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

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After a great pain, a formal feeling comes –
The Nerves sit ceremonious, like Tombs –
The stiff Heart questions was it He, that bore,
And Yesterday, or Centuries before?

The Feet, mechanical, go round –
of Ground, or Air, or Ought –
A Wooden way
Regardless grown,
A Quartz contentment, like a stone –

This is the Hour of Lead –
Remembered, if outlived,
As Freezing persons, recollect the Snow –
First – Chill – then Stupor – then the letting go –

Emily Dickenson, The Complete Poems of Emily Dickenson, 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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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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[1] See generally, Centers for Disease Control. Preventing Lead Poisoning in Young Children. Atlanta, Georgia: U.S. Department of Health and Human Services, 1991.

[2] U.S. Environmental Protection Agency, Strategy for Reducing Lead Exposures, p. 5, 1991.

[3] Centers for Disease Control, Preventing Lead Poisoning in Young Children. Atlanta, Georgia: U.S. Department of Health and Human Services, 1991, p.18.

[4] U.S. Department of Housing and Urban Development. Comprehensive and Workable Plan for the Abatement of Lead-Based Paint in Privately Owned Housing -- Report to Congress. Washington, D.C.: U.S. Department of Housing and Urban Development, 1990, p.xvii.

[5] U.S. Agency for Toxic Substances and Disease Registry. Nature and Extent of Childhood Lead Poisoning in the United States -- Report to Congress. Washington, D.C. Department of Health and Human Services, 1988., p. II-5.

U.S. case was reported in 1914.[6] 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 automobiles[9] while industry-funded medical experts asserted that lead exposure was "harmless" and "normal."[10]

"A CHILD LIVES IN A LEAD WORLD." --1924

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.

7 Ruddock JC, Lead Poisoning in Children, Journal of the American Medical Association 82:1682-84, 1924.

8 See Rosner D and Markowitz G., A 'Gift of God'?: The Public Health Controversy over Leaded Gasoline during the 1920s, American Journal of Public Health 75:344-352, 1985.

9 Nriagy JO, The Rise and Fall of Leaded Gasoline, The Science of the Total Environment, 92: 13-28, 1990.

10 See section 3 below.

11 See section 5 below.

12 See section 7 below.

world and millions of children across the United States are poisoned as a result.

1. EARLY HISTORY OF THE LEAD INDUSTRY

Romance and adventure fills 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 malleability, 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.

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]

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13 Rickard TA, A History of American Mining, New York: McGraw-Hill Book Co., 1932.

14 Rose CH, *The Lead Pigments* in Mattiello JJ, Ed, Protective and Decorative Coatings Volume II, New York, John Wileey & Sons, Inc., 1942.

15 Trigg ET, The Paint and Varnish Making Industry in Philadelphia, Philadelphia, Chamber of Commerce Educational Committee, 1917, p.20.

16 Ingalls WR., Lead and Zinc in the United States, New York: Hill Publishing Company, 1908, p.viii-ix.

17 After a Centruy of Operation, St. Joseph Lead Co. Plans for the Next, Engineering and Mining Jorunal 165:87, 1964.

18 See Section 8 below.

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 1888[19] 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 spasm 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 ores and ingots could be shipped readily by rail to manufacturing centers and markets on both coasts and to numerous points in between.

WITH THE OPENING OF THE WEST AFTER THE CIVIL WAR, ASTONISHING DISCOVERIES OF MINERALS IN THE ROCKIES THRUST THE INDUSTRY INTO A SPASM OF DEVELOPMENT.

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 Hecla 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

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19 Gregory CF. A Concise History of Mining New York: Pergamon Press, 1980, p.134.

20 Mines Register, New York, 1946, p.106.

21 Holley CD. The Lead and Zinc Pigments, New York: John Wiley & Sons, 1909, p.23.

22 Ibid., p. 24.

23 Forbes 128: 40, 42, 1981.

24 Rickard TA. A History of American Mining, New York: McGraw-Hill Book Co., 1932, p.170. Rickard cites Hourwich, IA., The Making of America, VI:273, 1905.

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."[25] 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.[27] 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.[28]

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.252; see also Holley, CD. The Lead and Zinc Pigments, New York, John Wiley & Sons, 1909, p.30

27 Mining International Yearbook, 1975, 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 poison-

29 Turner JA., Lead Poisoning Among Queensland Children, Australasian Medical Gazette 16:475-479, 1897.

30 Gibson JL., A Plea for Painted Railings and Painted Walls of Rooms as the Source of Lead Poisoning Amongst Queensland Children, Australasian Medical Gazette 23:149-153, 1904.

31 Gibson, JL., Plumbic Ocular Neuritis in Queensland Children, British Medical Journal 2:1488-1490, 1908; Gibson JL., The Importance of Lumbar Puncture in the Plumbar Ocular Neuritis of Children, Transactions of the Australasian Medical Congress 2:750, 1911; Breinl A, Young WJ., The Occurrence of Lead Poisoning Amongst North Queensland Children, Annals of Tropical Medicine and Parasitology 8:575-590, 1914; Gibson JL., The Diagnosis, Prophylaxis and Treatment of Plumbic Ocular Neurit is, Medical Journal of Australia 2:201-204, 1917.

32 Council of the Queensland Branch, British Medical Association, An Historical Account of the Occurrence and Causation of Lead Poisoning, Medical Journal of Australia 1:148-152, 1922.

ing due to lead paint over three decades of clinical work. Although the Australian literature is regarded as the first authoritative documentation of the dangers associated with lead paint, important reports and warnings from the U.S. medical and scientific community in the 19th century associated non-industrial ingestion of lead with serious illness in children, and documented children's special vulnerability to the effects of lead.

In 1889, W. Glenn reported in Science on the work of D.D. Stewart in discovering the cause of the outbreak of lead poisoning in Philadelphia.[33] According to Glenn, whose purpose it was "to spread wide the facts" about the toxicity of lead pigments, the Philadelphia experience with chrome yellow on baker's buns during 1887 established that such pigments "are often the sources of lead poisoning, which may exist largely in a community and yet escape detection." Medical writers frequently cited Stewart's 1895 report of the bakers' buns incident, "Lead Convulsions," as an important early contribution to the literature of lead poisoning and its effects on the central nervous system, since he demonstrated the greater vulnerability of children to ingested lead pigment (six out of seven with convulsions died).[34]

"WE WERE MUCH PUZZLED AS TO THE SOURCE OF THE LEAD, UNTIL HE WAS FOUND WITH HIS MOUTH COVERED WITH WHITE LEAD PAINT WHICH HE HAD BITTEN FROM THE RAILINGS OF HIS CRIB."

