best estimates, but others may represent upper bounds or lower bounds.

One should not overlook the limitations and caveats for these calculations, lest the estimates be misinterpreted and misapplied. In addition, source-based exposure estimates of children have different levels of precision. The estimated number of children potentially exposed to a given lead source at any level is necessarily greater than the number actually exposed at a level sufficient to produce a specified Pb-B value. Source-specific estimates of potentially and actually exposed children, based on the best available information and reasonable assumptions, are summarized as follows:

- o For leaded paint, the number of potentially exposed children under 7 years of age in all housing with some lead paint at potentially toxic levels is about 12 million. About 5.9 million children under 6 years of age live in the oldest housing, that is, housing with the highest lead content of paint. For the oldest housing that is also deteriorated, as many as 1.8 to 2.0 million children are at elevated risk for toxic lead exposure.

The number of young children likely to be exposed to enough paint lead to raise their Pb-B levels above 15 µg/dl is estimated to be about 1.2 million.

- o An estimated 5.6 million children under 7 years old are potentially exposed to lead from gasoline at some level.

Actual exposure of children to lead from gasoline, was projected, for 1987, to affect 1.6 million children up to 13 years of age at Pb-B levels above 15 µg/dl.

- o The estimated number of children potentially exposed to U.S. stationary sources (e.g., smelters) is 230,000 children.

The estimated number of children exposed to lead emissions from primary and secondary smelters sufficient to elevate Pb-B concentrations to toxic levels is about 13,000; estimates for other stationary sources are not available.

- o The number of children potentially exposed to lead in dust and soil can only be derived as a range of potential exposures to the primary contributors to lead in dust and soil, namely, paint lead and atmospheric lead fallout. This range is estimated at 5.9 million to 11.7 million children.

The actual number of children exposed to lead in dust and soil at concentrations adequate to elevate Pb-B levels cannot be estimated with the data now available.

- o Because of lead in old residential plumbing, 1.8 million children under 5 years old and 3.0 million children 5 to 13 years old, are potentially exposed to lead; for new residences (less than 2 years old), the corresponding estimates of children are 0.7 and 1.1 million, respectively.

Some actual exposure to lead occurs for an estimated 3.8 million children whose drinking water lead level has been estimated at greater than 20 µg/l.

EPA, in a recent study, estimated that 241,000 children under 6 years old have Pb-B levels above 15 µg/dl because of elevated concentrations of lead in drinking water. Of this number, 100 have Pb-B levels above 50 µg/dl, 11,000 have Pb-B levels between 30 and 50 µg/dl, and 230,000 have Pb-B levels between 15 and 30 µg/dl.

- o Most children under 6 years of age in the U.S. child population are potentially exposed to lead in food at some level.

Actual exposure to enough lead in food to raise Pb-B levels to an early toxicity risk level has been estimated to impact as many as 1 million U.S. children.

Despite limitations in the precision of the above estimates, relative judgments can be made about the impact of different exposure sources. Some key findings are:

- o As persisting sources for childhood lead exposure in the United States, lead in paint and lead in dust and soil will continue as major problems into the foreseeable future.

- o As a significant exposure source, leaded paint is of particular concern since it continues to be the source associated with the severest forms of lead poisoning.
- o Lead levels in dust and soil result from past and present inputs from paint and air lead fallout and can contribute to significant elevations in children's body lead burden (i.e., the accumulation of lead in body tissues).
- o In large measure, paint and dust/soil lead problems for children are problems of poor housing and poor neighborhoods.
- o Lead in drinking water is a significant source of lead exposure in terms of its pervasiveness and relative toxicity risk. Paint and dust and soil lead are probably more intense sources of exposure.
- o Greater attention must be paid to lead exposure sources away from the home, especially lead in paint, dust, soil, and drinking water in and around schools, kindergartens, and similar locations.
- o The phasing down of lead in gasoline has markedly reduced the number of children impacted by this source as well as the rate at which lead from the atmosphere is deposited in dust and soil.
- o Lead in food has been reduced to a significant degree in recent years and contributes less to body burdens in the United States than in the past.
- o Significant exposure of unknown numbers of children can also occur under special circumstances: renovation of old houses with lead-painted surfaces, secondary exposure to lead transported home from work places, lead-glazed pottery, certain folk medicines, and a variety of others unusual sources.

3. Section 118(f)(1)(C)

This subsection requires a statement of the long-term consequence for public health of unabated exposure to environmental sources of lead. Chapters III ("Lead Metabolism and Its Relationship to Lead Exposure and Adverse Effects of Lead") and IV ("Adverse Health Effects of Lead") address this issue.

Infants and young children are the subset of the U.S. population considered most at risk for excessive exposure to lead and its associated adverse health effects. In addition, because lead is readily transferred across the placenta, the developing fetus is at risk for lead exposure and toxicity. For this reason, women of childbearing age are also an identifiable, albeit surrogate,

subset of the population of concern, not because of direct risk to their health, but because of the vulnerability of the fetus to lead-induced harmful effects.

Direct, significant impacts of lead on target organs and systems are evident across a broad range of exposure levels. These toxic effects may range from subtle to profound. In this report, the primary focus has been on effects that are chronic and that are induced at levels of lead exposure not uncommon in the United States. Cases of severe lead poisoning are, however, still being reported, particularly in clinics in our major cities.

The primary target organ for lead toxicity is the brain or central nervous system (CNS), especially during early child development. Other key targets in children are the body heme-forming system, which is critical to the production of heme and blood, and the vitamin D regulatory system, which involves the kidneys and plays an important role in calcium metabolism. Some of the major health effects of lead and the lowest-observed-effect levels (in terms of Pb-B concentrations) at which they occur can be summarized as follows:

- o Very severe lead poisoning with CNS involvement commonly includes coma, convulsions, and profound, irreversible mental retardation and seizures, and even death. Poisoning of this severity occurs in some persons at Pb-B levels as low as 80 µg/dl. Less severe but still serious effects, such as peripheral neuropathy and frank anemia, may start at Pb-B levels between 40 and 80 µg/dl.
- o Numerous epidemiologic studies of children have related lower levels of lead exposure to a constellation of impairments in CNS function, including delayed cognitive development, reduced IQ scores, and impaired hearing. For example, peripheral nerve dysfunction (reduced nerve conduction velocities) have been found at Pb-B levels below 40 µg/dl in children. In addition, deficits in IQ scores have been established at Pb-B levels below 25 µg/dl. Preliminary data suggest that effects on one test of children's intelligence may be associated with childhood Pb-B levels below 10 µg/dl.
- o Adverse impacts on the heme biosynthesis pathway and on vitamin D and calcium metabolism, all of which have far-reaching physiological effects, have been documented at Pb-B levels of 15 to 20 µg/dl in children. At levels around 40 µg/dl, the effects on heme synthesis increase in number and severity (e.g., reduced hemoglobin formation).
- o Of particular concern are consistent findings from several recent longitudinal cover a period of years epidemiologic studies showing low-level lead effects on fetal and child development, including neurobehavioral and growth deficits.

These effects are associated with prenatal exposure levels of 10 to 15 $\mu\text{g}/\text{dl}$.

With regard to the long-term consequences of lead exposure during early development, the American Academy of Pediatrics (1987) has noted that utmost concern should be given to the irreversible neurological consequences of childhood lead poisoning. Recent findings from longitudinal follow-up studies of infants starting at birth (or even before birth) show persistent deficits in mental and physical development through at least the first two years of life as a function of low-level prenatal lead exposure. It is not yet known, however, whether deficits in later childhood development will continue to show a significant linkage to prenatal exposure or whether, at older ages, postnatal lead levels will overshadow the effects of earlier exposure. Human development is quite plastic, with well known catch-up spurts in growth and other aspects of development. On the other hand, even if early lead-induced deficits are no longer detected at later ages, this apparent recovery does not necessarily imply that earlier impairments are without consequence. In view of the complex interactions that figure into the cognitive, emotional, and social development of children, compensations in one facet of a child's development may exact a cost in another area. Very little information is available for evaluating such interdependencies and trade-offs, but at this point even "temporary" developmental perturbations cannot be viewed as inconsequential.

In addition, given the poor prospects for immediate improvements in the environments of many children (e.g., deteriorated housing occupied by underprivileged, inner-city children), lead exposure and toxicity often are, in practice, irreversible. Thus, the issue of persistence must encompass the reality of exposure circumstances as well as the potential for biological recovery.

4. Section 118(f)(1)(D)

This subsection asks for information on the methods and options available for reducing children's exposure to environmental sources of lead. Chapter IX ("Methods and Alternatives for Reducing Environmental Lead Exposure for Young Children and Related Risk Groups") addresses this issue. Abatement methods include primary as well as secondary measures. Primary abatement refers to reducing or eliminating lead's entrance into pathways by which people are

exposed; secondary abatement refers to ways of dealing with lead after it has already entered the environment or humans. Biological approaches such as improved nutrition may fall into either of these two categories, depending on whether they are intended primarily as prophylactic or treatment measures. Extra-environmental approaches to prevention (e.g., legal actions and strictures) are also discussed.

Here are some key points on the abatement of childhood lead exposure and poisoning :

- o Efforts in the United States to remove or reduce human lead exposure have produced notable successes as well as notable failures.
- o Effective primary lead abatement measures have included EPA's phase-down regulations for gasoline lead, EPA's national ambient air quality standard for lead, and cooperative actions between the Food and Drug Administration and the food industry to reduce lead in food.
- o A number of new initiatives are being implemented by EPA to reduce lead in the drinking water of children and other population segments. Of particular interest is water as it comes from the tap not only in homes but in public facilities such as kindergartens and elementary schools. The schools, in particular, present special exposure characteristics that have not yet been adequately assessed.
- o Existing leaded paint in U.S. housing and public buildings remains an untouched and enormously serious problem despite some regulatory action in the 1970s to limit further input of new leaded paint to the environment. For this source, corrective actions have been a clear failure.
- o Lead in dust and soil also remains a potentially serious exposure source, and remediation attempts have been unsuccessful.
- o Secondary prevention measures in the form of U.S. lead screening programs for children at high risk still appear to require improved standardization of screening methodology (criteria for populations, measurement techniques, data collection, data reporting and statistical analysis) and central coordination.
- o The effectiveness of screening children for lead poisoning is well demonstrated in terms of deferred or averted medical interventions, and in most settings is quite cost-effective.
- o Extra-environmental measures, such as comprehensive good nutrition programs, have a role in mitigation of lead toxicity, but they cannot be used as substitutes for initiatives to reduce lead in the environment.

- o At present, legal sanctions do not appear to be very effective; to be effective, sanctions have to be both meaningful and rigidly enforced. So long as it is cheaper to pay a fine than to remove lead from the child's environment, little progress is likely to be made on this front.
- o The "easiest" steps to lead abatement have already been taken or are being taken. These steps, not surprisingly, have involved reducing lead in large-scale sources, such as gasoline and food, with more-or-less centralized distribution mechanisms.
- o Enormous masses of lead remain in housing and public buildings, along with large amounts of lead in dust and soil. If these highly dispersed sources are to be abated, huge efforts will be required.

5. Section 118(f)(2)

Chapter X ("A Review of Environmental Releases of Lead as Evaluated under Superfund") was prepared by the U.S. Environmental Protection Agency (EPA) in response to Section 118(f)(2). The National Priorities List (NPL) of September 30, 1987, was reviewed to identify those sites containing lead. Of the 457 sites, 307 have lead as an identified contaminant and 174 have an observed release of lead to air, to surface water, or to ground water. In addition to describing facilities and lead releases for which remedial action was designed under the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA), EPA has also gathered data for an urban, non-CERCLA site in Boston where children are exposed to soil contaminated by lead-based paint. This site scored 3.56 under the current Hazard Ranking System (HRS). (The minimum HRS score needed for a site's listing on the NPL is 28.5.) Revisions of the HRS by EPA could change the urban site's score, depending on what revisions are made.

B. SUMMARY OF REPORT RECOMMENDATIONS

The report concludes with Chapter XI ("Lead Exposure and Toxicity in Children and Other Related Groups in the United States: Information Gaps, Research Needs, and Report Recommendations"), an overview of information gaps, research needs, and recommendations. Of key importance are the various general and specific recommendations of the report.

In view of the multiple sources of lead exposure, an attack on the problem of childhood lead poisoning in the United States must be integrated and coordinated, if it is to be effective. In addition, such an attack must incorporate well-defined goals so that its progress can be measured. For example, the lead exposure of children and fetuses must be monitored and assessed in a systematic manner if efforts to reduce their exposure are to succeed. A comprehensive attack on the lead problem in the United States should not preclude focused efforts by Federal, State, or local agencies with existing statutory authorities to deal with different facets of the same problem. Indeed, it is important that all relevant agencies continue to respond to this important public health problem, but they should do so with an awareness of how their separate actions relate to the goals of a comprehensive attack.

Specific recommendations, by category, are summarized below:

- o Coordinated efforts to reduce lead levels in sources that remain as major causes of lead toxicity, particularly paint and dust/soil lead, are strongly recommended.
- o Scientific assessments of lead levels in these sources, through strengthening of existing programs to monitor environmental levels of lead, should accompany removal/reduction efforts.
- o Major improvements in the collection, interpretation, and dissemination of environmental lead data on a national level are needed. In particular, lead screening data should be compiled in a uniform manner on a nationwide basis.
- o Precise and sensitive methodologies for environmental monitoring and in situ measurement of lead concentrations in various media are required.
- o An integrated assessment of all exposure sources for children is required, including those that are obvious and others that are not. Attention should be given to the lead exposure of children away from the home: paint lead, dust/soil lead, and lead in drinking water in schools, day-care centers, custodial care institutions, and similar sites. Particular attention should be given to the investigation of lead leaching into the drinking water of children in schools.
- o The report strongly recommends that lead abatement initiatives include careful consideration of lead movement to avoid simply shifting the lead problem from one part of the environment to another.

- o The report strongly recommends that much more attention be paid to exposure of the fetus with screening of Pb-8 levels in all high-risk pregnant women.
- o Key initiatives recommended by the American Academy of Pediatrics (1987) should be adopted. These initiatives include screening of every child in the United States at risk of exposure to lead.
- o The report recommends a careful examination of the role of improved nutrition in ameliorating lead toxicity.
- o Continuing large-scale assessments of lead burdens in children, including further national surveys and more regionally focused studies are required.
- o Continued support should be given to the highly productive prospective epidemiological studies now under way and to the development and refinement of metabolic models that are used to examine the quantitative relationship between source-specific lead exposure levels and the resulting lead levels in blood or other body compartments.

C. ISSUES, DIRECTIONS, AND THE FUTURE OF THE LEAD PROBLEM

1. Issues

A number of key scientific issues concerning lead as a major health problem are of special concern for the establishment of public health policy in the United States. These issues include:

- o The Indestructibility of the Problem. As an element, inorganic lead cannot be processed by current technology and destroyed. It will continue to be a potential problem in some form forever.
- o The Relative Non-Transferability of the Problem. Lead cannot be easily shifted from a hazardous setting to a nonhazardous setting without some concomitant increased potential risk elsewhere. Once removed from its geologically bound forms by human activities, lead poses a toxic threat for which there are no natural defense mechanisms.
- o The Environmental Accumulation Factor. Lead accumulates indefinitely in the environment so long as input continues -- no matter in how small a quantity.
- o The Human Body Accumulation Factor. The human body accumulates lead over the individual's active lifetime and does so even with "small" intakes from common sources. For hazards to exist, major exposures at given points in time need not occur.

- o The Risk Population Accumulation Factor. Estimates of exposure and toxicity based on data from particular points in time, such as the estimates provided in this report, greatly understate the cumulative risk for a population posed by a uniquely persistent and pervasive pollutant such as lead. This cumulative toll over extended time is of much greater magnitude, and hence concern, than the prevalence or total exposure estimates for any given year.
 - (a) An individual fetus is never counted more than once in any survey examining populations. In the absence of effective abatement of lead exposure, the estimate of 400,000 individual fetuses at risk for lead toxicity in a single year becomes 4 million individual fetuses in 10 years, or 20 million in 50 years, of lead exposure.
 - (b) Within a given time period, successive sets of preschool children are likely to move into the same housing unit, particularly in the case of deteriorated inner-city tenant housing. Thus, the number of infants and toddlers at risk for the exposure associated with such conditions (especially paint and dust/soil lead) is much greater than the number of deteriorated houses. If one assumes 3 to 5 years as the average period of residency, then perhaps 10 times as many children would be exposed to such conditions over a 30- to 50-year period.
- o The Pervasiveness of the Problem. As a pervasive toxicant, lead is shown in this report to affect totals of children that are high in all socioeconomic/demographic strata. The U.S. lead problem is not simply a problem of a generally neglected segment of society. At present, little or no margin of safety exists between existing Pb-B levels in large segments of the U.S. population and those levels associated with toxicity risk.
- o Absence of a Truly Optimal Blood Lead Level. As a toxicant serving no known physiological requirement, the presence of lead at any level in the body is less than optimal. Current average Pb-B levels in some U.S. population segments are 15- to 30-fold higher than the theoretical value of 0.5 µg/dl calculated for early, pre-industrial humans.

