The Hour of Lead

A brief history of lead poisoning in the United States over the past century and of efforts by the lead industry to delay regulation

Environmental Defense Fund

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After a great pain, a formal feeling comes—a
The Nerves sit ceremonious, like Tombs—a
The stiff Heart questions was it He, that bore,
And Yesterday, or Centuries before?
The Feet, mechanical, go round—a
of Ground, or Air, or Ought—a
A Wooden way
Regardless grown,
A Quartz contentment, like a stone—a
This is the Hour of Lead—a
Remembered, if outlived,
As Freezing persons, recollect the Snow—a
First—Chill—a then Stupor—a then the letting go—a

Emily Dickinson, The Complete Poems of Emily Dickinson, Johnson, TH, Ed. Boston: Little, Brown and Company, p.162. Though the poet was describing emotions rather than lead poisoning, the poem aptly describes some of the symptoms of lead intoxication.
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During the period of regulation, there was consolidation in the industry although smelting and refining ownership remained stable. The LIA continued its opposition to further regulation.
INTRODUCTION

In very small quantities, lead poisons the immature brain. Children with moderate long-term exposures -- but no immediate symptoms -- show reduced short term memory, delayed reaction time, reduced ability to concentrate, and diminished scores on IQ tests.1 Although no comprehensive data are currently available, the federal government estimates that about 15 percent of all U.S. preschoolers now have unacceptable levels of lead in their blood, levels that cause subtle but significant impairment of learning skills.2

Decades of use of lead in paint, gasoline, plumbing systems, and myriad other products have left these high blood lead levels as their poisonous legacy. Exposures most often result from dust contaminated by lead-based paint -- some of which contained up to 50% lead by dry weight in the early decades of this century.3 Nearly three quarters of U.S. homes constructed before 1980 contain some lead paint;4 an estimated three million tons of lead still coats the walls and woodwork of American homes.5

How did the lead get there?

For many decades, firms that made lead and sold lead products aggressively promoted the use of lead-based paint for the interiors and exteriors of homes. At that time the public perceived "White lead" -- which could be tinted a variety of colors -- to be the best protective coating, and it was available in abundant supply. Families used lead paint not only on their walls, but on their cribs, toys, woodwork, and furniture as well.

Infants and toddlers routinely place things in their mouths as a part of normal development. It is not surprising then that cases of lead poisoning in children caused by ingestion of lead paint began to appear in the English language medical literature before the turn of the century. In 1897, Australian researchers identified lead in paint as the cause of a "Toxicity of Habitation," and the first

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U.S. case was reported in 1914. By 1917, U.S. medical authorities had established that childhood lead poisoning from lead paint was a common problem. "A child," wrote a medical commentator in 1924, "lives in a lead world."7

Most lead paint then in use was based on lead carbonate, known as white lead. The product was manufactured by subjecting lead to corrosion, yielding a white powder. After some processing, the powder was sold as "dry white leads" to paint manufacturers, or ground with linseed oil and sold the product as paint.

After 1922, another important source of domestic lead wafted into the child's world: lead from automobile exhaust. When scientists discovered that a small amount of tetraethyl lead added to automobile fuel significantly improved performance and efficiency, the lead industry launched a campaign of medical research and political arm-twisting to assure that lead, this "Gift of God," would not be restricted.8 Despite warnings from a Yale University physiologist that poisonous dust from exhaust fumes would fill U.S. cities, the industry view prevailed. During the next half-century, about 7 million tons of lead churned into the air from automobiles9 while industry-funded medical experts asserted that lead exposure was "harmless" and "normal."10

Even though most of the industrialized world moved to control white lead paint by the turn of the century and curtailed its use soon after World War I, U.S. policymakers ignored medical and industrial labor reports from home and abroad. The lead industry proceeded to gain control over the conduct of medical research, the setting of public health priorities, and the dissemination of information to warn the public. Through a trade association, the nation's lead producers, refiners, and manufacturers disputed claims of lead poisoning and worked actively to discount such reports and thwart regulation.11 When competition from non-toxic paints became a problem in the 1930s, the association by-passed the marketplace and worked to assure that lead paint would be required in public housing projects and other public buildings.

The sheer weight of dead bodies of acutely lead poisoned children began to stir pediatricians and legislators into action in the 1950s, but federal regulation of lead paint was another two decades in coming.12 Today, despite significant restrictions on use of lead in paint and automobile fuel, the child still lives in a lead

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6 See section 2, below.
10 See section 3 below.
11 See section 5 below.
12 See section 7 below.
1. EARLY HISTORY OF THE LEAD INDUSTRY

Romance and adventure fill the lore of American mining and the record of industrial achievement. The trademarks, slogans and mythologies about lead perpetuated an image that grew out of the opening of the West.13 Mined steadily up and down the eastern seaboard from 1621, lead created an important local industry providing bullets, paint and pipe. Because of its maliceability, it was prized as a conduit for water. In addition, the industry claimed that lead surpassed all other substances as paint.

Although various methods for producing white lead were known from Roman times — Pliny described one in 77 A.D. — the particular process favored by manufacturers in this country was developed in Holland late in the 18th century. This process became known as the "Dutch Process," hence the famous brand name "Dutch Boy."14 The first American white lead business was established in 1752 in Philadelphia, followed in 1804 by the first white lead factory.15 White lead interests became a large, powerful force in the nation's economy.

Unimagined mineral wealth lay beyond the Appalachian frontier in Missouri where outcrops of galena, or lead-sulphate ore, gave frontiersmen and Indians a ready supply of lead. These deposits were first discovered by French explorers who made their way south on the Mississippi and began extracting lead late in the 17th century. The lead mines that were developed in Missouri after the 1820s proved to be extraordinary, and the concentration of lead-rich ores in a few geographic locations made it possible for ownership of the means of producing and marketing lead — mines, smelters and refineries — to be concentrated in a handful of large corporations that predominated in two sections of the state.16

In southeastern Missouri, the largest lead-producing district in the United States, the St. Joseph Lead Co. dominated the region for well over a century. The mines in St. Francois County had operated since the early 18th century, and became consolidated in the 19th century under St. Joe following its incorporation in 1864.17 After 1891, St. Joe's refinery at Herculaneum produced much of the nation's lead. Located about 50 miles southeast of St. Louis, the complex of mines, mills and refineries helped make that city into an industrial center. Today, the complex still operates, though the company is now called Doe Run.18

The second major lead region in Missouri was known as the Tri-State Region or the Joplin District, a 2,000 square mile area covering southwestern Missouri, southeastern Kansas, and northeastern Oklahoma. Eagle-Picher Mining Co. dominated the area. The firm organized in 188819 and was re-named the Eagle-Picher Mining and Smelting Co in 1919.20

The National Lead Company also had mines in Picher, Missouri and Baxter Springs, Kansas. The company had its origins in one of the great trusts, the National Lead Trust, capitalized at $90,000,000 in 1887.21 This trust was dissolved in 1891 and the National Lead Company formed, combining numerous smaller companies involved in the production, refining and manufacture of lead products.22 Until 1972, when a major reorganization occurred, the National Lead Co. dominated the manufacture and sale of lead products in the United States.23

With the opening of the West after the Civil War, astonishing discoveries of minerals in the Rockies thrust the industry into a span of development. In striking contrast to the stereotype of conestoga wagons creaking their way into the western sunset, mining historians boast that as early as 1847 the sight of Prairie Schooners lurching eastward from lead fields in Missouri and Illinois stimulated agricultural settlers to follow the trails west.24 After the Civil War, important lead deposits were found in Colorado, Utah, Idaho and Montana. Unlike Missouri lead, most western lead occurred in argentiferous ore, so that with specialized refining equipment, silver and other valuable metals could be recovered as well. The final ring of the hammer at Promontory Point, Utah, in 1869 signaled not only an historic railroad accomplishment, but the fact that ore and ingots could be shipped readily by rail to manufacturing centers and markets on both coasts and to numerous points in between.

By the turn of the century, a quartet formed by The American Smelting and Refining Company, The National Lead Co., The Anaconda Company and the Heca Mining Company controlled the mining and manufacturing of lead, copper, silver, gold and zinc in the Far West. These firms, along with Eagle-Picher and St. Joe in Missouri, maintained ownership of mines, mills, smelters, and refineries until the nineteen sixties.

The American Smelting and Refining Co. was founded in 1899 in New Jersey. The Guggenheim family became the dominant

17 After a Century of Operation, St. Joseph Lead Co. Plans for the Next, Engineering and Mining Journal 165:27, 1944.
18 See Section 8 below.
20 Mines Register, New York, 1946, p.106.
influence in American Smelting and Refining Co. in 1901 when the company "assumed control of the lead market, fixing the price both for producers and consumers, and regulating output by agreement with the large producers and by adjustments of its smelting charges in connection with small producers." In 1906, Guggenheim interests secured control of the National Lead Co. and the United Lead Co., thus bringing the major part of the lead-consuming industry of the United States into direct affiliation with the American Smelting and Refining Co. By 1906, the American Smelting and Refining Co. controlled about 85% of the white lead production.26

In Montana, the Anaconda Copper Mining Company, organized in 1895, held a large tract near Butte, and for most of the century produced copper, iron, aluminum and other minerals as well as lead and zinc. And in Idaho, the Hecla Mining Co., established in 1898, controlled properties and mills in Shoshone County that produced silver, lead and zinc. Hecla also held an interest in the United States Smelting and Refining Co., which owned a refinery in Idaho.27

Two major markets for white lead paint developed rapidly after the Civil War. At first manufacturers sold the paint to be used on the exteriors of the new farms, stores, and homes built in the wake of the expanding frontier. Later, the growth of large urban areas with tenements stimulated the use of white lead as an interior paint.

