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The Age of Lead: Politics, Science and Urban Ecology in Baltimore, 1900-1960s

Introduction

In the early 1900s, Baltimore began rapidly industrializing, a process that brought both wealth and problems to the city. The shift to an industrial economy had begun well before, especially after city leaders deliberately attempted to attract manufacturers after 1880. But after 1900, the pace of industrialization quickened and Baltimore attracted many industries, including a heavy presence of metal, shipping, transportation, glass, chemicals and clothing manufacturers.[1] Baltimoreans, like Americans generally, regard the proliferating industrial landscape with hope and fear, and their beliefs about the future, and what should be done to shape it, were varied.

These manifold emotions and beliefs were evident when Baltimoreans considered a material which was growing rapidly in use in industry and among consumers: Lead. "We are wont to speak of this era as the 'age of iron,'" the Baltimore *Afro-American* observed in 1906. "Nevertheless, few people realize how useful, if not absolutely necessary, to modern civilization is that other metal, lead." Lead's properties were complementary to iron's, the paper noted – lead was "soft, yielding, and flexible"– and its uses were myriad: Plumbing, printing type, solder, and paint, among others. "Verily, we live in an age of lead as well as of iron," the article concluded.[2]

For some Baltimoreans, however, the production of lead, and rampant industrialization more generally, was alarming. Standing before his congregation in industrial Locust Point, Baltimore, in 1908, one of the city's best known religious leaders, Reverend W.A. Crawford-Frost, recited a passage from Genesis. "And the Lord God formed man of the dust of the ground, and breathed into his nostrils the breath of life." But, the Reverend admonished, "Man has in the city of Baltimore largely substituted for this pure breath of life a mixture of gasoline, lead fumes," sulfur, carbon monoxide, fertilizers dust, smoke and soot.[3]

The *Afro-American* was right about a coming age of lead, but more for the reasons that the Reverend warned of than because of lead's industrial prominence. Lead, to be sure, was an extremely important part of many production processes and consumer goods. But it was not often "absolutely necessary" – i.e., there were technological substitutes – and it was not clearly more important than many others materials would be in the twentieth century, such as aluminum, plastic or silicon.
On the other hand, human exposure to lead in Baltimore increased tremendously over the course of the first half of the twentieth century with health effects that surpassed that of any other single material. This was both because lead exposure became very widespread, reaching virtually every child in the city in the middle of century, and because of its profound effects on bodies and brains. The use of lead first increased exposure in the work environment, leading to increasing attention to occupational health, and then increased in both the domestic and community environment as lead containing products deteriorated (in the case of lead paint), were recycled (in the case of lead batteries), or were wasted (in the case of lead gasoline).

Automobile-based suburbanized was at the center of increasing and unequal lead exposure in the first half of the twentieth century. Many different technological, political and personal choices made lead hazards worse or better in this time period, so lead exposure was complex and multi-causal. Automobility, however, was the most powerful and common underlying factor in increasing. In Baltimore, as in many American cities, automobile-based suburbanization boomed in the 1920s and then

[1] Joseph L. Arnold, "Baltimore Southern Culture and A Northern Economy," *Snowbelt Cities, Metropolitan Politics in the Northeast and Midwest since World War II*, 1990, 27.
[2] "The Age of Lead," *Afro-American* (hereafter *AA*), September 15, 1906, 7.
[3] "Poisoners of the Air," *The Baltimore Sun* (hereafter *Sun*), June 1, 1908, 9. The Genesis quote from the article uses the words "found man" rather than "formed man," but I assume this was a misprint.

resumed with gusto in the late 1940s after the Great Depression and World War II ended. More automobiles increased lead hazards through leaded gas and lead batteries. Traffic congestion increased automobile pollution even more. At the same time, the hollowing out of the city by suburbanization led to slum housing conditions, increasing lead paint hazards. Traffic congestion and a deteriorating urban core subsequently led to policies of urban renewal and interstate construction that massively restructured the built environment of Baltimore in the 1950s. These also increased lead hazards by creating large amount of lead-contaminated urban dust, although their effect tin the long run was more complicated. Weather also played a role, with years of high wind mobilizing lead-laden dust. Histories of lead poisoning have not usually tried to work out the multi-faceted aspects of exposure in this time period and for that reason have tended to recapitulate the simplistic models of lead poisoning that health professionals of the time worked with.[4]

Exposure to these lead hazards was not equal. Only certain kinds of work environments had lead hazard problems and these jobs were, at times, segregated by race and gender. Lead hazards also disproportionately affected those who were poor and those who lived in the urban core. Suburban automobiles funneled their lead hazards into the urban core, both in the form of commuter pollution and in the form of lead batteries that were recycled and re-smelted in the inner city. In contrast to work environments, where whites often experienced the most lead poisoning, African Americans suffered disproportionately from domestic and community exposures.

Knowledge production and action regarding lead poisoning in Baltimore in the first half of the twentieth century took place at many levels – public and private, expert and laity, neighborhood and national – and all levels played important roles and also had significant limitations. Health professionals played critical roles in producing and disseminating knowledge, and in some cases pushed for regulations, yet their solutions were not always effective and their knowledge was sometimes biased. Although the communities affected by lead poisoning in this period are often portrayed as ignorant and passive, they in fact often played critical roles in producing knowledge about, and solutions to, problems of lead poisoning.[5]

The mitigation of widespread lead poisoning, however, was not easy and was ultimately not very effective. In general, lead hazards and lead poisoning got worse in this time period and probably peaked in the 1950s. The effects of the massive exposure to lead in mid-century Baltimore will be taken up in Chapter X, but the exposure and disproportionate effects on the poor and black in Baltimore illustrates how much the putative Golden Age of the United States was not a Golden Age for all.[6]

Lead Science and Toxicology

As will become evident, lead poisoning was and is a complicated matter. Following its history requires a grounding in a few basic facts about lead.

Lead is an element normally present in minute quantities in the environment. Human use of lead in industry and consumer goods has greatly increased the amount of lead in the environment. Moreover,

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[4] This is true whether the historians are supportive or critical of the way health professionals and policy makers handled lead poisoning. For example, Christian Warren and the historian team of David Rosner and Gerald Markowitz have criticized the "victim blaming" aspects of public health policy while Peter English and John Burnham have generally supported how doctors and health departments approached lead poisoning. But all of these authors have focused their environmental analysis in this time period around lead paint without exploring other sources of exposure (even if they nominally acknowledge these other sources).

[5] Part of the reason that victims of lead poisoning come across as ignorant and passive in histories of this period is an artifact of the documentary trail – the classic social history problem of a lack of a paper trail from non-governmental, non-bureaucratic, and marginalized groups.

[6] For a powerful argument that the Golden Age of the 1950s was social limited see Thomas J. Sugrue, *The Origins of the Urban Crisis: Race and Inequality in Postwar Detroit*, Revised edition (Princeton: Princeton University Press, 2005). For Sugrue, however, the deteriorating urban environment is merely a symbol, rather than a cause or criteria, of inequality.

this use has also changed lead's chemical and bulk forms making it more "bioavailable," or easily absorbed into the body. Not only are the chemical formulation of lead in lead paint (lead carbonate and chromate, for example), more bioavailable than, for example, common lead ore (like lead-sulfide, also known as galena), but lead ore is usually encapsulated in other rock such as quartz. On the other hand, lead paint is not encapsulated in anything and becomes increasing bioavailable as it breaks down into finer particles, finally becoming a highly bioavailable lead paint dust.[7]

Lead in the body causes a host of problems, particularly in children, for whom lead toxicity can have permanent effects. The body burden of lead is measured by the amount of lead in blood, which is measured in micrograms per deciliter of blood. (A microgram is one millionth of a gram and a deciliter is one tenth of liter, or about half a cup. This measurement of blood lead in micrograms per deciliter is symbolized as PbB μ/dL, and this symbolization will be used throughout the article). The natural level of lead in human blood is about .001-.002μ/dL.[8] Lead causes a number of health issues, including significant renal problems, but it is lead's effect on children's developing nervous system that has garnered the most attention. As an indication of how profound the effects of small amounts of lead can be, the Centers for Disease Control no longer puts a lower limit on the child blood lead level that is cause for concern. Blood lead levels over 125 μ/dL are associated with acute encephalopathy (swelling of the brain) and death; above 80μ is associated with encephalopathy and renal toxicity; over 60μ is associated with lead colic (excruciating abdominal spasms); and over 20μ is associated with anemia and peripheral neuropathy (loss of nervous functioning in limbs). Blood lead below 10μ is associated with IQ deficits of up to 9 or 10 points and other psychological changes including irritability problems with what psychologists call "executive functioning" (which includes self-inhibition and the ability to plan). The neurobiological mechanisms involved in these effects from lead include reductions in gray matter (neurons), demyelination (interference with the connections between neurons), and reduced hippocampal development (a brain region involved in memory, among other things). Due to the severity of these health problems, lead is widely regarded as the greatest environmental threat to child health in the United States.[9]

Because having any amount of lead in the body is not "normal," and because no amount of lead absorption is considered safe, there is no categorical cut off for what constitutes "lead poisoning" in the present. Potentially, any amount of lead absorption can cause poisoning, if by poisoning we mean any of the above effects, and if we think of those effects in the statistical sense (meaning, those effects may not be discernible in any given individual but they are if you look at a big enough sample). This was not, however, how lead poisoning was understood in the past. As I describe, lead poisoning started out as a disease of that affected adults causing death or serious bodily issues like neuropathy (loss of nervous function in limbs). Lead poisoning in children was first associated with very extreme effects (death, convulsions, encephalopathy) and some simple signs like a lead gum line (a dark grey or blue line on the gums caused by . By the 1950s, doctors considered more subtle effects signs of lead poisoning, and they had developed a number of diagnostic tests and patient history cues to guide their diagnosis. To avoid confusion, when I use the term "lead poisoning" I am referring to how people were categorized at the time.

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[7] M. V. Ruby et al., "Advances in Evaluating the Oral Bioavailability of Inorganics in Soil for Use in Human Health Risk Assessment," *Environmental Science & Technology* 33, no. 21 (1999): 3697–3705.

[8] David C. Bellinger, "Very Low Lead Exposures and Children's Neurodevelopment," *Current Opinion in Pediatrics* 20, no. 2 (2008): 172–77.

[9] Donald T. Wigle, *Child Health and the Environment*, 1st ed. (Oxford University Press, USA, 2003); T. I. Lidsky and J. S. Schneider, "Adverse Effects of Childhood Lead Poisoning: The Clinical Neuropsychological Perspective," *Environmental Research* 100, no. 2 (2006): 284–93; Bellinger, "Very Low Lead Exposures and Children's Neurodevelopment."

Section I: Occupational Lead Hazards in Baltimore1870s-1930s

Until the 1930s, lead poisoning garnered the most attention in Baltimore as an occupational disease. In the late nineteenth century and early twentieth century, occupational lead poisoning gained sporadic public attention in Baltimore, mostly in the form of newspaper reports on incidents or studies outside of Baltimore. In the early 1900s, the first studies of lead poisoning in Baltimore were carried out by Johns Hopkins physicians, but were not publicized. The emergence of worker's compensation laws brought some specific occupational lead poisoning cases in Baltimore to light in the 1910s and 1920s, but it was not until the Baltimore City Health Department became formally engaged in industrial health in the 1930s that occupational lead poisoning garnered both sustained public attention and systematic studies.

The history of occupational lead poisoning in Baltimore City offers several important historical insights. Most studies of occupational lead poisoning, and occupational health in general, have examined its history through national or state politics, industries, organizations, and personalities. These histories have yielded important insights about the contributions and limitations of workers and scientists in creating occupational health knowledge and regulations, and the ways in which regulations and knowledge – and indeed the industrial hygienists themselves – have been vitiated by corporations.[10] However, rarely do these studies focus on a particular place and examine how a variety of workers living in that place were exposed to hazards. Because they do not take this approach, they do not indicate, for example, how great the burden of industrial disease was for a given place, how industrial hazards were interconnected with each other, or with domestic and community health. And they almost never mention city health departments. In major industrial cities such as Baltimore, however, it was eventually the city health department that became the most important organization for studying, influencing, and regulation occupational health.

I make two arguments about occupational lead poisoning in Baltimore in the first half of the twentieth century. First, I show that occupational lead poisoning had a different pattern than domestic/community lead poisoning, even though these types of poisoning were often connected. In contrast to the pattern of lead poisoning in communities and homes, especially among children, that I detail later, occupational lead poisoning was fairly stable over the first half of the twentieth century, yielding no big spikes. And in contrast to lead poisoning in homes and communities, occupational lead poisoning affected whites most. Second, I argue that the Baltimore City Health Department came to play the primary role in dealing with occupational health, including lead poisoning, in the 1930s. Although the city health department made important contributions to occupational health and faced significant constraints, just as labor organizations and industrial health scientists did, these contributions and constraints were unique to the institutional role of the health department. In particular, industrial hygiene at the city level was in constant interaction with other aspects of health. Sometimes it drew on the capacity or the public attention built up in other parts of the department and sometimes it competed with these other parts for funding and attention.

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The general fact that lead could cause poisoning has been known for thousands of years, since at least early Greek and Roman civilizations where the poisoning was associated with lead mining and smelting. By the end of the eighteenth century, lead poisoning was an acknowledged occupational disease of lead painters. Lead paint and other hazards of lead use became increasing problems in

[10] On the contributions of workers, see, for example Claudia Clark, *Radium Girls: Women and Industrial Health Reform, 1910-1935*, 1 edition (Chapel Hill: The University of North Carolina Press, 1997). On the politics of industrial health and the role of corporations, see David Rosner and Gerald E. Markowitz, *Dying for Work: Workers' Safety and Health in Twentieth-Century America* (Indiana University Press, 1987); Gerald Markowitz and David Rosner, *Deceit and Denial: The Deadly Politics of Industrial Pollution*, First Edition, With a New Epilogue edition (University of California Press, 2013); Christian Warren, *Brush with Death : A Social History of Lead Poisoning* (Baltimore; London: Johns Hopkins University Press, 2001). On the critical importance of industrial health scientists, see Christopher C. Sellers, *Hazards of the Job: From Industrial Disease to Environmental Health Science*, 1 edition (Chapel Hill: The University of North Carolina Press, 1999).

industrializing cities like Baltimore. The *Sun* carried stories on lead poisoning from various sources – lead cosmetics, a baker who used chrome yellow lead paint for his buns – as early as the 1840s, and lead poisoning was discussed as an industrial health problem in Baltimore as early as 1878.[11] In the late-nineteenth century, many public health and labor leaders called for lead paint prohibition, and by the early twentieth century many European countries did ban lead paint entirely.[12] Other trades that used lead also received study and drew concern from reformers, usually first in Europe or Britain and then later, in the early 1900s, in the United States with Progressive reformers like Alice Hamilton.[13]

Although Baltimore was not a dominant center for manufacturing anything that involved considerable lead exposure, capitalists from Baltimore were involved in developing lead resources and manufacturing, and capitalists in the city did become involved in many industries where lead poisoning was a problem for workers. Artisan and early industrial work in and around Baltimore sometimes entailed significant exposures to lead. Hand cutting of steel files, for example, required the use of grooved blocks of lead that could hold the file in place. The soft lead would protect the file teeth on one side as the worker chiseled teeth into the other side, but the process also created lead dust as the teeth cut into lead. Since workers pounded their files at a rate of 150 to 200 strikes a minute, this generated considerable dust.[14] Investigators in England examined this industry and found that 74 out of 100 file cutters examined had a blue line ("a very strong symptom of lead poisoning"), 28 had colic, and 20 had paralysis of wrists and fingers. The problem was prevalent enough that it was referred to as "file cutter's disease." The report noted that those with lead poisoning often had disorders of urinary and nervous systems, and some deaths were attributed to the poisoning. Moreover, the death attributed to them were according to one doctor's review "constitute but a small proportion of the deaths really due to poisoning by lead among workers who are exposed to its influence."[15] File cutters certainly existed in Baltimore in the late nineteenth century, but only appear to have served the local market and so were not a very large labor force. Mechanization helped end this kind of occupational exposure, though molten lead was still used to temper files and workers in this capacity sometimes endured poisoning. Baltimore capitalists facilitated the mechanization of file making in America by buying the patent rights from a French inventor in 1860. They built their factories in Rhode Island instead of Baltimore and the hand cutting trade died out slowly.[16]

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[11] "Caution," *The Sun* , August 5, 1840; Special Dispatch to the Baltimore *Sun*, "PHILADELPHIA AFFAIRS: The Lead Poisoning Case--Co-operation Among File Workers," *The Sun* , July 9, 1887. "Our Paris Letter -- Sanitary Subjects," *The Sun*, August 28, 1878.

[12] E. Fee, "Public Health in Practice: An Early Confrontation with the 'Silent Epidemic'of Childhod Lead Paint Poisoning," *Journal of the History of Medicine and Allied Sciences* 45, no. 4 (1990): 570–606; David Rosner and Gerald Markowitz, "'A Problem of Slum Dwellings and Relatively Ignorant Parents': A History of Victim Blaming in the Lead Pigment Industry," *Environmental Justice* 1, no. 3 (2008): 159–68.

[13] Robert Gottlieb, *Forcing the Spring the Transformation of the American Environmental Movement* (Washington, DC: Island Press, 2005); Christopher C. Sellers, *Hazards of the Job: From Industrial Disease to Environmental Health Science*, 1 edition (Chapel Hill: The University of North Carolina Press, 1999); Peter C English, *Old Paint : A Medical History of Childhood Lead-Paint Poisoning in the United States to 1980* (New Brunswick, NJ: Rutgers University Press, 2001); Christian Warren, *Brush with Death : A Social History of Lead Poisoning* (Baltimore; London: Johns Hopkins University Press, 2001).