2. Directions and Future of the Lead Problem

At the same time that progress is being made to reduce some sources of lead toxicity, scientific determinations of what constitute "safe" levels of lead exposure are concurrently declining even further. Thus, increasing percentages of young children and pregnant women fall into the "at-risk" category as permissible exposure limits are revised downward. Accompanying these increases is the growing dilemma of how to deal effectively with such a

widespread public health problem. Since hospitalization and medical treatment of individuals with Pb-B levels below approximately 25 µg/dl is neither appropriate nor even feasible, the only available option is to eliminate or reduce the lead in the environment.

In large measure, the more tractable part of the lead abatement effort in the United States is already underway, because the reduction of lead in gasoline, food, and drinking water is amenable to centralized control strategies. Lead in old paint, dust and soil, however, is pervasive and dispersed, and fundamentally different approaches to abatement will be needed. If the Nation is to solve these difficult facets of the lead problem, society must make a strong effort to do so. Without this effort, large numbers of young children in present and future generations will continue to be exposed to persistent and massive sources of lead in their environment.

2. Monitoring Lead Exposure in Young Children and Other Risk Populations

Health professionals determine the degree of lead exposure of children and other risk populations in three ways. The traditional method is to measure lead in external or environmental media (ambient air, workplace air, food, water, dust, soil, etc.) that are pathways for human exposure. Surveying lead in these media, termed environmental or ambient monitoring, provides information about the lead exposure sources and the potential risk for persons so exposed to lead. Environmental monitoring does not indicate the actual internal level of lead for any given individual, because individuals may respond differently to an external source of lead exposure. On a group basis, however, one can determine the probable internal exposure level, given knowledge of the concentrations in the external media and the mathematical relationship between the two variables (different levels in the media and different responses in groups of people). In many cases, one must use environmental monitoring, either because other types of exposure assessment cannot be carried out or because a more direct, systemic measure of exposure is considered unnecessary.

Lead exposure is also commonly assessed through biological monitoring and effects monitoring. Biological monitoring is the measurement of the concentration of lead in a biological sample, e.g., blood, from an exposed person. Effects monitoring involves measuring some endpoint, e.g., the amount of certain proteins or enzymes associated with lead exposure. For a more comprehensive examination of monitoring exposure to lead, see Elinder et al. (1987). Biological monitoring and effects monitoring have advantages for assessing health effects, giving an integrated picture of a person's uptake of lead from all external sources.

[To assess lead exposure in children and other risk groups, the level of lead in whole blood (Pb-B) is the most practical biological measure of ongoing lead absorption. This measure is used throughout Chapters IV-VIII as a means of relating lead exposure levels to adverse health effects. A Pb-B level is generally regarded as reflecting relatively recent exposure. However, Pb-B levels also give information on the relative level of exposure at more remote time points. In other words, a child who had the highest Pb-B level at one time will probably have the highest Pb-B level at a future retesting. Pb-B levels do not, however, indicate cumulative past exposure, as do lead levels of mineralizing tissue such as tooth or bone. Such cumulative measures cannot

yet be routinely employed in screening programs. The kinetic, toxicological, and practical aspects of biological monitoring and other approaches to assessing lead exposure have been extensively discussed by U.S. EPA (1986a).

In young children, effects monitoring for lead exposure is primarily based on lead's impact on the heme biosynthetic pathway, as shown by (1) changes in the activity of key enzymes delta-aminolevulinic acid dehydratase (ALA-D) and delta-aminolevulinic acid synthetase (ALA-S), (2) the accumulation of coproporphyrin in urine (CP-U), and (3) the accumulation of protoporphyrin in erythrocytes (EP). For methodological and practical reasons, the EP measure is the effect index most often used in screening children and other population groups.

Effects monitoring for exposure in general and lead exposure in particular has drawbacks (Friberg, 1985). Effects monitoring is most useful when the endpoint being measured is specific to lead and sensitive to low levels of lead. Since EP levels can be elevated by iron deficiency, which is common in young children, indexing one relationship requires quantitatively adjusting for the other.

An elevated Pb-B level and, consequently, increased lead absorption may exist even when the EP value is within normal limits, now ≤ 35 micrograms (μg) EP/deciliter (dl) of whole blood. We might expect that in high-risk, low socioeconomic status (SES), nutrient (including iron)-deficient children in urban areas, chronic Pb-B elevation would invariably accompany persistent EP elevation. Analysis of data from the second National Health and Nutrition Examination Survey (NHANES II) by Mahaffey and Annest (1986) indicates that Pb-B levels in children can be elevated even when EP levels are normal. Of 118 children with Pb-B levels above $30 \mu\text{g/dl}$ (the CDC criterion level at the time of NHANES II), 47% had EP levels at or below $30 \mu\text{g/dl}$, and 58% (Annest and Mahaffey, 1984) had EP levels less than the current EP cutoff value of $35 \mu\text{g/dl}$ (CDC, 1985). This means that reliance on EP level for initial screening can result in a significant incidence of false negatives or failures to detect toxic Pb-B levels. This finding has important implications for the interpretation of screening data, as discussed in Chapter V.

3. Environmental Sources of Lead in the United States with Reference to Young Children and Other Risk Groups

As graphically depicted in Figure II-1, several environmental sources of lead exposure pose a risk for young children and fetuses. Many sources not

Analysis (J. Schwartz and H. Pitcher) performed a statistical procedure called logistic regression analysis to update estimates of prevalences to 1984 and produce prevalences at the selected criterion values of >15, >20, and >25 µg/dl for SMSA populations of children in the required strata. A detailed discussion of this logistic regression/projection methodology is presented in the second part of Appendix G.

2. Results

Tables V-1 and V-2 give estimated prevalences of Pb-B levels in children above three selected levels; 15, 20, and 25 µg/dl within the strata. Tables V-1 and V-2, respectively, show these rates for "Inside Central City" and "Outside Central City" categories. Shown are three family income levels to indicate relative poverty, and each income category has two classifications by race. Each of these 6 strata is further divided into "SMSA with population ≥1 million" and "SMSA with population <1 million." In 122 SMSAs, we had 12 strata available to us, and each strata gave us three prevalence estimates.

TABLE V-1. PROJECTED PERCENTAGES OF CHILDREN 0.5-5 YEARS OLD ESTIMATED TO EXCEED SELECTED Pb-B CRITERION VALUES (µg/dl) BY FAMILY INCOME, RACE, AND URBAN STATUS,^a WHO LIVE "INSIDE CENTRAL CITY" OF SMSAs, 1984

Family Income/ Race	>15 µg/dl		>20 µg/dl		>25 µg/dl	
	<1 M	≥1 M	<1 M	≥1 M	<1 M	≥1 M
<u><\$6,000</u>						
White	25.7	36.0	7.6	11.2	2.1	3.0
Black	55.5	67.8	22.8	30.8	7.7	10.6
<u>\$6,000-14,999</u>						
White	15.2	22.9	4.0	6.1	1.1	1.5
Black	41.1	53.6	14.1	19.9	4.1	5.9
<u>≥\$15,000</u>						
White	7.1	11.9	1.5	2.5	0.4	0.5
Black	26.6	38.2	6.8	10.4	1.5	2.2

^aSMSA with population <1 million (<1 M) and SMSA with population ≥1 million (≥1 M).

TABLE V-2. PROJECTED PERCENTAGES OF CHILDREN 0.5-5 YEARS OLD ESTIMATED TO EXCEED SELECTED Pb-B CRITERION VALUES ($\mu\text{g}/\text{dl}$) BY FAMILY INCOME, RACE, AND URBAN STATUS,^a WHO LIVE "OUTSIDE CENTRAL CITY" OF SMSAs, 1984

Family Income/ Race	>15 $\mu\text{g}/\text{dl}$		>20 $\mu\text{g}/\text{dl}$		>25 $\mu\text{g}/\text{dl}$	
	<1 M	≥ 1 M	<1 M	≥ 1 M	<1 M	≥ 1 M
<\$6,000						
White	19.2	27.7	5.6	8.4	1.6	2.3
Black	45.9	57.8	17.9	24.5	6.1	8.4
\$6,000-14,999						
White	10.9	16.8	2.9	4.5	0.8	1.2
Black	32.4	43.7	10.7	15.4	3.2	4.6
$\geq \$15,000$						
White	4.7	8.1	1.0	1.7	0.2	0.4
Black	19.5	28.9	4.9	7.6	1.1	1.7

^aSMSA with population <1 million (<1 M) and SMSA with population ≥ 1 million (≥ 1 M).

Table V-3 shows the more limited number of strata and the relevant sets of prevalence estimates when Inside/Outside Central City status could not be ascertained. This applied to 196 SMSAs: 34 paired, 161 with populations below 200,000 (with very few exceptions), and Nassau-Suffolk, NY, with a population over 1 million but no central city.

The NHANES II Pb-B levels reported and used in calculating prevalences for criterion levels are based on Pb-B determinations for all cases--they are not influenced by initial erythrocyte protoporphyrin (EP) determinations and, for cases with elevated EP levels, subsequently selected Pb-B determinations.

In earlier analyses, we attempted to apply the national, urbanized composite prevalences to each of the SMSAs with the appropriate qualifications as to their reliability. This approach was attempted to best respond to the directive of Section 118(f) that asked for ranking of individual SMSAs. When the scientific community was reviewing this approach, however, problems were found with the level of permissible disaggregation due to the sample design of the NHANES II survey. For example, the national source-based differences that were implicit but not specific in the original analytic process could not be broken out and reassigned in disaggregation.

TABLE V-3. PROJECTED PERCENTAGES OF CHILDREN 0.5-5 YEARS OLD ESTIMATED TO EXCEED SELECTED Pb-B CRITERION VALUES BY FAMILY INCOME AND RACE WHO LIVE IN SMALL SMSAs,^a 1984

Family Income/ Race	>15 µg/dl	>20 µg/dl	>25 µg/dl
<u><\$6,000</u>			
White	23.9	6.9	1.8
Black	56.5	22.9	7.4
<u>\$6,000-14,999</u>			
White	13.2	3.4	0.9
Black	40.3	13.6	4.0
<u>≥\$15,000</u>			
White	5.8	1.2	0.3
Black	25.4	6.3	1.4

^aSMSAs with less than 1 million population.

In a second attempt to geographically specify lead exposure, projected prevalence estimates were calculated for the four major regions used in the original NHANES II survey: Northeast, Midwest, West, and South. However, statistical projection data could not be established for these regions. This was due in part to the small numbers of children with higher Pb-B levels in some regions, such as the West. If these small numbers had been used to calculate prevalences in the region, some prevalences would have had unacceptable margins of estimating error. Consequently, in this report, we provide the updated Pb-B prevalence calculations for selected Pb-B levels and the nation's urbanized childhood lead-exposure status for each of the 30 socioeconomic/demographic strata described earlier.

Tables V-4, V-5, and V-6 present the results of applying the estimated prevalences of the 30 strata of children in all SMSAs. Table V-4 depicts children living inside central cities for the SMSAs where such division was possible, and Table V-5 shows the children outside central cities in these SMSAs. Table V-6 shows the findings for smaller and paired SMSA child populations. A partial summary of these three tables for children with Pb-B levels above 15 µg/dl is presented in Table V-7. Table V-8 presents overall summary data.

of the SMSAs. But the ubiquity of the exposure to lead at $>15 \mu\text{g/dl}$ is the striking finding. There are no strata of children totally free of this potential health risk, which holds true for higher Pb-B levels as well.

B. NUMBERS OF LEAD-EXPOSED CHILDREN BY COMMUNITY-BASED SCREENING PROGRAMS

In the previous section, the number of lead-exposed children was identified and categorized by socioeconomic-demographic variables and prevalence of selected Pb-B levels. In this section, we will discuss U.S. communities that have screening programs for identifying young children at risk. Elevated Pb-B level plus elevated erythrocyte protoporphyrin (EP) in blood is the measure used for assessing this risk. Because these screening programs are defined geographically as to city, county, or state, these programs and their locales come within the general meaning of the directives of Section 118(f)(1)(A) of SARA. In general, children living in these screening sites are considered to be at highest risk for lead exposure/toxicity, as we presently understand it.

This section briefly discusses the Second National Health and Nutrition Examination Survey (NHANES II) to compare with the lead screening efforts used over the years.

1. Lead-Screening Programs

Before examining in detail the results of the various lead exposure screening programs operating in U.S. communities it is useful to consider how the screening programs developed and their current status for interpreting the results of these programs. An additional overview of the U.S. screening process is also presented later, in the chapter dealing with exposure abatement and related issues. A comprehensive history of the public health aspects and operational characteristics was presented by Lin-Fu (1985a,b).

Screening activities were first mandated and supported by the 1971 Lead-Based Paint Poisoning Prevention Act, and the actual project started in Fiscal Year (FY) 1972. Shortly thereafter, the U.S. Centers for Disease Control (CDC) was given administrative responsibilities for these activities. By FY 1981, under CDC's guidance, screening in the United States had expanded to more than 60 programs and represented eight regions of the Department of Health and Human

Services (DHHS). Part of CDC's overall efforts included establishing a laboratory proficiency testing service for Pb-B and EP measurements in screening and other child medicine services. This was under the aegis of the Center for Environmental Health.

In FY 1982, screening and other grant programs were incorporated into the Maternal and Child Health (MCH) Block Grant Program and administered by the Maternal and Child Health Division, Bureau of Health Care and Delivery Assistance, Health Resources and Services Administration, DHHS. Although Farfel (1985) proposes that this change was attended by funding reductions, it is not possible to identify an actual reduction figure. As noted by Lin-Fu (1987), each state receiving the block grant portion of money determined its own priorities within the MCH programs, including lead screening. Furthermore, in many cases lead-screening costs are included in budgets for comprehensive pediatric services (Lin-Fu, 1987).

At present, the Association of State and Territorial Health Officials' (ASTHO's) administrative unit, the Public Health Foundation, is collecting data on childhood lead-poisoning prevention efforts from the various states; however, participation is voluntary. A number of states and constituent programs within the states have attempted to maintain the same level of effort that prevailed under CDC administration of the programs. In general, this is the case for the major programs in New York City, Chicago, Baltimore, and Massachusetts.

In FYs 1982 and 1983, the numbers of reporting states were 26 and 33, respectively. Has the number of programs decreased? This question cannot be answered very well without detailed canvassing. During the present administrative period, each state agency reports their results, and a given state may have more than one program unit as defined under the former CDC system. In December 1986, ATSDR canvassed reporting states and other jurisdictions; 41 program units responded with usable data. For all responding programs, the count was over 45 units. This tally included the MCH projects in Massachusetts and counted the rest of the state as one screening unit.

Screening data for different geographic areas have been tabulated. For several reasons, including continuity across time and differences in program characteristics, shown are: (1) data for the final year of the original program administered by CDC, FY 1981; (2) screening results from the programs under the

Maternal and Child Health Block Grant as reported under the ASTHO program (FY 1983); and (3) survey results gathered by ATSDR in December 1986. The ATSDR survey include results from agencies reporting by fiscal and calendar years, reported within 1985 or 1986 or both, and from independent programs within the various states.

Within each program period and between program periods, several factors have influenced, and continue to influence, screening results. (1) The screening risk classifications have changed since the results set forth in the following tables were gathered. These various classification schemes are shown in Tables V-9, V-10, and V-11. (2) Targeting high-risk populations has probably changed over the years. From FY 1972 to 1981, the strategies for screening populations, under CDC guidance, were uniform. The main goal was to screen groups of children in the community judged at high risk and having high prevalence rates for elevated Pb-B levels and for lead poisoning serious enough to warrant medical or public health action. Although this screening may give the number of children exposed in high-risk areas, it does not necessarily reflect the nationwide status of the lead problem. This screening was appropriate for the original purpose, that is, to concentrate attention on those children who most urgently need screening.

TABLE V-9. CDC LEAD SCREENING CLASSIFICATION SCHEME, 1978-1985^a

Blood Lead ($\mu\text{g}/\text{dl}$)	Erythrocyte Protoporphyrin ($\mu\text{g}/\text{dl}$ Whole Blood)		
	50-109	110-249	>250
30-49	II	III	III
50-69	III	III	IV
≥ 70	b	IV	IV

^aClassification numbers increase with increased "toxicity" risk and need for diagnostic evaluation. A given class, e.g., Class III, can represent a combination of Pb-B/EP results. See CDC (1978) statement for more details.

^bNot commonly encountered in screened populations.

TABLE V-10. CDC LEAD SCREENING CLASSIFICATION SCHEME AS OF JANUARY 1985,
USING THE HEMATOFLUOROMETER^a

Blood Lead ($\mu\text{g}/\text{dl}$)	Erythrocyte Protoporphyrin ($\mu\text{g}/\text{dl}$ Whole Blood) ^b			
	<35	35-74	75-174	≥ 175
Not Done	I	- ^c	- ^c	- ^c
≤ 24	I	I ^a	I ^a	EPP ^e
25-49	I ^b	II	III	III
50-69	- ^d	III	III	IV
≥ 70	- ^d	- ^d	IV	IV

^aInstrument for field measurement of EP, see CDC (1985) statement.