25 Ingalls WR, op. cit., p.35
26 Ingalls WR, op. cit., p.35; see also Holley, CD, The Lead and Zinc Pigments, New York, John Wiley & Sons, 1909, p.30
27 Mining International Yearbook, 1925, p.90.
28 Mines Register, 1946, New York, 1946, p.139.

2. "TOXICITY OF HABITATION"

During the late 1890s and early years of the new century, physicians in Queensland, Australia, were perplexed and troubled by what was described in 1897 by A.J. Turner as a "Toxicity of Habitation," a mysterious disease of children.29 The key diagnostic signs were paralysis, notably "wrist drop" (inability to straighten the wrist); abdominal pains and pains in the limbs; ocular neuritis (inflammation, pain and paralysis of the optic); and convulsions. Chronic lead poisoning was the agreed-upon diagnosis in 76 cases at the Children's Hospital in Brisbane, but the source of this "nerve poison" eluded researchers. Subsequent articles recounting the quest for the source of the lead convey both urgency and frustration.

Then, in 1904, J. L. Gibson, an ophthalmic surgeon at Brisbane Hospital for Sick Children, reported in the first of several papers that lead paint had been identified as the source.30 The climate and regional architecture -- with large, painted verandas -- created an environment which increased the likelihood that dry, powdery, sweet-tasting paint would adhere to children's hot, sticky, frequently licked hands. The theory that lead-containing paint was the cause precipitated considerable debate, and Gibson and his colleagues published several articles after 1904 detailing the manner in which they had eliminated all other sources.31

They also demonstrated that the powdery paint from the verandas was tested by reliable government chemists and found to be a soluble carbonate of lead.32 After years of frustration Gibson seemed quite pleased in 1922 to have the Council of the Queensland Branch of the British Medical Association endorse proposed legislation to prohibit the use of lead paint on veranda railings and "outside surfaces within reach of children's fingers." While he expected opposition from "powerful monied interests," he was outraged to find that physicians could be recruited to the opposition.

From 1904, children were identified in English-language medical articles as being at risk of serious poisoning from ingesting lead pigment. Altogether, Gibson reported 299 cases of lead poisoning:

the case reported in 1914. Of another fatal case Blackfan wrote, "The father stated that the child would gnaw any painted article, and that he and his brother had repeatedly ruined a set of parlor furniture by eating the paint from it." The paper emphasized the particular vulnerability of children and the significance of convulsions as an indication of the severity of the lead poisoning. Blackfan concluded: "I would urge that energetic prophylactic measures be taken with children who habitually eat painted articles in order to guard against the development of lead poisoning... In all patients with convulsions in which the etiological factor is not clear, lead should be suspected." Another report of a child fatality from eating lead paint came from New Orleans in 1920.38

By the mid-twenties, the literature moved from case reports to commentary and warnings by authoritative physicians in textbooks, and to widely reported forums. For example, Abt's 1923 Textbook Pediatrics39 included the observation that "Poisoning with lead is probably more common in children than generally supposed." Abt states that "eating the enamel of iron beds and licking painted walls" are among the ways children are exposed. The eighth edition of Holt's Diseases of Infancy and Childhood 40 in 1923 also reported seven deaths out of eight cases: "The poisoning was caused in each instance by the child's nibbling and swallowing the paint from his crib or furniture." Holt himself underscored the seriousness of the phenomenon by publishing an article on lead poisoning in infancy.41 He presented a case of lead poisoning in a nursing infant due to lead acetate in a breast ointment, but described other sources of lead, including paint, boards, milk that had stood in lead containers, and medication.

The dangers of non-industrial sources of lead were highlighted at the fifth annual meeting of the Association for Research in Nervous and Mental Disease at New York City's Commodore Hotel and reported in The New York Times on December 31, 1924.42 The Times reported international lead poisoning incidents, and described the work of Carl V. Weller at the University of Michigan. Weller "told of a child who became a victim of lead poisoning after gnawing the paint from his crib." Weller emphasized that "lead poisoning continues to hold its place in the first rank of industrial hazards and to find its victims through the most unexpected sources among the non-industrial population as well."43 Weller attributed this to "phenomenal" growth of new industries using lead.

38 Strong RA, Meningitis Caused by Lead Poisoning, in a Child of Nineteen Months, Archives of Pediatrics 37:532-537, 1920
39 Abt AI, Pediatrics, W.B. Saunders Co. VII:246-249, 1923
42 The New York Times, December 31, 1924, p.42
43 Weller CV, Some Clinical Aspects of Lead Meningo-Encephalopathy, Archives of Clinical Medicine 23:604-613, 1925
Weller, a pathologist, was interested in the cerebral effects of lead poisoning and noted in a 1925 study that "Non-industrial lead poisoning has provided many of the cases of lead meningo-encephalopathy and the diagnosis is much more apt to be missed in these cases because the etiology is unsuspected." Giving examples of non-industrial lead poisoning, he cited Stewart's bakers' buns, carpet weavers, cosmetics and inks, and lead paints.

Shortly afterwards, in 1926, C.F. McKann at Harvard Medical School reported seventeen cases of lead poisoning and observed that the eponymous lead sickness, characterized by headaches, listlessness, and drowsiness, was frequently associated with picric acid, an unnatural craving for articles of food. Pica, from the Latin word magpie (a bird that eats anything and everything) was first associated with lead paint poisoning by Ruddock in 1924. Less than ten years after Thomas and Blackfan stated that lead poisoning in children was "not very common," McKann declared that "lead poisoning is of relatively frequent occurrence in children."44

By 1926, non-industrial exposure to lead was regarded as a serious hazard to infants and children, and eating lead paint from cribs, furniture, toys and woodwork was identified as a major source of the poison in 15 separate U.S. medical publications including journal articles, books, and textbooks.45

Underlying these U.S. case reports was substantial world literature indicating that lead had a particularly pernicious effect on the brain. The first authoritative treatise on lead poisoning was prepared by Tanquerel des Planches and published in France in 1838 and in the United States in 1842.46 It clearly associated lead poisoning with encephalopathy, an inflammation of the brain character


ized by violent seizures, coma, and death; his description of encephalopathy endured for decades.

Tanquerel was clearly frustrated by the difficulties he encountered in tracking lead's pernicious attack on the central nervous system. According to Tanquerel, Stockhausen (1666) and many others in the 17th century observed "cerebral affections in lead colic."47 The French physician described the passage of lead to the brain but seemed frustrated by limitations of "present state of science regarding intellectual lesions... The nature of the impression of lead upon the encephalitic edema would require investigation."48 Concerning the source of exposure, Tanquerel noted that "The use of playthings for children, colored with preparations of lead, intended to be placed in the mouth, such as trumpets, have caused serious diseases, among others colic."49 An eloquent and poignant appendix by Tanquerel's translator, Samuel L. Dana, M.D., of Lowell, Massachusetts, underscored the special vulnerability of children and warned about lead in water pipes.50

Decades later, observations by the authoritative Sir Thomas Oliver further clarified the insidious effects of lead on women who worked in white lead factories or potteries and their children. While his primary interest was occupational exposures, he expressed concern about the "direct transmission of lead as a poison to affected mothers to the offspring."51 His description of children in homes where pottery was a cottage industry sounded a clear, sharp warning that exposure to lead produced serious mental and physical deficits. Referring to a Dr. Prendergast of...
Hanley, Staffordshire, who had worked in the potteries, he reported that "Dr. Prendergast is of the opinion that the children of lead-poisoned potters do not grow up into capable men and women like other children, but that they are handicapped in their start in life, and that subsequently many of them exhibit signs of mental as well as physical deterioration."

Oliver saw the worst effects of lead poisoning in Hungary, and he described incredible familial devastation in sections of Hungary where pottery making was a cottage industry. At the close of this passage Oliver stated again "it is the repeated entrance of minute quantities of lead into the body and not of one or two large doses, which give rise to the worst types of plumbism." The contention of the paper is that there is a cumulative effect and "there are signs that the development of child life is to some extent being interfered with."52

In Britain, lead poisoning became a cause celebre, especially after vivid depictions of the suffering of women working in white lead factories from Charles Dickens53 and George Bernard Shaw.4 Both helped focus concern on white lead, concern that eventually prompted formation of the British White Lead Commission to examine the manufacture and use of white lead.54 Special rules for protection from occupational exposure in factories were issued in 1899 and strengthened in 1901.55 In Switzerland, an attempt to restrict the use of white lead failed in 1904,56 but Belgium prohibited the use of dry white lead (a major source of dust) in 1909.57 Germany and Austria also regulated the use of white lead, the latter explicitly banning white lead in domestic interiors.58

Even before World War I, international labor organizations were preparing a worldwide prohibition of white lead, and one of the provisions of the Treaty of Versailles called for the International Labour Organization to examine industrial working conditions in general.59 Lead poisoning was a high priority item and, as a result, the International Labour Organization in 1921 began a process to encourage national governments to ratify a ban. Through the 1920s many European nations ratified the ban on the use of white lead.60 In the United States the National Paint, Oil and Varnish Association successfully opposed the ban.61

Lucid and compelling accounts of lead's toll on children were etched clearly in medical and popular literature well before World War I, with special concern for the effects on children's mental capacity. In the United States, lead and paint interests simply ignored the medical reports, and induced public policy to follow suit.