The first United States report of a childhood case -- and fatality -- from lead paint came from the Harriet Lane Home of the Johns Hopkins Hospital. At the May, 1914 meeting of the American Neurological Association, H.M. Thomas and K.D. Blackfan described the case of a five year old boy who was admitted comatose with recurrent convulsions and treated for serous meningitis.[35] He recovered and seemed fine but returned to the hospital five months later with the same symptoms. After a more rigorous examination, the physicians finally noted a slight discoloration on a tooth which proved to be a "lead line."[36] Examination of the blood revealed the stippling that is characteristic of lead poisoning. "We were much puzzled as to the source of the lead, until he was found with his mouth covered with white lead paint which he had bitten from the railings of his crib." The child died a few weeks later.

Blackfan followed up in 1917 with another report on lead poisoning in four children, three of whom died.[37] One of these was

33 Glenn W., Chrome Yellow Considered as a Poison, Science 13:347-349, 1889.

34 Stewart DD., Lead Convulsions, American Journal of Medical Science 109:288-306, 1895.

35 Thomas HM and Blackfan KD., Recurrent Meningitis, Due to Lead, in a Child of Five Years, American Journal of Diseases of Children 8:377-380, 1914.

36 One of the classic signs of high dose lead poisoning is the "Burtonian Line," a thin bluish line along the gums, a secretion of lead sulphide, first described by Sir Henry Burton in 1840. Burton H., On A Remarkable Effect upon the Human Gums Produced by the Absorption of Lead, Medico-Chirurgical Transactions 23:64-79, 1840.

37 Blackfan KD., Lead Poisoning in Children, American Journal of Medical Science, 153:877-887, 1917.

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 recently 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 Pediatrics[39] 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, buns, milk that had stood in lead containers, and medication.

"THAT LEAD, IN CONTINUED SMALL DOSES, IS A CAUSE OF DISEASE AND DEATH, IS THE ACCUMULATED TESTIMONY OF TWO THOUSAND YEARS, YET THE METAL IS USED WHERE IT IS MOST DANGEROUS..."

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.

40 Holt, LE and Howland J., The Diseases of Infancy and Childhood, 8th Edition, New York: D. Appleton-Century Co., 1923.

41 Holt LE., Lead Poisoning in Infancy, American Journal of Diseases of Children 25:229-233, 1923.

42 The New York Times, December 31, 1924, p.6:2.

43 Weller CV., Some Clinical Aspects of Lead Meningo-Encephalopathy, Annals of Clinical Medicine 3:604-613, 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 ointments, and lead paints.

Shortly afterwards, in 1926, C.F. McKhann at Harvard Medical School reported seventeen cases of lead poisoning and observed that the eating of lead paint was frequently associated with pica, an unnatural craving for articles of food. Pica, from the Latin word for 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," McKhann 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 characterized 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 (1656) 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 concerning intellectual lesions . . . The nature of the impression of lead upon the encephalon . . . completely evade[s] 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 through affected mothers to the offspring."[51] His descriptions 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

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44 McKhann CF. *Lead Poisoning in Children*, American Journal of Diseases of Children 32:386-392, 1926.

45 Thomas HM and Blackfan KD, Recurrent Meningitis, Due to lead, in a Child of Five Years, American Journal of Diseases of Children 8:377-380, 1914. Graham EE. Diseases of Children, New York, Lea and Febiger, 1916, p.839. Blackfan KD., Lead Poisoning in Children, American Journal of Medical Science 153:977-887, 1917. Strong RA., Meningitis Causes by Lead Poisoning, in a Child of Nineteen Months, Archives of Pediatrics 37:532-537, 1920. Abt AI., Pediatrics W.B. Saunders Co., VII:246-249, 1923. Holt LE and Howland J., The Diseases of Infancy and Childhood, 8th Edition, New York: D. Appleton-Century Co., 1923, and 9th Edition, 1926. Holt LE, Lead Poisoning in Infancy, American Journal of Diseases of Children 25:229-233, 1923.Ruddock JC., Lead Poisoning in Children, Journal of the American Medical Association 82:1682-1684, 1924. "A Child Lives in a Lead World." Weller CV and Christensen AD., The Cerebrospinal Fluid in Lead Poisoning, in The Human Cerebrospinal Fluid, New York: Paul Hoeber, Inc., 1924, pp 266-269. Aub JC, Fairhall LT, Minot AS, Reznikoff P., Lead Poisoning, Medicine 4:1-250, 1925. Weller CV and Christensen AD, The Cerebrospinal Fluid in Lead Poisoning, Archives of Neurology and Psychiatry 14:327-345, 1925. Weller CV., Some Clinical Aspects of Lead Meningo-Encephalopathy, Annals of Clinical Medicine, 3:604-613, 1925. Holloway LW., Lead Poisoning in Children, Journal of the Florida Medical Association 13:94-100, 1926. McKhann CF., Lead Poisoning in Children, American Journal of Diseases of Children 32:386-292, 1926.

46 Tanquerel des Planches L., Lead Diseases, Dana SL, translator and editor, Lowell, Massachusetts: Daniel Bixby and Co., 1848.

47 Tanquerel des Planches L., op. cit. p265-266.

48 Ibid., p.324.

49 Ibid. p.49.

50 In a lengthy Appendix, p.352-422, derived from his own substantial experience with lead poisoning, Dana stated that poisoning from lead ingested by children from lead water pipes, not an uncommon phenomenon in northern New England. "Lead," wrote Dana, "in continued small doses, is a slow poison."

Dana included a report to the City Council of Lowell, Massachusetts, concerning lead water pipes which stated: "That lead, in continued small doses, is a cause of disease and death, is the accumulated testimony of two thousand years, yet the metal is used where it is most dangerous. Men are roused to a sense of its danger, only when the frequency of the disease, attended often by a fatal termination, has spoken in tones which chill the hearts of the bereaved, and alarm the living sufferers by well grounded fears, that the seeds of deathly disease may be germinating in their constitutions." Dana continued: "...there is reason to believe that a vast many cases of rheumatic and spasmodic and nervous disease, a general breaking up, as it were, of the foundations of the great deep of life, have occurred, which can be attributed only to the effects of small, daily doses of lead."

Dana made an especially poignant appeal concerning children: "In this sense of the constitution...of that agent called the vital force, it is found by experience, that the young, the delicate, soonest succumb, under the effects of lead drank [sic] in their daily drink, and like the dews of heaven, descending on all, the gentlest and fairest, first feel the chill which soon closes in death."

51 Oliver T., Lead Poisoning and the Race, British Medical Journal 1:1096-1098, 1911.

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]

"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."