^bZinc protoporphyrin measured with hematofluorometer and free EP with chemical method.

^cRequires a Pb-B measurement.

^dNot usually observed.

^eErythropoietic protoporphyria (EPP), a genetic disease, is a possibility.

TABLE V-11. CDC LEAD SCREENING CLASSIFICATION SCHEME AS OF JANUARY 1985,
USING CHEMICAL ANALYSIS OF EP^a

Blood Lead ($\mu\text{g}/\text{dl}$)	Erythrocyte Protoporphyrin ($\mu\text{g}/\text{dl}$ Whole Blood) ^b			
	<35	35-109	110-249	≥ 250
Not Done	I	- ^c	- ^c	- ^c
<24	I	I ^a	I ^a	EPP ^e
25-49	I ^b	II	III	III
50-69	- ^d	III	III	IV
≥ 70	- ^d	- ^d	IV	IV

^aSee CDC (1985) statement for more details.

^b"Free" erythrocyte protoporphyrin measured by chemical method.

^cRequires a Pb-B measurement.

^dNot usually observed.

^eErythropoietic protoporphyria (EPP), a genetic disease, is a possibility.

Screening programs are now conducted with methods that tend to under-report the true screening prevalences of Pb-B levels. Since an EP level is the first step in assessing lead exposure in young children, children who have a "normal" EP level but an elevated Pb-B level will not be counted as a subject risking toxicity. The rate of these "false negatives" was reported to be considerable (see earlier quantitative discussion in Chapter II). Furthermore, the true rate may be even higher than when the prevalence of lead exposure is determined mainly at the time of clinic visits or the equivalent, compared with, for example, intensive, door-to-door canvassing.

Tables V-9, V-10, and V-11 show changes in risk classifications that consist of lowering the Pb-B level for the lowest category by 5 μg (from 30 to 25 $\mu\text{g}/\text{dl}$) and lowering the EP level in whole blood by 15 units (from 50 to 35). As noted elsewhere, these changes represent a trade-off between the lead levels the pediatric health community sees as harmful and the logistics of screening and the technical limits of the EP measuring methods routinely used. In other words, Pb-B levels below the CDC level of 25 $\mu\text{g}/\text{dl}$ for whole blood should not necessarily be viewed as "safe."

In Table V-12, we have presented groups of children screened in the CDC program for FY 1981 and have given the number of children positive for lead toxicity based on the older action levels of 30 $\mu\text{g}/\text{dl}$ Pb-B and 50 $\mu\text{g}/\text{dl}$ EP. The numbers of responses currently defined as positive are separated further into two risk groups, Class II and combined Classes III and IV. Data in Table V-12 are as reported in the Morbidity and Mortality Weekly Report (CDC, 1982).

Table V-13 shows a summary of the more recent screening results that ASTHO collected from state health agencies. Twenty-seven state agencies provided numbers of young children screened for lead toxicity, but only 24 provided data on confirmed cases of toxicity (Public Health Foundation, 1986).

To determine the current scope and outcomes for various lead screening programs, ATSDR asked all state and known county or city screening units for information on the numbers screened, the numbers of confirmed lead toxicity cases, and the relevant time periods. Data from this ATSDR survey are in Table V-14. The survey period covers the time during which CDC distributed its January 1985 lead statement. Data in this table therefore represent numbers from some combination of the former and present screening classification schemes (For schemes see Tables V-9 through V-11.)

exposure cases was 1.5%. In terms of numbers, more children appear to be screened at present, although statistical comparisons with the older CDC framework would be difficult.

Toxicity responses for 1985-1986 ranged from 0.3% for four programs to 11.0% for the City of St. Louis program. The five highest prevalences are 11.0% (St. Louis), 9.0% (Augusta and Savannah, GA), 4.9% (Harrisburg, PA), 3.5% (Washington, DC) and 3.5% (Merrimac Valley, MA, a program within a MCH project). Most of the rates for confirmed toxicity cases were below 2%.

Prevalences reported for earlier years include those from state agencies, as summarized by the Public Health Foundation. For FY 1983, reporting agencies screened 675,571 children and recorded a prevalence of 1.5%. Similarly, the CDC data for FY 1981 indicated that 535,730 children were screened, with 21,897 meeting the toxicity risk criteria then in use--a prevalence of about 4%. For the reasons noted above, we cannot closely compare these three sets of results.

Trends in prevalences over time, at least in the years under CDC control, suggest a moderate decline in toxicity risk from 1973 on. Data from two programs for 1973 to 1985, and data from the St. Louis, MO, program show downward shifts, even with changes in risk classifications.

With the 1985 change in the CDC toxicity risk classifications, we might expect the number of toxicity risk positive results to increase, and the New York City screening program results show such an increase. In future years, then, the prevalences will reflect this change in classification, but they will also reflect the impact of reduced levels of lead in gasoline and food.

3. Comparison of Prevalences Found in NHANES II Updated Prevalences and U.S. Screening Programs

Because the NHANES II prevalences, and therefore the updated adjusted rates, are based on Pb-B determinations for the subjects, there is consequently no intervening problem of first determining EPs. These rates are not subject to systematic underreporting due to false negative results found when EP determinations are used to classify the initial population group being evaluated and identify who will then be tested for Pb-B levels. Screening programs, however, test for EP first.

Other factors in screening programs probably produce lower prevalences compared with those that can be predicted on the basis of results from NHANES II

and other surveys. Screenings are not always intensive, for example, house-to-house; in most cases, they are conducted in clinics. Any factor that affects the characteristics of clinic visits also affects Pb-B prevalences. For example, the mothers who bring children to clinics may be more concerned, informed, and motivated than those who do not bring their children.

Finally, communities with the highest prevalences may not have unique situations. Their approach to the problem simply may be more systematic than in other communities, and therefore, they report the highest rates. The more appropriate question may well be why all communities do not have higher rates rather than why certain communities have high rates. This type of question cannot be answered unless the screening programs are again centrally administered.

4. Children With Potential Exposure to Leaded Paint in the SMSAs

This section summarizes an attempt to identify potential lead exposure by each SMSA and by a specific source, leaded paint. It therefore combines approaches dealing with source-specific exposure described earlier in Chapter V and those in Chapter VI.

The number of children living in pre-1950 housing was obtained from the actual 1980 Census enumeration. Table V-20 and Appendix D present the ranking of SMSAs by the number of children in pre-1950 housing and show that young children are least often found in the newest housing, built in 1970-1980. This is probably due to the fact that young families are least able to afford newer housing and particularly newer units inside central cities. While children in families with the lowest incomes were found disproportionately in the older housing, large proportions of children in the highest income families were also found living in the oldest housing stock. Furthermore, the children with family incomes above the poverty levels frequently constituted the largest proportion in the oldest housing.

While there is no perfect correlation between the age of the housing and the presence of a leaded paint hazard, note that weathering and chalking occur even when there is no dilapidation or deterioration of the housing. We do not know how many pre-1950 housing units have been renovated or had high lead-content paint removed, but we can say with some confidence that the fraction is quite low, given data for Massachusetts cities described in Chapter IX.

Equally, we do not know the consequences of such paint removals in terms of increasing the raw lead content of the sites' dusts and soils within reach of these young children.

Another factor to consider when examining the significance of housing age is the presence of lead in plumbing and the potential contamination of the drinking water. Older housing may contain lead plumbing, and the most recent stock may contain lead in the solder used for copper piping. In either case, lead may leach into the drinking water, which is discussed more fully in Chapter VI.

The environment in which young children are housed, which depends on the income of young families at the start of the family phase of the life cycle as well as the availability of housing units of different ages, exposes a large proportion of young children regardless of family income to the older housing stock that in turn is likely to contain the paint with the highest lead content. The ubiquity of Pb-B levels representing health risks found by the NHANES II survey is supported by the finding that young children frequently live in the type of housing most likely to contain sources of lead: in paint, drinking water, and dust/soil.

Finally, one significant but often unrecognized point about childhood exposure sources such as leaded paint deserves emphasis. Until leaded paint and contaminated dust and soil are removed from young children's environment, the number of young children at risk for adverse health effects will accumulate over time and continue to present a serious public health problem since each new cohort of children will in turn be exposed to lead in the environment. In other words, the cumulative tally of exposed children becomes much larger as time passes than the specific counts given at one point in time. This is due to both the high mobility within the high leaded paint zones in which the high-risk families live and the sociological fact that poor families may be "locked" into such housing for many years.

5. Conclusions and Overview

This report represents the first systematic effort to quantify the extent of the U.S. child lead-poisoning problem and to place such numbers in some context of distribution of the children, the lead sources, the adverse health responses, and strategies for lead reduction or removal. This chapter is a key

component of this effort and is important both for the numbers provided and for helping answer the obvious questions "Which children have the problem?" and "What can we start to do about it?"

From the key findings of Chapter V, we conclude that the total number of U.S. children exposed to lead at unacceptable levels ($\text{Pb-B} > 15 \mu\text{g/dl}$), 2.4 million SMSA children or 17% of the SMSA child total, arise from many different socioeconomic and demographic strata.

It was to be expected that the "traditional" high-risk groups, e.g., poor, inner-city black children, would have figured prominently in the estimation outcomes, and they do. These high-risk groups are usually defined as such in terms of high prevalence rates of elevated Pb-B levels. Less well understood, perhaps, is the fact that the totals for exposed strata in this chapter are derived from both a prevalence for a given Pb-B and the base population by which the prevalence fraction is multiplied to give a stratum final total.

The consequences of an estimating exercise, across strata, for exposure totals is simply that large numbers in a stratum's base population can have quite low prevalences for certain Pb-B levels and still yield numbers that are comparable to those obtained from high-risk strata that have smaller base populations of children but quite high prevalences of elevated Pb-B levels.

In Chapter V, a variety of estimating strategies were employed, and provide quite different numbers for exposure estimates. These differences were explained earlier in the summary. Furthermore, some of the totals complement each other, providing different views of the same total population of U.S. children. For example, examining the very detailed U.S. Census Bureau counts (not estimates) of children in the 318 SMSAs reported in terms of housing age and family income (Section C) produces the unexpected finding that more children in older housing (high paint-lead levels) were also in noncentral-city, nonpoverty families than were children associated with the typical risk groups. This observation corresponds to this report's projected Pb-B distributions in the nation's children.

These distributions in high-risk housing might account for why certain Pb-B prevalences in the otherwise lower-risk strata of U.S. SMSA children are as high as they are. In other words, distribution of the nation's children into high lead-exposure risk housing is uniform enough that all strata of such children, when examined by means of a national composite survey such as NHANES II, will produce significant prevalences. Clearly, however, other sources also

have an impact on Pb-B levels and their prevalences in the general child population, and this point is established in Chapter VI.

At present, the third national survey of its type, NHANES III, is in the planning stage, and eventually results of this survey (expected in the mid-1990s) will produce more precise numbers for prevalences of Pb-B levels in strata used in NHANES II and in this report. In the interim, the 1990 U.S. Census will also be conducted. For the present, however, the estimates and U.S. Census counts of children in Chapter V are the best that can be done with the available data.

X. A REVIEW OF ENVIRONMENTAL RELEASES OF LEAD AS EVALUATED UNDER SUPERFUND

Section 118(f)(2) of the Superfund Amendments and Reauthorization Act (SARA) of 1986 requires this report to "score and evaluate specific sites at which children are known to be exposed to environmental sources of lead due to releases, utilizing the Hazard Ranking System of the National Priorities List." EPA has carried out this requirement in two ways: (1) by identifying proposed and final sites on the National Priorities List (NPL) that have been numerically scored under the Hazard Ranking System (HRS) and at which lead has been released into ground or surface waters or air, highlighting those sites where children are known to have been exposed to lead; and (2) by gathering data at an urban area in Boston where children are known to be exposed to lead in soil, and scoring one residence as a site under the HRS.

The HRS was designed to respond to section 105(a)(8)(A) of the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA). This section requires that the National Contingency Plan (NCP) include "criteria for determining priorities among releases or threatened releases throughout the United States for the purpose of taking remedial action,...." Section 105(a)(8)(B) requires that the criteria be used to prepare a list of national priorities for sites with known or threatened releases of toxic substances throughout the United States. This use of the HRS to form the NPL is a means of directing EPA response resources to those facilities believed to present the greatest magnitude of potential harm to human health and the environment.

The HRS is a means of comparing one site against others based on the estimated relative threat to human health and the environment. Relative threat is determined by assessing the likelihood of release or migration of waste contaminants from a facility, along with the consequences of such a release, such as effects on people. Migration of contaminants from a site occur through air, surface water, and groundwater. Consequences of such a release are

determined by the toxicity and other characteristics of the wastes and whether the release has affected or will affect people.

The HRS is not an assessment of the risks found at a facility. Such an assessment occurs only after a great deal of additional data have been gathered and is used to help determine the type and degree of cleanup necessary to reduce the risk to human health to an acceptable level.

A. NPL SITES--PROPOSED AND FINAL

To be listed on the NPL, a site must score at least 28.5 out of a possible score of 100 under the HRS in effect September 30, 1987. (EPA is now revising the HRS; see discussion later in this chapter.) Of the 957 proposed and final NPL sites as of September 30, 1987, 307 have lead as an identified contaminant, and 174 have an observed release of lead to air, to surface water, or to groundwater. An observed release is documented by monitoring data showing such a release from the site. The sites with only an identified contaminant had no data showing release of the contaminant from the site. All proposed and final NPL sites with an observed release of lead are listed (with their HRS scores) in Appendix F.

Exposure of Children to Lead at NPL Sites

EPA reviewed site files to obtain data regarding the exposure of children at each NPL site with an observed release of lead. In only a few cases was the Agency able to document exposure of children to lead from the site, since no records are kept that separate children from the general population exposed to releases from a site. In some cases, however, studies had been conducted around sites that showed that children were exposed to lead from the sites. These are documented below.

The Interstate Lead Company, an NPL site in Leeds, AL (HRS score 42.86), is a battery recycling and secondary lead smelting operation. Results of a March 1984 study of lead contamination conducted by the Jefferson County Department of Health and Bureau of Communicable Diseases, showed that children under 10 years of age living less than one-half mile from the lead plant had higher blood lead levels than children the same age living farther from the plant. Blood lead levels of all children ranged from 6 to 29 µg/dl.

The East Helena, MT, site (HRS score 61.65) is a primary lead and zinc smelter where 8.4 square miles of land have been contaminated, with lead in soil measuring more than 1,000 ppm. In a random sample of 90 children living near the smelter, blood lead levels of 6 to 25 $\mu\text{g}/\text{dl}$ were detected. In 1975 and 1978, cattle reportedly died from lead poisoning.

At the NL Industries/Taracorp Lead Smelter, an NPL site in Illinois (HRS score 38.11), the Illinois EPA measured high lead levels in the soil of residential areas near the smelter. Two soil samples exceeded 5,000 ppm lead. The Illinois EPA recommended that small children living nearby be restricted from playing in the dirt, from eating outside, and from placing dirt or dirty objects in their mouths.

The Sharon Steel Smelter, in Utah, has been proposed for the NPL (HRS score 73.49). It is an inactive smelter with 10 million tons of tailings piled on the site. People have taken some of the waste from these piles to use in sandboxes and gardens. Analyses by the State of Utah indicate elevated levels of lead and other heavy metals in edible portions of food grown on soil to which the waste from this site has been added.

At the Harbor Island Battery Recycling site in Washington State (HRS score 34.60), elevated levels of lead have been reported in workers and their children.

At the Brown's Battery Recycling site in Pennsylvania (HRS score 37.34), the Pennsylvania Department of Health measured elevated blood lead levels in four children. One child received treatment to reduce his body burden of lead. Soil from three residences adjacent to the primary disposal area had lead levels ranging from 1,120 to 84,200 ppm.

At the Bunker Hill Mining and Metallurgical site (HRS score 54.76), a lead smelter in Idaho, the Idaho Department of Health and Welfare found an epidemic proportion of children (98%) living within 2 miles of the smelter who had blood lead levels exceeding 40 $\mu\text{g}/\text{dl}$.

The Lackawanna Refuse site in Pennsylvania (HRS score 36.57) is a former strip mining site. It is near a residential area of 9,500 people, and local children use the site as a recreational area. EPA and the Pennsylvania Department of Environmental Resources found concentrations of 12,000 ppm lead in the waste contained in the thousands of drums found at the site.

As can be seen from the descriptions of the foregoing cases, the sites on the National Priority List with observed releases of lead consist of sites

where manufacturing, processing, or disposal of lead has taken place. This can be seen graphically in Table X-1, relating the twelve most common activities at NPL sites with observed lead releases from waste. Actual disposal of waste accounts for the largest proportion by number of sites, with manufacturing, ore processing, and battery recycling at the bottom of the list.