52 P.32: "Lead is particularly a cumulative poison, and post-mortem analyses of viscera show that it may be stored up in certain parts of the body, more especially in the bone and red bone marrow and brain." He cites several studies showing lead in the brain as well as other organs. P.33: "The quantity of lead present in the brain necessary to determine acute poisoning is not known and it is probable that an extremely minute quantity will produce very serious effects." P.36: "Malnutrition is recognized as a predisposing cause of practically all forms of disease, and with a chronic intoxication, such as lead poisoning, malnutrition and starvation, with its attendant depression of the vital force of the body, is essentially a predisposing cause of poisoning."

55 Holley CD, ibid.
56 Oliver T, Lead Poisoning: From the Industrial, Medical And Social Points of View, New York, Paul B. Hoeber, 1914, p.56.
57 Oliver T, ibid, p.56.
58 Oliver T, ibid, p.57.
60 International Labour Office, White Lead: Studies and Reports Series F (Industrial Hygiene), No. 11, Geneva, 1927, p.52. Austria, 1924; Belgium, 1926; Bulgaria, 1926; Chile, 1925; Czechoslovakia, 1923; Estonia, 1925; France, 1926; Latvia, 1924; Poland, 1924; Rumania, 1924; Spain, 1924; Sweden 1923.
61 International Labour Office, op. cit., p.36.

In 1922, a General Motors researcher in Dayton, Ohio discovered that the addition of tetraethyl lead to automobile fuel dramatically reduced the "knocking" that limited power and efficiency in automobile engines.62 General Motors, the DuPont Chemical Co., and Standard Oil of New Jersey campaigned long and hard to assure acceptance by the U.S. Government of industry-sponsored research showing that tetraethyl lead, a "Gift of God," was safe.63

After four years of studies, conferences and discussion, the U.S. Surgeon General put the stamp of approval on tetraethyl lead early in 1926. This marked the beginning of forty years of "enormous, indeed, hegemonic influence over the production and dissemination of knowledge about lead poisoning."64 The medical hegemony emanated from two principal sources of authoritative medical research: Joseph Aub, M.D., at Harvard Medical School, and Robert M. Kohoe, M.D., medical director of the Ethyl Corporation (a joint venture between General Motors and DuPont) and director of the Kettering Laboratory of Applied Phisiology in Cincinnati, Ohio.

The money to conduct the study of lead at Harvard had come from the lead industry, obtained by Alice Hamilton, the first female member of the Harvard Medical School faculty, and author of the authoritative handbook of industrial toxicology.65 Harvard handed the project to Joseph Aub, a brilliant young physician-scientist.

"I don't know how she did it," Joseph Aub later told an interviewer about Alice Hamilton's fund-raising, "but she extracted $25,500 from leading lead manufacturers for a three-year study. What was even more astounding was how she got it with no strings attached. Harvard had complete authority to investigate and publish its findings without submitting it to the industry for approval. What makes this performance all the more remarkable is that before Alice Hamilton came on the scene many leading people in the lead industry would not even admit the existence of lead poisoning."66

Aub had recruited a team and started publishing papers in 1922, culminating in the classic Lead Poisoning, which appeared in the Journal of Medicine in 1926, and in 1936 as a monograph.68 The Medicine article states that "the funds for this work were given to the Harvard Medical School by the National Lead Institute."69 Based on animal studies, human subjects, and the most exhaustive literature review to date, Aub and his colleagues investigated every possible aspect of industrial lead poisoning and presented it in a tidy package: On a good diet, lead would just tag along with calcium to the bones where it was essentially "harmless."70

Aub's analysis fit perfectly with developments in human nutrition. The appalling health status of the American youth who presented themselves for military service during World War I stunned the medical and public health communities and stimulated serious research into nutrition. By the mid-twenties, numerous vitamins had been discovered, and the miracle food of the decade was milk. Every major author on lead poisoning had addressed the protective effect of milk, but Aub described the mechanisms. A positive calcium balance hastened deposition of lead in the bones, minimizing the opportunity for absorption by other organs. Fluid milk, "Nature's Most Nearly Perfect Food" had been recently "purified" by the introduction on a large scale of pasteurization of public milk supplies.71 As fresh, clean milk made its way into everyday life during the 1920s, its reputation as a protective food may well have been nudged by policy makers who understood from reading Aub that lead followed the calcium.

To his credit, Aub was helpful in pointing out the risks to workers inhaling lead dust, and during the twenties, more protective measures were taken in mines and mills. Also, Aub took a conservative view on the question of levels of lead necessary to produce toxicity, referring to the work of Torald Sollman at the School of Medicine of Western Reserve University who concluded in 1921 that "Phenomena of lead poisoning appear after some weeks with daily oral doses of lead salts of 0.2 to 0.3 mg per kilogram in man ..." Sollman warned that smaller doses might produce non-clinical effects, and stated that there was a wide gap "between clinical disease and harmlessness."72


63 Ibid.

64 Graehner W., op. cit., p.46.

65 Hamilton A. and Hardy HL., Industrial Toxicology, New York: Hoeber, 1934.

66 Oral history taken by S. Benison 19 July 1957, Holmes Hall, Countway Medical Library, Archives GA4, Box 14, pp. 171-172.

67 Aub J.C., Fairhall LT, Minot AS, Reznikoff P. Section XX by Alice Hamilton, Lead Poisoning, Medicine 41:230, 1925.

68 Aub, J.C. et alia, Lead Poisoning, Baltimore, Williams & Wilkins, 1926.


70 Aub J.C. et alia, op. cit., 1926, p.68: "...after absorption has ceased, significant amounts of lead are to be found only in apparently harmless deposits in the bones."


Butressing Aub's view of lead as "harmless" came the steady stream of medical literature from R.A. Kehoe at the Kettering Laboratory in Cincinnati, Ohio. Kehoe was medical director of the Ethyl Corporation from 1925 to 1958 and also served as director of the Kettering Laboratory at the University of Cincinnati. Kehoe's work was characterized by an emphasis on the normalcy of lead in the human body, and the equivalency of adult and childhood exposures. Although his first publication on normal lead levels appeared in 1925, a major contribution was a series of articles published in the *Journal of Industrial Hygiene* in September, 1933. In these articles, entitled "On the Normal Absorption and Excretion of Lead, Parts I, II, III and IV," Kehoe established that ingestion of lead was a common, everyday occurrence for Americans, and that the exposure levels described by Aub and others as being toxic were normal. Even though the children showed lead excretion levels slightly higher than the "normal American adults," he insisted that processes of absorption and excretion were the same in adults and children. In none of his papers did he ever cite the Australian literature, or any of the U.S. papers concerning childhood lead poisoning.

Kehoe's contribution during the early 1930s was an important estimate of "normal" daily intake (2.0-35 mg/day) and excretion (25-30 mg/day). More significantly, Kehoe neutralized attempts to nail down a level of exposure that could be deemed toxic. He stated boldly in 1926: "There is at present no quantitative expression of lead secretion in man which may be said to be significant of impending or present lead poisoning." In effect, he simply declared that the level of lead ingestion viewed as poisonous by experts on two continents was "normal."

Together, Aub and Kehoe played down the potential effects of absorbed lead on children. Aub particularly seemed interested in minimizing the importance of encephalopathy; he favored a less dangerous view of lead's effect on the central nervous system, i.e., the view that the cerebral effects were meningitis, an inflammation of the brain's lining rather than encephalitis, an inflammation of the brain itself. He also implied that any child who chewed lead paint was already "defective," a bias that persisted for decades.

By declaring lead harmless, Aub provided the medical foundation for the Surgeon General's 1926 approval of tetraethyl lead. Kehoe's work put a lock on toxicology in general, and stalled pediatric research by asserting that absorption of lead was equivalent in adults and children. His assertion that child and adult exposures were equivalent endured into the seventies. Together, these bodies of scientific work from the Kettering Laboratory and from

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75 Aub JC et al., *op. cit.*, 1926, p.70.
4. "SAVE THE SURFACE"

From the time paint first became available as a substitute for wallpaper early in the century, white lead was offered to the American people as the only thing to use on interiors and exteriors. "The proper decoration of the interior of dwellings and public buildings has become of even greater importance than the protection and decoration of exteriors," wrote Henry Gardner in 1911 from his new Washington Office of the Educational Bureau of the Paint Manufacturer's Association. "Up until a few years ago... wall papers... were almost exclusively used... there seems to be no questions, however, that the use of wall paper is steadily declining, and that the art of interior decoration is undergoing a transition to the almost universal use of paint." Gardner touted paint as a conservation measure because it meant trees weren't ground into wallpaper. Gardner also noted proudly that in laboratory tests, walls painted with oil paints grew fewer bacteria, adding the sparkle of sanitation to paint as an alternative to wallpaper.

More importantly, in cities teeming with millions of new immigrants, the glossy durable finish of white lead meant walls could be easily washed. In one of the first large advertising campaigns, started in 1908, a statue of the Venus de Milo adorned an ad in the American Paint and Oil dealer describing how the statue inspired a brightening up of a filthy, dirty tenement. The ad apparently won the hearts of paint dealers as a way to move paint into the cities.77

White lead companies financed a "Save The Surface" campaign in a series of advertisements in The Saturday Evening Post during the 1920s. In one,78 an astronomic observatory peers into a star-studded universe over the headline "Bringing the Stars Down To Earth." The text explains that lead, in one of its more unusual properties, enhances the refracting power of glass lenses. "Most important of all, to modern civilization, is the use of white lead as the principle ingredient in good paint. Everywhere people are learning the importance of protection, the wisdom contained in seven short words -- Save the surface and you save all." The text goes on to invite the reader to try Dutch Boy paints and write for a free booklet "The Wonder Book of Lead," to learn "the hundred and one ways in which lead enters into the daily life of everyone." Another National Lead Company ad states "The highest protective power is found in those paints which contain the most white lead."79

A large two-page spread in The Saturday Evening Post sponsored by Acme White Lead and Color Works depicted a split-season picture of a bright upper middle class home, where a gentleman shovels the sidewalk in the winter half and sits casually reading the newspaper on his porch in the summer half. On the next page in smaller inserts, a man is shown painting wood trim and women are shown painting kitchen walls and a baby carriage with white lead.80

In the trade press, a National Lead Co. ad described "lead consciousness," a perception among painters that white lead was in a class by itself as a protective coating.81 Eagle-Picher also ran full page ads touting white lead. With an irony now painful to contemplate, the ad asked, "Is there enough LEAD in your Paints?"82 and proclaimed, "The more lead there is in your paint the more enduring will be your customer satisfaction."83

The remarkably successful ad campaigns of 1920s and 1930s -- the Dutch Boy, the "Save the Surface" campaign -- helped create a myth among painters that white lead was the real and only pigment. For painters, the process of mixing the white lead paste with linseed oil was part of the ritual of painting, as was testing the purity of the product on the base of the sheer weight of the can. The unmistakable weight of white lead was the surest mark of quality.