In Britain, lead poisoning became a cause celebre, especially after vivid depictions of the suffering of women working in white lead factories from Charles Dickens[53] 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

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.39: "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."

53 Dickens, C., The Uncommercial Traveller: A Collection of Short Stories, London, T. Nelson & Sons, 1861.

54 Holley CD., The Lead and Zinc Pigments, New York, John Wiley & Sons, 1909, p.143.

55 Holley CD.,Ibid.

56 Oliver T Lead Poisoning: From the Industrial, Medical And Sociial Points of View, New York, Paul B. Hoeber, 1914, p.56.

57 Oliver T Ibid. p. 56.

58 Oliver T Ibid. p. 57.

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.

59 Lead-Poisoning, Editorial in Mining and Scientific Press 123:491-492, 1921.

60 International Labour Office, White Lead, Studies and Reports Series F (Industrial Hygiene), No. 11, Geneva, 1927, p.52. Austria, 1924; Belgium, 1926; Bulgaria, 1925; Chile, 1925; Czechoslovakia, 1923;Estonia, 1922; France, 1926; Latvia, 1924; Poland, 1924; Rumania, 1924; Spain, 1924; Sweden 1923.

61 International Labour Office, op. cit., p.36.

3. "HARMLESS," "NORMAL," AND SAFE

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. Kehoe, M.D., medical director of the Ethyl Corporation (a joint venture between General Motors and DuPont) and director of the Kettering Laboratory of Applied Physiology 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 $52,500 from leading lead manufacturers for a three-year study. What was even more astounding 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]

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62 Superb descriptions of the campaign to introduce lead into U.S. automobile fuel have been prepared by Rosner and Markowitz, and by Graebner. Rosner D and Markowitz G., A 'Gift of God'?: The Public Health Controversy over Leaded Gasoline during the 1920s, American Journal of Public Health 75:344-352, 1985. Graebner W., Private Power, Private Knowledge, and Public Health: Science, Engineering, and Lead Poisoning, 1900-1970, in Bayer R, ed. The Health and Safety of Workers: Case Studies in the Politics of Professional Responsibility, New York, Oxford University Press, Inc., 1988, pp.15-71.

63 Ibid.

64 Graebner 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.

Aub had recruited a team and started publishing papers in 1922, culminating in the classic Lead Poisoning which appeared in the journal Medicine in 1925[67], and in 1926 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.

ON A GOOD DIET, LEAD WOULD JUST TAG ALONG WITH CALCIUM TO THE BONES WHERE IT WAS ESSENTIALLY "HARMLESS."

To his credit, Aub was helpful in pointing out the risks to workers of 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, deferring 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]

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67 Aub JC, Fairhall LT, Minot AS, Reznikoff P. Section XX by Alice Hamilton, Lead Poisoning, Medicine 4:1-250, 1925.

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

69 Apparently a predecessor of the Lead Industries Association. Aub, JC et. alia, op. cit. 1926, p.x.

70 Aub JC et. alia, op. cit. 1925, p.68: "...after absorption has ceased, significant amounts of lead are to be found only in apparently harmless deposits in the bones."

71 Reich P., How Pasteurization Became Public Policy, Hoard's Dairyman, March 10, 1982.

72 Sollman T., Studies of Chronic Intoxication on Albino Rats. VI. Lead Carbonate, Journal of Pharmacology and Experimental Therapeutics 19:375-384, 1922.

Buttressing 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 1926, 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.[73] 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.

IN EFFECT, HE SIMPLY DECLARED THAT THE LEVEL OF LEAD INGESTION VIEWED AS POISONOUS BY EXPERTS ON TWO CONTINENTS WAS "NORMAL."

Kehoe's contribution during the early 1930s was an important estimate of "normal" daily intake (.20-.35 mg/day) and excretion (.25-.30mg/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."[74] 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.[75]

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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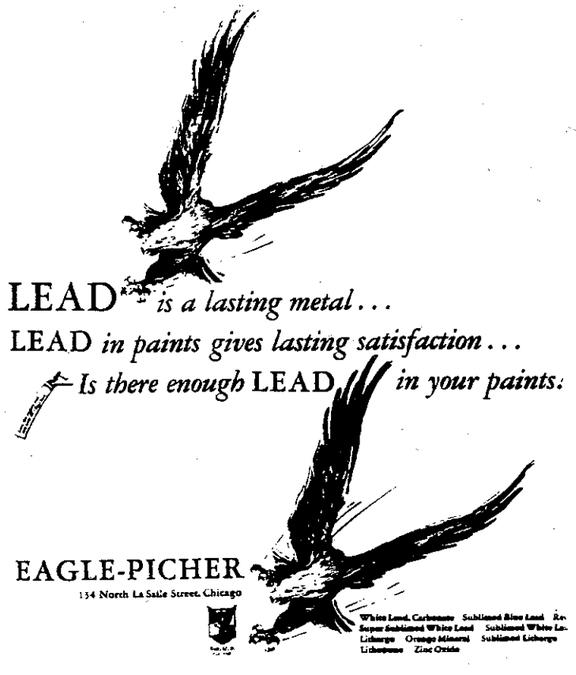
73 Kehoe RA., On the Normal Absorption and Excretion of Lead, Part I, Journal of Industrial Hygiene 15:257-272, 1933; Part II, 15:273-288; Part III, 15:290-300; Part IV, 15:301-305.

74 Kehoe RA, Edgar G, Thamann F, and Sanders L, The Excretion of Lead by Normal Persons, Journal of the American Medical Association, 87:2081-2084, 1926.

75 Aub JC et. alia, op. cit. 1926, p.70.

Harvard posed a formidable obstacle to anyone who wanted to learn more about lead poisoning for children *or* adults. In this benevolent climate, the lead industry proceeded to pump more lead than ever into the environment.



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."[76] 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.

"...EVERYWHERE PEOPLE ARE LEARNING THE IMPORTANCE OF PAINT PROTECTION, THE WISDOM CONTAINED IN SEVEN SHORT WORDS -- 'SAVE THE SURFACE AND YOU SAVE ALL.'"

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, enhance 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 paint 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

76 Gardner HA, Paint Technology and Tests, New York, McGraw-Hill Book Co., 1911, p.252.
77 Heckel GB., The Paint Industry, Reminiscences and Comments, St. Louis, the American Paint Journal Company, 1931, p.478-79.
78 March 25, 1922, p.5.