[14] Henry Disston & Sons, Inc, *The File; Its History, Making and Uses: A Description of the Development of the File from the Earliest Times to the Present Day; a Brief Statement of the Modern Methods of File Making; a Description of the Great Variety of Files and the Numerous Uses to Which the Tool Is Adapted* (Henry Disston & sons, inc., 1921), 21–22. Workers also used lead hammers to straighten files and molten lead baths to cool file tangs slowly so that they were soft. Erik Oberg and Franklin Day Jones, *Machinery's Encyclopedia: A Work of Reference Covering Practical Mathematics and Mechanics, Machine Design, Machine Construction and Operation, Electrical, Gas, Hydraulic, and Steam Power Machinery, Metallurgy, and Kindred Subjects in the Engineering Field* (The Industrial Press, 1917), 110.

[15] Edith Maynard, "File Cutting," *Journal of the Royal Sanitary Institute*, 1902, 408–409.

[16] Henry Disston & Sons, Inc, *The File; Its History, Making and Uses*, 25–26. Apparently, the Baltimore capitalists initially intended to build a factory in Baltimore but for unknown reasons decided New England would be a better

Other trades and manufacturing processes also created lead hazards in Baltimore in the late nineteenth and early twentieth century. Tinners, can makers and brass workers ended up at the Johns Hopkins Hospital with serious cases of lead poisoning around the turn-of-the-century, as did stereotypers.[17] Glass work was another area that was notorious for lead poisoning, and Baltimore was somewhat well-known as a center of cut glass production. Glass cutting workers who ground and polished glass – particularly cut glass or crystal – could get lead poisoning by inhaling the rouge or putty powder.[18] By 1904, at least one of these workers absorbed enough lead to send him to the hospital.[19] Another source of lead in this work was color pigment used in some types of decorative glass work. In 1930, six young women workers in a glass factory got lead poisoning after the East Baltimore factory introduced new glass decorating methods. After the women complained about getting sick from dry paint fumes, the company provided masks but these were not adequate and the workwomen still became sick. One 23 year old woman was hospitalized while the others were "not regarded as serious," according the *Sun*, and were recovery at home.[20]

These women, however, may be best grouped with workers who worked with lead paint in some manner who were by far the workers most in danger in Baltimore of getting lead poisoning in the late nineteenth and early twentieth century. These workers included those involved in producing white lead, red lead, and chrome leads that were used to make and color paints. It also included the workers who applied them to products in the factory – as glass workers, enamellers, and so on. And finally, painters who worked in construction.

Baltimore industries had a relatively long history of producing lead products for the paint and varnish industry, all of which were increasing in production in America generally in the late nineteenth and early twentieth century. Red lead and litharge were lead oxides used as inputs to various production processes and also, in the case of red lead, used as a paint for metal structures like bridges. Baltimore had a few businesses that specifically manufactured litharge and red lead (the latter made by heating the former), but much of litharge and red lead production was made in factories that focused on white lead production. The one area of lead production that Baltimore was truly a center of was lead chrome production. The wealthy Tyson family of Baltimore developed the area's first chromite (chrome iron) mine after discover it on and near their property near the city in the 1820 and 1830s. Initially, they shipped the ore to Glasgow, Scotland but eventually established the Baltimore Chrome Works in 1844, an endeavor that lasted until 1895. The Chrome Works did not itself make lead chrome, but its location allowed considerable lead chrome production at nearby white lead manufacturers.[21]

White lead production was far and away the most important lead paint product on the national level and in Baltimore. In America, the preservative power of paint was not as valuable given abundant and cheap timber. Thus, colonial American homes did not use paint on the inside or the outside. That began to change in the nineteenth century in urban areas where residents began using more paint for ornamental reasons. In 1810, Americans produced 369 tons of white lead; by 1850 they were producing 9,000 and by 1860 15,000. After the war production shot up to 35,000 tons, but the initial profitability of white lead led to overproduction and overbuilding of factories, making the business less profitable. Consolidation was one outcome of this; another was the formation of associations and trusts and ultimately, in 1891, the National Lead Company, which controlled a number of large white lead

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site, and so sited their company, the American File Company, in Rhode Island. John Leander Bishop, Edwin Troxell Freedley, and Edward Young, *A History of American Manufactures from 1608 to 1860...: Comprising Annals of the Industry of the United States in Machinery, Manufactures and Useful Arts, with a Notice of the Important Inventions, Tariffs, and the Results of Each Decennial Census* (E. Young, 1864), 731.

[17] Henry Thomas, "A Case of Generalized Lead Paralysis, With a Review of the Cases of Lead Palsy Seen in the Hospital," *Bulletin of the Johns Hopkins Hospital* 15, no. 159 (June 1904): 211–212.

[18] George Martin Kober and William Clinton Hanson, *Diseases of Occupation and Vocational Hygiene* (P. Blakiston's Son & Company, 1916), 637.

[19] Thomas, "Generalized Lead Paralysis," 212.

[20] "Sixth Woman Made Ill From Lead Poisoning," *Sun*, January 8, 1930, 26.

[21] Clayton Hall, *Baltimore: Its History and Its People* (Lewis Historical Publishing Company, 1912), 523–524.

businesses in the country. By this point, white lead production had reached 75,000 tons. As the director of the National Lead Company puffed in 1895, "With practically the same methods as those employed by the ancients" – mixing vinegar with lead to make white lead – "the industry has risen, through the sheer executive intelligence of the present age."[22]

White lead production in Baltimore increased over the nineteenth century and its major producer was eventually mixed in to the National Lead Company. The French General Reubel was apparently the first to make a foray into white lead production in Baltimore in the early 1800s, going into business with an "accomplished Professor Chemistry." This operation, however, was not big or long lasting.[23] By 1849, Baltimore had four, meagerly capitalized paint "establishments," with a total of eight workers.[24] In 1867, as part of a raft of white lead factory building in many cities in Northeast and Midwest , the Maryland White Lead Company built an "extensive plant" in the Locust Point area of Baltimore, inaugurating a new scale of lead paint production in the city.[25] Several other companies also established paint production businesses in the city after the Civil War, such as the Adams White Lead Company, although these were not as large. In the 1890s, the Maryland White Lead Company, which was absorbed into the large NLC, but the businesses was then liquidated in 1896.

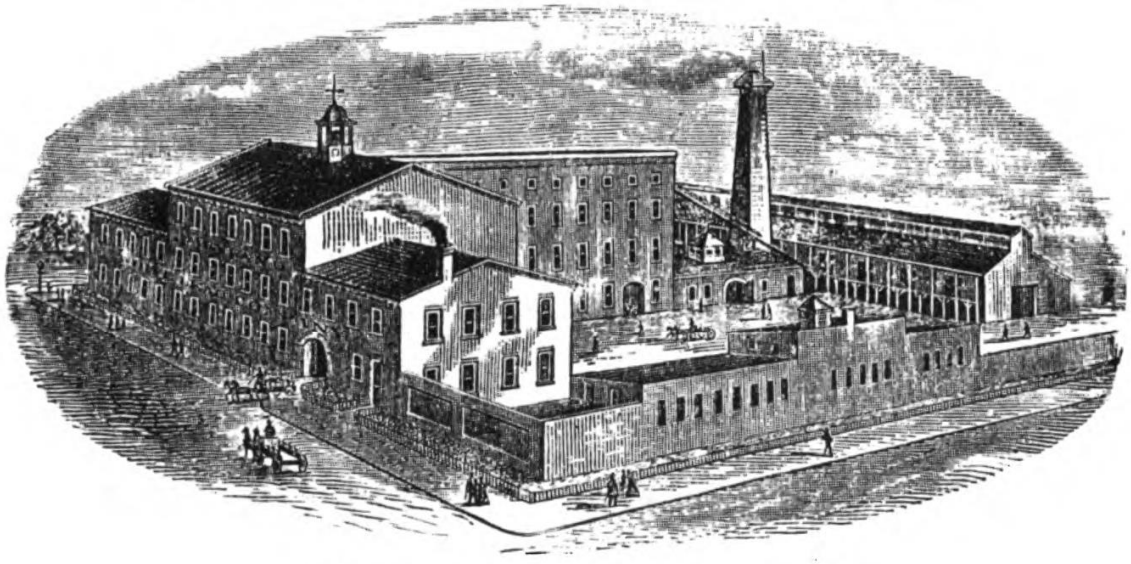

Figure 1: Maryland White Lead Works. Workers at lead paint factories like this one located in Locust Point, Baltimore, were at high risk for lead poisoning. Source: George Washington Howard, *The Monumental City: Its Past History and Present Resources* (JD Ehlers and Company, 1873).

Since there was virtually no systematic study of industrial work hazards in the nineteenth, it is difficult to say how many workers in these early paint industries were poisoned. A better picture began to emerge on both the local and national level in the early 1900s. Americans began paying more attention to studies from Europe, and Alice Hamilton began her famous studies of industrial hazards in American industry in 1907. Lead figured prominently in these studies and the Baltimore *Sun* carried several articles

[22] William P. Thompson, "The Lead Industry," in *1795-1895: One Hundred Years of American Commerce ... a History of American Commerce by One Hundred Americans, with a Chronological Table of the Important Events of American Commerce and Invention Within the Past One Hundred Years* (D. O. Haynes & Company, 1895), 436–441.
[23] Mary Barney, *A Biographical Memoir of the Late Commodore Joshua Barney: From Autographical Notes and Journals in Possession of His Family, and Other Authentic Sources* (Gray and Bowen, 1832), 242.
[24] Bishop, Freedley, and Young, *A History of American Manufactures from 1608 to 1860...*, 114.
[25] *Notes for a History of Lead: And an Inquiry into the Development of the Manufacture of White Lead and Lead Oxides* (D. Van Nostrand, 1888), 331.

related to lead poisoning and workers in the early 1900s.[26] Health professionals in Baltimore began their own studies in this time period. In the very early 1900s, this was primarily driven by doctors at Johns Hopkins who seeking to understand clinical cases that came their way at the hospital. In 1902, a doctor noted that an unusually high number of gout patients (10 of 32) worked in trades involving lead.[27] Two years later another Johns Hopkins doctor, Henry Thomas, reviewed the hospital's records of lead poisoned patients up to that point as a result of curiosity piqued by dealing with an unusual case of lead poisoning in which the patient became totally paralyzed. Of the 54 patients that had gone to Johns Hopkins Hospital for care since it was found in 1889, six got lead poisoning from food or medicine, four were undetermined, and the rest of the 44 patients got lead poisoning from their jobs. Of those workers, 28 worked in the paint industry and 3 worked as enamellers. These numbers suggest that the most serious cases of lead poisoning affected workers in the paint industry who were probably primarily adult, white, men.[28]

Further light was shown on the nature and extent of occupational lead poisoning in Baltimore as a result of worker's compensation laws. Maryland's first attempt at such a law in 1902 – the first in the nation – was declared unconstitutional by the Supreme Court in 1904, but the state passed a viable law in 1914. Initially, however, lead poisoning was not covered by the law. Around 1915 a Baltimore painter contracted lead poisoning, becoming "incapacitated," but the State Accident Commission determined that his case was not covered by the law. Although the point of workers compensation was to do away with determining who was at fault, another distinction remained: Whether lead poisoning was an "occupational disease" or an "accident." This distinction was a common, though disputed, aspect of workers compensation law at the time, and the Commission ruled that lead poisoning was a disease and disallowed the claim.[29] This distinction held until the late 1920s when the state began ruling that on the job poisonings did constitute an "accident" or "injury."[30]

Although the state government began receiving and granting claims related to lead poisoning in the nineteen teens and twenties, insurance companies that dealt in workers compensation (among other insurance fields), sought better information and helped reveal the extent and nature of lead poisoning. In 1913, Prudential's famous statistician, Frederick Hoffman, carried out an analysis of the Johns Hopkins Hospital that revealed 41 cases of lead poisoning that had led to admission to the hospital between 1902 and 1913. Almost all of these were white males – three were black males and one was a white female. Hoffman's study also suggests a slight increase in annual lead poisoning cases to Hopkins in the first decade of the twentieth century over the end of the nineteenth century.[31]

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[26] "The Dangerous Trades," *Sun*, October 22, 1902; Hamilton, "Lead Poisoning in 28 Trades," *Sun*, March 19, 1911.

[27] Dr. Futcher, "A Case Showing the Deformities of Chronic Gout," Johns Hopkins Hospital Bulletin, 13.134, 1902, 116. The association between lead poisoning and gout in fact went back to the early 1800s and continues to be an association that is noted if not fully understood. David S. Newcombe and Dwight R. Robinson, *Gout: Basic Science and Clinical Practice* (Springer, 2012), 142.

[28] Thomas, "Generalized Lead Paralysis," 212. Thomas' article does not give specific numbers on race, sex or age, but it does specify only one case of child lead poisoning and all of the cases that specify sex refer to males (except the child, who was female). It makes no mention of race.

[29] Although determining fault was no longer the issue, the Commission nevertheless made it clear that the painter was at fault for failing to wash his hands before eating lunch. Maryland State Accident Commission and Maryland Court of Appeals, *Reports of Cases under the Workmen's Compensation Act Decided by the State Industrial Accident Commission and the Court of Appeals of Maryland* (George W. King Printing Co., 1916), 141.

[30] Mark L. Matulef, "On-The-Job Lead Poisoning: Early Judicial Treatment of Claims for Recovery from Exposure to Workplace Lead," *U. Balt. J. Envtl. L.* 10 (2002): 1.

[31] Hoffman's study, unlike Thomas's, did not include lead poisoning victims who went to outpatient services but were not admitted, so the comparison between the two studies for admitted lead poisoning cases is: 33 from 1892 to 1903, and 41 from 1902 to 1911. Frederick Ludwig Hoffman, *The Statistical Experience Data of the Johns Hopkins Hospital: Baltimore, Md., 1892-1911* (Johns Hopkins Press, 1913); Thomas, "Generalized Lead Paralysis."

The Rise of Municipal Industrial Hygiene, 1910s-1930s

In the wake of increasing attention and legislation about workers health, the Baltimore City Health Department (BCHD) moved toward bringing industrial hygiene into its administrative orbit in the 1920s and 1930s. Industrial hygiene was a relatively new aspect of public health, and the BCHD was eager to become involved with it as its importance rose and as the importance of other aspects of public health declined. In 1931, Wilmer Schulze, the Chief of the BCHD's Chemical Technology for the Bureau of Chemistry and Food, defined industrial hygiene as "all the factors influencing the of industrial workers, such as occupational disease, industrial accident prevention and environmental conditions." These were important to think about, Schulze noted, because such a large proportion of the workforce was engaged in industrial work. He noted that most states had passed worker's compensation laws and employers were taking precautions to prevent health problems. The health department, he stated, would be targeting work environments that contained poisonous chemicals and harmful, noting that the constant innovation in chemical processing made public health monitoring a necessity. There were many industrial diseases known, but Schulze listed a few that showed the importance of industrial hygiene: carbon monoxide, benzol poisoning, silicosis and lead poisoning.[32]

Although the city health department's capacity was extremely limited in the early 1900s with regard to industrial disease, it was helped along by the decline in communicable diseases and its alliances and use of Maryland state law and capacity. Although many communicable diseases remained serious health problems in Baltimore in the first half of the twentieth century, several virulent disease were greatly diminished by the implementation of sanitary measures. Typhoid, in particular, went from XXX to XXX as a result of the building of a fresh water supply infrastructure and the use of chlorine treatment. The decrease in epidemics and communicable disease rates helped free the BCHD to concentrate on other aspects of health, shifting more funding and personnel to the sanitary division that would eventually encompass industrial hygiene.

The Health Department's move into industrial hygiene was also facilitated by state law and capacity. Beginning in 1912, Maryland law required physicians to report industrial diseases to the State Board of Health. The city health department, which often reminded physicians of this fact in its publications, was not a direct recipient of these reports initially.[33] But by the 1930s, the state had **"**deputized" the Baltimore Health Department to receive reports of these diseases. The Department was eager to collect this information, writing letters to hospitals and physicians and publicizing the new arrangement in its newsletter. While the reporting requirements and the BCHD's deputization did yield important information for the department, these industrial disease were chronically underreported. In 1932, the Department noted that there had been "some response" from physicians and hospitals.[34] The following year it was more candid, noting noted, there is a "laxity in compliance" with reporting legislation, a "negligence" the BCHD attributed in part to "the fact that compensation for occupational disease is not provided for in Maryland."[35] And while the BCHD could receive reports from the State Accident Commission these were "incidental," as Schulz put it, because they were only those cases that involved workmen's compensation claims. Thus Schulz lamented in 1932 that the BCHD still did not know the extent of lead poisoning in industries in Baltimore.[36]

While there was little the BCHD could do to force physicians and hospitals into better reporting of industrial disease, the department could produce its own knowledge about the industrial environments

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[32] BCHD *Health News*, February, 1931.

[33] "Physicians Columns," BCHD *Health News* 1925, September, 2.8, 58. Other reportable industrial diseases included poisonings from phosphorus, arsenic, mercury, other compounds, anthrax, or "compressed air illness" or "from any other ailment or disease contracted as a result of the nature of the patient's employment." From Annotated Code, 1924, Sec. 14, 1912, Ch. 165.

[34] BCHD *Annual* 1932, 220.

[35] BCHD *Annual* 1933, 286-288.