B. URBAN AREA SITE

EPA has carried out a preliminary assessment and site investigation of a Boston area to prepare an HRS package for scoring the site. The site consists of a rectangular area encompassing approximately 5 square miles where children are believed to be exposed to lead from both interior and exterior paint and from elevated lead concentrations in the soil surrounding the houses. This site was chosen because it contains areas that have been designated by the City of Boston as Emergency Lead Poisoning Areas (ELPAs). An ELPA is an area of one or more city blocks where a higher than average number of children were found to have elevated blood lead levels.

The areas consist mostly of triple-story houses of frame construction. Most have been converted to six apartments, causing a high population density in the area. Because the houses are separated by only a few feet, lack of sunlight inhibits the growth of grass or a suitable cover for the soil, and the lead is available to children playing in the dirt in these areas.

Data were gathered for two housing units within the area specifically to be evaluated under the HRS. Scoring was done for the unit expected to score highest under the HRS. This unit has greatly elevated soil lead levels both in front and behind the house, but no evidence of peeling paint. The lead concentrations in the front of the house near the street exceed the concentrations in the back of the house, indicating that a portion of the lead may have resulted from auto emissions.

The data for this area have been processed through the Hazard Ranking System as if it were a hazardous waste disposal site to be evaluated for the NPL. The data were collected by Region I personnel and scored prior to transmission to headquarters. This original scoring package passed through the quality assurance and quality control process without revisions, and the assigned score of 3.56 was affirmed. The minimum HRS score needed for listing on the NPL is 28.5.

TABLE X-1. MOST COMMON ACTIVITIES ASSOCIATED WITH LEAD WASTE
AT NPL SITES WITH LEAD RELEASE^a

Activity of NPL Site	With Observed Lead Release		All NPL Sites		
	Rank	Number of Sites	Rank	Number of Sites	Percentage With Lead Release
Landfill, commercial/ industrial	1	75	2	349	21
Surface impoundments	2	66	1	350	19
Containers/drums	3	49	3	261	19
Landfill, municipal	4	35	4	158	22
Waste piles	5	27	10	89	30
Spill	6	21	6	139	15
Other manufacturing/ industrial	7	18	5	142	13
Chemical process/ manufacturing	8	18	7	104	17
Battery recycling	9	15	24	17	88
Tank, above ground	10	14	9	94	15
Leaking containers	11	10	8	95	11
Ore processing, refining, smelting	12	9	19	29	31

^aThe release of lead is not necessarily attributed to the specific activity indicated. Sites often have more than one activity and EPA reporting requirements do not identify the activity to which the release of a specific substance is attributable. These activities are present at 162 of the 167 sites with an observed release of lead.

- (a) Field studies on the efficacy of broader lead removal from child environments, e.g., at a tract or neighborhood level or larger. Such efforts would include before-and-after evaluation of Pb-B levels, done with careful statistical and quality control/quality assurance protocols. In response to SARA, EPA is now addressing this problem systematically via three demonstration projects.
- (b) Field studies of the type detailed above with designs stratified to permit assessment of how such procedures relate to primary contributors, for example, lead paint versus urban air fallout of lead. Here, also, the EPA demonstration projects will be helpful, if sub-studies are made of these variables.
- (c) Further development of field methods is necessary to test lead in various exposure media. Of particular importance are improved in situ methods for lead in painted surfaces.
- (d) Assessment of the actual physical removal technology for removal of leaded paint, dust, and the like. A related examination of practical disposal plans is also necessary. As noted earlier, leaded paint consists of an aggregate burden of millions of tons, while other inputs also add up to millions of tons. Therefore, disposal is not inconsequential to the lead abatement problem. Moving the lead may also inadvertently shift exposure to another population. A draft report for a model site in Boston (Appendix E), discusses various scenarios and associated problems.
- (e) Further examination of the efficacy of lead screening programs for high-risk populations both for their scope and effectiveness and for the relationship between public financial support of screening and the ability to identify children at risk is needed.
- (f) Assessment of the relative costs of effective, if expensive, alternatives to the piecemeal abatement, the piecemeal enforcement, and the piecemeal follow-up for reexposure that appears to be the present status of remedial actions. Is it less expensive, in human and resource terms, to consider such measures as relocation?
- (g) Research that explores the feasibility of better biochemical screening measures beyond the use of erythrocyte protoporphyrin (EP) since this measure is not reliable and yields too many false negatives. Failure to detect positive cases makes such research urgent.

C. RECOMMENDATIONS

In view of the multiple sources of lead exposure, an attack on the problem of childhood lead poisoning in the United States must be integrated and

coordinated to be effective. In addition, such an attack must incorporate well-defined goals so that its progress can be measured. For example, the lead exposure of children and fetuses must be monitored and assessed systematically if efforts to reduce their exposure are to succeed. A comprehensive attack on the U.S. lead problem should not preclude focused efforts by Federal, state, or local agencies with existing statutory authorities to deal with different facets of the same problem. Indeed, all relevant agencies should continue to respond to this important public health problem, but do so with an awareness of how their separate actions relate to the goals of a comprehensive attack.

The following specific measures are recommended to support the general objective of eliminating childhood lead poisoning.

1. Lead in the Environment of Children

(a) We recommend that efforts be implemented to reduce lead levels in sources that remain major causes of childhood lead toxicity.

- (1) Leaded paint continues to cause most of the severe lead poisoning in U.S. children. It has the highest concentration of lead per unit of weight and is the most widespread source, being found in approximately 21 million pre-1940 homes.
- (2) Dust and soil lead, derived from flaking, weathering, and chalking paint plus airborne lead fallout over the years, is the second major source of potential childhood lead exposure.
- (3) Drinking water lead is of intermediate but highly significant concern as an exposure source for both children and the fetuses of pregnant women. Food lead also contributes to exposure of children and the fetuses.
- (4) Lead in drinking water is a controllable exposure source and state and local agencies should be encouraged to enforce strictly the Federal ban on the use of leaded solder and plumbing materials. Stronger efforts should also be made to reduce exposure to lead-based paint and dust/soil lead around homes, schools, and play areas.

(b) We recommend that efforts to reduce lead in the environment be accompanied by scientific assessments of the amounts in each of these sources through strengthening of existing programs that currently attempt such assessment. The largest information gap exists in determining which

housing, including public housing, contains leaded paint at hazardous levels. A similar information gap exists for information in distributions of soil/dust lead on a regional or smaller-area basis. Systematic monitoring of lead exposures from food and water is urgently needed. Programs, such as the FDA's Total Diet Study, must be supplemented. The data currently collected with the resources available do not yield sufficient information on the high risk strata of the population to support intervention measures. Water monitoring programs suffer from a paucity of systematically collected data.

- (c) Use of precise and sensitive methodologies is essential for environmental monitoring of source specific lead. More sensitive and precise techniques are required for in situ field testing of lead in painted surfaces.
 - (d) Major improvements in the collection, interpretation, and dissemination of environmental lead data on a national basis are required and recommended to assess the extent of remaining lead contamination and to identify trends. Data from screening programs should be compiled nationally and made uniform so that geographic differences in lead toxicity rates can be determined.
 - (e) The need to examine fully the extent of lead contamination in all parts of the child's environment remains. We recommend emphasis on examining the presence of lead in schools, day care centers, nurseries, kindergartens, etc., particularly the lead in paint, in drinking water, and in soil and dust in such facilities.
-
- (1) We recommend that all attempts at source-specific reduction in children's environments be accompanied by assessment of the long-term effectiveness and efficiency of such actions (see Section 2 of Recommendations also).
 - (2) Lead is both a ubiquitous and persistent pollutant. Planned lead reductions in any environmental compartment must be evaluated in terms of impacts on other compartments so that fruitless shifting of the problem from one source or medium to another is avoided. For example, when leaded paint or soil is removed from a child's environment, ultimate, safe disposal must be considered.
 - (3) The evidence is strong that in utero exposure of the developing fetus occurs at potentially toxic levels in some proportion of pregnant U.S. women. This risk population needs close attention to assess and reduce their most significant lead exposure sources, which should especially include occupational exposures.

- (4) Lead pollution is a health problem that involves almost all segments of U.S. society. Extra-environmental or legal measures should be explored to reduce lead levels in the environment by both public and private sectors.

2. Lead in the Bodies of Children

- (a) Children are being exposed to and poisoned by lead while environmental lead reduction is under way. Screening programs with sufficient funding to make a real and measurable impact are urgently needed.
- (b) There is a need to maintain screening programs extant in some states that currently identify children at risk from lead exposure at or above blood-lead levels of 25 µg/dl. Since current EP tests, used as the initial screen, cannot accurately identify children with blood lead levels below 25 µg/dl, screening tests that will identify children with lower blood-lead levels must be developed.
- (c) The 1987 statement of the American Academy of Pediatrics calling for lead screening of all high risk children should be supported by assistance in implementation.
- (d) Use of in vivo cumulative lead screening methods is recommended as soon as available. A quick, accurate, noninvasive screening test would be better accepted by parents, resulting in many more children being screened.
- (e) We recommend that screening be extended to all high-risk pregnant women, with particular emphasis on urban teenaged pregnant women, and that prenatal medical care providers be involved in this effort.
- (f) We recommend determination of the prophylactic role of nutrition in ameliorating systemic lead toxicity.
- (g) Further use should be made of metabolic models already developed and research to refine them should be done. This will enable their use to predict total body burden contributions from varying environmental sources of known lead levels.
- (h) Long-term prospective studies of lead's effects during child growth and development should continue to be supported through appropriate support mechanisms, beginning with the relationship of maternal lead burden to in utero toxicity and including children with neurological disabilities and genetic disorders, such as sickle cell anemia.

- (i) Nationwide assessments of lead toxicity status in U.S. children on a continuing basis are recommended. Efforts such as the planned NHANES III survey should be supported to maximize the data collected about lead exposure levels. Support as well should be provided for more geographically focused surveys, e.g., on the level of Metropolitan Statistical Areas (MSAs).

MUSHAK
EXHIBIT C

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PROCEEDINGS

of the

First National Conference on Laboratory Issues in Childhood Lead Poisoning Prevention

October 31 - November 2, 1991

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U.S. Agency for Toxic Substances and Disease Registry's
Report to Congress on Childhood Lead Poisoning in America
Review and Update

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SUMMARY

Lead poisoning is the major environmental threat to the health of children. The explosion in research documenting lead's status has been recorded and interpreted in a number of expert consensus documents. In response to mandate, the U.S. ATSDR sent to Congress in 1988 a key document of this type and a review with update is presented here. The ATSDR report set forth methodologies and results that answered the questions: How many U.S. children are at risk for lead poisoning by area? What are the sources of child Pb exposures? What are the health effects and their persistence? How effective are exposure reduction methods?

In 1984, 2.4 million metropolitan Black and White U.S. children had a projected Pb-B > 15 $\mu\text{g/dL}$. The national total for all races was 3 to 4 million. For children with Pb-B > 5 $\mu\text{g/dL}$ and > 10 $\mu\text{g/dL}$, post-report analysis yielded corresponding numbers (% of SMSA total) of 12.5 million (91) and 6.4 million (46). In 1984, 400,000 pregnant women were projected to have Pb-B levels > 10 $\mu\text{g/dL}$, a level linked to fetal toxicity risk. Numbers of children in screening programs show a 1.5% toxicity rate. The number of children < 6 years old in old housing, an indirect index of lead paint exposure, was 4.3 million in 1980.

The report to Congress quantified numbers of children exposed by source. Source-specific tallies are difficult for a multi-media contaminant like lead. Major sources and pathways are lead in paint, lead in dust and soil, airborne lead fallout, and lead in drinking water. Children exposed to lead paint enough to produce a toxic blood lead (> 15 $\mu\text{g/dL}$) number ca. 2 million. Dust and soil Pb exposures were estimated to be more than 10 million. Tap water potential exposure affects ca. 4 million children, 240,000 with Pb-B > 15 $\mu\text{g/dL}$.

Toxicity of Pb to the developing human CNS has been shown to be persistent in several studies. Persistence is also seen in exposed animals. Lead's effects are pervasive and occur at low exposures. Since the report's release, additional studies have appeared. The report articulates those sources for which abatement would reduce toxicity in U.S. children. The remedies are legislative and administrative and differ in their likelihood of effectiveness. Exposure reduction in America is basically a post-hoc process, since the large reservoirs of lead are already in place, and apt to remain in place indefinitely in some cases.

INTRODUCTION

The last 20 years has witnessed an explosion of scientific and public health data on lead contamination and its adverse effects. Such knowledge has not arisen in a vacuum, but represents a closed-loop interplay between research and policy activities, as seen in Fig. 1. First, the regulatory, legislative and public health sectors attempt to interpret and act upon new scientific knowledge. In turn, their conclusions and actions trigger support for further research, closing the loop. One mechanism by which science interacts with the policy area is the scientific consensus document. Described below are some elements of the interaction.

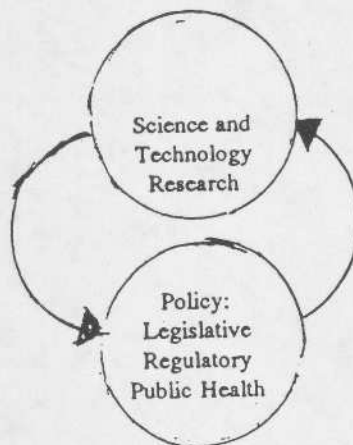


FIGURE LEGEND

Fig. 1. The interplay of science and the policy sectors

In the 1970s, Congress enacted the Clean Air Act (1970) and the Lead-Based Paint Poisoning Prevention Act (LBPPPA, 1971), two early major legislative anchors for regulatory initiatives against Pb sources. The newly-created U.S. Environmental Protection Agency (USEPA) also became involved with lead, both as a gasoline additive and as a criteria air pollutant. It produced its first comprehensive criteria document for lead, the 1977 "Air Quality Criteria for Lead" report (USEPA, 1977).

The Federal public health apparatus became involved in the lead problem, beginning with the Surgeon General's statement (1971). It set a blood lead guideline of 40 $\mu\text{g}/\text{dL}$ and recommended screening for lead poisoning in high-risk children. The U.S. Centers for Disease Control (CDC) issued two Statements on childhood lead poisoning in the 1970s (CDC, 1975; CDC, 1978). CDC's statements are intended to reflect the sense of the nation's pediatric community. Both Statements used blood lead (Pb-B) and erythrocyte protoporphyrin (EP) levels to classify risk.

The National Academy of Sciences (1972) produced the first of its reports on the lead problem, focused on airborne lead and gasoline lead combustion. This report included a set of recommendations for further research. Internationally, the World Health Organization produced its first health criteria report on lead (WHO, 1977). It covered sources of exposure, toxicology, environmental epidemiology of lead and an early attempt at risk assessment.

The first of a new series of assessments of asymptomatic childhood lead poisoning was published by Needleman and coworkers (1979). This study attempted to disentangle the effects of lead from confounding and interactive factors and also addressed the question of an appropriate biological marker of remote exposure, i.e., times when these children were most vulnerable.

In the 1980s, further Congressional actions were taken. These included the Comprehensive Environmental Remediation, Compensation and Liability Act (CERCLA/Superfund 1980), the Superfund Amendments and Reauthorization Act (SARA, 1986), 1986 amendments to the Safe Drinking Water Act and the Lead Contamination Control Act (1988). The USEPA issued its second lead criteria document as four comprehensive volumes (USEPA, 1986a) and the Agency for Toxic Substances and Disease Registry (ATSDR, 1988) released its report to Congress on childhood lead poisoning. The ATSDR report is the subject of this paper. Internationally, a royal commission in England issued its report on lead pollution, with emphasis on lead in air from leaded gasoline combustion (Royal Commission, 1983).

More recently, USEPA issued an update of the 1986 lead criteria document and its addendum material (USEPA, 1990). Two significant reports from the CDC have appeared in 1991. The first (CDC, 1991a) was a strategic plan for eliminating childhood lead poisoning. It placed heavy emphasis on paint-based childhood poisoning due to lead paint and included a cost-benefit analysis for lead paint abatement. The second report (CDC, 1991b), a revised Statement on childhood lead poisoning prevention, reduces the Pb-B action level to 10 $\mu\text{g}/\text{dL}$ and stratifies intervention into preventive and conventional forms.

Legislative Genesis of the Report to Congress

One reaction from Congress to the new data on lead was Section 118 (f) of the 1986 SARA. Section 118 (f) consisted of four subsections that attempted to quantify the scope and depth of the childhood lead problem in America.

ATSDR was directed to quantitatively evaluate four aspects of the lead problem:

- the number of American children exposed to toxic levels of lead by geographic unit;
- the number of children exposed to lead ranked by source and source type;
- the nature of childhood lead poisoning and persistence of toxic effects; and
- methods and alternatives for reducing lead exposure in children.

This article summarizes some of the key methodologies and findings in the resulting 1988 report to Congress and provides an update of material, 1988 to the present. For details, the reader is directed to the report itself (ATSDR, 1988) or publications in the literature based on the report (Mushak and Crocetti, 1989, 1990; Mushak et al., 1989; Crocetti et al., 1990a, b).