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 birght 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-Pitcher also ran full page ads touting white lead. With an irony now painful to contemplate, the ads 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 Public is "Lead-Conscious"

All intelligent paint buyers recognize white lead as the measure of paint value. For generations, from father to son, this belief—based on countless examples of white lead's long, economical surface protection—has been passed along. Why not cash in on this "lead consciousness"? Make it known that yours is a paint with a high lead content. Help your dealers reduce sales resistance—for quicker, easier profits.

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 basis 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

79 April 8, 1922, p.68.
80 November 3, 1923, pp. 144-145.
81 American Paint Journal, April 28, 1930, p.65.
82 Chemical Review, 89:1, 1930.
83 American Paint Journal, April 21, 1930, p.3.

paint for interior localities than basic carbonate white lead paint . . ." stated G.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, P.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]

THROUGH THE 1920S, THE NATIONAL BUREAU OF STANDARDS CONSISTENTLY RECOMMENDED WHITE LEAD FOR USE IN SCHOOLS.

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 Pigments 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]

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84 Hickson EF, Chemist, National Bureau of Standards, to George F. Wormrath, Board of Education, Minneapolis, Minnesota, May 30, 1930. National Archicves and Record Administration RG 167, Box 330.

85 Walker PH, Memorandum for Files, September 24, 1928, National Archives and Records Administration RG 167, Box 215A.

86 Letter from Office of Building and Grounds to a Baltimore firm, May 9, 1923, National Archives and Records Administration, RG 167, Box 42.

87 Hiers GO. and Rose CH., Lead in Building and Construction, Industrial and Engineering Chemistry 27:1133-1135, October 1935.

88 Dutch Boy Quarterly 15(2):14, 16, 1937.

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]

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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. 5535. [

90 LIA Annual Meeting (Jan. 24, 1939), Ibid.p. 5000, and LIA Annual Meeting (January 17, 1940), Ibid. p. 5355..

91 Meyer HM and Mitchell AW., U.S. Department of the Interior, Lead and Zinc Pigments and Zinc Salts, Minerals Yearbook, 1940, Washington, D.C., U.S. Government Printing Office, 1940, pp.152.

92 Ross WA., (Federal Security Agency) and Crutchfield D., (Lead Industries Association), Painting Farm Buildings and Equipment "Prepared and Published by Lead Industries Association in cooperation with the U.S. Office of Education, Federal Security Agency, 1943". Thanks given to "specialists in the U.S. Bureau of Standards," iv. LC No. TT305.R6.

93 Federal Trade Commission, In the Matter of National Lead Co., et al., Docket No. 5253, Trial Examiner's Recommended Decision of March 31, 1948, p.814.

94 Santiago v. Sherwin Williams, Civil Action 87-2799-T (D. Mass.),Plaintiff's Statement of Undisputed Facts Relevant to Plaintiff's Motion for Partial Summary Judgement (filed June 10, 1991), p.19.

95 Glidden Painting Specifications, (1951), GLD 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[96], and titanium had also become a significant substitute pigment. The U.S. Government published standards for white lead in 1919,[97] zinc oxide in 1922,[98] and titanium dioxide in 1924.[99] 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]

THE STRATEGY OF THE LIA FOR DECADES WAS THREE-FOLD: TO OPPOSE REGULATION OF LEAD, TO MINIMIZE THE SIGNIFICANCE OF LEAD POISONING, AND TO PROMOTE LEAD PRODUCTS.

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-Picher, 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 three-fold: 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

96 "...within the last twenty-five years, oxide of zinc gradually gained recognition among manufacturers and users of paint as being equally appropriate for the purposes for which theretofore carbonate of lead had alone been recognized as appropriate, and has come to be universally conceded as possessing important useful qualities as a white pigment not possessed by carbonate of lead..." Heath and MIlligan Manufacturing Co.v. Worst, 207 U.S. 338, 341 (1907) (concerning the North Dakota formula labelling law of 1906).

97 Interdepartmental Committee on Paint Specification Standardization, Recommended Specification for basic Carbonate White Lead, Dry and Paste, Washington, D.C., Government Printing Office, 1919.

98 Federal Specification Board, United States Government Specification for Zinc Oxide, Dry and Paste, Standard Specification No. 8, Washington: Government Printing Office, 1922.

99 Bureau of Standards, Department of Commerce, United States Government Specification for Titanium Pigment, Dry and Paste, Circular of the Bureau of Standards, No. 163, Standard Specification No. 115, 1924.

100 Robinson AHA., Titanium. Mines Branch, Canada Department of Mines, Ottawa: F.A. Acland, 1922, p.8.

101 Federal Trade Commission, In the Matter of National Lead Co., et al., Docket No. 5253, Findings as to the Facts and Conclusions, p. 1248.

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 spokesmen 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 labelling 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.[108] 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:

102 Wormser FE., "Cadmium," in Spurr JE and Wormser FE, eds., The Marketing of Metals and Minerals, New York, McGraw-Hill Book Co., 1925.

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

104 Trigg ET., Fifty-Five Colorful Years, The Story of Paint in America Stonington, Connecticut, The Pequot Press, 1954, 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.

105 "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 paint bill 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.321-23.

106 Heckel, GB. op. cit., p.328-33; 207 U.S. 338 (1907).

107 Santiago v. Sherwin Williams, Civil Action 87-2799-T, D. Mass., Plaintiff's Statement of Undisputed Facts Relevant to Plaintiff's Motion for Partial Summary Judgement (filed JUne 10, 1991)p.132.

108 E.g., Report of Committee on Legislation, Oil, Paint and Drug Reporter, 108:23, 25, 1925.

> "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 official 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."[109]

The relationship with Bowditch would endure for many years and he eventually became Director of Health and Safety for the LIA.[110]

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.

"...CHRONIC LEAD POISONING IN INFANCY AND CHILDHOOD IS BY NO MEANS A RARE CONDITION." --1930

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 traveller 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.[111] In a report later published by the U.S. Bureau of Labor, Hoffman in 1927 painted a gloomy picture of child health and safety.[112]

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.

109 LIA Annual Meeting (June 5, 1934), Federal Trade Commission, In the Matter of National Lead Co., et al., Docket No. 5253, p. 5535.

110 M. Bowditch Letter to Joseph Aub, June, 1950. Aub Papers, Homes Hall, Countway Medical Library, Box 5, folder entitled "Lead-Litigation - 1958".

111 Hoffman FL., The Frederick Ludwig Hoffman Papers, Rare Book and Manuscript Library, Columbia University Libraries, Box 12, Item 40.

112 Hoffman FL., Deaths from Lead Poisoning, Bulletin No. 426, Bureau of Labor Statistics, Washington, Government Printing Office, 1927.