In addition to the advertising campaigns, an influential federal agency disseminated the message that white lead was preferred for interiors of public buildings. Through the 1920s, the National Bureau of Standards (NBS) consistently recommended white lead for use in schools. In correspondence with citizens, firms, and public institutions, the Bureau made it clear that white lead was the paint of preference for interior surfaces. "[T]here is no more durable white

76 March 25, 1922, p.5.
77 April 8, 1922, p.68.
78 November 3, 1923, pp. 144-145.
80 Chemical Review, 89:1, 1930.
81 American Paint Journal, April 21, 1930, p.3.
paint for interior localities than basic carbonate white lead paint . . . stated C.K. Burgess, NBS Director, in 1926.

The Minneapolis Board of Education wrote to the Bureau of Standards in 1930 asking for standard specifications for the painting of schoolhouse walls and ceilings." E.F. Hickson, chemist for the NBS, responded by noting that the NBS had no specifications for school house walls, but strongly directed the reader to lead paints. Hickson described in detail the practice of one branch of the Government in painting walls and ceilings using entirely paste white lead.84 In response to an inquiry from the Board of Education, City of New York, F.H. Walker directed a division of the NBS to note in the letter that for painting plaster walls with lead and oil, "There is nothing better that can be used."85 Indeed, the NBS let people know that the White House was painted with white lead.86

Promotion of white lead for interiors continued for decades. In 1935 the National Lead Co. recommended lead for interior walls in a trade publication: "Lead Paints are used extensively for structural metal paints, exterior house paints, and many interior paints because these pigments impart to the paint films a marked degree of toughness and elasticity which is retained even after the films have aged for a long time." As the description of white lead noted, "The painter prepares a paint to meet his particular requirements by mixing the white lead paste with suitable paint vehicles and tinting colors. By using various vehicles, various types of paint can be made, including interior flat paints of great beauty as well as durability from the point of view of adhesion and washing properties."87

Two years later, the lead industry again promoted white lead interior paints. As part of a Lumber Products - Better Paint Campaign, an exhibit in a car-drawn trailer toured Kentucky, Ohio and Indiana early in 1937. The novel idea was designed to "promote the wider use of lumber by educating lumber dealers and the public in general to the importance of good paint.... Outstanding among the exhibits are six model house exteriors and six interiors, the color schemes for which were carried out by National Lead Company's Dept. of Decoration." The article also reported in a photo caption that the Palatine Hotel in Newburgh, New York "was redecorated on the interior with Dutch Boy materials - White Lead ...."88

85 Walker PH, Memorandum for Files, September 24, 1929, National Archives and Records Administration RG 167, Box 205A.
86 Letter from Office of Building and Grounds to a Baltimore firm, May 9, 1929, National Archives and Records Administration, RG 167, Box 42.

Although the Lead Industries Association received newspaper clippings almost on a daily basis about reports of lead poisoning,89 it embarked in 1939 on a $250,000 White Lead Promotion Campaign, the largest in LIA history.90 A model home covered with lead paint was featured in the July issue of Better Homes and Gardens and other publications. As part of the campaign, white lead paints became widely available in colors.91

As late as 1943, a publication produced jointly by the U.S. Government and the Lead Industries Association recommended white lead for farm buildings and domestic interiors.92 While the publication mentions other paints, it provides precise instructions for using 100% white lead for exteriors. Simple, clear line drawings show how to mix the paint. The section on interior paints explicitly recommends white lead. The booklet also provides handy formulas for making "home-mixed interior paint."

During the thirties and into the mid-forties, market share in lead was heavily skewed to National Lead Co., which held about 55 percent of the white lead market.93 Sherwin-Williams and Glidden held about 20 percent between them; Eagle-Picher accounted for 13 percent, and eight percent was produced by International Smelting and Refining, a subsidiary of Anaconda. Sherwin Williams Co. produced and sold white lead until 1947.94 Glidden until 1957, and National Lead Co. into the 1960s. A 1951 Glidden painting guide recommended lead paint for interior and exterior surfaces including walls, trim and doors.95

89 LIA Annual Meetings (January 24, 1939), In Federal Trade Commission. In the Matter of National Lead Co., et al., Docket No. 5253 at p. 5555. 1
90 LIA Annual Meeting (Jan. 24, 1939), Ibid.p 5000, and LIA Annual Meeting (January 37, 1940), Ibid, p. 5355.
95 Glidden Painting Specifications, (1951), CLD 32166.
5. THE LEAD INDUSTRIES ASSOCIATION 
AT WORK, 1928-1946

By the late twenties, white lead experienced increasing competition from substitutes, both zinc and titanium. Zinc had been recognized by the U.S. Supreme Court in 1907 as being essentially equal to white lead, and titanium had also become a significant substitute pigment. The U.S. Government published standards for white lead in 1919, 1924, 1929, and 1936. However, lead producers dominated the zinc mining and refining process and the National Lead Company quickly assumed control of titanium pigments. That the National Lead Company would control the market for substitute pigments seemed obvious to some observers. 100

To assure the continued protection of the market for white lead and other lead products, the principal producers of lead together established in 1928 the Lead Industries Association, with headquarters in New York, representing virtually all of the United States white lead production. Key sponsors of the Association were The National Lead Company and the St. Joseph Lead Co. Other firms that joined included Hecla Mining Co., the Anaconda Company, the American Smelting and Refining Co., Eagle-Ficher, the Sherwin Williams Co. and the Glidden Co. 101

To judge from Lead Industry Association minutes, medical literature, and industry publications, the strategy of the LIA for decades was threefold: to oppose regulation of lead, to minimize the significance of lead poisoning, and to promote lead products. The key player for the LIA was Felix Wormser, a former writer and editor who spent the next four decades managing medical research, legislative work and promotion. Joining the industry after a stint

as assistant editor of Engineering & Mining Journal Press, 102 Wormser coordinated anti-legislative activities and medical research from the early 1930s until his retirement to join the St. Joseph Lead Company in 1947 as an Assistant to the President. He came out of retirement in 1953 to serve as Assistant Secretary of the Interior for Mineral Resources, a position he held until returning to the St. Joseph Lead Company in 1957. In June, 1962, he testified before Congress on matters of lead pricing policies, and in 1966 he still served as the industry's spokesman on health and safety issues. 103

On legislative matters, Wormser's role would supplement a well-established network that had been in place for nearly three decades: National Paint Varnish and Lacquer Association (NPVLA). Its predecessor, the National Paint, Oil and Varnish Association 104 had had a nationwide legislative committee in place since the first decade of the century. 105 Even though the state of North Dakota enacted a formula labeling law in 1906, which the paint industry took all the way to the Supreme Court, 106 there was virtually no state or local regulation until the forties and fifties. Major white lead firms such as National Lead Co., Sherwin-Williams Co. and Glidden Co. served on the NPVLA's executive committee through the thirties. 107

Reports of the legislative committee's effort to thwart regulation were common in industry publications. A good example of how the system worked occurred in 1933, when a new director of occupational health, Manfred Bowditch, threatened to adopt regulations concerning the reporting of lead poisoning in Massachusetts. Wormser went into action. He met with Bowditch and reported success in his 1934 annual report:


101. LIA Board of Directors Meeting (1967), LIA 00826.

102. Triggs ET, Fifty-Five Colorful Years, The Story of Paint in America Stonington, Connecticut, The Pequot Press, 1984, p.251-52. The National Paint, Oil and Varnish Association was formed in 1888, and was consolidated with other trade associations in 1933 to form the National Paint, Varnish and Lacquer Association, Inc.

103. "From 1900 onward bills for proposed paint laws appeared regularly in a dozen or more states." The "regular procedure" for G. B. Heckel, "was for my correspondent (usually a paint man at the State Capital) to mail me a copy of any bills then introduced and on its receipt I would reproduce it and mail it to the Legislative Committee and to manufacturers in the State. Frequently also I would write the chairman of the committee having charge of the bill, pointing out its objectionable features. And until North Dakota stepped into the limelight not a single paint law in any State got past the Legislature." Heckel GB, The Paint Industry: Reminiscences and Comments, St. Louis, American Paint Journal Company, 1931, p.221-23.


"During the year an effort was made by the Massachusetts Department of Labor to establish regulations which would have seriously affected the use of white lead in painting buildings. This subject was discussed by the Secretary (Wormser) with the State officials having the matter in hand and a satisfactory adjustment procured. It was particularly important to obtain a hearing and settlement in Massachusetts otherwise we might have been plagued with an extension of similar restrictive painting legislation in other States, affecting the use of white lead."