ORGANIZATION OF THE REPORT

General Approach

The report posed a number of challenges. First, there was no similar report in the lead literature, qualitatively or quantitatively. Second, source data were scattered in various places and contained in different disciplines. Finally, the report's structure and findings required an extensive analysis and interpretation in a form understood by a broad readership.

A further complication was the non-specific nature of the mandate's directives. An adequate scientific response to the mandate required going beyond the language of the mandate in some cases, as in the inclusion of numbers of women of childbearing age and pregnant women, so as to address the full spectrum of lead's impact on development.

Report Authorship and Peer Review

The report's principal authors were provided additional data by Federal lead specialists and produced six interim drafts and a final version. The drafts were critically evaluated through rather exhaustive peer review, including input from a Federal ad-hoc group of lead experts meeting with the co-authors in four workshops, review by 19 outside lead experts and over 100 other Federal reviewers.

NUMBERS OF AMERICAN CHILDREN EXPOSED TO TOXIC LEAD LEVELS BY AREA

Data sets available for this portion of the report included: (1) enumeration information lodged in the user data tapes of the U.S. Bureau of the Census's 1980 Census, including residents in U.S. Standard Metropolitan Statistical Areas (SMSAs) stratified by socioeconomic and demographic categories, (2) post-Census vital statistics data for updating to 1984, the most recent year for the childhood age band selected, (3) data in the Second National Health and Nutrition Examination Survey (NHANES II; Annett and Mahaffey, 1984), which contained aggregated blood lead levels in socioeconomic and demographic strata for the entire U.S. population, (4) lead screening programs supported of, first, the CDC categorical program from 1973 to 1981 and then the block grant program for states since then, and (5) age-stratified housing data for young children in the American Housing Survey of the 1980 Census.

These data sets allowed three estimation and enumeration exercises for numbers of lead-exposed children at potential or actual poisoning risk arrayed by geographical unit:

- numbers of children < 6 years old estimated to have blood lead (Pb-B) levels above selected values as a function of 30 national socioeconomic and demographic strata in 1984
- numbers of total children enumerated in lead screening programs around the nation and numbers enumerated as having confirmed toxicity risk

- numbers of children <6 years old enumerated as living in housing around the nation built before 1950

Numbers of Children in Various National Strata Having Pb-B Levels Above Selected Values

Estimation of numbers of children with elevated Pb-B in strata involved combining a prevalence for a selected Pb-B with a base population. This gave the numbers of children above a Pb-B level. A base population was obtained by enumeration of the total base population for the national metropolitan strata. The NHANES II survey data required statistical adjustments to 1984, owing to a continuing decline in population Pb-B levels in response to source Pb reductions. These adjustments entailed use of linear and logistic regression analysis techniques, described in detail in an appendix in the report.

Three blood lead ceilings were selected for the report, representing the thresholds selected by various public agencies for onset of adverse effects or a maximum ceiling to safe level: 25 $\mu\text{g/dL}$ (CDC, 1985); 20 $\mu\text{g/dL}$ (WHO, 1987) and 15 $\mu\text{g/dL}$ (USEPA, 1986a).

Detailed tabulations are in the report to Congress and published material (Crocetti et al., 1990a). Table 1 presents prevalences > 15 $\mu\text{g/dL}$ for strata at highest risk for lead exposure and toxicity, i.e., children in inner-city areas of major metropolitan areas. Low-income, inner-city Black children have the highest rates, over 50%, followed by Black children in higher family income categories. Differences across income strata for Black children are lower than for Whites. The most population-dense areas had higher prevalences than smaller cities.

We estimated that 2.4 million metropolitan Black and White children <6 years old had a Pb-B > 15 $\mu\text{g/dL}$ in 1984. We also estimated that the total number of all U.S. children would be 3 to 4 million children above this value. Elements of overestimation and underestimation are present in the prevalence modelling and these are described in the report.

Updated Material on Elevated Pb-B in Children by Area

The lowest level of Pb-B selected for prevalence projection modelling in the report to Congress was 15 $\mu\text{g/dL}$. Since then, recommendations of the US EPA's Science Advisory Board (CASAC, 1990) and the guidelines of the newly released CDC 1991 Statement (CDC, 1991b) identify a Pb-B level of 10 $\mu\text{g/dL}$ as the new value of concern. Prevalences were calculated for this Pb-B level and 5 $\mu\text{g/dL}$ as well. Prevalences of Pb-B above 5 $\mu\text{g/dL}$ in children in most of the high-risk strata described in the report to Congress are virtually 100% for 1984 (Table 2). Corresponding prevalences for > 10 $\mu\text{g/dL}$ in Black children are also very high, exceeding 90% in the inner-city segment of large and smaller U.S. cities (Table 2). Even strata representing minimal Pb exposure show quite high percentages (Table 3). Table 3 shows the affluent segment of White children living in smaller areas were still projected to have about 80% > 5 $\mu\text{g/dL}$. The corresponding figure for 10 $\mu\text{g/dL}$ was 22.1%.

The total numbers of metropolitan Black and White young children projected as having Pb-Bs > 5 $\mu\text{g/dL}$ in 1984 amounted to 12.5 million, or 91% (Table 4). The total number of children with

Pb-B $> 5 \mu\text{g/dL}$ outside the inner-city areas of SMSAs was higher than those inside, due to larger base populations in the latter.

It has been claimed in at least one USEPA source (USEPA, 1989) that the current Pb-B level as a U.S. population geometric mean has declined to about $5 \mu\text{g/dL}$, determined from crude projections to account for exposure reduction. It is difficult to compare such estimates to the 1984 projection prevalences at the $5 \mu\text{g/dL}$ and $10 \mu\text{g/dL}$ levels. Results of the Third National Health and Nutritional Survey, NHANES III, should be available in several years.

Numbers of Metropolitan Pregnant Women and Women of Childbearing Age

A full description of the scope of developmental lead toxicity in populations requires an estimate of in-utero exposures. The methodology for this is detailed in the report and published material (Crocetti et al., 1990b). Four age bands were selected and it was necessary to estimate both the number of pregnant women in the reference year, 1984, as well as numbers of women of childbearing age, since any member of this group can become pregnant at any time.

Results are summarized in Table 5. As seen in Table 5, over 400,000 pregnant women were projected to have a Pb-B level $> 10 \mu\text{g/dL}$, a level linked to fetal effects. The corresponding number for women of childbearing age was about 4.5 million.

Table 1. Projected % of Children, 0.5-5 y old To Exceed Pb-B Values ($\mu\text{g}/\text{dl}$) Who Live "Inside Central City" of SMSAs, 1984*

Family Income/Race	> 15 $\mu\text{g}/\text{dl}$		>20 $\mu\text{g}/\text{dl}$		>25 $\mu\text{g}/\text{dl}$	
	< 1 M	≥ 1 M	< 1 M	≥ 1 M	< 1 M	≥ 1 M
< \$6,000						
	White	25.7 36.0	7.6 11.2	2.1 3.0		
	Black	55.5 67.8	22.8 30.8	7.7 10.6		
\$6,000-14,999						
	White	15.2 22.9	4.0 6.1	1.1 1.5		
	Black	41.1 53.6	14.1 19.9	4.1 5.9		
$\geq$ \$15,000						
	White	7.1 11.9	1.5 2.5	0.4 0.5		
	Black	26.6 38.2	6.8 10.4	1.5 2.2		

* By family income, race and urban status; 1 M = 1 Million

Table 2. Projected % of Children, 0.5-5 y. Old To Exceed Pb-B Values ($\mu\text{g}/\text{dl}$)* Who Live "Inside Central City" of SMSAs, 1984

Family Income/ Race	> 5 $\mu\text{g}/\text{dl}$		> 10 $\mu\text{g}/\text{dl}$	
	< 1 M	$\geq$ 1 M	< 1 M	> 1 M
< \$6,000				
	99.0	99.9	67.3	80.4
	99.9	99.9	91.6	96.5
\$6,000-14,999				
	96.0	99.0	49.8	65.1
	99.9	99.9	83.5	92.0
$\geq$ \$15,000				
	89.0	96.6	31.9	47.2
	99.7	99.9	72.7	85.5

* By family income, race and urban status

Table 3. Projected % of Children 0.5-5 y. Old To Exceed Pb-B Values ($\mu\text{g/dl}$) Who Live "Outside Central City" of SMSAs, 1984*

Family Income/ Race	> 5 $\mu\text{g/dl}$	> 10 $\mu\text{g/dl}$
	< 1 M	< 1 M
< \$6,000		
	97.0	55.7
	99.0	85.2
\$6,000- 14,999		
	90.4	38.3
	99.4	74.2
≥ \$15,000		
	77.9	22.1
	98.5	60.4

* For SMSAs less than 1 Million

Table 4. Numbers of Children in U.S. SMSAs 0.5-5 y. Old Exceeding a Pb-B of 5 $\mu\text{g/dl}$ By Race, Income and Urbanization, 1984

Stratum	In Central City		Outside Central City	
	< 1 M	> 1 M	< 1 M	> 1 M
< \$6,000				
	129,600	256,800	138,700	254,600
	142,100	346,100	92,000	76,600
\$6,000- 14,999				
	275,800	488,400	387,100	487,200
	137,500	341,600	53,700	114,200
≥ \$15,000				
	646,600	1,015,400	1,013,000	2,703,500
	156,500	394,900	72,600	212,300
TOTALS	1,488,000	2,843,200	1,757,100	3,848,400
	Grand Total: 12,526,000 % Total SMSA: 91%			

* Does not include smaller SMSAs; estimates of these total 2.59 million > 5 $\mu\text{g/dl}$.

TABLE 5
Estimated Numbers of SMSA Women of Childbearing Age and Pregnant Women
and Projected Numbers Above 10 $\mu\text{g/dL}$, 1984

Childbearing Age			Pregnant Women		
Race/Age	Number > 10 $\mu\text{g/dl}$		Race/Age	Number > 10 $\mu\text{g/dl}$	
White 15-19	5,478,000	504,000	White 15-19	433,000	39,800
20-44	29,740,000	2,884,800	20-44	2,380,000	230,900
Black 15-19	1,090,000	90,000	Black 15-19	187,000	15,300
20-44	4,984,000	981,000	20-44	595,000	117,200
			44		
TOTAL	41,300,000	4,460,600	TOTAL	3,595,000	403,200

Numbers of Young Children Identified in U.S. Lead Screening Programs

Criteria for selection of toxic exposures were the risk classification guidelines in either the 1978 or 1985 CDC Statements (CDC, 1978, 1985). That is, either a Pb-B of 25 $\mu\text{g/dL}$ or higher confirmed in children with an EP level of 35 $\mu\text{g/dL}$ or higher, or a Pb-B of 30 $\mu\text{g/dL}$ /above and an EP of 50 $\mu\text{g/dl}$ /above. The methods for data gathering and the results are described in the report and published material (Crocetti et al., 1990a).

Using screening data obtained from U.S. programs operating in the 1985-1986 period by ATSDR staff, a total annual screening tally for the interval was enumerated at 785,285, of whom 11,739, or 1.5%, met CDC criteria for confirmed lead toxicity. This number cannot be related to earlier screening activity very easily.

Update on Numbers of Pb-Exposed Children Identified in Screening Programs

Preliminary summary lead screening statistics for FY 1989 have been provided to this author (Public Health Foundation, 1991). Table 6 summarizes the total number screened, total number of confirmed toxicity cases and toxicity % in states reporting both results. The ten largest reporting programs are also tabulated. The total preliminary number screened in the 30 reporting states was 1,228,798, ranging from a low of 34 (Mississippi) to 357,129 (New York). Of these states, 25 reported both a screening count and the number of confirmed toxicity cases. For these states, the total number of toxicity cases was 9,305 out of a total screening tally of 1,129,948, or 0.8%. The range of toxicity cases was 1 (Mississippi) to 2,510 (New York).

Numbers of Children Exposed to Paint Lead in Housing

Details as to enumeration methodology and the resulting SMSA-based enumerations are in the report and the paper of Crocetti et al. (1990a). The total number of children <6 years old living in pre-1950 housing in the 318 SMSAs documented in 1980 Census tapes was 4,374,600, which is 30.6% of the total count of 14,278,000 SMSA children. Size of the SMSA does not necessarily correlate with size of child population living in pre-1950 housing. The older sections of the nation contained the largest fractions of young children in the oldest housing. The newest housing stock is to be found in the West and Southwest. Thirty-three SMSAs had 50% or more of young children in pre-1950 housing, while 14% had 70% or more in the oldest dwellings.

Numbers of Children Exposed to Lead by Source or Source Type

Little in the way of specifics was provided to the authors in Section 118 (f) (1) (B) of SARA. Available data required a multi-tiered response to permit the broadest policy use of such information. Data sets available for quantifying this response to the mandate included numbers of children in age- and condition-stratified U.S. housing. Estimates of children potentially exposed to lead via household plumbing were available, as were estimates of tap water lead levels exceeding safe limits. One analysis was available showing the number of children having a toxic Pb-B level due solely to water Pb exposure. EPA estimates were available showing projected reductions in numbers of exposed children due to leaded gasoline phase-down action. Also available were estimates of children exposed to lead from smelters and battery plants, the major stationary sources.

Lead in soil and dust is generally due to contamination via paint and/or atmospheric fallout. Atmospheric lead arises from leaded gasoline combustion and lead emissions from stationary sources. No information is available showing numbers of children at risk due only to dust/soil lead. One must employ summed estimates from the pathway inputs: paint lead, gasoline lead and emissions from stationary operations.

Available dietary lead data showed percentile distribution of childhood lead intake from measurements obtained in the 1970s.

Methodologies and strategies in responding to this directive involved exposure numbers for children at three levels of precision: potential exposures to lead in a given source or pathway, exposures to lead in different media sufficient to induce elevation in Pb-B, and, finally, numbers of children exposed in source-specific and pathway-specific ways sufficient to induce blood lead levels associated with toxic effects.

Numbers of Children Exposed to Lead in Paint

Any young child in a home with lead paint accessible through ingestion of peeling paint chips or the chewing and gnawing of accessible paint surfaces is potentially at risk for lead paint toxicity. It was estimated that 12 million children <7 years old lived in the approximately 42 million residential units build during the period when lead paint was used, i.e., pre-1977.

The highest risk for lead paint poisoning occurs in the most deteriorated housing, where paint lead is relatively available as chips or powdered/weathered particles. The number of children in the approximately 6.2 million deteriorated, pre-1940 units was estimated to be 1.8 million. We also found that an estimated 1.2 million children <7 years old had Pb-B > 15 $\mu\text{g/dL}$ due principally to lead paint.

Numbers of Children Exposed to Lead from Leaded Gasoline

All communities with heavy traffic density have been impacted by leaded gasoline over the years, especially in past years when lead additive levels were highest. The number of potentially exposed children in the case of leaded gasoline was chosen to be the number of children <7 years old in the nation's 50 largest metropolitan areas. This figure was 5.6 million. Children actually exposed to lead exhausts at toxic levels were estimated by EPA for children up to 13 years of age (USEPA, 1985). In EPA's approach, we cannot identify actual Pb-B levels in children but only that reduction in Pb-B is sufficient to fall below selected Pb-B levels. Using a toxic Pb-B criterion value of 15 $\mu\text{g/dL}$, the corresponding reductions for the six years employed were:

<u># Children</u>	<u>Year</u>
0.7 million	1985
1.7 "	1986
1.6 "	1987
1.5 "	1988
1.4 "	1989
1.3 "	1990

These declines continue past the phase-down year due to the residual effect of past leaded gasoline fallout onto soils and as dusts.

Numbers of Children Exposed to Lead From Stationary Sources

Several estimates of children potentially exposed to stationary source lead were available and these base numbers were combined with prevalence of elevated Pb-Bs at stationary source sites to yield numbers of children with elevated Pb-B. Prevalences ranged widely, producing a range in total numbers. EPA found that the total number of children <7 years old living around primary and secondary smelters and battery plants is 230,000. Of this base population, the number of children with Pb-B above 20 $\mu\text{g/dL}$ was estimated to be 7,500 for those living around secondary smelters. The corresponding estimate of total numbers for primary smelter exposures with Pb-B > 25 $\mu\text{g/dL}$ is a broad range, 210 to 5500 children.

Numbers of Children Exposed to Lead in Dusts and Soils

Estimates of children exposed to lead in dust and soil are derived from exposures to paint, gasoline and stationary source lead. Owing to simultaneous exposures, simple summing across sources gives an over-estimate because of double counting. Summing yields an upper bound to the overall estimate (because of overlap) of 11.7 million children.

Numbers of Children Exposed to Lead in Drinking Water

Lead enters the drinking water of children both at home and away from home, e.g., at kindergartens, elementary schools, day care centers, etc. Lead contamination in household or building plumbing is the source rather than from the water source or distribution (USEPA, 1986b).