The other source of statistical information was Louis I. Dublin at the Metropolitan Life Insurance Co.. Dublin administered a survey of "prominent pediatrists" in 1930 and published results in the firm's bulletin.[113] 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 [McKhann] 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 in to deal with both McKhann's medical report and Dublin's survey. In a 1933 follow-up to his earlier articles on lead poisoning in children, McKhann 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.[114] 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."[115]

THE LIA DECLARED AT ITS DECEMBER 1946 MEETING THAT IT WOULD SEEK TO CONVINCE THE GOVERNMENT TO USE LEAD MORE LIBERALLY IN GOVERNMENT BUILDINGS.

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.[116] 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

113 Metropolitan Life Insurance Co., Chronic Lead Poisoning in Infancy and Early Childhood, Statistical Bulletin 11(10):4, 1930, pp.4-5.

114 McKhann CF. and Vogt EC., Lead Poisoning in Children, Journal of the American Medical Association 101:1131-1135, 1933. "The lead industry and the manufacturers 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, 1933. National Archives and Record Administration, RG 102, Box 498, File # 4-5-17.

116 Reported in Hoffman, F.L., Lead Poisoning Legislation and Statistics, Prudential Press, 1933. Columbia University, Rare Book and Manuscript Collection, Box 19, Item 149.

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 labelling 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. Damm, 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. Damm 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

117 For example, in a July 1953 "Report of the President to the Executive Committee," Joseph F. Battley, President of the NPVLA 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 base 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 painted surfaces." National Archives and Record Administration, RG 102, Box 609, File # 4-5-17.

118 LIA Directors Meetings (Dec. 12, 1946), Federal Trade Commission, In the Matter of National Lead Co., et al.,Docket No. 5235, p.5692.

119 Oil, Paint and Drug Reporter, October 21, 1935, 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 #3, File PWA. Undated. Estimated to be 1934-1937.

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

lead poisonings in Chicago,[122] Wormser and the LIA made an inquiry. Wormser later reported that "[o]n 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 Aub was an essential ally, advising Wormser when reported cases were probably not lead poisoning. In 1940, Aub wrote a letter to the editor of the prestigious Journal of the American Medical Association protesting a recently published report on lead poisoning. "I write this vigorous letter," he stated, "because too much publicity is being given to minor exposures to lead these days. This is particularly true with regard to lead taken by mouth, which is so much less toxic than inhaled lead. That lead may produce deleterious effects there can be no question, but there is no evidence that a small fraction of a milligram can produce the effects ascribed to it . . . ."[126]

Aub served as the outside expert on cases of occupational and childhood lead poisoning referred to the Lead Industries Association. For example, Wormser forwarded to Aub 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." Aub's analysis: the man "died of peritonitis, probably starting somewhere around the appendix . . . these are the things he died from."[127]

Twice in 1945 Wormser wrote Aub asking the Harvard physician's opinion on cases of childhood lead poisoning and Aub 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 paralysis fit in with a possible diagnosis of lead poisoning. Still I am suspicious of the fact that

122 Lead Toys -- Lead Paint -- Lead Poisoning, Safety Education 22:74, 1942.

123 Wormser FE., Facts and Fallacies Concerning Exposure to Lead, Occupational Medicine 3:155-144, 1947.

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 100:1485-1489, 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 labelled "the depression disease." In view of the widespread poisonings over two winters, the City of Baltimore undertook vigorous protective 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 23:1943.

126 Aub JC., Lead as a Hazard (Letter), Journal of the American Medical Association 114:2237, 1940.

127 Aub JC to Wormser FE, Aub Papers, Holmes Hall, Countway Medical Library, Aub JC, Archives, Box 5.

"TOO MUCH PUBLICITY IS BEING GIVEN TO MINOR EXPOSURES TO LEAD THESE DAYS."
--LEAD INDUSTRY SPOKESMAN, 1940

this child died of an infection, just barely possibly of a meningococcus."[128] In the case of a ten month-old infant who died in Texas in July, 1945, the medical report showed a leadline, anemia, and stippling 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."[129] 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 Byers 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," Byers 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

128 Ibid.
129 Ibid
130 LIA Executive Committee (Jan. 15, 1943), in Federal Trade Commission, IN the Matter of National Lead Co., et al., Docket No. 5253, p. 5355.
131 Byers RK and Lord EE, Late Effects of Lead Poisoning on Mental Development, American Journal of Diseases of Children 66:471-494, 1943.
132 TIME, December 20, 1943, p.49.
133 The Children's Bureau, "Paints, Pigments and Dyes," U.S. Dept. of Labor, Washington, D.C., December 1945, CB 46-352, National Archives and Records Administration, RG 102, Box 103, File No. 4-5-17.

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 withdrew 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 AMA 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.

134 Federal Trade Commission, (April 26, 1946),In the Matter of National Lead Co.,et al., Docket No. 5253, p. 5692.
135 LIA Executive Committee (Dec. 28, 1945), Federal Trade Commission, In the Matter of National Lead Co.,et al., Docket No. 5253, p. 5435.
136 [Glass Menagerie] Wormser FE to Aub JC, February 4, 1946. Aub Papers, Holmes Hall, Countway Medical Library, Aub JC, Archives, Box 5.
137 Meeting Minutes February 7, 1946. Aub Papers, Holmes Hall Countway Medical Library. Aub JC, Archives, Box 5.
138 P. Drinker to Harvard School of Public Health Deans Huber and Burwell, Feb 12, 1946. Aub Papers, Holmes Hall, Countway Library, Boston, Massachusetts, Archives, Box 5, folder "Lead 1927-1965".
139 LIA Annual Meeting Reports, 1929-1941, FTC.

"THE LEAD HAZARD IN INDUSTRY AND TO THE PUBLIC IS RELATIVELY SMALL AND CAN BE EFFECTIVELY CONTROLLED WHEN IT CANNOT BE ELIMINATED." --LEAD INDUSTRY SPOKESMAN, 1946.

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."[140]

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 quantitative 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"[141] -- the latter figure twice as high as the upper limit he had described in 1933.[142]

KEHOE CHOSE TO OMIT REFERENCES TO LEAD PAINT POISONING THAT WERE CLEARLY IN POSSESSION OF HIS LABORATORY.

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.[143] Aub referred queries about lead poisoning to the American Public Health Association position papers.[144] 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, McKhann, Blackman and others, making it clear that the Kettering Laboratory had access to the core literature on childhood lead poisoning.[145]

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140 Wormser FE., Facts and Fallacies Concerning Exposure to Lead, Occupational Medicine 3:135-144, 1947.