[36] Memo Schulz to Jones, March 18, 1932, in Folder Lead Poisoning by Burning Battery Cases 1932-1934, Box 3.3-3.4 Housing and Lead Paint Poisoning, Huntington Williams Papers (hereafter, HW Papers), Alan Mason Chesney Medical Archives, Johns Hopkins (hereafter, JHMA).

that people worked in. Doing so, however, would require administrative reorganization and help from the state. Administrative restructuring had been going on continuously in some ways for years, but it proceeded rapidly in the 1930s, especially under Huntington Williams who became the Commissioner of Health in 1932. By 1933, the BCHD had consolidating activities relating to sanitation, gas and water services, and industry into a Bureau of Environmental Hygiene in the hopes of increasing the profile of public health in these arenas. It pushed hard into industrial health, creating a training class for inspectors and assigning three of its inspectors to industrial health. It also began carrying out systematic surveys of industries. A smaller one in 1932 targeted plants deemed to be the most hazardous including laundries, paint and enamel manufactories, and clothing producers. The cases brought to the attention of the BCHD included one or two cases for dust, hydrogen sulphide, arsinine, mercury, volatile solvents, and acid fumes, and 14 cases of skin infections. The most numerous problem was lead poisoning, with 16 total cases. The plurality of these came from the scrapping of ships with six, six from "miscellaneous" industries, one from lead smelting, and three from scrapping storage batteries.[37]

In 1933, together with the State Commission of Labor, the BCHD carried out a larger study of 2938 establishments employing about 50,000 people. The study found 36 hazardous substances in use in various industries, including chromium, carbon monoxide, dusts, lacquer and paint fumes, and lead. A few others hazardous chemicals, such as mercury and arsenic, were also found but only in a few industries. The study also examined the work environment and safety and sanitation measures, including lighting, ventilation, washing facilities and so on. In terms of occupational diseases, the BCHD found out about 38 cases. Twenty eight of these were some form of dermatitis. Additionally, there was one case each of benzol, carbon monoxide, and nitrous gas poisoning. The remaining seven cases were lead poisoning cases.[38] Most of the lead cases again came from the scrapping of old ships – a burgeoning industry that Baltimore was at the center of[39] – that had been painted with lead paint and which workers cut into pieces using acetylene torches and little protection.[40]

The surveys still did not fix the problem of underreporting because the BCHD was still at the mercy of what diseases physicians reported or what problems made their way to the Accident Commission. In addition, the surveys of industries took place during the depths of the Great Depression when industry was at a "low ebb" of activity.[41] Nevertheless, the conditions were bad enough and there were enough cases of health problems to catalyze action. In the end, the surveys proved to the BCHD what it had expected – that work environments were a health problem – and provided a justification for its involvement as a monitor, educator, and regulator of these environments.

Despite asserting the need for regulation, however, the BCHD focused almost exclusively on educating industrialists about better working conditions. The department did, with the help of the city Buildings Engineer, prevent the building of a lead recovery plant because of inadequate protections against lead poisoning. But primarily it urged businesses to change practices and fielded requests from businesses about how to improve industrial hygiene.[42] The department was encouraged that better ventilation and better sanitary facilities for workers had been accomplished through education rather than legal action.[43] For example, in 1932, the BCHD had begun investigating and insecticide factory where an employee had become sick after being exposed to lead dust. The company had a history of lead

[37] BCHD *Annual* 1932, 220-221.
[38] The BCHD did not state the percentage of industries studies. BCHD *Annual* 1933, 286. BCHD *Annual* 1934, page 201, clarifies that the survey was begun in 1933 and completed in early 1934, so was not a continuation of earlier surveys include the preliminary one carried out in 1932.
[39] "New Metals for Old: Aged Ships, Cars and Junk Are Base of Huge Industry," *Sun* July 12, 1936, SM6. Curtis Bay was a "leading center for scrapping ships," including ocean liners and obsolete military ships from World War I.
[40] BCHD *Annual* 1933, 286-288. The BCHD also made special studies of dermatitis in the vegetable packing industry and carbon monoxide in poisoning in storage garages.
[41] BCHD *Annual* 1933, 286.
[42] BCHD *Annual* 1934, 202-203.
[43] BCHD *Annual* 1932, 221.

poisoning, and this was the second in a year, but the company was, according to Schulz, "very cooperative," and had provided filter masks, hygiene education, and time to for workers to change clothes and shower. It had also forbidden eating during work hours and had moved the worker who became sick to another position.[44] Schulz likewise managed to eliminate (for a time) the lead poisoning cases from cutting old ships up by providing masks and education to workers. In other cases, like a lead smelting factory, the BCHD met with a less responsive employer who did little to improve conditions. Occupational lead poisoning cases did fall considerably from 1933 to 1934 perhaps in response to the department's efforts. But by that time, the face of lead poisoning had changed considerably from the painter and lead smelter worker to the child in the home.

Section II: The "Depression Disease": Lead Batteries and Child Poisonings in the 1930s

In 1932, when Baltimore City Health Department was diving head first into industrial hygiene, lead poisoning in occupations trumped other industrial diseases. But in that year, industrial diseases were themselves outshined by a new problem. The health department reported a "most interesting and insidious development": The mass poisoning of children in their homes by burning the wooden cases that held and separated the lead plates of lead acid batteries, before these cases were replaced by synthetic materials in the post-war era. In total that year, the department reported 36 cases of lead poisoning by this exposure. Thirty five of these cases were children, almost all of them poor and African American.[45]Before the 1930s, lead poisoning was not considered a community public health problem in the United States. It was an occupational health problem and a problem that occasionally afflicted other individuals in a rather unsystematic way. Then, in the nadir of the Great Depression, poor families in Baltimore and other cities began using discarded lead acid battery casings for home fuel, leading to dozens of serious cases of lead poisoning in children. The outbreak of lead poisoning was one of the first in which a discrete disease event was linked to a particular source of chemical pollution (as opposed, for example, to industrial air pollution being generally recognized as unhealthy and thus regulated) and the first in which the focus was on child health. Although the problem was not particularly congruous to the division of labor in the department between child welfare, epidemiology, and industrial health, the Baltimore City Health Department, under the leadership of an ambitious new commissioner, Huntington Williams, aggressively confronted the problem in the city and successfully publicized it nationwide.

Although historians frequently mention the battery case poisoning in Baltimore, the story has not been told in any detail. Doing so contributes to our understanding of how community environmental health problems arose, were understood, and were approached, and also as a comparison with later lead poisoning issues that surrounded lead paint, gasoline, and dust. First, I argue that it illuminates the often double-sided issue of energy justice: The ways in which communities, by virtue of their poverty and geography, are at once deprived of critical energy resources and at the same time often disproportionately bear the burden of negative externalities (e.g.., pollution) caused by energy production. In this case, the community that was poisoned lived in a gas/electric desert in the middle of Baltimore while they were simultaneously poisoned – in their homes, neighborhoods, and workplaces – by the recycling of batteries to run automobiles and other devices.[46] Second, I argue that it shows the important contributions of community knowledge and action in the discovery and mitigation of environmental health problems.

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The understanding of the lead poisoning epidemic in 1932 emerged over the course of the summer as the result of a number contributions, from local community knowledge to the latest lab testing techniques. Patient zero was a seven year old African-American girl. On June 29, 1932, concerned

[44] Memo Schulz to Jones, March 18, 1932, Folder Lead Poisoning by Burning Battery Cases 1932-1934, Box 3.3-3.4 Housing and Lead Paint Poisoning, HW Papers.
[45] BCHD *Annual* 1932, 220.
[46] On energy justice, see Benjamin K. Sovacool, Roman V. Sidortsov, and Benjamin R. Jones, *Energy Security, Inequality and Justice* (Routledge, 2013).

relatives brought her to the Harriet Lane Home. The girl was unconscious, and had previously experienced convulsions followed by a state of "stupor." Her signs pointed toward tubercular meningitis, a common problem in poor and African American communities in Baltimore, but a lumbar puncture test for this disease was more indicative of lead poisoning, even though other tests also showed that she did have tuberculosis.[47]

In order to get a better handle on what disease or diseases the child had, and where they might have come from, Miriam Brailey, an intern at the Harriet Lane Home, visited the child's house. At the house, Brailey found the child's mother who "moved about with difficulty" and seemed "confused mentally." A neighbor, Melrose Easter, helped answer Brailey's questions and took the doctor on a tour of the house to look for fresh or loose paint and plaster, which Brailey knew to be a potential source of lead poisoning for children. After a "fruitless search" and the denial of Easter and the child's mother of the child having access to this potential source of poisoning, Easter suggested that maybe the family had become sick from breathing in the bad smelling vapors put off from burning battery casings – a practice common in the poor, East Baltimore neighborhood according to Easter. Brailey took a sample of the battery casings for lab work and they proved to be contaminated with lead. Within the same week, another child poisoning case was admitted to Provident Hospital. Physicians from these hospitals then contacted the Baltimore City Health Department based on the belief that the cause – a cause suggested by a community member – might be a widespread practice that public health experts would best handle.[48]

The Baltimore City Health Department threw itself into the new problem with great energy, organizing both the public health response in Baltimore as well as using the incident to better understand lead poisoning in general. Wilmer Schulz, the Director of Environmental Hygiene, carried out most of the on-the-ground work in Baltimore. The new health commissioner, Huntington Williams, meanwhile, enthusiastically pushed a research and education agenda that would extend beyond the particular Baltimore lead poisoning cases. In this, Williams had something of a successful template to work from: His own work on a public health poisoning issue in New York. In 1928 Williams, a district health officer, had gotten to the bottom of a mysterious mass poisoning that followed a convention dinner at a hotel in Utica. It was the cyanide-laden silver polish in the kitchen with the plates! Williams and his family were themselves poisoned in this way at a hotel in Atlantic City the following year. Because the cyanide poisonings were hard to distinguish from food poisoning, many hotels had been unknowingly serving poison on a silver platter to their guests for years. Williams' public health sleuthing, in which he investigated the kitchen and noticed the almond-smell of cyanide from the polish, was followed by more research and public health education and regulation of cyanide polish. He then published his research findings and public health action in the *Journal of the American Medical Association*.[49] Faced with the lead battery casing issue, Williams explicitly sought to replicate his past success in uncovering, mitigating, and publishing on an emerging public health poisoning mystery.[50] Williams' research ultimately took a two pronged approach, including both a study of the extent and dynamics of battery casing use in Baltimore and a study of lead poisoning victims themselves.

In the fall of 1932, Williams directed Schulz to carry out a number of surveys in the city of residents and those involved in the lead battery business and found significant, if spatially concentrated, recycling and reuse of both batteries, lead, and battery casings. Schulz found six scrap and junk shops that dissembled batteries. Five of these were concentrated in a relatively small five by seven block area in East Baltimore. One other was located in north central Baltimore. At the local level, the way the system worked was that automobile owners would take their used up batteries to numerous service stations and

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[47] Huntington Williams et al., "Lead Poisoning from the Burning of Battery Casings," *Journal of the American Medical Association* 100, no. 19 (1933): 1485–89.
[48] Ibid.
[49] Huntington Williams, "Cyanide Poisoning, Acute and Nonfatal, Apparently from Hotel Silver Polish," *Journal of the American Medical Association* 94, no. 9 (1930): 627–30.
[5050] Letter HW to Park, October 10, 1932, in Folder Lead Poisoning by Burning Battery Cases 1932-1934, Box 3.3-3.4, HW Papers.

battery repair shops that were scattered throughout the city and suburbs. The shops would then either deliver or sell at pick up batteries to the smaller number of junk shops in the city who would break the batteries apart and the sell the lead plates to a re-smelting factory in Locust Point. The battery casings were then sold or given to nearby residents in order to raise a little more money or at least avoid hauling the casings to the dump. In some cases, they were burned in the junk yards to dispose of them.[51]

Of course, this local system was part of a much bigger national sociotechnical system of lead mining, automobile use, and battery recycling. Recovery of lead from batteries increased rapidly in the nineteen teens and twenties, and then leveled off during the depression of the 1930s. Some small scale lead recycled took place in the nineteenth century and the early twentieth century.[52] In 1910, industries recovered 55,422 short tons of lead. This more than doubled to 124,650 in 1920 and then more than doubled again to 255,800 in 1930.[53] These increases reflected the rise of the automobile in American life: By this point, cars batteries dominated an emerging, if highly incomplete, lead recycling loop: Most lead was used to make automobile batteries, and most of the supply of secondary lead was automobile batteries.[54] While the Depression hurt many scrap processors, putting some out of business, some metals, like iron, fared particularly bad.[55] Although the production of storage batteries declined considerably, secondary lead recovery only fell to 198,300 in 1932, and climbed back up after that.[56] The reason was that lead recovery was evidently generally more economical than lead mining (primary production). In 1931, secondary lead production equaled 53% of primary production, but by 1932 it had risen to 69%. The stock of batteries "kept up remarkably well in 1932," the Bureau of Mines reported, especially in urban areas, and there was an "active demand from smelters all year." Backlash against the rebuilding of batteries, which were often faulty, also increased battery scrapping, as consumers began avoiding rebuilds and metal trade associations pressed scrap dealers to break up batteries so that they could only be smelted, not rebuilt.[57]

In Baltimore, however, lead battery recycling at the junk shops Schulz investigated actually appears to have increased during the early years of the Great Depression. Several of the smaller operators reported increases between 1929 and 1931 and Berg Bros, who was by far the largest dealer in batteries, doubled the amount they received from about 1000 per week in 1929 to 2000 per week in 1932.[58] It is unclear if the increase represents an increase in battery recycling in urban regions (in contrast to the national patterns), or whether some junk dealers had gone out of businesses and those that remained received more batteries. Either way, more lead from Baltimore's "lead shed" was funneled into the urban core during the Great Depression.

This was an urban core that was not only relatively poor, but also had a far more circumscribed set of energy and heating options that the rest of the city and suburbs. Baltimore had been a very early innovator in gas lighting systems in the nineteenth century, and in the early twentieth century, under the direction of the Consolidated Baltimore Gas and Electric Company, it had become famous for expansive development of gas and electric service. The CBGE had heavily subsidized rates in the nineteen teens for residences in an effort to get residents to adopt gas over wood and electric heating, and by the 1920s its coverage spread impressively well beyond the city limits into the suburban Baltimore County. The

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[51] Memo Schulze to HW, October 29, 1932 and Letter "A. Citizen, A. Voter, A. Tax Payer," to HW, September 15, 1932, both in Folder Lead Poisoning Cases 1932, Box 3.3-3.4, HW Papers.

[52] Geological Survey (U.S.), *Flow Studies for Recycling Metal Commodities in the United States* (U.S. Dept. of the Interior, U.S. Geological Survey, 2004), F–1.

[53] *Statistical Abstract of the United States* (U.S. Government Printing Office, 1939), 725.

[54] United States Bureau of Mines, *Mining Industry of the United States of America* (U.S. Government Printing Office, 1922), 39.

[55] Carl A. Zimring, *Cash for Your Trash: Scrap Recycling in America* (Rutgers University Press, 2009), 83.

[56] *Statistical Abstract of the United States*, 725.

[57] United States. Bureau of Mines and Geological Survey (U. S.), *Minerals Yearbook* (Washington : Bureau of Mines : Supt. of Docs., U.S. G.P.O., 1933), 168. For problems with rebuilt batteries, see "Battery Gyp," *Popular Mechanics* (Hearst Magazines, 1933), 675.

[58] Memo Schulze to HW, October 29, 1932 in Folder Lead Poisoning Cases 1932, Box 3.3-3.4, HW Papers.

coverage, however, was purely theoretical for many residents of the urban core. These residents either could not afford hookup fees for their homes or, more likely, lived in poor rentals that had not been hooked up to gas or electric service by their owners. These residents thus lived a donut hole of energy service, as Figure 2, shows. This game them fewer options for heating, did not allow them to benefit from some systematic attempts at relief during the Great Depression such as the CBGE's voluntary rate reductions, and generally required them to burn dirtier fuel to cook and heat, whether that was coal or ultimately used lead battery casings.[59] According to junk dealers, residents started using these casings in small numbers in the late 1920s, but as the Depression hit and as word of mouth spread about the casings, use of them increased considerably.[60] Burned in crowded houses with leaky stoves, the fumes from these casings poisoned families over the course of many months of exposure.