The relationship with Bowditch would endure for many years and he eventually became Director of Health and Safety for the LIA.

Wormser and the LIA also had a role in focusing the poisoning issue around children's toys and furniture and placing responsibility on the parent. This strategy appears to have emerged as articles on childhood lead poisoning moved out of the medical literature into insurance company reports, and as the U.S. Children's Bureau began a period of anti-lead poisoning activism.

Two statisticians working for leading insurance companies produced especially convincing data. One was Frederick Hoffman, whose work for the Prudential Life Insurance Company spanned three decades, ending with his retirement in 1935. A world traveler and expert on public health, Hoffman may have unwittingly sparked the concept of "A piece of the rock" when he returned from Gibraltar in the early years of the century with 2,000 pounds of it. In a report later published by the U.S. Bureau of Labor, Hoffman in 1927 painted a gloomy picture of child health and safety.

Hoffman declared his report to be "the first definite statement of its kind ever published revealing the true extent of the mortality from chronic lead poisoning among the American People." With statistics from the Metropolitan Life Insurance Co., the U.S. Navy, and 14 cities and states, Hoffman's report listed occupations most frequently associated with mortality from lead poisoning. Painters had about eight times the mortality of other occupational groups. Significant by their presence in the non-industrial grouping were the 19 deaths from lead poisoning among youths under 18, including seven from eating lead paint.

The other source of statistical information was Louis J. Dublin at the Metropolitan Life Insurance Co. Dublin administered a survey of "prominent pediatricians" in 1930 and published results in the firm's bulletin. A majority of these physicians, reported Dublin, "agreed that chronic lead poisoning in infancy and childhood is by no means a rare condition, and almost all believed that wide publicity should be given to this fact through the press or the 'popular' literature of health departments and private health agencies, with special insistence upon the dangers inherent in cribs and toys painted with material that contains lead." Dublin observed that lead poisoning "would be a more prominent item in both morbidity and mortality records but for the fact that the condition is often unrecognized by physicians." The Bulletin quotes a Boston physician [McKann] who reported 50 cases at a single hospital beginning in 1924, noting that the diagnosis of lead poisoning from chewing paint from cribs, woodwork, or toys, was "proven beyond a doubt."

The LIA stepped into deal with both McKann's medical report and Dublin's survey. In a 1933 follow-up to his earlier articles on lead poisoning in children, McKann cited Wormser in a footnote to the effect that cooperation between the lead industry and manufacturers of toys and cribs was helping to minimize risks. In correspondence, Dublin warned that the October Bulletin article "received a great deal of publicity against which there was strong remonstrance by the Lead Industries Association. You will readily understand that we wish to avoid any controversy with the lead people. Please, therefore, do not mention the Metropolitan [hand written insert 'either directly or by inference'] in connection with whatever releases you may make." In 1930, apparently in anticipation of President Hoover's National Conference on Child Health, the Lead Industries Association conducted its own survey of manufacturers of children's cribs, beds and furniture. Of twelve respondents, most indicated they did not use lead paint. The industry position was that reputable manufacturers no longer used lead paint on children's toys or furniture.

The poisoning problem from lead-painted toys and cribs thus having been "solved" by an unwritten code of honor in industry

114 McKann CP, and Vogt DC, Lead Poisoning in Children, Journal of the American Medical Association 101:3134-3135, 1933. "The lead industry and the manufaturesters of cribs and toys, informed of the danger to small children from the ingestion of lead paint, have cooperated by substituting other types of pigments for the lead pigments formerly used. New cribs are seldom painted with lead paint, the better grades of toys are largely free from lead pigment."
115 Dublin LI. Letter to Dr. Ella Oppenheimer, M.D. of the Children's Bureau, September 14, 1930, National Archives and Record Administration, RG 102, Box 498, File 8-4-5-7.
concerning toys and cribs, the burden remained on the consumer to find non-poisonous paints. Doing so was greatly hampered because the trade associations had long blocked enactment of any formula labeling provisions.\(^{117}\)

The organization's apparent resolve was to preserve its markets as long as there was a substantial production capacity, and one way to do that during the Depression was through government contracts. Just a few weeks after the Lead Industries Association survey concerning lead paint in toys and children's furniture, the LIA declared at its December 1946 meeting that it would seek to convince the government to use lead more liberally in government buildings and contracts, for interior and exterior surfaces.\(^{118}\)

When widespread public housing programs became a priority of the Roosevelt Administration, the paint industry saw the potential for more business. Describing opportunities in post-Depression public works projects, the National Paint, Varnish and Lacquer Association's representative in the Federal Housing Administration, T.E. Dann, asked "paint manufacturers to once again consider the various phases of the national housing act, with but one end in view, namely, 'How can we profit from it.'" Mr. Dann exhorted his colleagues to win some of the $200,000,000 in predicted expenditures on painting.\(^{119}\)

When the Public Works Administration published instructions to private builders, the architect's instructions specified: "All wood and metal, inside and out, 2 coats lead and oil or enamel over priming ..." \(^{120}\) Rent estimates for public housing were to "include wall painting with lead and oil every four years."\(^{121}\)

Wormser continued as the industry's liaison to the medical community, and he seems to have seen himself as a corporate troubleshooter whose primary purpose was to follow up reports of lead poisoning and cast doubt upon them. For example, when in 1942 the National Safety Council published a report on fatalities due to lead poisonings in Chicago,\(^{122}\) Wormser and the LIA made an inquiry. Wormser later reported that "[i]n an investigation by the Lead Industries Association, it was shown that there was no lead poisoning of the kind described, nor were lead toys or lead painted cribs involved. ..." \(^{123}\) The children had died from inhaling the fumes from burning battery casings.\(^{124}\) The Council published a correction a year later.\(^{125}\)

Joseph Aeb served as the outside expert on cases of occupational and childhood lead poisoning referred to the Lead Industries Association. For example, Wormser forwarded to Aeb material concerning death of a black employee at the John R. MacGregor Lead Company. The coroner's report stated that the cause was "peritonitis with obstruction of the jejunum associated with chronic lead poisoning ... Said poison received during the course of 16 months employment at the John R. MacGregor Lead Co." Aeb's analysis: the man "died of peritonitis, probably starting somewhere around the appendix ... these are the things he died from."\(^{126}\)

Twice in 1945 Wormser wrote Aeb asking the Harvard physician's opinion on cases of childhood lead poisoning and Aeb responded in carefully worded replies that minimized the likelihood of lead poisoning: "... I would not consider it a clear cut case although, of course, the bones and parathyroid fit in with a possible diagnosis of lead poisoning. Still I am suspicious of the fact that ..."

\(^{117}\) For example, in a July 1953 "Report of the President to the Executive Committee," Joseph F. Basley, President of the NFPLA described state and municipal efforts to regulate lead: "The New York City Board of Health on July 14 ordered the preparation of an amendment to the City Sanitary Code to require warning labels on lead based paint containers. We have been granted the privilege of a hearing on the amendment and will oppose it. The Department is distributing pamphlets warning parents to prevent their children from chewing paint surfaces." National Archives and Record Administration, RG 102, Box 609, File # 4-5-17.

\(^{118}\) LIA Directors Meetings (Dec. 17, 1946), Federal Trade Commission, In the Matter of National Lead Co., et al., Docket No. 5235, p.5092.\(^{119}\)

\(^{119}\) Oil, Paint and Drug Reporter, October 21, 1945, pp.27, 42B.

\(^{120}\) Specified in "Instructions to private architects for low rent housing projects by the housing division, Public Works Administration (Plans and Specifications Branch), National Archives and Record Administration RG 190, Series 43, File PWA, Undated.Estimated to be 1936-1937.

\(^{121}\) U.S. Housing Authority, Interoffice Memoranda, National Archives and Record Administration RG 196, Series 14, Interoffice Memoranda Thru February 1941, May 8, 1939.

\(^{122}\) Lead Toys -- Lead Paint -- Lead Poisoning, Safety Education 22:74, 1942.


\(^{124}\) Williams H, Schulze WH, Rothchild HB, Brown AS, Smith FR, Lead Poisoning from the Burning of Battery Casings, Journal of the American Medical Association 100:480-489, 1933. It had become a common practice among Blacks in Baltimore to burn battery casings as a source of heat. The ensuing lead poisoning was labeled "the depression disease." In view of the widespread poisonings over two winters, the City of Baltimore undertook vigorous preventive measures, including radio warnings. Several other states issued warnings, and an additional national warning was sent out by the Lead Industries Association.

\(^{125}\) Error in Report on Lead Poisoning, Safety Education 23:943.

\(^{126}\) Aeb JC, Lead as a Hazard (Letter), Journal of the American Medical Association 142:237, 1940.

\(^{126}\) Aeb JC to Wormser FE, Aeb Papers, Holmes Hall, Countway Medical Library, Aeb JC, Archives, Box 5.
this child died of an infection, just barely possibly of a meningococcus. In the case of a ten-month-old infant who died in Texas in July, 1945, the medical report showed a leadline, anemia, and stipping of the blood cells, classic signs of lead poisoning. Wormser wrote indignantly "Frankly, I do not see how you can call this a genuine case of lead poisoning, do you? To which Aub replied "... autopsy ought to prove the problem of whether or not the child had lead poisoning, if they do lead analysis of the liver and bones. Up to now, the evidence is inadequate." The LIA Board noted at its January 15, 1943 meeting that "without the... assistance we have received from Dr. Aub and his associates, many of our lead poisoning problems would be unsolved." 130

As World War II drew to a close, the LIA began to address the serious publicity given to lead poisoning and its effects on mental development. In 1943, an important medical paper concerning the effects of lead on children's mental development appeared. Although the effects of lead on mental development had perplexed physicians since the seventeenth century, had particularly troubled Tanquerel des Planches in the nineteenth century, and had alarmed Oliver early in the twentieth, the first scientists to analyze quantitative data from mental and behavioral tests of exposed children were Byrns and Lord. 131 The Harvard-Yale team's study examined 20 children who had been poisoned by lead in infancy. Referring to Aub's physiological studies and his assertion that lead was "harmless," Byrns and Lord suggested that "under chemical shifts common in childhood, concentrations of lead known to be significant may be recurrently liberated into the circulation." The study also explicitly refuted Aub's contention that children who eat lead paint are mentally deficient to start with.