141 Kehoe RA,. Exposure To Lead, Occupational Medicine 3:156-171, 1947.

142 Kehoe RA, On the Normal Absorption and Excretion of Lead Part I, Journal of Industrial Hygiene, 15:257-272, 1933.

143 American Public Health Association Year Book, 1935-1936, American Journal of Public Health, Supp. 26:25, 1936; American Public Health Association Year Book, American Journal of Public Health, Supp. 31:36, 1941.

144 Aub JC. Letter to AMW Hursh, 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.

145 Conway N., Lead Poisoning, Industrial Medicine 9:471-477, 1940.

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.[146] 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.

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146 Committee on Lead Poisoning of the Industrial Hygiene Section of the American Public Health Assocaition, 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 lead" pigments, which were widely used in automotive and transportation paints (not necessarily red in hue).

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.

147 Mines Register, New York, 1946, p.199.

148 Mines Register, New York, 1946, p.106.

149 Mines Register, New York, 1946, p.34.

150 Mines Register, New York, 1946, p. 31-32 and p. 571.

151 Ibid.P. 571.

152 Mines Register, New York, 1946, p. 199. National Lead Co. "is said to do about 50% of the lead products business in America."

153 There were twelve white lead plants in 1945: Eagle-Picher Co., Cincinnati, Ohio; Euston Lead Co., Scranton, PA; W.P. Fuller & Co., San Francisco, Calif.; International Smelting & Refining Co., East Chicago, Ind.; John T. Lewis & Bros. Co., Philadelphia, Pa.; National Lead Co., Melrose, Ca., Chicago, Ill. (2), St. Louis, Missouri, Perth Amboy, N.J., Brooklyn, N.Y.;The Sherwin-Williams Co., Chicago, Ill. Meyer HM and Mitchell AW. Lead and Zinc Pigments and Salts, Minerals Yearbook 1945, U.S. Bureau of Mines, Washington, D.C.: Government Printing Office, 1945, p.213.

Along with the white lead businesses that controlled the Lead Industries Association, manufacturers of the other lead pigments joined the fight to stave off adverse reports. Red lead continued to be produced in amounts greatly exceeding white lead.[154] Although the red lead people were not as organized as the white lead industry, they too took up the case against the Children's Bureau.

Soon after publication of the 1946 symposium papers,[155] U.S. paint and pigment manufacturers developed a strong campaign to quash the Children's Bureau warning. Beginning in June 1948, several firms began a letter-writing campaign with the apparent goal of protecting their markets. The campaign ended with removal of the publication from circulation.

Documents from the Children's Bureau reveal that during the summer of 1949, numerous firms corresponded with Dr. Marian M. Crane, assistant director of the Division of Research in Child development.[156] The correspondence shows that by the end of the summer, Dr. Crane was worn down by the technical questions posed to her by the various paint companies and perhaps by superiors.

A letter from Dr. Crane to P. E. Sprague, vice-president of The Glidden Company summarizes the position of the Children's Bureau after a summer of tiptoeing through a mine field of complex pigment terminology:

> Over a period of years the Children's Bureau has received many inquiries from parents regarding types of paint that are safe to use on children's toys, furniture, etc. For this reason, in 1945 we undertook to prepare a statement that could be used in answering such questions ... the fact that the Children's Bureau published this statement resulted in technical questions being referred

154 Mote, RH., Lead, Minerals Yearbook 1945, U.S. Bureau of Mines, Washington D.C.: Government Printing Office, 1945, p.164.

155 See Section 5, above.

156 National Archives and Records Administration, RG 102, Box 398, File # 4-5-17. Letters in the file are from F.A. Putnam Mfg. Co., Inc., Keene, N.H. (May 13, 1949); Holland Color and Chemical Company, Holland, Mich., (June 6, 1949); the National Paint, Varnish and Lacquer Association, Washington, D.C. (June 24, 1949); The Glidden Company, Cleveland, Ohio, (June 13, 1949); E. I. Dupont de Nemours & Company, Wilmington, Del., (July 12, 1949).

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 HANDFUL OF CITIES AND STATES HAD ADOPTED LEAD PAINT LEGISLATION OR REGULATION.

157 Crane MM, Assistant Director, Division of Research in Child Development, The Children's Bureau to P.E. Sprague, Vice President, The Glidden Company, September 22, 1949. National Archives and Records Administration, RG 102, Box 398, File #4-5-17.

158 Williams H, Kaplan E., Couchman CE, Sayers RR., Lead Poisoning in Young Children, Public Health Reports 67:230-236, 1952.

159 Byers RK., Urinary Excretion of Lead in Children, American Journal of Diseases of Children 87:548-558, 1954.

160 Chisolm JJ and Harrison HE., The Exposure of Children to Lead, Pediatrics 18:943-958, 1956; Chisolm JJ and Harrison HE., The Treatment of Acute Lead Encephalopathy in Children, Pediatrics 19:2-20, 1957.

161 McLaughlin MC., Lead Poisoning in Children in New York City, 1940-1954, New York State Medical Journal 56:3711-3714, 1956.

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

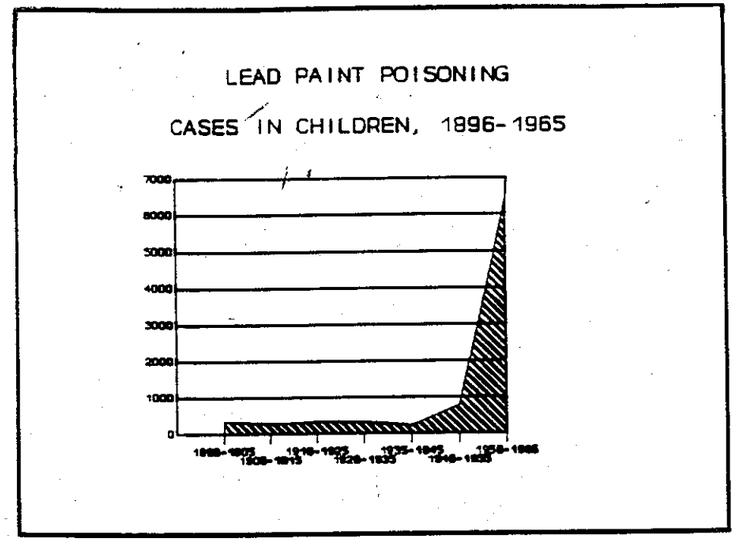

Figure 1

The first response to the crisis was a voluntary -- and unenforceable -- national standard for paint to be used on children's toys and interior surfaces. Adopted by the American National Standards Institute (ANSI) in 1955, the standard called for a lead content of less than one percent of the total weight of solids in surface coatings.[163] By the mid-fifties, as pressure from consumer groups and physicians increased, a handful of cities and states had adopted lead paint legislation or regulation.[164]

Meanwhile, industry continued through organized symposia to downplay the risks of any type of lead exposure. In 1963, Kehoe convened a lead symposium at the University of Cincinnati College of Medicine. Joining Kehoe on the symposium agenda was a relative newcomer to the issue, Robert L. Ziegfeld, who had represented the LIA as an alternate on the committee that developed the ANSI standard in the mid-fifties. In his 1963 presentation, Ziegfeld noted that lead consumption in 1962 was 1,077,000 short tons, of which 16% went into tetraethyl lead gasoline additives, 36% to storage batteries, and less than three percent to paints and varnish.[165] Only 11,000 tons of white lead were consumed. He stated emphatically that "None of the white lead pigments is today employed in the manufacture of indoor paints."