[59] Thomson King, *Consolidated of Baltimore, 1816-1950: A History of Consolidated Gas Electric Light and Power Company of Baltimore* (The Company, 1950), 271.
[60] Memo Schulze to HW, October 29, 1932 in Folder Lead Poisoning Cases 1932, Box 3.3-3.4, HW Papers

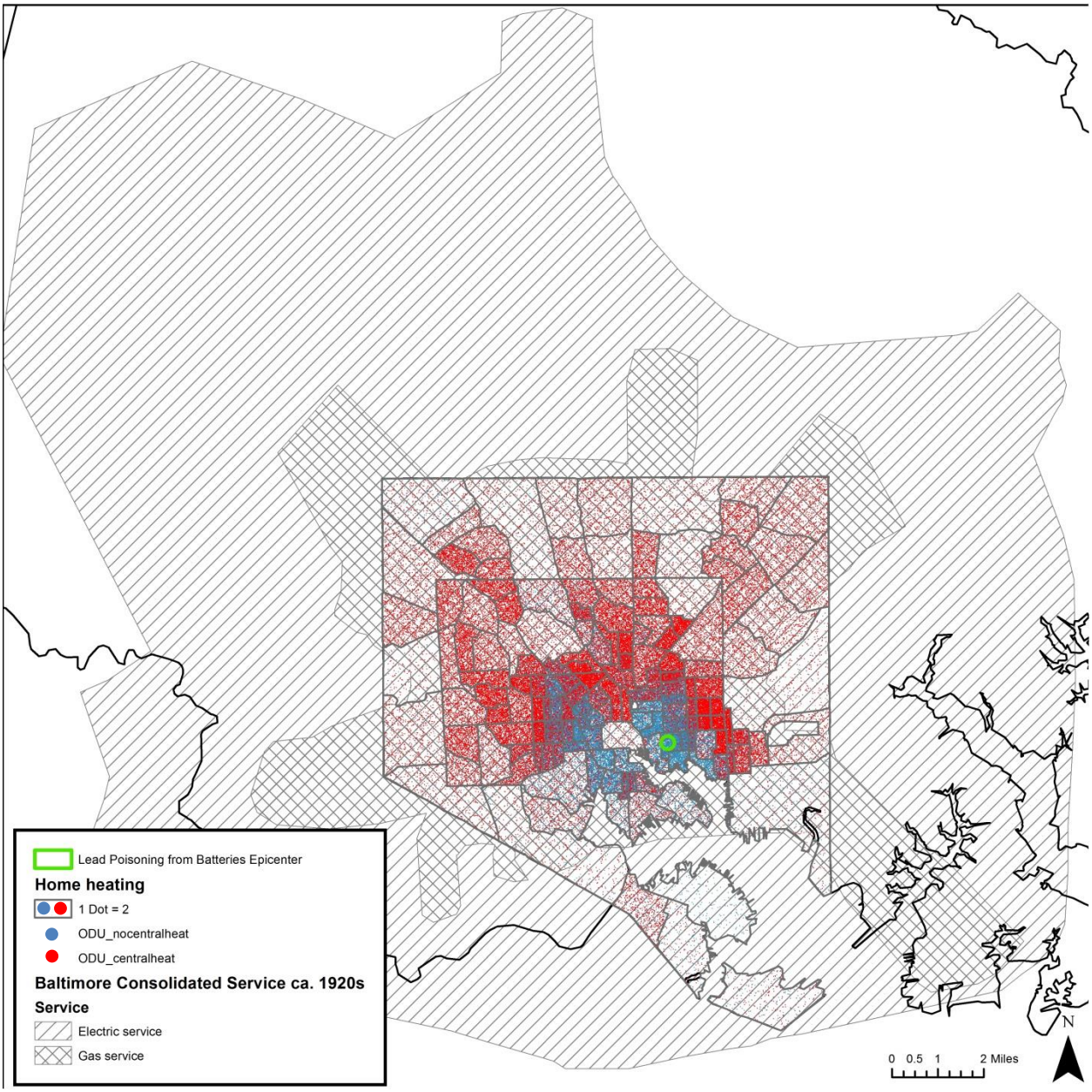

Figure 2:Map of Baltimore City and surrounding area showing gas and electric utility coverage compared with homes (occupied dwelling units or ODUs) with and without central heat. As the map illustrates, despite wide coverage, many homes in the urban core had no central heat and it is in one of these gas/electric "deserts" that the battery case burning epidemic was centered. Source: Census data on central heating from NHGIS Minnesota Population Center; battery burning cases locations from Folder Lead Paint Poisoning Cases 1931-1932, Box Restricted from Series III, HW Papers; Delbert B. Lowe, *History of the Consolidated Gas, Electric Light and Power Company of Baltimore*, January 6, 1928.

Although the most serious cases of lead poisoning affected families who burned battery casings, lead batteries exposed people in the urban core in other ways as well. Some of the workers from these neighborhoods, for example, were directly employed in the lead battery recycling business. The battery burning cases brought increased scrutiny to these business, but even before they arose workers in both battery junking businesses and lead re-smelting businesses had come to the attention of the department for lead poisoning cases. Berg Bros, the major battery breaking facility, and Chesapeake Smelting and Refining Corporation had had several cases of occupational lead poisoning. The businesses were owned by the same man, Mr. Berg, who appears to have been far less amenable to the educational approach of the health department to mitigating lead poisoning.[61] "From witnessing the procedure it is evident that considerable lead adheres to the hands of these employees," Schulz wrote to Williams. "Although we have cautioned employers of the possible dangers of these men getting lead poisoning little attention appears to be given toward preventative measures."[62]

But the funneling of lead batteries to the urban core also exposed the broader community to lead hazards, beyond the specific workers in the lead industry and families that used casings for fuel. When a public health nurse went to investigate the homes of children with lead poisoning in 1932, she arrived in a neighborhood she noticed the "atmosphere heavily laden with smoke." Residents told her that the smoke came from the chimneys and "to a great extent from an open fire in the rear yard of the Philadelphia Rubber and Metal Company," where the casings were burned after the lead plates were salvaged.[63] After the battery case burning gained public notice in the newspapers in September 1932, a resident wrote to Huntington Williams to inform him that while burning these cases might be problem, worse was the community pollution from the Chesapeake Smelting and Refining Corporation that re-smelted the lead batteries. The writer claimed the plant was "working night and day," smelting 25 tons of battery plates a day and "filling the city with these fumes." The plant's proximity to water brought the fumes closer to the ground and on rainy days "the fumes are terrible and as we breath the air our lungs are filled with this lead fumes [sic]". The writer asked to Williams to investigate, but there is no indication the BCHD did except for occupational lead poisoning.[64]

Not surprisingly, given the spatial concentration of the lead battery recycling industry, the effects of lead exposure were borne disproportionately by certain populations. Of the 57 lead poisoning cases that eventually resulted from battery case burning all, except one, involved African American families. This was because the battery junk shops were mainly located in predominantly African American neighborhoods and also because relative whites, blacks in the same neighborhoods probably had fewer resources. In terms of occupational hazard, every single one of the battery breaking laborers was African American. This was notable to the BCHD, which probably indicates that it was an intentional segregation of work by race and lead hazard on the part of employers.[65]

At the community level, lead exposure was the result of spatial segregation by both race and class. Baltimore had been a pioneer in racial segregation, passing the first racial zoning ordinance in 1911. Although these was overturned by the Supreme Court, realtor agreements, neighborhood association covenants, and less organized racial intimidation helped keep African Americans in precisely the sections of Baltimore that emerged as areas of lead poisoning.[66] In addition, many working class Baltimoreans,

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[61] Letter Schulze for Mr. Berg, Chesapeake Smelting and Refining Corporation, September 16, 1932, Folder Lead Poisoning by Burning Battery Cases 1932-1934; Memo Schulze to HW, October 29, 1932, Folder Lead Poisoning Cases 1932, both in Box 3.3-3.4, HW Papers.

[62] Memo Schulze to HW, October 29, 1932, Folder Lead Poisoning Cases 1932, Box 3.3-3.4, HW Papers.

[63] Letter William Warthen, Director Bureau of Child Welfare, to HW, September 7, 1932, Folder Lead Poisoning by Burning Battery Cases 1932-1934, Box Restricted from Series III, HW Papers.

[64] Letter "A. Citizen, A. Voter, A. Tax Payer," to HW, September 15, 1932, Folder Lead Poisoning by Burning Battery Cases 1932-1934, Box 3.3-3.4, HW Papers.

[65] Memo Schulze to HW, October 29, 1932, Folder Lead Poisoning Cases 1932, Box 3.3-3.4, HW Papers.

[66] Sherry H Olson, *Baltimore : The Building of an American City* (Baltimore: Johns Hopkins University Press, 1981).

both black and white, could not afford to live far from their work because housing outside of the urban core was expensive and they could not afford automobiles or mass transit. Thus both white and black working class Baltimoreans were concentrated in the urban core that was polluted by lead battery processing. The biggest polluter was probably the Chesapeake Smelting Corporation located in Locust Point, a community that had large numbers of both whites and blacks. The informant for the public health nurse that noticed heavy smoke in the neighborhood she visited was white. So it likely that lower level (but still very significant) amounts of lead exposure happened to a larger and more diverse swath of Baltimoreans. Many of these Baltimoreans who were concentrated in the urban core lacked affordable access to heating and transportation – and ultimately energy – even though they lived and worked in the midst of industries devoted to energy.

The class, race and poverty aspects of the battery burning cases made an effective response to the problem totally different from the silver polish incident, even if the epidemiological investigation had some similarities. Lead and silver might have been mined together, but the way these metals made their way out into American society and became implicated in the poisoning of people could hardly have been more different. The silver polish poisonings happened in expensive hotels at the height of roaring twenties, the lead battery poisonings at the depths of the Great Depression in the poorest parts of cities.[67] The BCHD, however, essentially took the same tack as Williams had in dealing with silver polish: research, education, and mild regulation. Williams was a master of public relations, and put his skills to use quickly and effectively. The BCHD helped publicize the issue at the local level using its own Health News, helping run stories in Baltimore's newspapers,[68] and broadcast warnings on the radio. It also carried out door to door surveys that informed many residents and businesses of the problem. At the national level, Williams sent close to a hundred letters and copies of reports to other health departments, physicians, and those working in the battery and lead industry. Although a few other cities like Detroit and Philadelphia also had poisonings from battery cases, Williams aggressive networking probably helped save the health and perhaps lives of children in many cities that took preemptive measures. Williams also placed a news item in the *Journal of the American Medical Association* and ultimately a short article.[69]

In addition, the BCHD helped to remove access to battery casings themselves. Williams made a sort of *ad hoc* regulation against selling or giving away battery casings, bringing the police commissioner on board to help with education and enforcement. After junk dealers complained that the garbage dumps had raised their prices on battery casings in response to increasing need to dispose of them, Williams arranged with the city engineers to have casings delivered and incinerated for free at the city incinerator. For all this, however, Williams' approach would have had very limited effectiveness on its own. Unlike silver polish, technological substitutes were not easy to come by – one could not, for example, supply gas or electric heat given that these residences had never been hooked up. And unlike silver polish, substitutes were necessary. Although some households who were use battery casings did switch to coal once they learned of the lead poisoning danger, others were reticent about saying whether they used casings. Others, like one man with 400 casings in his cellar, refused to give them up until another fuel was delivered.[70]

Although BCHD memos sometimes expressed surprise that households continued to use or hold on to battery casings, the reason is not hard to explain. It was not that residents were unconcerned with lead poisoning – they often expressed concern – rather it was that heating was quite literally a matter of life and death for some of these families.[71] During the harsh winter of 1933-34, a reporter in a poor part of Baltimore relayed stories of a mostly bed-ridden woman who "burned paper and rags in the egg beater"

[67] United States Bureau of Mines, *Mining Industry of the United States of America*, 38.
[68] The Afro-American did not carry a story on it, though Williams did contact the paper about it. But the Sun and the Baltimore American covered the story.
[69] Williams et al., "Lead Poisoning from the Burning of Battery Casings."
[70] Handwritten note, from Schulze, no date (probably circa March, 1933)
[71] XXX

to keep her room warm after she ran out of coal. Another women believed she would have frozen to death if the grocery store had not extended emergency credit to her husband. A family literally in rags with no way to heat their house despaired at what would happen to them. And a single father begged for coal to heat the "icebox" where he cared for his young girl.[72] Hospitals reported several cases of frostbite, and in December 1933, a 40 year old African American who lived, a few blocks east of the battery burning cases, froze to death *in his home*.[73]

A solution to the problem, in other words, required providing another fuel source so that households would not either freeze to death or continue to seek out battery casings. This solution was provided by the Family Welfare Association, Baltimore's largest charitable organization that had, by 1932, been almost completely overwhelmed by the economic hardship of the Great Depression.[74] Nevertheless, by March of 1933, Schulze found that virtually all the families he surveyed had stopped using casings. A few stated they were purchasing their own coal from the store (perhaps with credit extension as in the case mentioned above). But the majority were receiving coal from the FWA, for which several families worked.[75] Even the man with 400 cases in his cellar yielded them after receiving promises that the FWA would continue to supply fuel.[76] After 1933, there were no more cases of lead poisoning from battery cases for the remainder of the 1930s.

The twentieth century was a period of incredible expansion in energy use, energy quality (the form of energy), and thus the flexibility that Americans had in terms of places to live, their daily movements, and their ability to heat their homes. Yet the benefits and costs of this new and expanded energy sources and technologies were not shared equally. This was evident in Baltimore even by the 1930s as automobile suburbanization at once greatly increased the stock of lead batteries that were funneled into the urban core for processing where they poisoned the bodies and brains of many residents. This lead exposure happened most famously in homes, but also in work places and in the community generally. Those affected not only bore a burden of pollution from the energy and material consumption of those on the periphery of Baltimore who depended on automobiles, they often lacked basic access to energy to heat their homes (especially during the Depression) and this privation was itself a catalyst for poisoning by using battery casings.

The solution to the battery casings required knowledge about the cause, education, and a material alternative to casings fuel. The BCHD played a crucial role in developing knowledge of the problem, in educating on a local and national level, and in helping removing casings from the urban core. But other played crucial roles as well, especially the Family Welfare Association with provided a viable alternative to battery casings.

Members of the community also played crucial roles, and their actions generally indicated informed concern rather than passive ignorance. Melrose Easter first suggested the cause of the disease, for example, and to their credit, a number of historians and public health researchers who recounted this episode of public health history have noted Easter's role. Nevertheless, they are more likely to quote Brailey on how Easter had bloodshot eyes and smelled of whiskey than they are to note what Easter said about himself: "Melrose had had a year or so at Tuskegee Institute and confided to the doctor that he had had ideas of studying medicine," Brailey wrote.[77] Similarly, although the historian Christopher Sellers has emphasized Easter's role, he describes Easter's suggestion of "vapors" as the cause of lead poisoning as "a-medical" and "worlds away" from Brailey's understanding of the world. Brailey by virtue of being a doctor, would likely not have made the connection between the vapors and lead poisoning, Sellers

[72] "Reporter Finds Much Distress in Baltimore's Poorer Streets," *AA*, February 17, 1934.
[73] "Cold Wave Here Claims Victim," *AA*, January 6, 1934, 12; "Negro Is Frozen to Death in Room," *Sun*, December 28, 1933, 20.
[74] Jo Ann E. Argersinger, *Toward a New Deal in Baltimore: People and Government in the Great Depression* (University of North Carolina Press, 2011).
[75] Memo Schulze to HW, March 30, 1933
[76] Handwritten note, from Schulze I think, but maybe HW, no date
[77] Williams et al., "Lead Poisoning from the Burning of Battery Casings."

suggests.[78] While these descriptions are intended to give dignity to local knowledge and highlight Brailey's prejudices, they come close describing community members as something like idiot savants. It seems like Easter considered his hypothesis medical, since he went on to discuss medical school and what organ systems he had studied. The community member who complained about the Chesapeake Smelter not only understood the fumes to contain lead, but understood how the rain and the nearby water created microclimates that made the pollution worse. The reverse was also true: The public health nurse visiting the neighborhood of lead poisoning victims was not precluded from thinking that the heavy smoke in the air was related to lead poisoning despite her medical background.

Community knowledge and the knowledge of health professionals can certainly be different, but, in cases like those here, they are not worlds apart. The distinctions between either local ignorance and health expertise, or between savant local knowledge and science, are more a product of how health professionals have portrayed or ignored community knowledge than categorical differences. It is worth considering how close Easter was, for example to being completely excluded from the lead battery poisoning story. Huntington Williams, at times a near ideal-type of progressive but elitist expert, was reluctant to give Easter credit for the discovery. The first *Health News* story credited Brailey with the "brilliant" discovery. Brailey reminded Williams twice, in a letter and phone call, that while she was flattered, it was Easter, not her, who suggested the cause. Williams' subsequent *JAMA* article still credited Brailey with the discovery, although it also included Brailey's own account that related the story about Easter. The community member who wrote asking for an investigation of the lead fumes from the smelter *was* essentially erased. The BCHD appears to have never investigated it and it never entered the public discourse or documentary record. It's hardly the case that

Community members affected by lead poisoning could make inferences about what was happening, they could understand and were concerned about the health consequences, and they took action to make their situation with the broader structural constraints they lived in. This is important to keep in mind, for as lead poisoning from battery casings faded from the center of attention, a new lead poisoning epidemic emerged and with it a more flagrant image of ignorant and passive victims of lead poisoning.

Section III: The Rise of Childhood Lead Paint Poisoning, 1890s-1950s

Going by the records of hospitals and the Baltimore City Health Department, child lead poisoning cases emerged as an annual fact in the 1920s, spiked in the 1930s, only to drop a little and plateau in the 1930s and 1940s, before spiking several times in the 1950s, and then, finally, dropping down for one last big spike in the 1970s (Figure 3). The records of cases are, of course, a reflection of many factors, including changing definitions of lead poisoning, changing diagnostic technology, changing awareness, attention, and budgeting among health professionals for lead poisoning, changing behaviors of individuals, and a changing social and environmental context. Historians have made a number of claims about trends in child lead poisoning like, and including, the trends from Baltimore, and have focused in particular on the rise in child lead poisoning cases in the 1950s. In this section, I will engage with several aspects of these claims.

[78] Christopher C. Sellers, "The Dearth of the Clinic: Lead, Air and Agency in Twentieth-Century America," *Journal of the History of Medicine and Allied Sciences* 58, no. 3 (2003): 255–91.