Potential exposure of children <5 years old in old housing stock with a significant fraction of lead lines or connectors amounts to 5.2 million subjects. The corresponding number for children 5-13 years old is 8.7 million. Estimates of children in dwelling units built before 1920 total 2.7 million children, while 0.84 million lived in those built in the past several years before the analysis.

It has been estimated that 42 million U.S. residents consume tapwater lead at a level >20 $\mu\text{g/dL}$ (USEPA, 1986b). Of these individuals, 3.8 million are children <6 years of age. This level of contamination will elevate Pb-B by a minimum of several units. In its cost-benefit analysis for control measures associated with water lead, EPA has estimated (USEPA, 1986b; Levin, 1987) that about 240,000 children <6 years old will have a Pb-B >15 $\mu\text{g/dL}$. Of this figure, 230,000 have a Pb-B level between 15 and 30 $\mu\text{g/dL}$.

Numbers of Children Exposed to Lead in Food

The report to Congress quantified toxicity risk for dietary lead among children by (1) determining the level of Pb intake at the 95th percentile in children 2 to 5 years old, adjusted for decline in contaminant level from the period 1973-1978 to time of the study and (2) translating this percentage to a total number. The 95th percentile of lead intake was 130 μg diet Pb/day. Use of a 50% reduction to account for reduced Pb content in foods over recent time yielded a 95th percentile level of 65 $\mu\text{g/day}$. This percentile intake translates to a Pb-B of about 10 $\mu\text{g/dL}$ (Mushak and Crocetti, 1989). This level is linked to early toxicity (CDC, 1991b). As a portion of American children under six years old, 5 percent, or about 1 million children, are therefore above the 95th percentile.

Quantitative Ranking of Pb Exposure Sources for American Children

It is not possible to provide a precise, rigid ranking of lead sources and pathways, as requested in the Congressional directive. First, the level of precision in generating the numbers of exposed children is variable across sources and pathways, ranging from potential exposures to estimation of numbers with toxic body lead burdens. Second, there is some degree of unavoidable overlap, given the multimedia nature of lead exposure in human populations.

It is more useful to rank these sources and pathways by relative magnitude of impact on children's lead exposures. We confine ranking to what would be the most pervasive sources. We also recognized that a myriad of miscellaneous sources existed for ethno-specific or other settings. These are not trivial sources of exposure, per se. Quantification of their impact is not feasible given available data.

At present, lead paint ranks first. It is the most pervasive source for exposure of children and affects those who are also at risk for health problems because of poverty, nutritional deficiencies and more likely contact with this source. Paint lead may be ingested directly or after its transfer as particulate matter to dusts and soils (e.g., Charney et al., 1983; Clark et al., 1985; Bornschein et al., 1987). The report estimated that over 3 million tons of lead remain distributed among the millions of housing units with lead paint. Lead in dust and soil can be ranked second. Dust/soil lead intake is a pervasive exposure pathway for children in both urban and certain non-urban settings (USEPA, 1986a).

Lead in tapwater is a pervasive, potentially troublesome source for childhood exposures, and this occurs both in the home and away from home, e.g., lead in water fountains at schools.

Lead in gasoline is a declining source of lead relative to other current sources, and should be in the second rank of sources. Since the 1920s, however, there was a cumulative input through fallout from leaded gasoline combustion of 4 to 5 million tons. Similarly, lead in children's diet is presently a low-level source. Lead loadings in diets are highly variable, however, and the above discussion only refers to a typical dietary profile. Many idiosyncratic sources for lead entry into food still exist. For example, there remains a problem with canned foods which are imported from countries which have no controls on lead-seamed cans. Acidic fruit juices and beverages can still leach sizeable quantities of lead from poorly glazed vessels serving as storage and preparation containers. The extent to which these sources remain troublesome depends on changes in control of entry into the market.

Update of Source-Specific Material in the Report to Congress

The magnitude of lead paint as a source has not diminished since the release of the report given the huge, dispersed inventory of lead paint-impacted housing units. Since the report's release, a subsequent appraisal by the Department of Housing and Urban Development in its report to Congress (HUD, 1990) noted that there were more units in deterioration than identified in the ATSDR report, i.e., 14 million units nationwide are in unsound condition and 3.8 million of these were occupied by young children. HUD's report to Congress expanded ATSDR's figure of 42 million units having some amount of lead paint to 58 million units, representing about 74% of all housing stock. Studies show the potential for additional paint lead exposure in the course of paint lead abatement, discussed in Chapter 3 of CDC's 1991 Statement (CDC, 1991). The Statement also makes it clear that even partial abatement is effective in reducing children's body lead burdens.

Lead in dust and soil remains a pervasive, lingering exposure pathway for children. While the dust component can be affected by household variables such as cleaning practices (Yankel et al., 1977), the refractory nature of dust/soil lead is obvious, because the major inputs, particularly lead paint, remain (CDC, 1991a; HUD, 1990).

Leaded gasoline combustion since the report to Congress continues to make less of a real-time input to childhood lead exposure. The 1990 Amendments to the Clean Air Act prohibit any lead in gasoline beginning in 1992, concluding the phaseout by the end of 1995 (CDC, 1991b).

Diet as a source of lead continues to decline for the typical American child. It is estimated that by 1988 the daily dietary lead intake was on the order of 5 $\mu\text{g/day}$ (CDC, 1991b). This decline reflects reduced food contamination during production and preparation than in earlier years. This is due to lowered atmospheric fallout and the continued decline of domestic lead-seamed can production, estimated to be 1.4% of all can production by 1989.

LEAD TOXICITY AND ITS POTENTIAL FOR PERSISTENCE IN AMERICAN CHILDREN

Lead exposure over a broad range induces a large variety of acute, subacute and chronic toxic effects in young children. This topic was discussed and analyzed at length in the full report and in the open literature (Mushak et al., 1989). The topic and an update with illustrative studies are briefly reviewed here.

The concern in childhood lead exposure is the long-term, persisting impact of low-level lead exposures on the developing CNS, due to the grave nature of such effects and the reality that injury to the CNS is apt to be irreversible (American Academy of Pediatrics, 1987).

Prospective Assessments of Developmental Toxicity in American Children

The most persuasive evidence supporting neurotoxicity at low lead exposures in children is to be found in results from a group of international prospective (longitudinal) studies. These studies followed development of neurotoxicity and other effects in child cohorts, from gestation through at least the first four to five years of life. While studies are not identical, they generally evaluated common toxic endpoints and exposure assessment instruments. These studies are based in Boston, Cincinnati, Cleveland and Port Pirie, Australia. Other studies are either not as far along or their results are difficult to evaluate relative to the above-mentioned studies.

Infants in the above studies showed decrements in the Bayley Mental Development Index (MDI) as a variable function of prenatal (Cincinnati study), cord (Boston and Cleveland studies) or early postnatal (Australian and Boston studies) Pb-B levels. The MDI is commonly used as an early measure of neurobehavioral development in infants. The magnitude of the deficit varied from 2-8 MDI points in studies where reported. The public health implications of this level of MDI decrements have been examined by Grant and Davis (1989) and Davis and Svendsgaard (1987). Average decrements do not reflect the full impact across the full distribution of decrements, i.e., those infants at the lower tail and the higher tail of the distributions sustain proportionately greater impacts.

There were also reported from the Boston Study results showing that child Pb-B at two years of age was statistically associated with decrements in McCarthy scales at almost five years (57 mo.) of age (Bellinger et al., 1987).

These prospective studies also identified effects of prenatal lead exposure on perinatal indicators of development in the newborn. Effects on gestational age and birth weight were noted in the

Cincinnati study (Dietrich et al., 1986) while the Australian study indicated that preterm delivery was significantly associated with maternal blood Pb at delivery (McMichael et al., 1986).

Cross-Sectional Studies of Lead's Developmental Toxicity in American Children

Cross-sectional studies point, in the aggregate, to a causal role for lead in the identified effects. Such studies began in earnest with the pioneering general-population report of Needleman et al. (Needleman et al., 1979). Using secondary dentine lead levels as the exposure marker in a group of Boston-area children, they reported that verbal IQ and classroom behavior were significantly affected by lead. As lead level increased, IQ decrement increased and classroom behavior was more disturbed. Four years later, grade retention of the high-dentine lead children was significantly greater than in the low-dentine group (Bellinger et al., 1984).

A number of subsequent, well-done cross-sectional studies confirmed neurobehavioral effects of lead exposure. For example, Fulton et al. (1987) and Hatzakis et al. (1987) reported that there were significant IQ or IQ-related effects with increases in blood lead levels below 25 $\mu\text{g}/\text{dL}$ for Scottish and Greek children, respectively.

Cross-sectional studies of other neurotoxic outcomes of lead exposure have been described in the report. Schwartz and Otto (1987) described the inverse association of Pb-B in the NHANES II survey with decrements in hearing acuity, while Otto et al. (1985) described diverse neuroelectrophysiological effects in children with relatively low lead exposures.

Other cross-sectional studies of lead were noted. Schwartz et al. (1986) reported that analysis of NHANES II data showed an inverse relationship between Pb-B and anthropometric measures, i.e., chest circumference and height. Lauwers et al. (1986) reported the inverse relationship of Pb-B and growth in children up to about eight years of age. Effects on heme biosynthesis and calcium/vitamin D functions were also described.

Dose-Effect and Dose-Response Relationships for Lead's Effects in Children

The various prospective and cross-sectional studies report neurotoxic and other effects which are associated with a blood lead of 10 and in some cases even $< 10 \mu\text{g}/\text{dL}$. As Pb-B levels rise above 10-15 $\mu\text{g}/\text{dL}$, both the severity of an effect and the multiplicity of adverse effects increase. At Pb-B levels above 100 $\mu\text{g}/\text{dL}$ a fatal outcome becomes more probable.

Persistence of Lead's Effects in Young Children

In the Boston prospective study, blood lead levels at two years of age produced effects that persisted at five years of age in the form of impaired performance on the McCarthy Scales (Bellinger et al., 1987). Bellinger et al. (1984) reported marginally significant inverse relationships of dentine lead in the Needleman et al. (1979) study with teacher's ratings and student IQs in schools. Effect persistence has a biological or intrinsic definition and one that is extrinsically or exposure-driven. Effects that are biologically reversible would still require that

exposures be removed or reduced for reversibility to occur. If exposure persists for years, then so will the effects.

Update of Lead's Low-Level Neurotoxicity in Children

Since the release of the report to Congress, a large amount of published material has appeared providing further support of results noted therein. New material includes statistical ways of examining validity of studies, the cumulative statistical impact of otherwise limited studies via meta-analysis, and appearance of new studies. Evidence for lead as a potent, pervasive health threat to children is supported by a spate of recent studies describing animal models of lead's developmental neurotoxicology. Space limits discussion to a few illustrative studies.

Prospective Studies Update

The above-described prospective studies are ongoing. Recent studies have been published, including updates from the Boston and Cincinnati groups.

Bellinger et al. (1989a) examined the role of lead, IQ and social class (SC) in their study cohort in the form of Pb-IQ, Pb-SC and IQ-SC relationships. These workers also reported that early postnatal Pb-B levels $> 10 \mu\text{g/dL}$ were associated with MDI scores, but only among children in lower socioeconomic strata (Bellinger et al., 1989b). The Boston cohort was also examined with regard to cord blood and fetal growth (Bellinger et al., 1991). Cord blood was found to be inversely related to fetal dysmaturity and weight gain at Pb-B levels of $15 \mu\text{g/dL}$ and above.

In the Cincinnati cohort, Shukla et al. (1989) found that postnatal growth was related to the Pb-B increment, 3 to 15 mo., but only in infants whose mothers had a prenatal median Pb-B of $7.7 \mu\text{g/dL}$. Dietrich (1990) noted a significant inverse association between maternal Pb-B (mean = $8.1 \mu\text{g/dL}$) and speech/language abilities in three-year-olds. Four-year-olds were subsequently found to show an inverse relationship between neonatal Pb-B and all subscales of the Kaufman-Assessment Battery for Children, but this was for the poorest subset of children (Dietrich et al., 1991). A weak association was noted between postnatal Pb-B and the SIM subscale, i.e., the one that measures visual-motor integration and visual-spatial skills. This subscale is analogous to the portion of the McCarthy Scales found to be linked to postnatal Pb-B in the Australian and Boston cohorts. Neurotoxicity other than impaired cognitive performance was reported for this cohort by Bhattacharya et al. (1990). These workers noted that maximum postnatal Pb-B at two years of age in a group of 63 children (mean age = 5.74 y) was significantly associated with impaired postural balance. This suggests that lead impairs interconnections of the vestibular and/or proprioception systems.

Update of Cross-Sectional Studies

A considerable number of new cross-sectional studies were published after the ATSDR report. Several of the most rigorously controlled studies or study groups are noted below. Also presented is a meta-analysis of a number of individual studies.

Winneke et al. (1990) have summarized results of the European Multicenter Study on Lead Neurotoxicity in Children, a cross-sectional multi-group set of neurobehavioral studies under the aegis of the World Health Organization and the Commission of the European Communities. Jointly, the studies involved 1879 school-age children and four measures of neurobehavior. Psychometric intelligence was affected, but the consistency across studies was poor. Other, more consistent findings were effects of lead on visual-motor integration and reaction performance.

Hansen and co-workers (1989) reported that Danish children with circumpulpal dentine levels $>18.7 \mu\text{g/g}$ in shed teeth showed a significant decrease, after controlling for confounding variables, in the verbal and full-scale IQ components of the Wechsler Intelligence Scale for Children (WISC). Impairments were also noted in a behavior rating scale and a measure of visual-motor integration.

Needleman and Gatsonis (1990) carried out a meta-analysis of 12 of the more recently published studies exploring the relationship of childhood lead exposure to IQ via multi-regression analysis with controlling for covariates and using two forms of such analysis. The joint P values for the studies using Pb-B ($N=7$) were 0.0001 for both approaches within the meta-analysis, while for those studies using dentine lead as the exposure index ($N=5$), the two subanalyses gave P values of 0.0005 and 0.004. The approach of meta-analysis in environmental epidemiology theoretically gets around the chronic problem of single studies that have poor statistical power to isolate an association with an endpoint affected by covariates.

Update in Persistence of Lead's Neurotoxicity

Needleman et al. (1990) reported that a subset of subjects in the earlier investigation who showed behavioral problems and lowered IQ as a function of secondary dentine lead were subsequently found (mean age = 18.3 y) to have a seven-fold higher risk of failure to graduate from high school and a six-fold higher risk of having reading problems. These Pb neurotoxic effects therefore persist at least 11 years.

METHODS AND ALTERNATIVES FOR REDUCING LEAD EXPOSURE IN CHILDREN

The history of efforts to reduce lead exposure in children is one that records few successful results in terms of actual prevention or reduction of childhood lead poisoning. Our detailed evidence for this is contained in the full report and the paper of Mushak and Crocetti (1990).

Basic Approaches in Response to the Directive

The language of the relevant subsection in Section 118 (f) of SARA gives little specific direction as to what Congress meant with respect to the level of lead exposure to be prevented. Does it mean reducing exposures that would simply lower body lead burdens below some toxicity threshold? Alternatively, is there implicit in Congress' intent the notion that reductions in

exposure be such as to also produce some margin of safety? To what level is toxicity risk to be reduced? Answers to these questions lie to some extent in the differing forms of lead exposure prevention or reduction that are applicable.

It is common practice in epidemiology to classify exposure prevention methodologies into primary, secondary and tertiary forms. These distinctions are linked in part to preventing the incidence of a disease and avoiding subsequent complications of that disease. In the case of lead exposure prevention, we considered primary and secondary approaches as specifically defined below.

Primary and Secondary Lead Exposure Prevention Classifications

Under the classification of primary prevention, we noted two subcategories: environmental or source control strategies and a mixture of environmental and biological, i.e., nutritional augmentation. The first was source-driven: lead in paint, ambient air, dust and soil, drinking water and food. The second included consideration of calcium and iron supplementation programs.

We employed three subcategories under secondary prevention: environmental, environmental plus biological, and extra-environmental. The first of these included case finding, screening programs, environmental follow-up and event-specific exposure abatement. Nutritional augmentation also appears in the second one, but implemented on a case-specific basis. Legal actions and strictures comprise the final subcategory.

Primary prevention strategies range from controls on the entry of a potentially toxic substance such as lead into commercial channels to removal of the substance from human exposure pathways. With lead paint, this includes banning sale of lead paint and removal of paint from the child's environment. Secondary prevention approaches to lead paint are basically reactive in nature, i.e., are responses to existing and identified problems. Screening programs, although often considered a mechanism for primary prevention, e.g., reducing the incidence of lead poisoning, are also ranked here as secondary. That is, it was often required that areas at high risk for lead poisoning first be identified before screening programs were introduced.