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.

164 In 1954, New York City required a warning label on paint containing more than 1% of lead. Sanitary Code, chapter 22, para. 230d. Baltimore barred the use of leaded paint for residential interiors in 1951, and required labeling of lead paint in 1958. See Agency for Toxic Substances and Disease Registry, Report to Congress, op. cit., p. I-26.

165 Ziegfeld RL., Importance and Uses of Lead, Archives of Environmental Health 8:202-221, 1964.

Sticking by his figures from 30 years earlier, Kehoe 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 .03 mg/day.[166] 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 50 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."

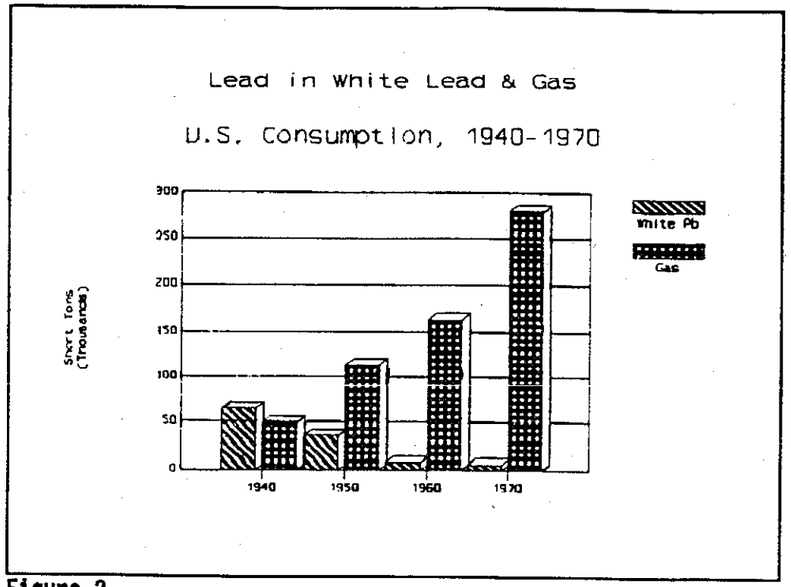

Figure 2

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 white 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.

166 Kehoe RA., Normal Metabolism of Lead, Archives of Environmental Health 8:232-235, 1964.

7. LEGISLATION & REGULATION 1965-1992

A 1965 paper by geochemist Clair Patterson of the Massachusetts Institute of Technology seriously challenged Kehoe's "lead is normal" doctrine and -- eventually -- triggered federal controls. Carefully documenting the tonnage of lead dispersed into the environment from industrial sources, automobiles, contamination 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.[167] This calculation was later confirmed.[168]

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 0.002 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.

"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."[169]

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

167 Patterson CC., Contaminated and Natural Lead Environments of Man, Archives of Environmental Health 11:344-360, 1965.

168 Ericson JE, Shirahata MS, Patterson CC., Skeletal Concentrations of Lead in Ancient Peruvians, The New England Journal of Medicine 300:946-951, 1979; Piomelli S, Corash L, Corash MB, et al., Blood Lead Concentrations in a Remote Himalayan Population, Science 210:1135-1136, 1980; Flegal AR, Smith DR, Lead Levels in Preindustrial Humans (Letter to the Editor), New England Journal of Medicine 326: 1293-1294 (1992).

169 Ziegfeld RL. Importance of Lead (Letter), Archives of Environmental Health 12:134, 1966.

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 [Kehoe]. The press asked him what his salary was and who paid it 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]

"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."

Patterson's article ignited 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

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."

171 Fuller, W.R., Understanding Paint, St. Louis, The American Paint Journal Company, 1965, p.19.

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 were 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 6 to 28 percent of urban children had blood lead levels greater than 50 ug/dl, above the 40 ug/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 legislatiors 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 intercurrent infectious illness . . .[176]

172 Smith HD., Pediatric Lead Poisoning, Archives of Environmental Health 8:256-261, 1964.

173 Perlstein MA. and Attala R., Neurologic Sequelae of Plumbism in Children, Clinical Pediatrics 5:292-298, 1966.

174 Lead-Based Paint Poisoning, Hearing Before the Subcommittee on Health of the Committee on Labor and Public Welfare, November 23, 1970. Washington, U.S. Government Printing Office, 1970, p.1-2 (Statement of Senator Ralph W. Yarborogh).

175 Public Health Service, Bureau of Community Environmental Management, Control of Lead Poisoning in Children (pre-publication draft) Washington, D.C., Department of Health, Education and Welfare. Dec. 1970, p.2.

176 Lead-Base 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."[177]

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."[178] The responsibility of prohibiting "the application of lead-based paint to any toy or furniture article" was given to the Consumer Product Safety Commission.[179]

"NONE OF THESE SYMPTOMS ARE SPECIFIC FOR LEAD POISONING, SO THAT THEY ARE OFTEN ATTRIBUTED TO OTHER DISEASES."

However, funding fell far short of expectations[180] 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,[181] 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.[182] 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.[183]

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,[184] this time to 30 ug/dl.

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177 Ibid. p.220-21.

178 42 CFR Section 4831.b.

179 42 CFR Section 4831.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."

181 U.S. Comptroller General, HUD Not Fulfilling Responsibility to Eliminate Lead-Based Paint Hazard in Federal Housing, Washington D.C., U.S. General Accounting Office, 1980.

182 Ashton v. Pierce, 726 F.2d 70 (D.C. Cir. 1983)

183 The New York Times February 25, 1975, p.15:1.

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.

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.[185] 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.[186] 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."[187] Further attacks on Needleman's work were pressed -- with a conspicuous lack of success -- in 1991-92 by two scientists with links to the lead industry.[188]

Another research finding was that, contrary to Kehoe's belief, childrden absorb and retain far more ingested lead than adults -- some 40-50% for children, as opposed to about 10% for adults.[189] 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 30mg/dl, with greater prevalence of elevated levels in blacks (12.2%) than in whites (2%).[190]

COLE EXPRESSED INDIGNATION THAT "THE ANTI-LEAD ADVOCATES...WOULD HAVE US BELIEVE THAT LOW-LEVEL LEAD EXPOSURE POSES A HAZARD OF BRAIN DAMAGE TO CHILDREN."