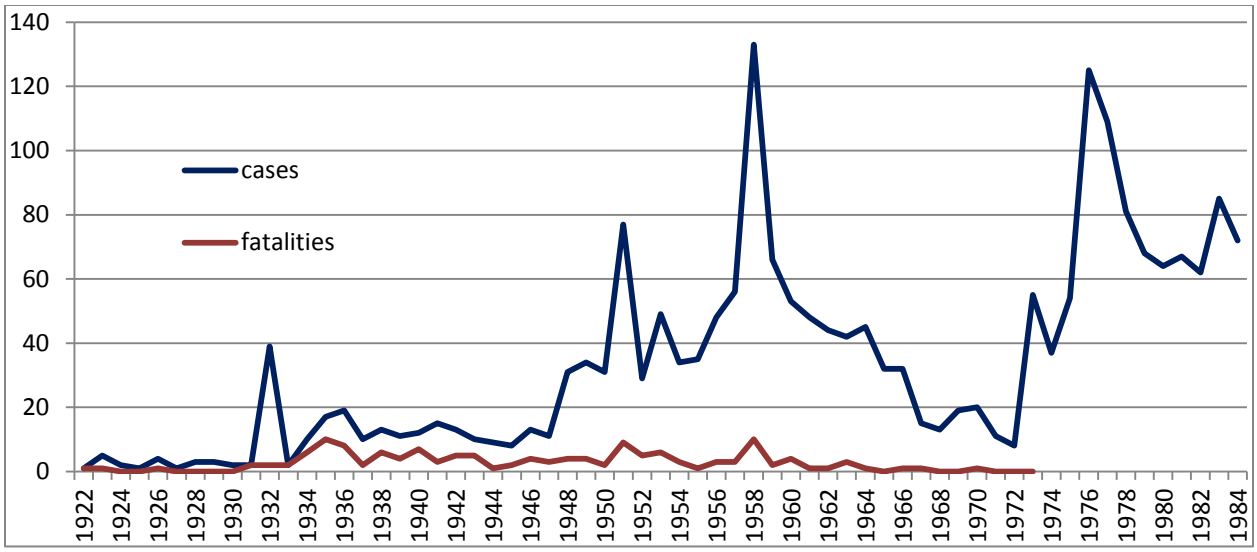

Figure 3: Child lead poisoning cases and deaths in Baltimore City, 1922-1984. The patterns of childhood lead poisoning are a product of changing exposures, changing diagnostic definitions, changing diagnostic technologies, and changing medical awareness and attention. The spike in the early 1930s is from the lead battery case burning epidemic while the spike in the immediate post-war period was attributed by the Baltimore City Health Department to deteriorated housing and pica. The spike in the 1970s is the result of city-wide screening and a more liberal definition of lead poisoning. Graph by author using the following sources: BCHD *Annuals* and *Vital Statistics*; "Pediatric Index," Edwards Park Collection, JHMA.

Historians can be roughly divided into two camps regarding the rise of child lead poisoning. The first camp argues that health professionals were basically correct in their diagnoses of lead poisoning and in their causal attribution of lead poisoning. There were few child lead poisoning cases reported in the early twentieth century because there were few actual cases. Cases exploded after World War II because there really were many more cases. And these cases were primarily the result of what health professionals at the time attributed the rise in poisoning to: deteriorated housing combined with "pica" – the compulsive eating of non-food materials like paint chips and plaster.[79] Other historians have argued that child lead poisoning was a "silent epidemic," in time and place. Child lead poisoning burst on to the scene in places like Baltimore in the 1950s because public health professionals started really looking for it. If they had had the same knowledge, technology and motivation in the early 1900s, or in other cities that reported far less lead poisoning than Baltimore, they would have found child lead poisoning in great numbers there, too.[80] Moreover, the strong emphasis on pica was wrong either because pica was simply a social construction, or because pica was a real, but non-pathological conditions, and pathologizing it was simply a way of "blaming the victim."[81]

In this section I make two arguments relevant to this historiography. First, I argue that the increase in child lead poisoning in Baltimore in the 1950s was multi-factorial. There is a great deal of evidence that deteriorated housing increased in this period in parts of the city and that this contributed to child lead poisoning by making more peeling and chipped paint available. But this was not the only way that lead exposure increased, and since lead absorption is cumulative all of these exposures need to be considered. Others that I argue were important include high summer winds, a factor that is well-recognized as a contributor to blood lead levels but that no other historian has considered for this period. I also argue that demolitions from urban renewal projects may have played a role, and that, perhaps most

[79] English, *Old Paint*, 2001; John C. Burnham, "Unraveling the Mystery of Why There Was No Childhood Lead Poisoning," *Journal of the History of Medicine and Allied Sciences* 60, no. 4 (2005): 445–77.
[80] Warren, *Brush with Death*; Fee, "Public Health in Practice."
[81] Warren, *Brush with Death*; Rosner and Markowitz, "'A Problem of Slum Dwellings and Relatively Ignorant Parents.'"

importantly, increasing traffic and traffic congestion from suburbanization contributed to the rise in child lead poisoning cases in the 1950s.

Understanding the multiple, increasing, cumulative lead exposures at mid-century is important because it tells us something about the broader processes contributing to lead poisoning. Automobile based suburbanization did not change wind patterns in the 1950s, but it did contribute indirectly to deteriorating housing and urban renewal projects, and it contributed directly to traffic congestion in the urban core. Understanding these exposures is also important because it can tell use more about the extent of lead poisoning in the city, including what the extent of non-diagnosed, chronic lead poisoning may have been – and where it may have been happening.

Finally, it is important because it gives us a better purchase on the extent to which professionals at the time were correct in their diagnoses and explanations. And this brings me to my second argument: While pica does seem to have played an important role in child lead poisoning in Baltimore, its explanatory power was over-played at the time. Although there was definitely a victim-blaming dynamic to the pica-based explanation, this was not the main reason it came to have such a hold on health professionals. Rather, I argue, health professionals had almost no other explanation and they had limited, practical diagnostic tools. The latter problem led them to rely on the finding of pica in children as a diagnostic indicator, which in turn led to selection bias in their findings.

* * *

Until the lead battery case burning epidemic in Baltimore, childhood lead poisoning was not really regarded as an important public health issue. In Australia, child lead poisoning from paint had produced a large public health problem, as early as the late nineteenth century. These cases came to the attention of American health professionals when the physician J. Lockhart Gibson published a 1904 paper describing lead poisoning in a number of children in Queensland, Australia. In these cases, old lead paint on porches had crumbled to a fine dust, which then got on the sweaty hands of children, where it was subsequently ingested when children licked their fingers.[82] About a decade later, in articles published in *JAMA* in 1914 and 1917, Kenneth Blackfan, a physician at Johns Hopkins, famously described several cases of childhood lead poisoning from interior paints in the United States. These children, who ingested lead paint by chewing on cribs and furniture, lived at the Harriet Lane Home for Invalid Children in Baltimore.[83] After Blackfan's articles, physicians in Baltimore and in other cities began recording child lead poisoning cases more regularly, even if not in large numbers, almost all of which involved the ingestion of lead paint. Like all diseases, lead poisoning has certainly under-reported, although how much it was underreported for child cases in the late nineteenth and early twentieth century is unclear.

Did doctors just become more aware of lead and better at diagnosing it in the twenties or were child lead poisoning cases increasing?[84] Depending on how one defines "child," child lead poisoning had probably been going on for some time because people under the age of 18 were employed in some of the work that had lead poisoning problems. In the nineteenth century Baltimore, for example, job postings for a file cutter's apprentice sought workers between the ages of 15 and 17. In United States as a whole, according to historian Peter English, most of the very young cases of child lead poisoning in the nineteenth and early twentieth century involved food or water, with a few cases stemming from ingestion of fresh lead paint.[85] In Baltimore in 1904, the only case of child lead poisoning at Johns Hopkins was a five and a half year old girl who was "in the habit of eating the remains of food left in tin cans." She was poisoned enough that she became unable to walk and was hospitalized.[86] After this, there are no more cases discussed from Johns Hopkins until Blackfan's articles.

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[82] J. L. Gibson, "A Plea for Painted Railings and Painted Walls of Rooms as the Source of Lead Poisoning amongst Queensland Children," *Australian Medical Gazette* 23 (1904): 149–53.
[83] Fee, "Public Health in Practice."
[84] Warren, for example, suggests something like this. Warren, *Brush with Death*.
[85] English, *Old Paint*, 2001, 10–11.
[86] Thomas, "Generalized Lead Paralysis," 211.

Although lead poisoning was difficult to diagnose, doctors were certainly aware of it – it was a major issue in occupational health and policy at the time – and they did diagnosis it.[87] But it is also certain that some serious cases did go undiagnosed and also that some were diagnosed but were not well-recorded or remembered. For example, while every history of child lead paint poisoning usually traces the first cases to the Blackfan cases in 1914, a similar case took place in San Francisco in which a girl of about six years got lead poisoning after she ate white lead paint coating the "crib" she slept in. She lost the ability to walk.[88] More than these sort of extremely serious cases – i.e., where children literally died or became paralyzed – it's likely that many serious, but less extreme cases went undiagnosed. As one publication reporting on the San Francisco case noted, "There is probably more of the sub-acute lead poisoning in children than is diagnosed." There was after all plenty of lead paint in the world of children by this point – on their walls, furniture, and toys – and that paint would have found its way into the exploratory and rather indiscriminate mouths of children.[89]

Given the diagnostic techniques of the early twentieth century for lead poisoning, however, it was very unlikely that sub-acute cases would come to the attention of physicians. For this reason, knowledge about childhood lead poisoning in the early part of the twentieth century was confined by, and indeed defined by, lead poisoning cases at the extreme end of the poisoning spectrum. The more mild (relatively speaking) aspects of lead poisoning were not visible to researchers.[90] In this era, children with lead poisoning, if they were not dead, were children with encephalopathy, seizures or lead colic. Achieving these extreme symptoms required ingesting or inhaling a lot of lead.

The fact that child lead poisoning cases were extreme poisoning cases shaped the way physicians understood the cause of lead poisoning when they began thinking and publishing more on the subject in the 1920s.[91] These articles were all case studies of two or three patients, unlike the public health studies of Gibson in Australia and the subsequent study by Williams of the battery cases in Baltimore in the 1930s that looked at dozens of patients. Among the most influential of these early case study articles was Los Angeles physician John Ruddock's "Lead Poisoning in Children With Special Reference to Pica," published in *JAMA* in 1924. In the article, Ruddock described a strange condition called pica, which he defined as "a craving for unnatural articles of food – a depraved appetite." Following other researchers, he suggested that pica manifested itself in two cases: 1) individuals of any age who suffer from diseases and malnutrition; and 2) children with no particular maladies who develop this "morbid craving" out of an exaggeration of the "normal habit in young infants of invariably placing everything within reach of their mouths." The latter form of pica was the more common. Although pica was generally harmless, for children who had access to lead objects –Ruddock noted that "a child lives in a lead world" – it could be dangerous and even deadly.[92]

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[87] Some in the paint industry, indeed, argued that doctors had "lead on the brain" and were over-diagnosing lead poisoning. "It is not even necessary to be in a factory" to be diagnosed with lead poisoning, the report fretted. *The Chemical Trade Journal and Oil, Paint and Colour Review* (Davis Bros., 1898), 245.

[88] The crib was painted in the summer or fall of 1892. It was not varnished and took "many months" to dry, during which time the girl "used to scratch it off with her finger nails." It was not until December of 1893, that she exhibited serious symptoms, so it is likely she continued eating paint after the paint was dry. Leo Newmark, "Lead Palsy in Children," in *Medical News* (Henry C. Lea's Son & Company, 1895), 505–506.

[89] Horatio C. Wood, *American Medico-Surgical Bulletin* (The Bulletin Publishing Company., 1896), 534. This publication noted specifically that lead paint was used on children's toys. A few other publications also noted Newmark's study, including William A. Edwards, *Diseases of Children, Medical and Surgical: Supplement to Keating's Cyclopaedia of the Diseases of Children* (Lippincott, 1901), 1203.

[90] Even the extreme effects were not straightforwardly indicative of lead poisoning. They might be caused by other diseases, such as tuberculosis. If clinicians suspected lead poisoning, they would then check for other diagnostic signs, such as a black line on the gums or stippling of red blood cells.

[91] Fee, "Public Health in Practice."

[92] John C. Ruddock, "Lead Poisoning in Children with a Special Reference to Pica," *Journal of the American Medical Association* 82, no. 21 (1924): 1682–84. Ruddock was a privately practicing physician and active member

The association of child lead poisoning with lead paint and pica was fortified over the 1920s and 1930s with other studies and ultimately became a cornerstone of how the Baltimore City Health Department understood child lead poisoning in the 1930s after the subsistence of the battery burning cases. Before the 1930s, the BCHD took no notice of child lead poisoning. Some Baltimore physicians did, however, especially Edwards Park, a pediatrician at Johns Hopkins who included them in an index of child diseases that he began in the early 1920s. Although the number of cases was never large in any year in the 1920s, they did grow slightly, and Park became concerned enough that when Huntington Williams took the helm of the BCHD he contacted Williams to urge him to take action. Williams apparently agreed, but was subsequently swamped by child lead poisoning cases from battery case burnings in 1932.[93]

The campaign against battery casing burning was a great success for Williams. No doubt emboldened by this success as well as troubled by the continuation of child poisoning cases from lead paint, Williams set the Baltimore City Health Department on permanent crusade against childhood lead poisoning. The Baltimore Health Department also gained important public health expertise when the Rockefeller Foundation funded a five year public health collaboration between the Johns Hopkins School of Public Health and Hygiene (itself a product of Rockefeller money) and the city in 1932. Although the grant expired after five years, the city and JHU continued to work closely together on public health projects, especially lead poisoning, for the remainder of the twentieth century.[94]

Through the energetic work of Huntington Williams and with the expanded scientific capacity afforded by the connection with Johns Hopkins, the Baltimore City Health Department began producing a number of research articles and reports. Virtually all of these established a strong connection between pica, paint and lead poisoning. In the first scientific article on the prevalence of lead paint poisoning in a U.S. city, J.M. McDonald from the Bureau of Occupational Diseases and Emmanuel Kaplan from the Health Department claimed that of the children lead poisoned between 1931 and 1940 (excluding the battery casing burning victims) "practically all had a history of pica associated with chewing of objects painted with lead-containing paints." In 1943, the Health Department's *Annual Report* stated that for the 11 cases of lead poisoning in 1942 an "investigation of the source of poisoning was made in each case and the usual history of pica was obtained."[95]

As clear as these connections between pica and lead poisoning appeared to researchers, the Health Department never emphasized the role of pica in public, for the 1930s and early 1940s were a time of relative optimism about lead poisoning. Indeed, the Health Department constantly suggested that the eradication of the childhood lead poisoning scourge was imminent. But then Baltimore, like many other cities, experienced an upsurge in childhood lead poisoning cases beginning in the late-1940s, which then spiked even higher in the 1950s (Figure 3). This significant rise in cases, accompanied by a steady but less pronounced rise in deaths, presented a serious challenge to how public health officials understood and responded to childhood lead poisoning.

The crisis of rising lead poisoning cases resulted in an intensified research effort to understand patterns of lead poisoning, ways of screening and diagnosing the disease, and ways of treating and educating people about it. A major outcome of this research, not always intentional, was an increasingly public and scientific emphasis on the role of pica in lead poisoning and, consequently, an effort to understand and subdue pica. Ironically, while these studies showed that pica was indeed a risk factor for childhood lead poisoning, they also showed that the factors leading to lead poisoning were much more complex than emerging public discourse pica and lead suggested.

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of the American Medical Association. See C. B. Pinkham, "Medical Licensure in California," *California and Western Medicine* 35, no. 3 (September 1931): 167–169.

[93] Fee, "Public Health in Practice."

[94] John Duffy, *The Sanitarians: A History of American Public Health* (University of Illinois Press, 1992), 261.

[95] John M. McDonald and Emanuel Kaplan, "Incidence of Lead Poisoning in the City of Baltimore," *Journal of the American Medical Association* 119, no. 11 (1942): 870–872; Baltimore City Health Department, *Annual Report*, 1943, Baltimore City Health Department Archives.

In 1956, when the pediatrician J. Edmund Bradley published his study of childhood lead poisoning in Baltimore, the disappointment of an unfulfilled scientific hypothesis was barely detectable in the customary stoicism of peer reviewed science: Bradley summarized "that the use of the [coproporphyrin urine] test alone for screening will result in many children with abnormal amounts of lead in blood escaping detection." Bradley had hoped urine tests would be a cheaper and easier way to screen children for lead poisoning than blood tests. But no luck. He had not, however, come away empty handed. He also concluded that a history pica – the deliberate ingestion of non-food substances – was recorded in about 70% of the children with abnormal blood lead levels. "This one finding gave a higher correlation with blood lead than any other single test or symptom," Bradley noted, ringing a small note of triumph.[96]

Bradley's study was far from the first to draw a strong relationship between pica and child lead poisoning, but as the first cross-sectional study of blood lead levels and pica, it added an aura of specificity and objectivity to that relationship. Among the earliest of and most influential of these was a study by Edmund Bradley and several other researchers at the University of Maryland published in 1956. Examining the blood lead levels of hundreds of patients at the U of M's Pediatric Outpatient Clinic and the Baltimore Health Department's Well-Baby Clinic, the researchers' major finding was that a history of pica "gave [a] higher correlation with the blood lead than any other single test or symptom." 70% of children with a dangerously high blood lead level (over 50μ/dL), Bradley reported, also had a history of pica.

Although Bradley's finding was often interpreted as such, it was clearly not a straightforward validation of pica-centered explanation of child lead poisoning (Figure 4). If 70% of high PbB children with pica was an indication of a strong connection between the two phenomena, it showed that 30% of the cases of children with very high lead levels had *no* pica.[97] While there was clearly a significant relationship between pica and lead poisoning in Baltimore in the 1950s, there are several reasons why Bradley's seminal article gave an exaggerated impression of the importance of pica. First, Bradley did not report the pica prevalence of the population alongside pica prevalence by lead poisoning. According to Bradley's study, the pica prevalence was an astoundingly high 54%. In other words, the rate at which children with high PbB had pica was only about 15% higher than average amount of children with pica. For those children with a PbB in the 50-60 μ/dL range, pica prevalence was slightly *below* the average pica prevalence in the population (51% and 54% respectively). By analogy, if Bradley had reported that about 50% of children with PbBs in the 50-60 range were boys, this would not seem particularly surprising, since the average percentage of boys in the population is about 50%.