Primary Prevention of Lead Exposure

Lead in Paint

Control of lead paint exposure in young children has largely been a dismal failure. Lead paint was introduced into commercial use in the United States with little concern for any hazard to the health of children in homes with lead paint. Few controls were imposed long after lead paint was known to be a poisoning source. The time between the lead poisoning review of McKhann et al. (1926) and passage of the LBPPPA was almost half a century. Since Congress enacted the LBPPPA in 1971:

- The Consumer Product Safety Commission in 1977 banned sale of leaded paint having leaded content $>0.06\%$ in interstate commerce. This act did nothing for

the approximately 3+ million tons of lead sequestered in dispersed paint in the 58 million living units noted above. It did not affect intrastate production and sale of lead paint, nor did it ban paint already in the national marketing pipeline.

- Housing and Urban Development, in 1973 and 1976, took several steps that were generally reactive in nature. It provided a warning to purchasers and tenants of pre-1950 housing about lead paint and it banned lead in paint at $>0.5\%$ (5,000 ppm). In response to court action (Ashton vs. Pierce, 1983), HUD issued a set of interim guidelines for public and Indian housing. These were described in detail in the report and in Mushak and Crocetti (1990).
- Municipal and state actions against lead paint exposure have entailed both sale of leaded paint within jurisdictions and diverse laws and ordinances to ameliorate such exposures, with little overall success. Some cities were active quite early, e.g., Baltimore, but the magnitude of the problem remains relatively undiminished. Among states, Massachusetts was identified in the report as representing a typical case study of the complex interplay of opposition from vested interests which render seemingly comprehensive regulatory language ineffective in practice.

Lead in Ambient Air

Control of lead in ambient air entails regulation of lead in gasoline and lead emissions from such stationary sources as lead smelters and battery manufacturing plants. Unlike the abysmal record for lead paint, there has been a relatively successful effort to reduce lead emissions to air from leaded gasoline combustion. The USEPA has had regulatory authority over leaded gasoline since 1974. In 1975, EPA classified Pb as a criteria pollutant. This classification required that an enforceable ambient air standard be issued. The growing use of lead-sensitive pollution control equipment in new automobiles of the 1970s also produced a practical, compelling reason for removing lead from gasoline. Effective January 1, 1986, lead in gasoline was reduced to a level of 0.1 g/gal.

Under provisions of Section 108 of the 1970 Clean Air Act and 1977 amendments, EPA reduced the ambient air lead standard to 1.5 micrograms/cubic meter air, averaged quarterly.

Lead in Soils and Dusts

At present there are no specific legislative or regulatory actions directed towards dust and soil lead. Regulatory control is indirectly through the primary input sources, lead paint and stationary/mobile sources of air lead emissions.

Lead in Drinking Water

USEPA is required by the 1974 Safe Drinking Water Act to set primary and secondary drinking

water standards for lead and other pollutants. For human health, this was done via the Maximum Contaminant Level (MCL), which for lead was an enforceable level of 50 $\mu\text{g/L}$.

Lead in Food

Control of lead in the U.S. food supply has rarely taken the form of specific promulgation of food lead standards. The U.S. Food and Drug Administration (FDA) has had control over lead in food dating to use of lead-based pesticides for food crops. There are a number of steps at which lead can enter the food supply for children. These steps have involved FDA in the form of action goals to reduce lead intakes in children below certain levels. Lead contamination reduction from lead-seamed cans entailed non-regulatory cooperative efforts between FDA and the can manufacturers. FDA does control, albeit with mixed results, the import of poorly glazed food containers which are known to be hazardous in typical use.

Through various cooperative measures, the lead in daily diet of children has been declining. In the report to Congress, we noted that major reductions had occurred, when comparing 1982-1984 numbers with 1984-1986 data (Table 6).

Primary Prevention Measures Using Environmental and Biological Approaches

In theory, biological control methodologies can suppress lead entry into the bodies of exposed children and assist in reducing the level of toxicity risk. As part of primary prevention, this entails community-wide nutritional intervention actions. Given the available information on likely effective nutritional modalities, use of iron and calcium supplements would be particularly helpful. Such adjuncts are no substitute for effective environmental lead abatement.

Secondary Prevention Measures

The nationwide lead screening programs that were set up in response to the 1971 LBPPPA under the aegis of CDC, and lasting until 1981, screened approximately 4 million children. Of these, 250,000 children had been identified as lead-toxic cases. About 30% of high-risk children were estimated to have been screened in this time.

There is considerable support for the argument that lead screening has been very effective at present levels of case-finding. It will be even more effective as medical intervention costs continue to rise. The report actually estimated a ratio of >50 for the benefit of averted health costs to unit screening cost.

Legal sanctions as an adjunct to secondary prevention have been grossly ineffective. The report described a case study in which it was shown that in one municipality with a large lead paint poisoning problem, legal measures consisted of minor fines and were largely ineffective.

Update of Alternatives and Methods for Reducing Childhood Lead Exposure

A number of encouraging developments toward reducing childhood exposure have been occurring on the legislative, regulatory and public health policy fronts.

Recently, CDC (1991 a, b) issued two major documents that directly address the issue of exposure abatement and responses to unabated exposures. CDC's strategic plan (1991a) makes it clear that lead paint abatement is cost-effective and the plan offers a schedule for abatement of the worst housing. The 1991 CDC Statement on preventing childhood lead poisoning is a major departure from past statements, in that environmental control and interventions are explicitly recommended on a community as well as individual case basis. The new action level of $10 \mu\text{g/dL}$ will result in a large number of new children at risk, requiring more wide-spread abatement measures.

HUD has produced a report to Congress. It has also issued a set of interim (September, 1990) and revised (May, 1991) guidelines for lead-based paint hazard identification and abatement for public and Indian housing (HUD, 1991). These guidelines, legislatively mandated by the 1989 HUD-Independent Agencies Appropriations Act and the Housing and Community Development Act of 1987 and the Stewart B. McKinney Homeless Amendments Act of 1989, deal with testing, abatement, clean-up and disposal of lead-based paint in public and Indian housing.

Topics in the HUD guideline document include risk reduction for lead-based paint poisoning (Ch. 2), responsibilities in testing (Ch. 3), abatement considerations and methodologies (Ch. 5-7, 9), worker protection (Ch. 8) and waste disposal (Ch. 11).

The USEPA has released a lead exposure reduction plan similar to that of CDC, "Strategy to Reduce Lead Exposures," issued February 21, 1991. In June, 1991, EPA issued its final rule on lead in drinking water [56(110) FR 26480-26564; June 7, 1991]. Lead in drinking water has enforceable regulation at a level of 5 ppb at the water source. Tap levels in homes are to be covered under action level procedures, depending on the outcome of community tapwater sampling. Further steps entail corrosion control followed by removal of lead lines, if necessary, with a 21-year total grace period. The long period for lead plumbing line removal means chronic Pb exposures of infants and toddlers in communities with such systems.

EPA is also proposing rulemaking [ANPR; 56(92) FR 22098-22098; May 13, 1991] with reference to regulating uses of lead under Section 6 of the Toxic Substances Control Act (TSCA). Notable topics for special attention include consideration of the phase-out of current uses of lead posing unreasonable risks and assessment of overall Pb production and use.

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DECLARATION OF J. ROUTT REIGART, M.D.

I, J. Routt Reigart, M.D., declare as follows:

1. The matters stated herein are true of my own personal knowledge. If called as a witness, I would competently and truthfully testify consistent with the following.
2. I am an Associate Professor of Pediatrics at the Medical University of South Carolina in Charleston, South Carolina.
3. Since July 1991, I have served as the Chairman of the American Academy of Pediatrics' Committee on Environmental Health ("Committee"). This Committee is reviewing the Academy's policy on lead testing and treatment, which is currently contained in a five-year-old document entitled Statement of Childhood Lead Poisoning. We are currently reviewing this document because recent information about lead toxicity in children requires revised recommendations to pediatricians. I also serve as Chairman of the Childhood Lead Poisoning Prevention Advisory Committee of the Centers for Disease Control ("CDC").

PROFESSIONAL BACKGROUND

4. As a pediatrician I have devoted much of my professional career to the study, screening and treatment of pediatric lead poisoning. For over 20 years I have been researching, writing, teaching and lecturing on lead poisoning issues. In my medical practice I have also screened and treated several thousand children who have been lead poisoned.
5. I have authored and co-authored numerous articles and papers dealing specifically with pediatric lead poisoning issues, including: Chisolm, Currant, Finbert Hopkins, Lin-Fu, Piomelli, Reigart, Houk, "Increased Lead Absorption and Lead Poisoning

in Young Children. A Statement by the Centers for Disease Control," J. Pediatrics, 87:824 (1975); Penny, Reigart, Loadhold, Taylor, "Variability in Response to Lead Exposure: Demonstration of a Genetic Influence," (Abstract), Pediatric Research, 11:438 (1977); Whitlock, Reigart, Priester, "Lead Poisoning in South Carolina," SCMA Journal, 73:378 (1977); Reigart, "Lead Poisoning," Hospital Medicine, 17:161 16J, 16N, 16P: August, 1981; Reigart, "Future Directions," Childhood Lead Poisoning — Current Perspectives (1988) and Reigart, "Recommendations of the American Academy of Pediatrics on Lead Screening in Children," Proceedings of the First National Conference on Laboratory Issues in Childhood Lead Poisoning Prevention, October 31 - November 2, 1991.

6. Last month I addressed members of the Annual Academy of Pediatrics at its annual conference in San Francisco on the issue of "Childhood Lead Poisoning." This month I lectured at the University of Medicine & Dentistry of New Jersey, School of Osteopathic Medicine, in Atlantic City, New Jersey, on the topic of "Pediatric Lead Poison Prevention Continuing Education for Physicians," and in December I will facilitate a panel at the National Childhood Lead Poisoning Prevention Conference discussing "Medical Management Issues."

7. I serve on numerous committees dedicated to pediatric environmental health issues, including: Chairman, Committee on Environmental Health, American Academy of Pediatrics; Member, Lead Poisoning Education Task Force, Presidents Council on Environmental Quality; Chairman, Centers for Disease Control Childhood Lead-Based Paint Poisoning Advisory Committee; and as a Member of the Pew Charitable Trust's Advisory Committee on Lead Poisoning. In the past I have served on the Centers for

Disease Control Childhood Lead-Based Paint Poisoning ad hoc Advisory Committee and worked with the Environmental Protection Agency as a Medical Consultant for Acute Pesticide Poisoning.

LEAD POISONING AND MEDICAID ELIGIBLE CHILDREN

8. Speaking as a pediatrician, I am someone who has actually worked for many years in lead poisoning prevention out in the field. The situation in my city, Charleston, South Carolina, illustrates the enormity of the lead poisoning problem in young children. In 1972, when we began screening in Charleston, we were detecting that somewhere in the neighborhood of 39% of our children had blood lead levels greater than 40 $\mu\text{g/dL}$. By 1975, I was following over 1,250 children with lead levels greater than 30 $\mu\text{g/dL}$. Today the Centers for Disease Control has decreased the level of concern to 10 $\mu\text{g/dL}$, so that there are many more children out there who are potentially injured by dangerous blood lead levels.

9. Although the American Academy of Pediatrics' revised report is not yet completed, one recommendation that will certainly come from the Committee is that there is a need for more testing. Lead poisoning cannot be detected absent a blood test; a series of questions cannot verify blood lead levels. Nor will a series of questions guarantee that a child will be appropriately classified as low risk. In other words, a child can answer all risk-identifying questions in the negative and still be poisoned.

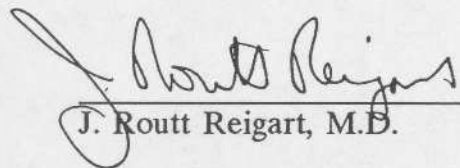
10. Pediatricians obviously want to test for lead using a test that will accurately measure blood lead at the levels of concern announced by the CDC ($\geq 10 \mu\text{g/dL}$.) The erythrocyte protoporphyrin or "EP" test (which really does not measure blood lead levels

at all) is simply incapable of accurately predicting lead levels below 40 µg/dL.

11. In my opinion, as a pediatrician with extensive experience in lead poisoning prevention, Medicaid eligible children represent a high risk group since the environment of low-income children often contains numerous sources of lead exposure.

12. As noted in the CDC's October 1991 statement, "[a]lthough all children are at risk for lead toxicity, poor and minority children are disproportionately affected. Lead exposure is at once a by-product of poverty and a contributor to the cycle that perpetuates and deepens the state of being poor." U.S. Dep't of Health & Human Services ("HHS"), Public Health Service, Centers for Disease Control, Preventing Lead Poisoning in Young Children, 12 (Oct. 1991) ("CDC Statement").

13. The CDC Statement concludes, and I concur, that accurate measurement of blood lead levels can only be determined through the use of blood lead tests. Id. at 41. Accordingly, if the lead testing program is to be effective and if children are to be saved from lead poisoning's devastating effects, it is essential that HHS immediately mandate that States replace the EP test with the blood lead test as the primary lead screening method.


J. Routt Reigart, M.D.

DECL. OF BILL LANNN LEE

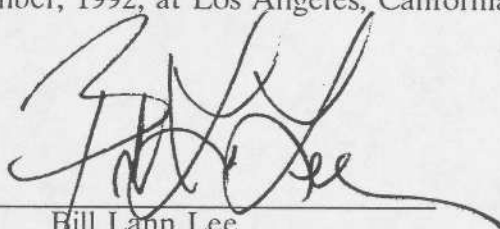
DECLARATION OF BILL LANN LEE

I, BILL LANN LEE, declare and say:

1. I am one of the counsel for the *amici curiae* and the proposed plaintiff-intervenors People United for a Better Oakland, *et al.*
2. Attached hereto as Exhibit A is a true and accurate copy of McElvaine, M.D., Orbach, H.G., Binder, S., Blanksma, L.A., Maes, E.F., & Krieg, R.M., Evaluation of the Erythrocyte Protoporphyrin Test as a Screen for Elevated Blood Lead Levels, 119 *J. of Pediatrics* 548 (1991).
3. Attached hereto as Exhibit B is a true and accurate copy of Report of the House Budget Comm. on H.R. 3299, 101 Cong., 1st Sess. (September 20, 1989), *reprinted in Medicare and Medicaid Guide (CCH)*, Extra Edition No. 596 (October 5, 1989) (pp. 398 - 410.

I declare under penalty of perjury that the foregoing is true and correct.

Executed the 23rd day of November, 1992, at Los Angeles, California.



Bill Lann Lee

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LEE
EXHIBIT A

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Evaluation of the erythrocyte protoporphyrin test as a screen for elevated blood lead levels

Michael D. McElvaine, DVM, MPH, Hyman G. Orbach, PhD, Sue Binder, MD, Lorry A. Blanksma, PhD, Edmond F. Maes, PhD, and Richard M. Krieg, PhD

From the Division of Environmental Hazards and Health Effects, Center for Environmental Health and Injury Control, Centers for Disease Control, Atlanta, Georgia, and the City of Chicago Department of Health, Chicago, Illinois

To study the effect of lowering the definition of an elevated blood lead level on the performance of the erythrocyte protoporphyrin screening test and the number of children who would require follow-up, we collected laboratory data from a screening program. The estimated sensitivity of an erythrocyte protoporphyrin level $\geq 35 \mu\text{g/dl}$ for identifying children with elevated blood lead levels was 73% when we used 1985 Centers for Disease Control guidelines (elevated blood lead level $\geq 25 \mu\text{g/dl}$). Eight percent of the tests showed positive results. When we redefined an elevated blood lead level as $\geq 15 \mu\text{g/dl}$, the sensitivity estimate was reduced to 37% and the number of positive test results increased fourfold. (J PEDIATR 1991;119:548-50)

The most recent statement of the Centers for Disease Control (CDC), published in 1985,¹ defined an elevated blood lead level in children as $\geq 25 \mu\text{g/dl}$ ($1.21 \mu\text{mol/L}$), a threshold exceeded by an estimated 250,000 children in 1984.² The 1985 CDC statement recommended screening for elevated BPb levels by measuring erythrocyte protoporphyrin levels and considering an EP level of $\geq 35 \mu\text{g/dl}$ ($0.62 \mu\text{mol/L}$) as a positive screening test result. EP is a precursor of heme that accumulates when lead interferes with normal heme synthesis.

Few data are available on how well the EP test, with a positive test result defined as $\geq 35 \mu\text{g/dl}$, would perform in identifying children with BPb levels of $\geq 25 \mu\text{g/dl}$ in actual clinic screenings. Among children tested in the Sec-

ond National Health and Nutrition Examination Survey, the sensitivity of the EP test in detecting elevated BPb levels was 26% (unpublished data). For low-income black children in NHANES II who lived in the central city (i.e., were at high risk for lead poisoning), the sensitivity estimate was 42%. A more recent survey of urban children in a community with characteristics associated with high rates of lead poisoning, Oakland, Calif., yielded a sensitivity estimate of 50%.³

BPb	Blood lead
EP	Erythrocyte protoporphyrin
NHANES II	Second National Health and Nutrition Examination Survey

Presented in part at the 1990 Epidemic Intelligence Service Conference, April 23-27, 1990, Atlanta, Ga.

The authors of this article are solely responsible for all analysis and interpretation of the data presented here.