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

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185 Needleman HL, Gunnoe C, Leviton A, et. alia., Deficits in Psychologic and Classroom Performance of Children with Elevated Dentine Levels, The New England Journal of Medicine 300:689-695, 1979.

186 See U.S. Centers for Disease Control, Statement on Preventing Childhood Lead Poisoning, Atlanta, Georgia, 1991, pp. 9-10.

187 Cole JF, (letter), The New York Times, June 3, 1980, p.5:1.

188 Putka G, PRofessor's Data on Lead Levels Cleared by Panel, Wall Street Journal, May 27, 1992, p. B9; Hilts P, Hearing vs.. Held on Lead Poison Data, The New York Times, April 15, 1992, p. D28.

189 Alexander FW, Clayton BE, Delves HT, MIneral and Trace-Metal Balances in Children, Quarterly Journal of Medicine 43:89, 1974;Ziegler FW, Edwards BB, Jensen RL, et alia., Absorption and Retentionof Children with Increased Lead Absorption, Pediatric Research 12:29-34, 1978.

190 Mahaffey KR, Annest JL, Roberts J, Murphy RS, National Estimates of Blood Lead Levels: United States, 1976-1980, The New England Jornal of Medicine 307:573-579, 1982.

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 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 seventies.[196] Ironically, these regulations were initially developed to protect the newly developed catalytic converter in automobiles -- a pollution-control device that happens to be rendered inoperative 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

192 Cole J, executive vice-president of the International Lead Zinc Research Organization, Inc. Old Paint, Not Gasoline, Is the Problem in Lead Poisoning, The New York Times, August 23, 1982.

193 Marshall E., Legal Threat Halts CDC Meeting on Lead, Science 223:672, 1984.

194 Centers for Disease Control. Preventing Lead Poisoning in Young Children. Atlanta, Georgia: Department of Health and Human Services, 1985.

195 Centers for Disease Control, Preventing Lead Poisoning in Young Children. Atlanta, Georgia: Department of Health and Human Services, 1991.

196 For a detailed review of this topic, see Silbergeld, EK and Percival, RV, "The Organometals: Impacts of Accidental Exposure and Experimental Data on Regulatory Policies," in S Sparber and H Tilson, eds., Neurotoxicology of Organometals, New York, Wiley Interscience, pp. 328-352 (1987).

197 Small Refiner Lead Phasedown Task Force v. EPA, 705 F.2d 506 (D.C. Cir. 1983) (upholding numeric standards though remanding to correct procedural flaws).

198 705 F. 2d at 531.

199 The New York Times, September 8, 1984, p.42.

200 Pub. Law No. 101-549, amending 42 U.S.C. 211 (n).

201 55 Federal Register 14556 (April 18 1990).

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]

202 Associated Press, August 2, 1990. Dialog AN:02145409.

8. THE LEAD INDUSTRY, 1965-1992

From the time Congress began rumbling 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]

TOTAL LEAD CONSUMPTION REMAINED FAIRLY CONSTANT.

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, most 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 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]

203 Moulds DE., Mineral Facts and Problems, 1965 Edition, Washington, U.S. Department of the Interior, Bureau of Mines, 1965, p.500.
204 Minerals Yearbook, 1971, p.682.
205 After a Century of Successful Operation, St. Joseph Lead Co. Plans for the Next, Engineering and Mining Journal 165:87-92, 1964.
206 Mining Yearbook, 1971, p.534.
207 Forbes, 120:53,55, 1977.
208 Jaffe T., Forbes 142:210, 1988.

The Bunker Hill Co. merged with Gulf Resources & Chemical Corp. (GRC) 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 GRC'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."[213]

The National Lead Co. changed its name to NL Industries in 1971.[214] Big changes began after 1972 when Ray C. Adam came on board as chief operating officer. "At the time, the company had 79 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.[215]

THE MOVE AWAY FROM LEAD PRODUCTION WAS PERCEIVED AS AN IMPORTANT RESPONSE TO ENVIRONMENTAL CONCERNS.

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,[216] and soon a series of corporate mergers ensued. In August, 1981 St. Joe Minerals Corp., merged with Fluor Corp., based in Irvine, California.[217] St. Joe and Homestake merged their lead operations in 1986,[218] and established the Doe Run Co. which owned the St. Joe mines and the Herculaneum refinery.[219] Homestake sold out to Fluor in 1990, 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

209 Financial Times Mining International Yearbook, 1983, p.73.
210 The New York TImes September, 14, 1975, p.53:5.
211 The New York Times October 24, 1981, p.7:6.
212 Mining International Yearbook, 1975, p.90.
213 Stern RL., Denny's Always the Low-Cost Producer, Forbes 143:87, 1989. Dialog AN: 07561857.
214 The Wall Street Journal, April 16, 1971, 21:3.
215 American Painting & Coatings Journal, Jan. 3, 1977, p.29.
216 Mining Yearbook, 1971, p.534.
217 Financial Times Mining International Yearbook 1983, pp.338-39.
218 Zipf P., St. Joe Minerals and Homestake Merging US Lead Operations, American Metal Market, 94:2, 1986.
219 San Francisco Chronicle, November 1, 1986, p.50.

Fluor to find a buyer for Doe Run, which holds a 60-percent share of the domestic lead market."[220] 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, yeilding 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 deferring 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

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220 Farricker M., Doe Run Banking on Lead Recycling, American Metal Market, May 14, 1990, p.2. Dialog AN: 02565600.

221 American Metals Market, Oct. 10, 1991 p.1. Dialog AN 11356046.

222 Bureau of Mines, Lead, Annual Report 1990, , April 1992, p.4.

223 Ibid. ASARCO also operated a smelter in EAst Helena, MT and a refinery in OMaha, NE, which produced 61,000 tons of refined lead in 1992. Much of the feedstock is apparently secondary.

224 Ibid.

225 Ibid.

226 Ibid.

227 Archer C., Lead Group Fights New EPA Regs: Inadequate Funding Cited as LIA Obstacle, American Metal Market 92:1, 1984.

228 The victory may prove at least in part pyrrhic one, as a growing number of lawsuits seek to hold the lead and paint industries liable for harm caused by lead paint. City of Philadelphia v. Lead Industries Association, (E.D. Pa.), City of New York v. Lead Industries Association (New York Supreme Court).

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."