[96] J. Edmund Bradley et al., "The Incidence of Abnormal Blood Levels of Lead in a Metropolitan Pediatric Clinic: With Observation on the Value of Coproporohyrinuria as a Screening Test," *The Journal of Pediatrics* 49, no. 1 (1956): 1–6.

[97] In a subsequent article, Bradley suggested (as many researchers did) that parents under-reported pica in their children, either because of ignorance or shame. Although he did not say so explicitly, other authors (Greenberg?) suggested that this may have accounted for why there was still a significant portion of children with high PbB with no pica. Given Bradley's already very high overall pica prevalence, however, this seems extremely unlikely, as Byers pointed out in his review. R. K. Byers, "Lead Poisoning: Review of the Literature and Report on 45 Cases," *Pediatrics* 23, no. 3 (1959): 585–603.

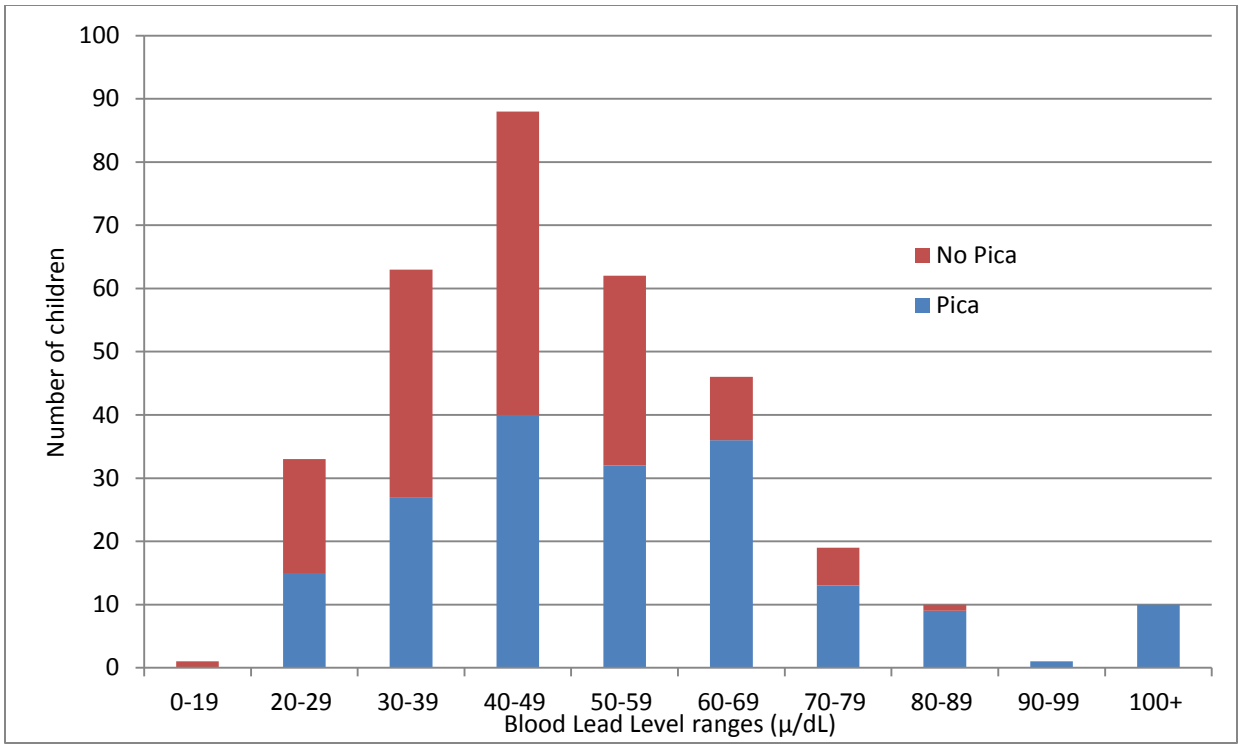

Figure 4: Number of children with pica by blood lead level. This data, based on children from a Baltimore clinic in the 1950s, indicates that a significant number of children with what was then considered a high blood lead level (over 50 µ/dL) did *not* have pica. Those with very high blood lead levels almost all had pica, however. It also shows that a very large proportion of children had pica – *if* the method of determining pica was reliable. Graph by author, based on data from Bradley, J. Edmund, et al. "The incidence of abnormal blood levels of lead in a metropolitan pediatric clinic: With observation on the value of coproporohyrinuria as a screening test." *The Journal of Pediatrics* 49.1 (1956): 1-6.

A related reason that Bradley's study exaggerated the importance of pica was that it reported only the pica prevalence for high PbB cases. The pica prevalence for the low PbB children (below 50µ/dL) was about 44%, a number that can be calculated based on Bradley's statistics but was not reported. This is still a very high number of children with pica, and is only 10% below the pica prevalence average for all children in the study. Putting the pica prevalence of all children alongside that for low and high PbB gives a considerably different impression of the importance of pica.

Finally, the 70% number did not give an impression of how the children were distributed in different blood lead level groups. Most of the children fell into the PbB levels between 20 and 60 µ/dL, and in this PbB range the pica prevalence only varied from 42% to 51%. An actual statistical correlation of pica and PbB suggests that pica "explains" only about 6.8% of the pattern (the variance, in technical terms) in PbB levels. This, of course, may in part be because the "model" does not include whether these children with pica lived in houses with lead paint, especially deteriorating lead paint. Even the most generous reading of Bradley's data (that for every child with lead poisoning and pica, the pica caused the lead poisoning), however, cannot escape the evidence that at least 30% of children with abnormal blood lead levels – levels that Bradley noted could result in symptomatic lead poisoning – did not have pica.[98]

[98] This correlation was run by making pica a dummy variable and taking the median value of each blood lead level group. Bradley categorized the last group as 90µ/dL or greater so the value for this was estimated by taking 300 µ/dL as the upper end of that range for calculating the median value. The regression gives a slope of 15.7 for pica (which is just the average difference between the blood levels of the groups) with a standard error of 3.2 and t-value

Overall, Bradley's data suggests that pica was probably very important for producing extremely high blood lead levels (above 80μ/dL); that pica could not account for a significant proportion of the overall lead poisoning cases; and that on its own, pica did not explain the overall pattern in blood lead levels very well at all.

If the invariable connection between lead poisoning and pica was questionable, so were the broader explanations that emerged as attempts to understand the etiology of pica itself and to use this etiology to explain the pattern of lead poisoning. When physicians connected pica to childhood lead poisoning in the 1920s, 30s, and 40s, they reflected these long-held notions of pica as pathological. Ruddock referred to pica as a "depraved appetite." The physician from Boston, Charles McKhann, who wrote a series of articles on childhood lead poisoning in the twenties and thirties argued that children with pica were mentally defective, and McKhann and several subsequent researchers suggested that the mental deficiencies associated with lead poisoning may have been the cause of lead poisoning. That is, mentally deficient children with pica ate lead paint and got poisoning. However, virtually all studies that actually tested this hypothesis found no difference in intelligence between children with pica and those with no pica.[99]

Another explanation for pica that emerged in the early-twentieth century was also folded into the lead poisoning literature beginning in the 1940s. In 1909 the Rockefeller Foundation initiated and funded a large scale campaign was to eradicate hookworm in the South. Public health researchers part of this campaign who sought to both outline the effects and ways of detecting hookworm found one putative symptom to be particularly useful: dirt eating. The "dirt eaters" were poor, rural whites who had previously been understood as depraved and lazy. But the discovery of high incidence rates of hookworm in their population at once lifted the charge of laziness against them and explained their dirt eating behavior: Hookworm resulted in anemia and dirt eating was hypothesized to be a way to recover nutrients, especially iron.[100]

The hookworm eradication campaign lasted into the 1930s when many public health researchers declared victory. The publicity the program had generated over the years, not least of which was tied to the fascination with the "dirt eaters" themselves, and the apparent success of the program helped to popularize the nutritional explanation of "depraved" eating habits. (Not to mention foreshadowing the use of pica a screening/diagnostic tool). In the 1950s, many public health researchers drew this explanation of pica into the literature on childhood poisonings, particularly lead poisoning. Marcia Cooper, a professor at Johns Hopkins, wrote one of the most comprehensive studies of pica in this period (the only full monograph on the subject). Her book documented the history of pica (which dated back at least to ancient times) and theories about its cause. She also carried out a large study of 784 children who visited the Mothers' Advisory Service. Cooper found that 21.9% of these children had pica; the prevalence for White children was 16.8%, and that for Black children was 27.2%. She found no difference in intelligence or family income between children with and without pica. The only significant difference was that children with pica had poorer nutrition, more illness and physical defects than the no-pica children. Cooper took this to be validation of the nutrition hypothesis about the cause of pica.[101] While other researchers also supported the relationship between malnutrition (especially anemia) and pica, other researchers called this explanation into question, both because of experimental studies that did

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of 4.9. Correlating blood lead and pica yields an R^2 of about .068. The correlation is statistically significant, but low.

[99] Get Needleman and other references and check these. McKhann CF, "Lead Poisoning in Children: The Cerebral Manifestations," *Archives of Neurology & Psychiatry* 27, no. 2 (February 1, 1932): 294–304; Marcia Mann Cooper, *Pica: A Survey of the Historical Literature as Well as Reports from the Fields of Veterinary Medicine and Anthropology, the Present Study of Pica in Young Children, and a Discussion of Its Pediatric and Psychological Implications* (Thomas, 1957).

[100] Frances Maule Björkman and Charles Wardell Stiles, *The Cure for Two Million Sick: The Discovery of the Hookworm Disease* (Doubleday, Page, 1909); John Ettling, *The Germ of Laziness: Rockefeller Philanthropy and Public Health in the New South*, First Edition (Harvard University Press, 1981).

[101] Cooper, *Pica*.

not support the nutrition hypothesis and because of the inability of the nutrition hypothesis to explain Black-White differences in pica prevalence.[102]

Pica's etiological grounding in dysfunctional families, particularly African Americans who had recently moved to urban areas from the South, originated in a 1956 study of six lead poisoning cases from the Children's Hospital in Washington, DC. In this seminal study, researchers Frances Millican, Reginald Lourie and Emma Layman employed a Freudian psychoanalytic frame to analyze pica. The researchers argued that children normally go through a stage of putting objects in their mouths beginning at six months, but this normal period ends at 12 months. Children with pica continue chewing and eating non-food items after this point, they argued, because of a dysfunctional mother-child relationship. The dysfunction consisted of either the mother not providing proper and adequate personal relationship for the child, and instead encouraging (or not dissuading) the child to seek satisfaction orally through pica. The mother acted this way because she had a number of personality deficiencies, including anxiety, aggression, and immaturity. These personality traits in turn, were the result of unspecified "economic and cultural factors." The upshot was that, "until about the end of the first year of life, lead poisoning must be regarded as accidental," but lead poisoning after that was the result of a social pathology mediated by motherhood.[103]

Millican and Lourie followed up on this line of research throughout the 1960s. In a 1962 of poor African Americans, Lourie, Millican and Layman directly confronted the nutritional hypothesis of pica. Though the researchers did find that anemia and poor diets associated with pica, but they continued to reject the nutritional hypothesis of pica because poor nutrition was also associated with aspects of the home environment. These aspects "might well contribute to causing pica" and included "unmarried mothers, more frequent changes of residence, more siblings farmed out to relatives, less adequate play resources and play mates, more homes with peeling plaster and paint, and more families with major emotional problems." In general, children with pica came from "disorganized" families. In 1963, these authors argued that pica developed in four ways: 1) as an attempt to meet oral needs when the mother was unavailable; 2) excessive oral gratification due to overstimulation by the mother; 3) aggression toward the mother due to earlier conflicts over feeding; and 4) brain damage. Pica, in this theory, was a dysfunctional coping mechanism for dealing with a dysfunctional social environment, and it was a coping mechanism that was culturally specific, often being learned by the child from the mother. The cultural origins were in a Black rural cultural that was particularly prone to eating clay, and this explained Black-White differences in pica prevalence. In the city, however, Black were either less discriminant or not able to get clay, and substituted other substances such as paint chips. More studies followed with similar conclusions body of work became the standard way in which pica was explained by public health researchers in Baltimore, New York, and other cities.[104]

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[102] For example, the Whites that Cooper studied were actually more malnourished than the Black, suggesting they should have had higher pica prevalence. Cooper suggested post hoc that the Black may have had more anemia, even if they had better overall nutrition. Ibid. For support of the nutrition hypothesis, see Philip Lanzkowsky, "Investigation into the Aetiology and Treatment of Pica," *Archives of Disease in Childhood* 34, no. 174 (1959): 140–48.

[103] Frances K. Millican, Reginald S. Lourie, and Emma M. Layman, "Emotional Factors in the Etiology and Treatment of Lead Poisoning: A Study of Pica in Children," *Archives of Pediatrics & Adolescent Medicine* 91, no. 2 (1956): 144. At about the same time in Chicago, researchers suggested that pica was an emotional disorder connected to mother –child relationships. The researchers found, in particular, that lead poisoned children with pica often exhibited aggression and tried to bite their peers and parents. Irritability, however, was also a consequence of lead poisoning itself. C. David Jenkins and Robert B. Mellins, "Lead Poisoning in Children: A Study of Forty-Six Cases," *Archives of Neurology and Psychiatry* 77, no. 1 (1957): 70.

[104] Frances K. Millican et al., "The Prevalence of Ingestion and Mouthing of Nonedible Substances by Children.," in *Clinical Proceedings-Children's Hospital of the District of Columbia*, vol. 18, 1962, 207; Margaret F. Gutelius et al., "Treatment of Pica with a Vitamin and Mineral Supplement," *The American Journal of Clinical Nutrition* 12, no. 5 (1963): 388–93; John G. Howells, *Modern Perspectives in International Child Psychiatry* (Oliver & Boyd, 1969).

While Millican, Lourie, and Layman emphasize the socio-emotional aspects of the home environment as the primary culprits in causing or fostering pica, they also mentioned another aspect of the home environment that exacerbated the problems with pica: "homes with peeling plaster and paint." For public health researchers, in fact, this aspect of the home environment had become the key part of explaining why the incidence of childhood lead poisoning had begun to increase around 1950. As lead poisoning cases increased in the late 1940s in Baltimore, the Health Department sent out public health nurses to survey houses. These nurses found was not only greatly deteriorated housing conditions in slums with peeling and flaking lead paint. But slums had existed in Baltimore long before the 1950s, so what had changed? Health officials found that most of the lead poisoning cases were in formerly middle-class homes that had been abandoned by the movement to the suburbs and subsequently turned into tenements for the poor. Unlike the old slums homes in alley houses and the newer public housing units, these formerly middle class homes had considerable amounts of lead paint in them.[105]

The triad of factors in the pica-centered explanation of lead poisoning that emerged in the 1950s was consonant with a broader sociological zeitgeist surrounding Black and White differences and the social consequences of the great waves of Black migration from the South to the urban north. Beginning in the 1930s, many sociologists, particularly those of the Chicago School, had come to see the combination of racism and the shock of moving to the city (which was the spatial proxy for the modern world) as deeply devastating to African Americans. The city, as the prominent sociologist E. Franklin Frazier argued, was a "zone of deterioration" that contributed to a "disorganized" and matriarchal family form in Black. This cultural form, which was a product of unequal economic opportunity, then perpetuated poverty itself. And this family situation's effect on children was highly pernicious. A dearth of motherly security, "affectional as well as economic," beset poor, Black children who then sought "satisfaction in [their] individualistic impulses and wishes." Although pica researchers like Lourie, Millican and Layman did not cite any social science like this specifically, their descriptions of the Black family are so similar that it is highly likely they were influenced by this body work.[106]

The pica-centered explanation of lead poisoning that emerged in the 1950s was thus shaped by many factors. It was spurred by the increasing incidence of childhood lead poisoning. The proximate reason for that poisoning – children eating lead paint – was shaped by a body of case studies that connected pica to lead poisoning and was then bolstered by epidemiological studies that connected (if imperfectly) pica to lead poisoning on a larger scale. Meanwhile, a pathological and socio-emotional etiology of pica helped explain why pica occurred more in African Americans (and offered an alternate explanation to harried nutritional hypothesis), consonant as it was with sociological theory. And deteriorating housing in combination with these other two factors explained why lead poisoning incidence rose when it did.

The actual prevalence of pica is also difficult to judge. As Figure 5 shows, the estimation of pica prevalences varied considerably. Cities besides Baltimore and D.C. also produced varying prevalences. These differences may reflect some difference in the population (like percentage African American or percentage from the South), but they also surely reflect the varying definitions of pica used by researchers. For some researchers, no child under one year old could have pica, but other researchers clearly categorized some children of this age as having pica. Prevalence across racial groups also varied. Most researchers believed that African Americans were more prone to pica than whites and the poor were

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[105] H. Williams et al., "Lead Poisoning in Young Children," *Public Health Reports* 67, no. 3 (1952): 230. Several historians, including English and Burnham, have essentially concurred with the BCHD on this point, arguing that deteriorated buildings were not a source of lead poisoning before the late-1940s. Other historians, such as Christian Warren, have argued that the increase in cases reflected changes in lead poisoning diagnosis, not fundamental material changes that increased lead hazards. Peter C English, *Old Paint : A Medical History of Childhood Lead-Paint Poisoning in the United States to 1980* (New Brunswick, NJ: Rutgers University Press, 2001); John C. Burnham, "Unraveling the Mystery of Why There Was No Childhood Lead Poisoning," *Journal of the History of Medicine and Allied Sciences* 60, no. 4 (2005): 445–77; Warren, *Brush with Death*.