Buyers' and Lord's work received wide publicity in a TIME magazine article. 132 Entitled "Paint Eaters," the article warned that "If your child is slow with building blocks, but quick on tantrums, he may be a lead eater." The article indicated that children "may start chewing paint off window sills and other places," but went on to place blame on parents who re-paint cribs with leaded paint. The result of eating paint, reported TIME, was "stupidity."

Within two years, the Children's Bureau decided to undertake a major public education campaign. Late in 1945 or early in 1946, the Children's Bureau published a two-page flyer entitled "Paints, Pigments, and Dyes." 133 The text warned that some paints were poisonous when swallowed and actually listed types of paints — but not brand names — that were usually considered harmless or poisonous. Lead carbonate topped the list of poisonous paints. "With this list as a guide," the flyer noted, "a harmless paint can be selected in most paint stores." The flyer warned the reader that "it is not safe to take the word of the salesman as to whether it is harmless or not because he may not know."

In his report to the Lead Industries Association in April, 1946, Wormser warned that if attacks on lead go unchallenged, "they may very easily lead to the sponsoring of totally unwarranted state and federal legislation of a regulatory or prohibitive character...this is an unending battle from which we can only withdraw at our peril." 134 At one point, the LIA board considered funding medical research "to prove that the ingestion of extremely minute amounts of lead, contrary to public conception, may actually be beneficial to public health." 135

Soon after the flyer was published, Wormser began planning a medical conference to be jointly sponsored by the Lead Industries Association, the American Medical Association, and Harvard Medical School. For a February 7, 1946 planning meeting, Wormser registered Aub in a New York hotel, paid the deposit and secured tickets for Dr. and Mrs. Aub to The Glass Menagerie. 136 At the meeting, an agenda for the symposium was developed and the American representative asked the LIA to prepare a paper "on the general subject of the occurrence of lead in the United States so as to give the doctors a background on the subject and express the industry's viewpoint." Wormser agreed to undertake this task. 137

The next week, Harvard faculty member Philip Drinker wrote to Harvard School of Public Health Deans suggesting Harvard host the symposium, adding "You will remember that the studies on lead poisoning which resulted in the publications by Dr. Aub and others were sponsored by the Lead Association. They have consistently been our very good friends." 138 During the thirties and forties, LIA contributions to Harvard were on the order of several thousand a year, averaging about $3,000 with a break during the Depression. 139

The Symposium was conducted on September 30, 1946 at Harvard. Several papers from the symposium were published in Occupational Medicine in 1947. The first article, by Felix E.

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137 Meeting Minutes February 7, 1946. Aub Papers, Holmes Hall Courtyard Medical Library. Aub JC, Archives, Box 5.
138 P. Drinker to Harvard School of Public Health Deans Huber and Burns, Feb 12, 1946. Aub Papers, Holmes Hall, Courtyard Library, Boston, Massachusetts, Archives, Box 5, folder "Lead 1927-1965".
139 LIA Annual Meeting Reports, 1929-1941, FTC.
Wormser, is entitled “Facts and Fallacies Concerning Exposure to Lead.” In it, Wormser pointed out that not all paint contains lead, and that “Prepared interior paints, furniture paints and enamels are usually free of lead.” Exterior paints usually contain white lead, he said, because “it is necessary.”

Wormser carefully reviewed the various sources of lead in society, including storage batteries, pigments, and automobile fuel, and concluded that “it is apparent today that despite the large amounts of lead used in everyday life . . . the lead hazard in industry and to the public is relatively small and can be effectively controlled when it cannot be eliminated.” Dealing specifically with reports of childhood lead poisoning, Wormser ridiculed and dismissed Byers and Lord. “Certainly there was no proof here of lead exposure.”

Another speaker at the Symposium was Robert Kehoe, who presented data from numerous studies purportedly demonstrating that some lead intake is normal. He denied other medical reports that lead accumulates in the body, stating that “no such accumulation occurs or it is so slight as to be insignificant in the course of a lifetime”. Kehoe was convinced, and tried to convince his audience, that lead posed no public health threat to the general public. He also offered a qualitative analysis, stating that “the safe level for the ingestion of lead in food and in drink . . . is greater than 0.3 mg. and less than 0.6 mg. per day” the latter figure twice as high as the upper limit he had described in 1933.

Kehoe also made it difficult for individuals, communities and schools to obtain information about lead poisoning from what should have been a good source, the American Public Health Association. Both Kehoe and Aub were senior members of the Association’s committee on lead poisoning. Aub referred queries about lead poisoning to the American Public Health Association position papers. Although Kehoe’s bibliographies consistently ignored the considerable literature from reputable institutions, one paper from the Kettering Laboratory in 1940 listed nine references including Thomas and Blackfan, McKann, Blackman and others, making it clear that the Kettering Laboratory had access to the core literature on childhood lead poisoning.

144 Aub JC, Letter to AMW Hurst, Chief Medical Examiner of the Pennsylvania Railroad Voluntary Relief Department, July 23, 1947. Aub Papers, Holmes Hall, Countway Medical Library, Aub JC Archives, Box 5.

Nevertheless, Kehoe omitted reference to these core papers from a American Public Health Association publication. In a lengthy report on occupational exposure, Kehoe provided a separate category for “Non-Industrial Lead Poisoning,” which included two articles on battery casings, one on snuff, three on water, three review articles, and an historical article. Here, he chose to omit references to lead paint poisoning that were clearly in possession of his laboratory. Anyone turning to the American Public Health Association for information on childhood lead poisoning would come up empty handed or learn that Kehoe and Aub were cited heavily as the underlying authorities.

In sum, for almost two decades LIA successfully dominated public — and legislative — perceptions of lead use and toxicity, with Aub and Kehoe playing key roles. During that period, millions of tons of lead found their way into residential paint, a legacy of hazard with us still today.

146 Committee on Lead Poisoning of the Industrial Hygiene Section of the American Public Health Association, Occupational Lead Exposure and Lead Poisoning, New York, American Public Health Association, 1943, p.66.
6. THE POSTWAR LEAD INDUSTRY, 1946-1963

During and after the war, lead mining and the production of pig lead (from which white lead was manufactured) remained in the hands of the same firms that had dominated since the turn of the century. As the decade progressed and the reports concerning childhood lead poisoning accumulated, the white lead industry was joined in the campaign to control information by other lead pigment manufacturers. By the mid-fifties, however, the mounting evidence of lead’s toxicity prompted some changes: initially, adoption of an unenforceable national code, and, eventually, enactment of federal legislation.

In 1945, nearly half of the domestic mine output came from southeastern Missouri, where St. Joe owned most of the mines. National Lead Co. also had mines in the southeastern Missouri fields through its subsidiary, the St. Louis Smelting and Refining Co.147 Eagle-Picher Company operated mines in the tri-state district in Oklahoma, Missouri and Kansas.148 The Anaconda Copper Mining Co. operated lead mines in Montana, Idaho, Utah, Nevada, and California, as well as in Mexico and Chile.149

The Smelting and refining of lead ore was similarly concentrated. Major smelting and refining interests were held by American Smelting and Refining Co., which operated lead smelters and refineries in California, Colorado, Illinois, Montana, Nebraska, New Jersey, Texas, and Utah and accounted for about 80 percent of production.150 Less than ten percent was accounted for by Bunker Hill & Sullivan (partially owned by Hecla); International Smelting and Refining Co. (a subsidiary of Anaconda); and United States Smelting, Refining, and Mining Co.151 The National Lead Co. controlled about half of all U.S. lead manufacturing.152

National Lead Co. and Eagle-Picher were in first and second place in production of lead pigments; together they consumed about 60% of the pig lead used for pigment production.153

By the late 1940s, the market for white lead had decreased dramatically since its heyday in the 1920s, when it constituted the largest market share of major lead products. U.S. consumption of white lead declined steadily during the century, and by 1945, the 35,600 tons of white lead consumed were less than 20 percent of the nearly 200,000 tons consumed in 1923. Chief outlets for lead in the forties and fifties were storage batteries, tetraethyl lead, and pigments. An emerging industry made 'red' pigments, which were widely used in automotive lacquers and varnishes.
to us that we are not competent to answer. It was therefore finally decided to withdraw the statement. 157

To judge from Children's Bureau correspondence in the early 1950s, frustration over attempts to publish a warning about what was becoming recognized as a common poison was a force in the development of national poisoning prevention programs. That cause was furthered by the appearance of numerous medical articles from several cities during the early fifties.

Reports of widespread lead poisoning came from Baltimore, 158 from Byers at Harvard in 1954, 159 and from a pediatrician who would become a pre-eminent authority on lead poisoning, Julian J. Chisolm. 160 Among the startling findings reported by Chisolm in 1956 was that Baltimore children exposed to lead were excreting six times more lead than industrial workers with lead exposures. Important epidemiological reports came from New York City in 1956 161 and from Chicago in 1957. 162 The numbers of cases and fatalities reported in the medical literature continued upwards.