Submitted for publication April 3, 1991; accepted June 20, 1991.

Reprint requests: Michael D. McElvaine, DVM, MPH, Division of Environmental Hazards and Health Effects, Center for Environmental Health and Injury Control, Centers for Disease Control, Mail Stop F-28, 1600 Clifton Rd. NE, Atlanta, GA 30333.

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Recent research has shown that significant adverse effects result from BPb levels of 10 to 15 $\mu\text{g/dl}$ or even lower,^{2,4} and the CDC is reconsidering its definition of an elevated BPb level. There is poor correlation between BPb and EP when BPb levels are less than 18 to 20 $\mu\text{g/dl}$.^{5,6} Accordingly, to assess how lowering the definition of an elevated BPb level would affect EP test performance and the numbers of children needing follow-up care, we analyzed laboratory data from a childhood lead poisoning prevention

Table I. Population blood lead and erythrocyte protoporphyrin values by age group, Chicago, Ill., 1988-1989

Age group (yr)	n	Mean BPb (range) ($\mu\text{g/dl}$)	Mean EP (range) ($\mu\text{g/dl}$)	Children with BPb $\geq 25 \mu\text{g/dl}$ (%)
<2	211	12.1 (3-83)	29.8 (2-146)	4
2-3	219	14.8 (2-49)	31.8 (1-234)	12
4-5	147	13.4 (3-50)	28.1 (4-214)	8
TOTAL	577	13.4 (2-83)	30.1 (1-234)	8

program that screens children at high risk for lead poisoning.

METHODS

The City of Chicago Department of Health Division of Laboratories measures EP and BPb levels in about 50,000 venous blood samples each year. The EP level is measured by hematofluorometry.⁷ The BPb level is measured by atomic absorption spectrophotometry⁸ or by anodic stripping voltammetry. This laboratory is one of six reference laboratories used by the proficiency testing program run jointly by the CDC, the Health Resources and Services Administration, and the University of Wisconsin to establish target values for BPb testing and is also a regular participant in the proficiency testing program for EP.

The population that we studied consisted of children who visited city-run clinics that operate in low-income neighborhoods. Every child received lead screening as part of the routine health program. In addition, the city also provided mobile screening services to underserved areas.

Laboratory and demographic data were abstracted from a 2% systematic sample of laboratory test records ($n = 1057$) for the period of Sept. 1, 1988, through Aug. 31, 1989. A two-digit random number was used as a starting point; then every fiftieth record was selected. From this sample, we included all children from 6 months through 5 years of age who were tested at the city's screening centers. For the four children who appeared twice in the sample, only the first set of results was used. A total of 577 records met these criteria and were included for analysis.

RESULTS

The study population was 53% male. The ethnic distribution was 68% black, 29% Hispanic, and 3% other. The highest mean BPb and EP levels were in the 2- and 3-year-old age group (Table I).

Performance of the EP test to identify various levels of BPb elevation is shown in Table II. Twenty-three percent of the EP tests were $\geq 35 \mu\text{g/dl}$. Using the 1985 CDC definition of an elevated BPb level, we estimated the sensitivity to be 73%. If an elevated BPb level was redefined as ≥ 15

Table II. Performance of the erythrocyte protoporphyrin test to identify samples with elevated blood lead levels, with an EP level $\geq 35 \mu\text{g/dl}$ used as a screening cutoff point, Chicago, Ill., 1988-1989

Definition of elevated BPb ($\mu\text{g/dl}$)	Children with elevated BPb ($n = 577$) (%)	Sensitivity (%) (95% CI)	Specificity (%) (95% CI)
≥ 25	8	73 (60-86)	81 (78-85)
≥ 20	15	49 (39-60)	82 (78-85)
≥ 15	32	37 (30-44)	84 (80-87)
≥ 10	66	26 (21-30)	82 (77-88)

CI, 95% Confidence intervals for estimated values.

$\mu\text{g/dl}$, the proportion of samples with elevated BPb levels increased four times and the sensitivity of the EP screen was reduced by half to 37%. When a definition of $\geq 10 \mu\text{g/dl}$ was used, the proportion with elevated BPb levels increased to 66% and the sensitivity decreased further. For this sample population, the EP test (at $\geq 35 \mu\text{g/dl}$) identified all subjects with BPb levels of $35 \mu\text{g/dl}$ or greater.

DISCUSSION

The results show that the sensitivity of the EP test in this high-risk population (73%) is higher than that found from NHANES II overall (26%), from a high-risk subset of NHANES II (42%) and in Oakland, Calif. (50%).³ The higher sensitivity estimates seen in the Chicago data may be the result of several factors. Among subjects with elevated BPb levels, those levels were relatively higher in Chicago than in the other populations. In NHANES II, 55% of the children with elevated BPb levels had levels in the 25 to 29 $\mu\text{g/dl}$ range. In Chicago, of the tests showing elevated BPb levels 67% showed levels of $\geq 30 \mu\text{g/dl}$. Because EP increases exponentially with increasing BPb values, sensitivity is greater for children with higher BPb levels.^{5,6}

Iron deficiency also can cause an increase in the EP level in children.^{9,10} The apparent sensitivity of the EP test for identifying elevated BPb levels should be greater in a population in which iron deficiency is more prevalent. Iron deficiency and lead poisoning often coexist.⁹ The prevalence

of iron deficiency in the Chicago children (a largely minority, inner-city population) may have been higher than in the NHANES II children (a representative sample of U.S. children); unfortunately, in this study, no data were available to assess the iron status of the Chicago children.

These data indicate that the EP test performed acceptably as a screening test for BPb levels $\geq 25 \mu\text{g/dl}$ in the high-risk Chicago population but that when the definition of an elevated BPb level was lowered to $\geq 15 \mu\text{g/dl}$, the sensitivity of the EP test decreased to 37%. This decrease makes it a less useful screening test for BPb levels in the 10 to $24 \mu\text{g/dl}$ range.

The EP test has several practical advantages: it is inexpensive and easy to perform, and it can identify iron deficiency in children.⁹ The sensitivity of the EP test is also good for identifying children with very high BPb levels, those who most urgently need follow-up. For identification of the children with BPb levels of 10 to $24 \mu\text{g/dl}$, another screening method, probably BPb measurement, will be needed. Such screening would be facilitated by the development of cheaper, easier-to-use, portable instruments for measuring BPb lead levels accurately at the new, lower levels of concern.

Information on the Childhood Lead Screening Program was provided by Charles R. Catania, City of Chicago Department of Health. Thomas D. Matte, MD, and Jeffrey J. Sacks, MD, of the Centers for Disease Control, provided valuable editorial assistance in the preparation of this manuscript. The NHANES II data tapes were supplied by the National Center for Health Statistics. Data management was assisted by Mary Boyd, of CDC, and Nance Dulaj, of the City of Chicago Department of Health Laboratory.

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EXHIBIT B

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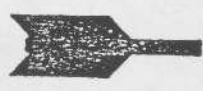
—EXTRA EDITION—

Omnibus Budget Reconciliation Act of 1989

H. R. 3299

Report of the
House Budget Committee
September 20, 1989

Explanation of the Energy and Commerce
and Ways and Means Committees
Affecting Medicare-Medicaid Programs

 ***CCH Special 1*** 

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cient time for a mother to make the transition from welfare to a job that offers health insurance coverage for her and her children.

To further encourage welfare families to work, the Committee bill would allow the States, at their option, to extend the current 12-month transitional coverage period for an additional 12 months or 3, 6, or 9 months, as the State elects. Thus, a State could offer a working welfare family a total of 24 months of transitional Medicaid coverage (12 mandatory, 12 optional). Under the bill, the structure of the current mandatory benefit would remain unchanged. Thus, States could, at their option, impose the same income-related premium during this optional 12-month period that they are allowed to impose during the 2nd mandatory 6-month period. The Committee bill would also repeal the sunset.

The Committee bill would also make some technical corrections to current law. It clarifies that Medicaid transition coverage terminates at the close of the first month in which the family ceases to include a child, whether or not the child is a dependent child under part A of Title IV, or would be if needy. The Committee bill also clarifies that families who, prior to April 1, 1990, are receiving Medicaid extension coverage under the current law 9-month provision are entitled to continue receiving this extension coverage after that date until their 9-month coverage period expires.

Section 4213—Early and periodic screening, diagnostic, and treatment services

(a) *In general.*—Under current law, States are required to offer early and periodic screening, diagnostic, and treatment (EPSDT) services to children under age 21. States are required to inform all Medicaid-eligible children of the availability of EPSDT services, to provide (or arrange for the provision of) screening services in all cases when they are requested, and, to arrange for (directly or through referral to appropriate agencies or providers) corrective treatment for which the child health screening indicates a need.

The EPSDT benefit is, in effect, the nation's largest preventive health program for children. Each State must provide, at a minimum, the following EPSDT services: assessments of health, developmental, and nutritional status; unclothed physical examinations; immunizations appropriate for age and health history; appropriate vision, hearing, and laboratory tests; dental screening furnished by direct referrals to dentists, beginning at age 3; and treatment for vision, hearing, and dental services found necessary by the screening. These services are available to children under EPSDT even if they are not available to other Medicaid beneficiaries under the State's plan.

The EPSDT benefit is not currently defined in statute. In the view of the Committee, as Medicaid coverage of poor children expands, both under current law and under the Committee bill, the EPSDT benefit will become even more important to the health status of children in this country. The Committee bill would therefore define the EPSDT benefit in statute to include four distinct elements: (1) screening services, (2) vision services, (3) dental services, and (4) hearing services. Each of these service elements would have its own periodicity schedule that meets reasonable practice standards. These items and services must be covered for children

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even if, under the State Medicaid plan, they are not offered to other groups of program beneficiaries.

Under the Committee bill, screening services must, at a minimum, include (1) a comprehensive health and developmental history (including assessment of both physical and mental health development), (2) a comprehensive unclothed physical exam, (3) appropriate immunizations according to age and health history, (4) laboratory tests (including blood lead level assessment appropriate for age and risk factors), and (5) health education (including anticipatory guidance). The Committee emphasizes that anticipatory guidance to the child (or the child's parent or guardian) is a mandatory element of any adequate EPSDT assessment. Anticipatory guidance includes health education and counselling to both parents and children.

Under the Committee bill, vision services must, at a minimum, include diagnosis and treatment for defects in vision, including eyeglasses. Dental services must, at a minimum, include relief of pain and infections, restoration of teeth, and maintenance of dental health. Hearing services must, at a minimum, include diagnosis and treatment for defects in hearing, including the provision of hearing aids. While States may use prior authorization and other utilization controls to ensure that treatment services are medically necessary, these controls must be consistent with the preventive thrust of the EPSDT benefit. For example, States may not limit dental care to emergency services only. *Mitchell v. Johnston*, 701 F.2d 337 (5th Cir. 1983).

The Committee bill also clarifies the periodic nature of EPSDT services. With respect to screening services, the bill requires that they be provided at intervals which meet reasonable standards of medical and dental practice, as determined by the State after consultation with recognized medical and dental organizations. The Committee intends that these health examinations be provided at intervals that are no greater than those described for well-child care in the "Guidelines for Health Supervision" (1981) of the American Academy of Pediatrics. The Committee is informed that some States use periodicity schedules for medical examinations to govern the frequency with which children may receive dental examinations. The Committee intends that, among older children, dental examinations occur with greater frequency than is the case with physical examinations.

The Committee bill also requires States to provide screening services at intervals other than those identified in their basic periodicity schedule, when there are indications that it is medically necessary to determine whether a child has a physical or mental illness or condition that may require further assessment, diagnosis, or treatment. These interperiodic screening examinations may occur even in the case of children whose physical, mental, or developmental illnesses or conditions have already been diagnosed, if there are indications that the illness or condition may have become more severe or has changed sufficiently, so that further examination is medically necessary. The Committee emphasizes that the determination of whether an interperiodic screening is medically necessary may be made by a health, developmental, or educational professional who comes into contact with a child outside of the

health care system (e.g., State early intervention or special education programs, Head Start and day care programs, WIC and other nutritional assistance programs). As long as the child is referred to an EPSDT provider, the child would be entitled to an interperiodic health assessment (or dental, vision, or hearing assessment) or treatment services covered under the State plan.

These same considerations apply with respect to vision, dental, and hearing services, all of which must be provided when indicated as medically necessary to determine the existence of suspected illnesses or conditions. For example, assume that a child is screened at age 5 according to a State's periodicity schedule and is found to have no abnormalities. At age six, the child is referred to the school nurse by a teacher who suspects the child of having a vision problem. Under the Committee bill, the child can—and should—be referred at that point to a qualified provider of vision care for full diagnostic and treatment services, and the State must make payment for those services, even though the next regular vision exam under the State's periodicity schedule does not occur until age 7.

While States may, at their option, impose prior authorization requirements on treatment services, the Committee intends that, consistent with the preventive thrust of the EPSDT benefit, both the regular periodic screening services and the interperiodic screening services be provided without prior authorization.

The Committee notes that Medicaid-eligible children are entitled to EPSDT benefits even if they are enrolled in a health maintenance organization, prepaid health plan, or other managed care provider. The Committee expects that States will not contract with a managed care provider unless the provider demonstrates that it has the capacity (whether through its own employees or by contract) to deliver the full array of items and services contained in the EPSDT benefit. The Committee further expects that, in setting payment rates for managed care providers, the States will make available the resources necessary to conduct the required periodic and interperiodic screenings and to provide the required diagnostic and screening services.

The Committee bill clarifies that States are without authority to restrict the classes of qualified providers that may participate in the EPSDT program. Providers that meet the professional qualifications required under State law to provide an EPSDT screening, diagnostic, or treatment service must be permitted to participate in the program even if they deliver services in school settings, and even if they are qualified to deliver only one of the items or services in the EPSDT benefit.

(b) Report on the provision of EPSDT.—In order to assess the effectiveness of State EPSDT programs in reaching eligible children, the Committee bill would require the States to report annually to the Secretary, in a uniform form and manner established by the Secretary, the following information, broken down by age group and by basis of eligibility for Medicaid: (1) the number of children receiving child health screening services; (2) the number of children referred for corrective treatment (the need for which is disclosed by the screening); and (3) the number of children receiving dental services. These reports would be due April 1 of each year (beginning with April 1, 1991) and would apply to services provided

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or special education. WIC and other child is referred to an interperiodic assessment) or

to vision, dental, and when indicated of suspected illness child is screened and is found to be referred to the of having a vision—and should—be vision care for full must make pay-ular vision exam occur until age 7. authorization re-ntends that, con-benefit, both the eriodic screening

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during the Federal fiscal year ending the previous September 30 (beginning with FY 1990).

Section 4214—Extension of payment provisions for medically necessary services in disproportionate share hospitals

(a) *Coverage of medically necessary services for children.*—Under current law, States may impose reasonable limits on the amount, duration, and scope of covered services. However, effective July 1, 1989, States are prohibited from imposing any fixed durational limit on Medicaid coverage of medically necessary inpatient hospital services provided to infants under age 1 by disproportionate share hospitals. As of January, 1989, according to the National Association of Children's Hospitals and Related Institutions, 12 States imposed durational limits on inpatient hospital services for children (Alabama, Alaska, Arkansas, Florida, Kentucky, Louisiana, Mississippi, Missouri, Oregon, Tennessee, Texas, and West Virginia).

The purpose of the current law exception to fixed durational limits is to prohibit States from using arbitrary length of stay limitations (e.g., 20 days per year) to reduce payments for medically necessary services provided by hospitals, including many public and children's hospitals, that serve a disproportionate number of low-income patients. The Committee bill would extend this current law prohibition to any fixed durational limits on payment for inpatient services provided to children under age 18 by disproportionate share hospitals. The requirement is effective for inpatient hospital services furnished on or after July 1, 1990.

(b) *Assuring adequate payment for inpatient hospital services for children in disproportionate share hospitals.*—Under current law, States may reimburse hospitals for inpatient services on a prospective basis. If they choose to do so, States must, effective July 1, 1989, provide for an outlier adjustment in payment amounts for medically necessary inpatient services provided by disproportionate share hospitals involving exceptionally high costs or exceptionally long lengths of stay for infants under 1 year of age. According to the National Association of Children's Hospitals and Related Institutions, as of January, 1989, a total of 44 States pay for inpatient hospital services on a prospective basis; only 17 of these do not make outlier adjustments for high cost or long-stay cases (Alabama, Alaska, California, Colorado, Connecticut, D.C., Florida, Kentucky, Mississippi, Missouri, Nevada, New Hampshire, New Mexico, Oklahoma, Tennessee, Texas, and Washington).

The Committee bill would extend this current law requirement to cases involving children from age 1 up to age 18. States that pay for inpatient hospital services on a prospective basis would be required to submit to the Secretary, no later than April 1, 1990, a State plan amendment that provides for an outlier adjustment in payment amounts for medically necessary inpatient services provided by disproportionate share hospitals after July 1, 1990, involving exceptionally high costs or exceptionally long lengths of stay for children age 1 up to age 18.

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