[106] Frazier, quoted in Alice O'Connor, *Poverty Knowledge: Social Science, Social Policy, and the Poor in Twentieth-Century U.S. History* (Princeton University Press, 2002), 64, 83–84.

more prone than the middle class. Cooper, however, found no difference across income groups. And in the only randomized study of pica in this period, carried out in Boston from 1958 to 1962, epidemiologist Donald Barltrop found no statistical difference in pica prevalence across racial or income groups, and no effect of recent migration from the South.[107]

pica prevalence	population	place	source	note
54%	90% black, poor	Baltimore	Bradley, 1956	from clinic, not random sample
50%	2-3 years age	Baltimore	Chisolm, 1968	unclear where data is from, possibly Bradley 1956
34%	black	D.C.	Millican, 1962	from clinic, not random sample
22%	6 mo. or older	Baltimore	Cooper, 1957	from clinic, not random sample
27%	black, 6 mo. or older	Baltimore	Cooper, 1957	black-white difference statistically significant
17%	white, 6 mo. or older	Baltimore	Cooper, 1957	black-white difference statistically significant
19%	age 1-6	Boston	Barltrop, 1966	random sample
25%	white	Boston	Barltrop, 1966	black-white difference NOT statistically significant
23%	black	Boston	Barltrop, 1966	black-white difference NOT statistically significant

Figure 5: Mid-century pica studies. These show the wide variability in findings, but the generally much lower findings for studies based on random samples. Table by author from the following sources: Bradley, J. Edmund, et al. "The incidence of abnormal blood levels of lead in a metropolitan pediatric clinic: With observation on the value of coproporohyrinuria as a screening test." The Journal of Pediatrics 49.1 (1956): 1-6; Chisolm, J. Julian, and Eugene Kaplan. "Lead poisoning in childhood—comprehensive management and prevention." The Journal of pediatrics 73.6 (1968): 942-950; Millican, Frances K., et al. "The prevalence of ingestion and mouthing of nonedible substances by children." Clinical proceedings-Children's Hospital of the District of Columbia. Vol. 18. 1962; Cooper, Marcia Mann. Pica: A survey of the historical literature as well as reports from the fields of veterinary medicine and anthropology, the present study of pica in young children, and a discussion of its pediatric and psychological implications. Thomas, 1957.

Even accepting a higher prevalence of pica in African Americans, Cooper's study of pica in Baltimore presents difficulties for the trope of the pica-prone rural Black moving to the city and eating paint chips instead of clay. According to Cooper, while African Americans had a higher overall pica prevalence, Whites had a much higher prevalence for paint. African Americans were more likely seek out dirt and clay, but Whites were *three times* more likely to seek out paint than African Americans. (Paint eaters made up 24% Whites with pica and 8% of Black with pica). This means that there should have been more Whites in Baltimore with pica for paint than Blacks. The notion that Blacks suffered from more lead poisoning because they were more likely to eat paint was often stated at the time, and has been repeated by scientists and historians since, but it is not supported by the most comprehensive study of pica in Baltimore.[108]

[107] Donald Barltrop, "The Prevalence of Pica," *Archives of Pediatrics & Adolescent Medicine* 112, no. 2 (1966): 116.

[108] Cooper, *Pica*. The historian Peter English, for example, has made the claim about Blacks and paint eating. English, *Old Paint*, 2001. Black and Whites were about equally likely to seek out plaster. Plaster, of course, could also be source of lead paint because it had paint on it (or in some cases, in it). Likewise, dirt could have lead in it

If the pica-centered explanation had such serious problems, why did health researchers not produce alternative, or more complex, theories of child lead poisoning? Perhaps a better question to begin with is: What was known and what could they have known about alternative ways that children absorbed lead into their bodies?

Part of the difficulty in conjuring an another explanation was that the alternative was often literally not visible. Lead in the air from automobile exhaust is not visible, and dust contaminated with lead is, if not invisible, easily overlooked. Moreover, dust is not visible in bodies at all, unlike paint chips that would show up in x-rays – among many other things that a child with pica might have. Similarly, the effects of a child with pica on a wall or furniture could be quite dramatic and visible – holes in plaster, chairs and cribs stripped entirely of paint. The actions necessary to ingest left no such marks. Moreover, the exposure routes of dust are much more complex than ingesting paint – lead emitted from vehicles, which then falls on dirt that might be tracked into a house and get on a child's hands.

Nevertheless, making these connections were clearly not beyond the reach of scientists at this time – indeed that had been mentioned early in the twentieth-century. In several publications in the 1920s, the Yale physiologist Yandell Henderson had pointed out lead oxide from automobile exhaust would fall on dirt that would then get blow into dust and cause "slow poisoning" in people. Even more pertinent, the very first discussion of childhood lead poisoning by A.J. Gibson had been entirely centered on dust, not paint chips or pica, as the route by which children were seriously poisoned.[109]

But these early publications and theories were not widely known among the pediatricians who dealt with lead paint poisoning at mid-century. The debate about leaded gasoline had become largely one of occupational health, and along with many articles about fathers bringing poisonous lead dust to their homes, the occupational health and pediatric literature carried on without much crossover. The main explanation of Gibson's paper, meanwhile, appears to have been lost in the early American case studies of pica. When it was discussed in American literature on childhood lead poisoning, dust was virtually never mentioned, and by mid-century Gibson himself was virtually never mentioned.

So as American public health researchers grappled with the rising cases of childhood lead poisoning at mid-century, they really had one theory available to them: pica. And despite the fact this hypothesis fit increasingly less well as research moved away from the case study approach, and as the threshold for lead poisoning became more liberal, researchers continued to apply it with gusto. As some psychological research suggests, the lack of alternative hypotheses causes people to express over-confidence in their own hypothesis, and numerous publications on childhood lead poisoning did this.[110] One report in a congressional hearing, for example, stated that lead " ingestion by young children is invariably associated with the phenomenon of pica."[111] Department of Health officials in New York City claimed that there were some cases of lead poisoning unrelated to pica – such as those that occurred as a result of burning battery casings and contaminated water – but "in aggregate they do not add up to a significant percentage of the total cases." But 30%, even 10%, is a significant number when one are discussing a population that consists of all the children in the United States.[112]

The reach of this sort of simplistic, monolithic understanding of childhood lead poisoning, along with the pathological understanding of pica, is best seen in Figure 6, a diagram presented by Julian

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either from exterior lead paint that had chipped or because of automobile exhaust. But eating paint directly would have been the most hazardous form of pica for lead poisoning.

[109] Some researchers also believed the creation of the paint dust was peculiar to the particular Australian climate, and so hence the concern about paint dust did not apply to the United States.

[110] Craig A. Anderson, B. Lynn New, and James R. Speer, "Argument Availability as a Mediator of Social Theory Perseverance," *Social Cognition* 3, no. 3 (1985): 235–249

[111] Committee of Hospitals of Brooklyn to Eradicate Lead Poisoning, "The Eradication of Childhood Lead Poisoning," United States Congress Senate Committee on Labor and Public Welfare Subcommittee on Health, *Lead Based Paint Poisoning Amendments of 1972: Hearings, Ninety-Second Congress, Second Session, on S. 3080 ...* (U.S. Government Printing Office, 1972), 229.

[112] Morris Greenberg et al., "A Study of Pica in Relation to Lead Poisoning," *Pediatrics* 22, no. 4 (1958): 756–60. For other examples, see the discussion of congressional hearings on lead poisoning below.

Chisolm, widely considered the foremost expert on childhood lead poisoning, to a congressional hearing on lead paint and subsequently reprinted in a pediatrics textbook.

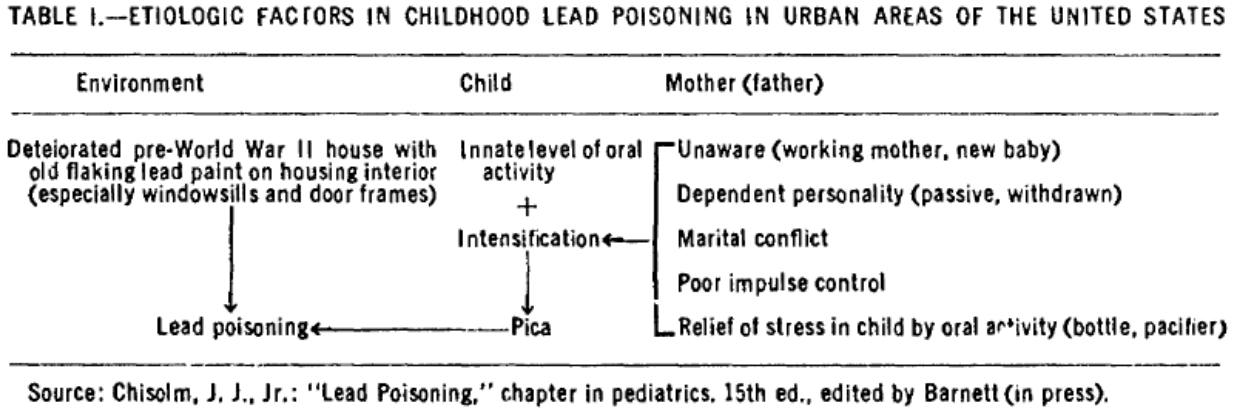

Figure 6: The pica-centered theory of child lead poisoning. The diagram, from a presentation given by Julian Chisolm to the Senate in 1970 and published in a pediatrics textbook, shows the singular focus on pica and the understanding of the pathology of pica based on the works of Millican and Lourie. Other diagrams reprinted in congressional hearings were similar to this in indicating that pathological nature of pica and in showing pica as the only route by which lead got into children bodies. Source: United States Congress Senate Committee on Labor and Public Welfare Subcommittee on Health, *Lead-Based Paint Poisoning: Hearing, Ninety-First Congress, Second Session, on S. 3216 ... [and] H.R. 19172 ... November 23, 1970* (U.S. Govt. Print. Off., 1970), 210; Henry L. Barnett, ed., *Pediatrics*, 15th ed. (Appleton-Century-Crofts, 1972).

By this point – 1970 – researchers had already cemented the pica-centered explanation of lead poisoning with a cascade of research and action that reinforced their beliefs. Beginning in the 1950s, researchers suggested using pica as screening and/or diagnostic mechanism for lead poisoning. The search for these individual screening and diagnostic tools was in turn part of a broader and longer movement of the "New Public Health." The New Public Health began emerging in the nineteen-teens and slowly eclipsed the older sanitarian approach to public health that had focused on fixing the unsanitary environment. Instead, the New Public Health made use of new diagnostic technologies in the laboratory to try to pinpoint diseases in particular individuals. For different diseases, the scientific and technological tools necessary for individual diagnosis emerged at different times for different diseases.[113]

But even when the technology and scientific knowledge was available, screening and diagnosis of individuals was not always practical on the large scale that public health worked at. Although blood lead tests were available in the 1930s and were considered the best method for determining lead exposure, they were expensive and extremely hard to take in young children. Public health officials thus sought alternative ways that children could be screened, ones that were cheap, easy and fast. This was the impetus for Bradley's study comparing blood lead levels and urine tests. Not finding the urine test to be useful at all, Bradley and other researchers latched on to a variable that, though it was not their main concern, they clearly thought was important: pica. When their hoped for diagnostic method failed, pica began to emerged from their research as not only the key cause of lead poisoning, but a potential method of screening or diagnosis.[114]

But using the risk factor, in this case pica, as a way of screening and diagnoses children also served to create highly biased samples of data for obvious reasons. The lead poisoning division of New York's Health Department had the motto was "look and ye shall see" – in other words, if you look for lead poisoning, you will find it. But the same was true of the pica-centered explanation of lead poisoning. In 1955, New York began aggressively screening children with for pica history in order to find lead

[113] Duffy, *The Sanitarians*.
[114] J. Edmund Bradley and Samuel P. Bessman, "Poverty, Pica, and Poisoning," *Public Health Reports* 73, no. 5 (1958): 467.

poisoning cases. They found many poisoning cases, and not surprisingly, also found that very high numbers of lead poisoned children had pica.

Researchers in New York even appear to have used the pica screening mechanism for a randomized study of lead poisoning in the city. In a 1963 study, researchers randomly selected children and then took blood lead levels from "suspected cases." Who were suspects? Although the researchers did not specify what this meant for the particular study, they described the general approach of the health department the article thus: "Where pica history is positive the… physician conducts [an] examination" for lead poisoning signs and symptoms and gives a blood test. The result of this random study was the highest finding of any city for the association between lead poisoning and pica: 91%. This finding, along with Bradley's article, became one of the most influential in pica-centered explanation of lead poisoning.[115]

Researchers not only saw what they were looking for, they heard what they were asking for. In a door to door survey in Cleveland, Robert Griggs and colleagues found highly variable rates for pica in census tracts that were otherwise socially similar. In the tract that was in a poor area, with dilapidated housing with lots of lead paint, 50% of children were reported to have pica, and 45% of those had pica for paint. In two other tracts with similar poverty and dilapidated housing, but no lead paint, pica prevalence was reported to be 22 and 28%, with pica for paint being 1 and 3% respectively. The authors offered several suggestions for these very odd numbers, including the unreliability of the informants with regard to pica (a common claim). Perhaps. But it seems more likely (given, for example, the much lower findings of pica prevalence in randomized studies) that it was the *interviewers* who were unreliable – that the interviewers felt they should be finding pica along with lead poisoning and pressed for it. Other studies struggled with interviewer bias, and some discarded their findings on pica as result of it.[116] There was one final way in which these researchers reinforced their own belief in the pica-centered theory: by trying to educate the masses. In Baltimore, public health education about lead poisoning had been ongoing from the 1930s, but in the 1950s it took on a different tone, and in the 1960s it took on a different scale.

In lay literature on childhood lead poisoning in Baltimore in the 1930s and early 1940s, pica was not presented as abnormal, even if researchers thought it was. Pica was not even discussed. News reports from the Baltimore *Sun* that interviewed Huntington Williams and J.M. McDonald of the Bureau of Occupational Diseases made no reference to pica. In these news articles, health officials warned parents about children eating or chewing objects with lead paint, often noting the special danger to children going through normal teething stages, and never suggesting the problem lay with abnormal eating habits.[117]

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[115] Like Bradley's paper, this was cited and accepted by many researchers, and included in all of the major congressional reports on child lead poisoning circa 1970. Harold Jacobziner, "Lead Poisoning in Childhood Epidemiology, Manifestations, and Prevention," *Clinical Pediatrics* 5, no. 5 (May 1, 1966): 277–286. The authors also note that if a child had high blood lead levels (60μ/dL was their cutoff) and pica, but no other lead poisoning signs or symptoms, the child was diagnosed as a "possible" case. In other words, pica was part of the diagnosis process, not just a screening mechanism.

[116] Sunshine I Griggs RC, "Environmental Factors in Childhood Lead Poisoning," *JAMA* 187, no. 10 (March 7, 1964): 703–707. Other researchers on lead pica suspected interviewer-bias in questioning about pica. See Anthony J. Yankel, Ian H. von Lindern, and Stephen D. Walter, "The Silver Valley Lead Study: The Relationship Between Childhood Blood Lead Levels and Environmental Exposure," *Journal of the Air Pollution Control Association* 27, no. 8 (1977): 763–767.

[117] "Reports 24 Cases of Lead Poisoning: Health Department Shows Summer Months Are Worst Here Official Warns Parents Against Repainting Cribs Children Chew," *The Sun* , August 9, 1937; "Lead Poison Kills 22 Here in Three Years: Thirty-Five Others Made Ill, According To Health Department Nearly All Cases Due To Chewing Paint From Cribs And Chairs," *The Sun* , April 4, 1937; "Warns Against Child's Chewing Painted Object," *The Sun* , August 20, 1939; "3 Children Dead of Lead Poisoning: 3 Others Ill After Nibbling Newly Painted Objects," *The Sun* , August 3, 1946. The lack of emphasis on pica in news articles and educational material at this time appears to be common in other cities as well, although pica was discussed in the following newspaper articles: W.A. Evans,

By the 1950s, as childhood lead poisoning cases were on the rise, the medical, scientific, and moralistic attitude about pica began to change. In 1951, for example, in the first article in the *Sun* that mentioned pica, Johns Hopkins pediatrician Francis Schwentker suggested that lead poisoning (which he thought was not receiving enough attention) was caused mainly by pica – "the manifest tendency in a child to eat abnormal things," as the *Sun* put it – and not the normal habit of teething. "For this reason," the *Sun* stated, "he considers the problem not so much one of removing the paint as a matter of educating parents to watch their children."[118] Likewise, J. Edmund Bradley and Samuel Bessman, for example, called for only one type of action for the "entirely preventable" malady: educating parents about the problems of pica, many of whom they bemoaned thought of pica was a "harmless manifestation of normal infantile development."[119]

In 1962, the Baltimore City Health Department initiated a large "hard sell" program, a three-year "intensive education effort with a person-to-person approach" to be "directed to the parents and other responsible for the care of children under four." To gauge the effectiveness, the department set up a control group of similar census tracts and compared the number of cases of lead poisoning and the number of elevated blood lead levels. After three years, they found no statistical difference between the experiment and control tracts.[120]

The historian Elizabeth Fee has interpreted this to mean that the earlier education efforts had reached a saturation point. But there is another reason it may have had little effect: pica may not have been the pervasive cause that health professionals believed it to be. And whether it was or not, the effect of such a campaign was to place blame on parents, especially mothers, who then closed themselves off to the education efforts.