BY THE MID-FIFTIES, AS PRESSURE FROM CONSUMER GROUPS AND PHYSICIANS INCREASED, A HANDBOOL OF CITIES AND STATES HAD ADOPTED LEAD PAINT LEGISLATION OR REGULATION.


162 Jenkins CD and Melios RB, Lead Poisoning in Children, American Medical Association Archives of Neuropsychiatry 77:70-78, 1957.

163 American National Standards Institute, American National Standard Specifications to Minimize Hazards to Children from Residual Surface Coating Materials, Z66.1-1955, revised 1964. The Children's Bureau and the LIA were represented on the committee.


Sticking by his figures from 30 years earlier, Kohoe asserted that the intake of the average adult lead intake from food, beverages and air was about 33 mg/day, that excretion was about 30 mg/day, and absorption was about 33 mg/day. He stated that "no effective absorption of lead occurs in the alimentary tract under ordinary circumstances." He noted that 5 to 10 percent of what is ingested is absorbed, and not more than 10 percent of what is inhaled is retained. "There is little or no indirect evidence of the retention or accumulation of lead in the body of the 'normal' individual."

![Lead in White Lead & Gas](image)

**Figure 2**

A large number of the papers in this symposium were devoted to airborne lead, an indication that public and industry concern was now focused on atmospheric pollution. With while lead production capacity essentially shutting down (Fig. 2), the industry seemed eager to shift the attention of policy makers away from tetraethyl lead to the regulation of paint.

7. LEGISLATION & REGULATION 1965-1992

A 1965 paper by geochemist Clair Patterson of the Massachusetts Institute of Technology seriously challenged Kohoe's "lead is normal" doctrine and -- eventually -- triggered federal controls. Carefully documenting the tonnage of lead dispersed into the environment from industrial sources, automobiles, consumption of food crops, lead solder, lead arsenate insecticides, ceramics, pipes and paint, Patterson argued forcefully that the existing average lead levels were due almost entirely to environmental releases. He proposed that truly natural lead levels, in the absence of all industrial sources, would be lower by several orders of magnitude. This calculation was later confirmed.

Patterson asserted that:

- acceptance of typical lead levels in humans in the United States today as normal and therefore safe or natural is founded on nothing more than an assumption that these terms are equivalent. No acceptable evidence exists which justifies this assumption. On the contrary, as this report shows, such an assumption may be in gross error. The 0.25 ppm level of lead in the blood, which has been and still is regarded with ill-founded complacency, actually seems to lie between an average natural level of about 0.001 ppm and an acute toxic threshold of 0.5 to 0.8 ppm. This suggests that the average resident of the United States is being subjected to severe chronic lead insult.

Patterson's paper was a bombshell. The outpouring of letters to the editor of the Archives of Environmental Health was unprecedented. Among the responders were the LIA's Ziegfeld, then executive vice-president of the LIA, who described the paper as "completely conjectural."

The tone of the furor was captured by Harriet Hardy, the physician who co-authored the well-known *Industrial Toxicology* with Alice Hamilton, in her autobiography:

About this time (1965) C.C. Patterson attempted to publish his paper on environmental lead pollution. It quickly became clear that the lead industry was angry. Only by heroic means did Katherine Boucot, the editor

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of Archives of Environmental Health, persuade the editorial board to publish this paper. Several industry-supported research workers and plant doctors who read the now-famous Patterson paper threatened dire consequences if it were published, as did Patterson if it were not. . . . Since the U.S. government was soundly scolded by both sides (industry and medical profession), a meeting was called while Patterson was at the South Pole to, so I was told, "calm industry's fears of restrictive legislation." A few clinicians like myself were asked to a government-sponsored meeting, supposedly to talk informally . . .

The meeting was held in a huge room in Washington. It was no "shirt-sleeve" session for there were two tables of press and more than 100 senior officials of the lead industry . . . The discussion was noisy, angry, and sometimes incoherent because of emotion. After lunch there were more talks by government staff trying to make the atmosphere less tense, and the meeting broke up. A few of us were asked to stay for the press conference, a very cold-blooded affair. I almost felt sorry for one of my industrially hooked, very senior colleagues [Kchoe]. The press asked him what his salary was and what he paid and what money supported his laboratory. Unhappily, all his funds came from one large industry. This meeting was, I think, a small-stage warning of the restrictions to come in control of environmental lead pollution in the United States. 170

Patterson's article ignored concern about environmental pollution that had been smoldering since the publication of Rachel Carson's Silent Spring in 1959. The New York Times reported in September 1965 that average blood lead levels had increased sharply during the past decade. The report was based largely on Patterson's work and strongly implicated lead in automobile fuel as a major source. The market for lead in automobile fuel was still growing at astounding speed, and haggling over terms of paint legislation was a convenient way to protect that market. "Straight white lead and oil is almost dead," 171 the paint industry acknowledged.

From the mid-sixties on, the medical literature continued to document the epidemic of lead poisoning from both automobile emissions and paint. The number of relevant articles indexed by the National Library of Medicine grew from 21 in 1965 to 112 in 1969, and there was increasing attention to the effects of small doses. Even physicians at the Kettering Lab acknowledged that lower IQs were an outcome of lead poisoning. 172 Another medical article stated that mental retardation was among the "most common sequelae" and that lead paint was the single biggest source. 173

The lead paint poisoning issue came before Congress in 1970. On November 23, 1970 the Senate Committee on Labor and Public Welfare opened hearings on the Lead-Based Paint Poisoning Prevention Act. The best estimates in 1970 that 200 children died each year from lead poisoning, and of the 12,000 to 16,000 poisoned children who didn't die, half were left mentally retarded.

As the Subcommittee Chairman noted in his opening remarks, "We know that lead poisoning in children is caused by the repeated ingestion of chips and flakes of lead-containing paint and plaster from the walls, window sills, and woodwork of old and poorly maintained pre-World War II houses." 174 The committee learned that in many U.S. cities, as much as 80% of old houses in slum areas contained dangerous quantities of flaking paint. From across the nation's cities, surveys revealed that from 0 to 28 percent of urban children had blood lead levels greater than 50 μg/dl, above the 40 μg/dl level viewed by the Public Health Service as "undue lead absorption." 175

Much of the medical data was presented by Dr. J. Julian Chisolm, Jr., an associate professor of pediatrics at Johns Hopkins Medical School. Chisolm explained to the legislators the following:

Lead poisoning in children is exceedingly difficult to diagnose. . . . Clinical symptoms in early childhood are subtle, non-specific and insidious in onset. During the first four to six weeks of abnormal ingestion, no symptoms are apparent. Thereafter, over the next four to six weeks there is the insidious onset of decreased appetite, unwillingness to play, increased irritability, sporadic vomiting and delay in development. None of these symptoms are specific for lead poisoning, so that they are often attributed to other diseases. Indeed, the child may be thought to have a behavior disturbance or some minor intermittent infectious illness . . . 176

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170 Hardy, HL, Challenging Man-Made Disease. New York: Praeger, 1983, 121-122. Hardy also notes that "My favorite letter of many received (following the meeting) was one from the president of a New York brewery, threatening a libel suit. I had reported a measurable amount of lead (0.1 mg/liter) in beer. I was pleased to supply the source of this statement since it came from a brewer's journal. The suit was dropped."


176 Lead-Based Paint Poisoning Hearing, op. cit.
Chisolm identified the source of the problem as "Deteriorated pre-World War II house with old flaking lead paint on housing interior (especially window sills and door frames)."

As the hearings continued, John M. Montgomery, general counsel of the National Paint, Varnish and Lacquer Industry, acknowledged the Committee's purpose in dealing with children ingesting chips of old lead-based paint in the form of "flaking" chips of old paint and crumbling plaster from the interior surfaces of dilapidated residential housing built prior to World War II. ... It is true that, prior to World War II, many structures were painted with paints which contained large amounts of basic carbonate or sulfate of lead (white lead). This type of paint has not been used on interior surfaces for more than thirty (30) years."

Congress passed the Lead-Based Poisoning Prevention Act in 1971, restricting residential use of lead paint. It directed the Secretary of Housing and Urban Development "to prohibit the use of lead-based paint in residential structures constructed or rehabilitated by the Federal Government, or with Federal assistance in any form after January 13, 1971." The responsibility of prohibiting "the application of lead-based paint to any toy or furniture article" was given to the Consumer Product Safety Commission.

However, funding fell far short of expectations and both agencies were taken to court for failing to carry out the statute's mandates. HUD was chastised by the U.S. Comptroller General in 1980, and in 1983 the Court of Appeals in Washington ruled that HUD failed in its duty to protect children from the poisoning effects of lead-based paint. Consumers Union sued the Consumer Product Safety Commission in 1975 for allowing unsafe levels of lead in paints destined for household use and children's toys.

Throughout the seventies and eighties, additional medical and public health reports continued to underscore the severity of the lead poisoning problem. In 1978, the Public Health Service's Centers for Disease Control again lowered the blood-lead level defining undue lead absorption, this time to 30 μg/dl.

A major new contribution to the medical literature in the late seventies was that of Herbert L. Needleman and colleagues, whose large-scale studies correlated elevated lead levels in dentine (tooth) lead with psychologic and academic performance. Subsequently, a number of researchers have reported that neurobehavioral effects were observed at levels at least as low as 10 micrograms per deciliter or less. The LIA's Jerome Cole in a letter to The New York Times took issue with a Jane Brody column on lead poisoning, describing Needleman's work as "flawed and irrelevant." Cole expressed indignation that Brody and "the anti-lead advocates...would have us believe that low-level lead exposure poses a hazard of brain damage to children." Further attacks on Needleman's work were pressed — with a conspicuous lack of success — in 1991 by two scientists with links to the lead industry.

Another research finding was that, contrary to Kekoo's belief, children absorb and retain far more ingested lead than adults — some 40-50% for children, as opposed to about 10% for adults. Huge numbers of children were carrying elevated blood lead levels. Four percent of U.S. children between six months and five years of age carried lead burdens above 30μg/dl, with greater prevalence of elevated levels in blacks (12.2%) than in whites (2%).

Despite the compelling medical evidence and reports showing that declines in leaded fuel correlated positively with reductions in blood lead levels, the lead industry continued to oppose controls on lead in gasoline. It developed what Science described as a "two-pronged campaign to fend off controls, with the ILZRO [International Lead Zinc Research Organization] handling the scientific front and the Lead Industries Association (LIA) managing the legal blockade."

The industry tried in 1982 to pin the blame exclusively on "old lead paint which poor children eat either in

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177 Ibid., p.220-21.
178 42 CFR Section 403.1.b.
179 42 CFR Section 403.1.c.
180 Congressional Record - House, July 13, 1971, p.H6694 (Statement of Rep. B. Siegel). The FY 1972 budget included no funds for lead poisoning ...in spite of all the rhetoric on prevention of disease and health maintenance, this Administration has not been willing or even interested in providing the necessary resources to fight this obviously preventable disease.
184 Centers for Disease Control, Preventing Lead Poisoning in Young Children, A Statement by the Centers for Disease Control, April 1978, Atlanta, Georgia, Department of Health and Human Services, 1978.

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186 See U.S. Centers for Disease Control, Statement on Preventing Childhood Lead Poisoning, Atlanta, Georgia, 1991, pp.5-10.
191 Marshall E, Senate Considers Lead Gasoline Ban, Science 225:34-35, 1984. "The campaign has not been entirely successful, but neither has it failed, for it has kept leaded gasoline on the market longer than might have been expected."
the form of paint dust or chips."192 The Centers for Disease Control postponed a meeting in 1984 to update its policy statement on "Preventing Lead Poisoning in Young Children" after the LIA threatened to sue,193 but within the year lowered the threshold for "elevated" blood lead levels to 25 micrograms per deciliter.194 A further revision of the Statement, released in October 1991, lowered the threshold still further, to 10 micrograms.195

A protracted phasedown of leaded fuel began in the seven-tees.196Ironically, these regulations were initially developed to protect the newly developed catalytic converter in automobiles—a pollution-control device that happens to be rendered inoperable by lead—rather than to safeguard human health. Although the Task Force on Regulatory Relief targeted the lead gas restrictions for repeal in the early 1980s, vociferous objections from the public health and environmental advocacy communities convinced EPA to abandon its thoughts of weakening the standards; instead, the Agency strengthened them. The subsequent industry challenge to the substantive standards was unsuccessful.197 Indeed, the reviewing court concluded that the case against lead-gas was so strong that it would support restrictions even more severe than those developed by the EPA.198 Chicago became the first city to ban leaded gasoline in September 1984.199 Under the 1990 amendments to the Clean Air Act, a total ban on lead in most fuel is scheduled for the end of 1995.200

In April 1990, the Department of Housing and Urban Development issued the first federal guidelines for abatement of lead-based paint in public housing.201 A few months later, the presidential spaniel was diagnosed as poisoned. "Lead poisoning," said the President of the United States, "flaking the paint, licking her toes. The paint falls— you know, they’re re-doing the White House and she’s licking her feet and she’s ingested lead."202

198 705 F.2d at 531.

8. THE LEAD INDUSTRY, 1965-1992

From the time Congress began tumbling toward legislation in the early seventies, the lead producing and manufacturing industry has undergone some notable changes, with a greater emphasis on secondary recycled lead and a consolidation in mining and refining operations.

In the mid nineteen sixties, domestic mine production was about half what it had been twenty years earlier, but production of secondary lead more than doubled. Total lead consumption remained fairly constant at about 1.1 million tons annually.203

The basic configuration of mine ownership remained more or less stable during the early seventies. But in the wake of the federal lead gas restrictions of the late 1970s, there were, for the first time in decades, important changes.

In the far west, the major players were mostly familiar names: Bunker Hill Co., Hecla Mining Co., U.S. Smelting, Refining and Mining Co. and American Smelting and Refining Co. (ASARCO).204

In Missouri, St. Joe had celebrated its centennial by upgrading the Herculaneum plant and opening new mines, more than half of them within 50 or 60 miles of Herculaneum.205 There were expanding operations at Indian Creek and Viburnum, and the Fletcher mine in Reynolds County was opened in 1967. Yet another mine at Brushy Creek was to be operational in 1972. The Herculaneum plant was upgraded in 1969, bringing the company's productive capacity to 200,000 tons of pig lead per year.206 Missouri mines continued to dominate domestic production, but the Tri-State District of Oklahoma/Kansas/Missouri was out of the picture in terms of mining production.

Mining activity in the tri-state district that had been dominated by Eagle-Picher ended in the late fifties. After 1968, Eagle-Picher was no longer among the top lead producers, and after 1971, it was no longer carried as an entry in Walter Skinner's Mining Yearbook. The firm diversified and by 1977 it was divided into three groups: basic materials and chemicals, machinery and allied parts, and transportation products, including storage batteries.207 During the 1980s, Eagle-Picher became a defendant in thousands of lawsuits involving asbestos injury.208

The Bunker Hill Co. merged with Gulf Resources & Chemical Corp. (GRD) in 1968.209 Environmental woes hit Bunker Hill in 1975, when it was determined that the smelter at Kellogg, Idaho had contributed to extremely high lead levels in area children.210 Bunker Hill began a program to reduce emissions, but a lawsuit contended that the company's lead emissions had poisoned and permanently disabled nine children; in 1981, the company settled out of court in the $20 million case.211 That same year, 1981, "a decision was made to discontinue GRD's operations in the lead, zinc, and silver mining, smelting and refining business. Accordingly, immediate steps were taken which began the orderly shutdown of Bunker Hill Co.'s operations at Kellogg, Idaho."

A similar fate awaited Anaconda, which had enjoyed the output of copper, zinc, silver, lead, gold and manganese at the 4,000 acre site in Butte County, Montana.212 Atlantic-Richfield (ARCO) bought Anaconda in 1977 and "by 1983, having lost over $100,000,000 ARCO closed the mines."

The National Lead Co. changed its name to NL Industries in 1971.213 Big changes began after 1972 when Ray C. Adam came on board as chief operating officer. "At the time, the company had 70 major divisions. Today there are 26 fewer divisions, 5,000 fewer employees, and the company is over 7 times as profitable. NL Industries currently concentrates in metals, chemicals, and petroleum services and equipment. Restructuring of the company, which took 8 years, is almost complete." One of the divisions sold in the process was Dutch Boy paints, sold to ELT DNC in Baltimore.214

Several of St. Joe's principal mines continued in operation, but ownership of St. Joe had changed. In 1970, St. Joseph Lead Company changed its name to St. Joe Minerals Corporation,215 and a series of corporate mergers ensued. In August, 1981 St. Joe Minerals Corp. merged with Fluor Corp., based in Irvine, California.216 St. Joe and Homestake merged their lead operations in 1986,217 and established the Doe Run Co. which owned the St. Joe mines and the Herculaneum refinery.218 Homestake sold out to Fluor in 1950, which in turn expressed interest in selling Doe Run. American Metal Market reported that Fluor was planning to exit the lead business, and the consolidation would "make it easier for

"THE MOVE AWAY FROM LEAD PRODUCTION WAS PERCEIVED AS AN IMPORTANT ENVIRONMENTAL CONCERN."

206 Mining Yearbook, 1971, p.534.
Flour to find a buyer for Doe Run, which holds a 60-percent share of the domestic lead market. The move away from lead production was perceived as an important response to environmental concerns, and indicated that Doe Run was eyeing the secondary (recycled) market which had been growing steadily. In mid-1991 a $38-million secondary smelting with an annual capacity of 60,000 tons came on line near Boss, Missouri.221

As of 1990, Doe Run, together with ASARCO Inc. and Cominco American Inc., controlled 90% of domestic lead mining. All three of Doe Run’s mines are located in Missouri and its integrated smelter-refiner produced about 231,000 tons of refined lead.222 Missouri hosts most of ASARCO’s operations as well; producing 112,000 tons of lead in 1990.223 Cominco American Inc. operated one mine in Missouri that produced 87,000 tons of concentrate as a joint venture with Dresser Industries and opened a enormous new mine in Alaska which, after its official opening in February, processed 904,000 tons of ore, yielding 51,400 tons of lead concentrate.224

All told, Missouri continued to overshadow the other states in terms of lead mining. That state represented 78% of domestic production for 1990. Another 18% of the lead produced domestically was mined in Alaska, Colorado, Idaho, and Montana.225

Overall, secondary production (recycling) continues to outstrip primary production by more than two to one.226

The LIA acknowledged in 1984 that it hadn’t won a major victory over the Environmental Protection Agency in the past ten years, and the Association’s president, Werner Mayer, said “Our victories have been in the deferral of implementation of certain regulations.”227 Even so, the record of success by the lead industry and paint manufacturers in deterring regulations for well over half a century may well be unparalleled in this nation’s history.228

Julian Chisolm still supervises a Baltimore clinic that treats lead-poisoned children. More than twenty years after his

1971 Senate testimony, children are still being poisoned by the lead on decaying paint surfaces and in contaminated soils. After taking a visitor on a tour of his labs and talking about the frustrations of 35 years, Chisolm shrugged. “When the lead is all gone,” he said, “they’ll outlaw it.”