That was apparently the case in New York. By the early 1970s, public health officials in New York, who had been the most aggressive proponents of pica history as a diagnostic tool, moved completely away from this approach because of its effect on education and compliance. Vincent Guinee, the Director of the Lead Program, maintained that "practically by definition, lead poisoning must be associated with pica and yet, paradoxically, when a history of pica has been used as a case-finding method more cases were lost than found." Why? Because pica "might not be considered good behavior and we would be asking the mother to tell us her child was bad." And second, because they wanted to avoid forcing a mother to "put her reputation as a mother on the line when she brings her child to be tested" – to give her the impression "she has been proven 'scientifically' to be a bad mother."[121]

But if educating mothers about pica was challenge, the educational effort may have educated the educators. In letters to the mayor, interviews with newspapers, and in public health education campaigns, researchers constantly regularly recited the causal connection between pica and lead poisoning. The very act of doing this, a great deal of psychological research suggests, increased their confidence in their singular theory of childhood lead poisoning.[122]

"How To Keep Well: Lead Poisoning in Children," Chicago Tribune, February 9, 1927 (citing the work of McKhann).

[118] R. H. Gardner, "Lead Poison Takes Toll: Second Only To Pneumonia In Child Mortality," *The Sun* , October 3, 1951.

[119] Bradley and Bessman, "Poverty, Pica, and Poisoning."

[120] George W. Schucker et al., "Prevention of Lead Paint Poisoning among Baltimore Children. A Hard-Sell Program.," *Public Health Reports* 80, no. 11 (1965): 969.

[121] Vincent F. Guinee, "Pica and Lead Poisoning," *Nutrition Reviews* 29, no. 12 (1971): 267–69.

[122] Morgan P. Slusher and Craig A. Anderson, "Using Causal Persuasive Arguments to Change Beliefs and Teach New Information: The Mediating Role of Explanation Availability and Evaluation Bias in the Acceptance of Knowledge," *Journal of Educational Psychology* 88 (1996): 110–122; Derek J. Koehler, "Explanation, Imagination, and Confidence in Judgment.," *Psychological Bulletin* 110, no. 3 (1991): 499–519.

Urban Ecology and Lead Poisoning: Leaded Gas, Dust, and Climate

So if the pica-centered theory was incomplete, what other lead exposures might help account for lead poisoning cases and patterns?

One important factor air polluted with lead that resulted from both an increase in automobile use and an increase in lead in gas in the post-war period (Figure 5). As rationing was removed after World War II and factories re-oriented toward private good production, and affluence increased, people bought cars in Baltimore at a much higher rate than at any other time before. Moreover, they bought the worst polluting cars in the United States of all time, before they were regulated at the federal and state level in the 1960s.[123] Finally, the 1950s was a period of extraordinary traffic congestion in Baltimore, which would have exacerbated the pollution effects of automobiles, since they would have burned more gas in a less efficient way to go the same distance.

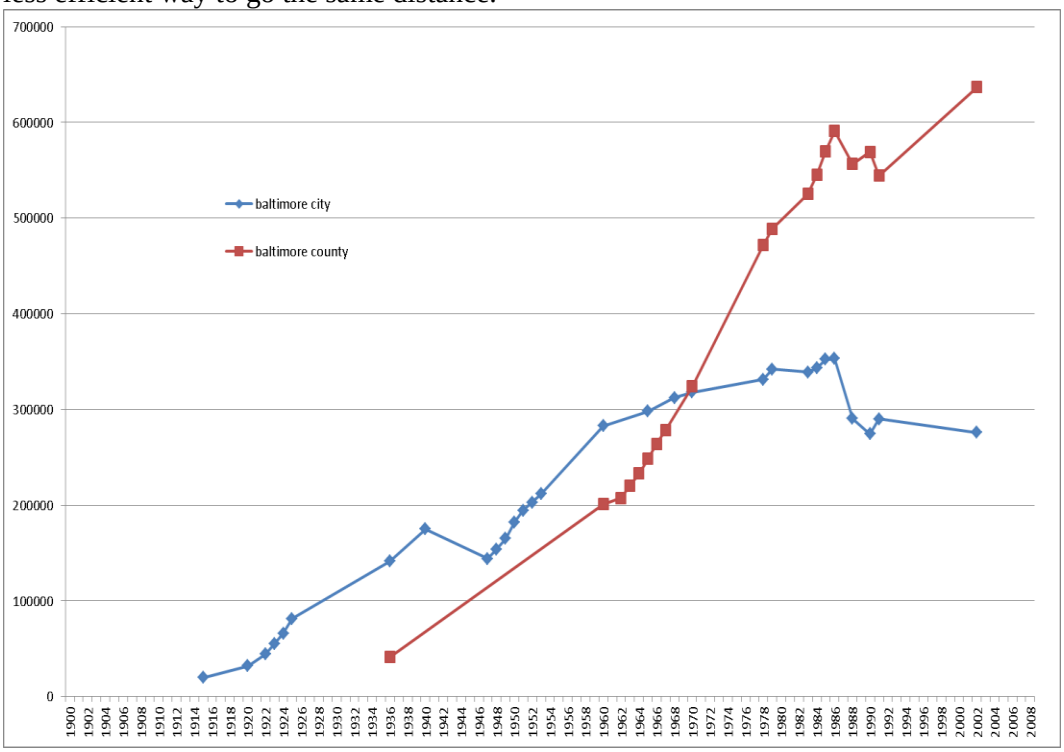

Figure 6: Vehicle registrations in Baltimore city and county. This graph shows the surge in automobile ownership, and hence, use, after World War II. Graph by author from the following ources: Baltimore *Sun*; State Roads Commission (1938); *Report of the Baltimore City Engineer* (1942), *Report of the Baltimore Grand Jury* (1957), *Report of the Task Force on Motor Vehicles* (1971); Maryland Department of Transportation *Annual Reports*; Maryland State *Statistical Summaries*.

[123] Nationally, vehicles released on average about 13 g/mile of HC, 87 g/mi CO, and 3.6 g/miles of NOx in the 1950s. At the national level this was not curbed until the 1965 Motor Vehicle Air Pollution Control Act. California acted earlier, but not Maryland. *Atmospheric Pollution: History, Science, and Regulation* (Cambridge University Press, 2002), 211. By contrast, vehicles today emit .25, 3.4, and 0.4 respectively. In California, light vehicles (i.e., cars) hydrocarbon (HC), NOx and carbon monoxide (CO) emissions decreased by about half from 1957-1962 averages to 1963-1967 averages. But both of these emission averages were much higher than subsequent years. By about ten times compared to 1975 and about twenty times by 1991. Timothy J. Wallington, John L. Sullivan, and Michael D. Hurley, "Emissions of CO2, CO, NOx, HC, PM, HFC-134a, N2O and CH4 from the Global Light Duty Vehicle Fleet," *Meteorologische Zeitschrift* 17, no. 2 (2008): 109–16. It is still unclear to me whether total emissions in Baltimore would have been higher in the 1950s, 1960s, or 1970s (pretty sure they were lower after 1980). National total emissions resulting from vehicles are not the highest in 1950s. Gerhard Gschwandtner et al., "Historic Emissions of Sulfur and Nitrogen Oxides in the United States from 1900 to 1980," *Journal of the Air Pollution Control Association* 36, no. 2 (1986): 139–49.

Moreover, this congested traffic was concentrated in the urban core, since in the 1950s many suburbanites still used the city for work, shopping and play. Thus much like the lead acid battery recycling system, automobile exhaust was also funneled and concentrated in the. And like the battery recycling system, within the urban core, this pollution was concentrated in particular areas that were often low income and African American communities. (Figure 7, a map of child lead poisoning cases in Baltimore in 1959 mapped along with roads and their average daily traffic, is a preliminary analysis that suggests some association between lead poisoning cases and high traffic areas.) In the early 1960s, as the result of interstate building and an overhaul of the city's traffic and parking system by famed traffic engineer Henry Barnes, congestion decreased considerably. Thus the peak in the 1950s may be partly explained by a combination of high traffic, heavily leaded gas, and traffic congestion that rose and fell in the 1950s.

Automobile exhaust could also contribute to another source of lead pollution: dust. There is now a very large amount of data showing that dust of any kind with lead in it is an important factor, and often the most important factor, in predicting blood lead levels in children. This dust – which is far more bioavailable than paint chips – gets into children's bodies by being inhaled, eaten on food, or through any number of "normal" behaviors like licking or mouthing objects and sucking thumbs. So the question is: is there a reason to believe that dust would have been a problem at mid-century and that, in fact, it might have been particularly bad at that point?

Like many post-war cities, Baltimore's urban landscape was massively reshaped due to urban renewal and interstate highway building, and these activities would have greatly increased exposure to exterior dust. Contemporary studies have shown that demolition increases lead dust in the environment and has shown an association between demolition activity and children's blood lead levels.[124] A second source of exterior dust was the massive destruction of building through slum clearance. Although Baltimore had carried out a few slum clearance projects before the 1950s, clearance and demolition was undertaken on an unprecedented scale in the 1950s with the passage of successive FHA Acts in 1949 and 1954 that target slums for urban renewal and provided federal assistance for doing so.[125] In this period, the Baltimore Housing Authority and the Bureau of Redevelopment tore down thousands of dwellings on hundreds of acres of land in the central city. The first of these began in 1951 and was followed by dozens of others.[126] Not only was dust created during the destruction, but often nothing was put in place for some time, leaving, as one commenter put it in 1955, "only a jungle of rumble."[127] This was especially true of the urban renewal projects in the 1950s, which often left acres of demolished building and bare ground in the middle of dense neighborhoods for years. The city prohibited contractors from bringing in clean fill, using the demolished materials for fill.[128] The city burned unwanted demolished materials, creating local lead hazards, a practice it only stopped in 1962 in response to a broader movement for cleaner air in the city. The city also tried to keep these areas clear of weeds, apparently in an attempt to make them look better, though this would also have increased dust from these sites. Though the city briefly discussed fencing the sites because children were walking through them on the way to school they decided against

[124] Mark R. Farfel et al., "A Study of Urban Housing Demolitions as Sources of Lead in Ambient Dust: Demolition Practices and Exterior Dust Fall.," *Environmental Health Perspectives* 111, no. 9 (2003): 1228; Mark R. Farfel et al., "A Study of Urban Housing Demolition as a Source of Lead in Ambient Dust on Sidewalks, Streets, and Alleys," *Environmental Research* 99, no. 2 (2005): 204–13; F. A. Rabito et al., "The Association between Demolition Activity and Children's Blood Lead Levels," *Environmental Research* 103, no. 3 (2007): 345–51.
[125] John F. Bauman, Roger Biles, and Kristin M. Szylvian, *From Tenements to the Taylor Homes: In Search of an Urban Housing Policy in Twentieth-Century America* (Penn State Press, 2000), 145.
[126] City Planning Files, University of Baltimore Archives
[127] For jungle of rubble quote, see "Those Empty Acres Facing Hopkins Hospital," *Sun*, January 19, 1955.For other accounts of slum clearance, especially in the 1950s, see "City Agencies Consider Plans to Destroy South Baltimore Slum Area," *AA*, October 6, 1945; "Going: 47 Acres Of Slum," *Sun*, July 13, 1950; "24 More Acres For Slum Clearance," *Sun*, May 17, 1952.
[128] Planning Department Files, Baltimore City Archives.

it.[129] Thus the sites became not only travel routes for children but – in poor neighborhoods that at the time had virtually no recreational facilities – also places for children to play, as Figure 5 shows.

Figure 7: The Broadway Urban Renwal Project, ca. 1950s.. African American children play in the rubble of demolished houses, which may have been contaminated with lead paint dust. Source: Citizen Planning and House Association, University of Baltimore Archives.

Urban renewal projects that demolished neighborhoods to build new housing or shopping centers in Baltimore were dwarfed by an even larger project in the 1950s: The construction of the Jones Falls Expressway. Like clearance projects, the building of highways was not unprecedented, but the scale was, and that scale was made possible by the large influx of federal funds resulting from Interstate Highway Acts in the 1950s. The Jones Falls Expressway was the larger road building project in Baltimore's history – began construction in 1956 and opened in 1962. The peak years of construction were 1958 and 1959, when project razed houses in numerous neighborhoods and tore up old roadsides that would have been heavily laden with lead contaminated dust. Figure 6 shows a picture of one of the bridges under construction in the summer of 1959, when the Baltimore *Sun* praised the workers who "toiled in the 90-degree plus, and humidity and dust all summer." The peak construction years thus roughly coincide with the large spike in lead poisoning cases in the late 1950s.

The *Sun* article raises another question about dust exposure: Was there any role for weather? Current lead research has produced remarkably accurate model of blood lead based on the theory that blood lead levels are driven by exterior dust with lead in it. The factors that are most important for prediction are soil particle size, temperature, precipitation and wind speed.[130] There is no historical data on soil particle size and little reason to think it changed, but climatic data suggests the weather was different and almost certainly made exterior dust exposure worse. In the mid-Atlantic, the 1950s experienced a "zonal regime" that brought slightly above average temperatures and rainfall.[131] The higher temperatures fit with an increase in dust, while higher precipitation would have mitigated against increased dust. But wind speeds in the 1950s were extraordinarily high. In general the mid-Atlantic

[129] Ibid.
[130] Mark AS Laidlaw et al., "Seasonality and Children's Blood Lead Levels: Developing a Predictive Model Using Climatic Variables and Blood Lead Data from Indianapolis, Indiana, Syracuse, New York, and New Orleans, Louisiana (USA)," *Environmental Health Perspectives* 113, no. 6 (2005): 793.
[131] Colin Polsky et al., "The Mid-Atlantic Region and Its Climate: Past, Present, and Future," *Climate Research* 14, no. 3 (2000): 161–73.

region experienced a greater frequency of storms, and a the local level, according to newspaper report, Baltimore experienced an abnormal amount of wind events.[132] But probably more important than these particular events were the average differences in wind speed, which were much higher in the 1950s and whose large peak in that decade -- which was the highest on record for Baltimore – corresponds to the large peak in lead poisoning cases in 1958 (Figure7). A *Sun* article recorded a pitch-perfect example of lead dust exposure in that year: "Showers accompanied by dust-stirring winds sent scores of people spending the afternoon in the open scurrying for cover. [The wind did not cause damage] aside from blowing dust into the eyes and onto sticky lollipops and ice cream cones."[133]

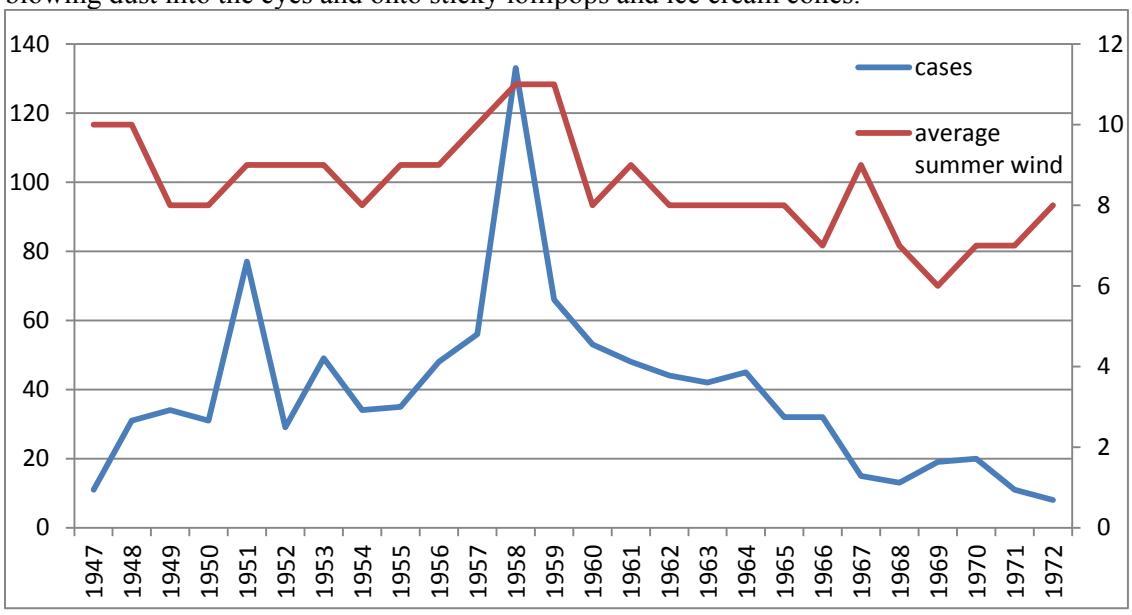

Figure 8: Relationship between wind and lead poisoning cases. The very high summer winds correspond to the spike in lead poisoning cases in the late 1950s, consistent with other scientific studies showing a correlation between high winds and average blood lead levels. Graph by author from following sources: BCHD *Annuals*; Historical Weather Data, National Oceanic and Atmospheric Administration.

Precise predictions of lead levels from these sources are obviously impossible and would vary considerably for different individuals, living in different houses, by different roads, and so on. But these sources have all been shown to be significant sources of lead poisoning, and thus there is good reason to think, especially at a time when the overall amount of lead in the environment would have been quite high, that these could have contributed to lead levels at or above what public health researchers in the 1950s considered dangerous (50 or 60 micrograms).[134]

Dust can also explain the seasonality in lead poisoning, something that the pica theory had trouble with. As Huntington Williams stated in 1952, "Although pica does not exist on a seasonal basis, a striking number of lead-poisoning cases resulting from this habit occur in the hot summer months. For

[132] For Baltimore wind events see Baltimore Sun ; for region see Keqi Zhang, Bruce C. Douglas, and Stephen P. Leatherman, "Twentieth-Century Storm Activity along the US East Coast," *Journal of Climate* 13, no. 10 (2000): 1748–61.

[133] *Sun*, April, 1958

[134] Demolition is the least understood of these, but there is an emerging literature. F. A. Rabito et al., "The Association Between Demolition Activity and Children's Blood Lead Levels," *Environmental Research* 103, no. 3 (2007): 345–351; B P Lanphear et al., "Lead-contaminated House Dust and Urban Children's Blood Lead Levels.," *American Journal of Public Health* 86, no. 10 (October 1996): 1416–1421; Isabelle Romieu et al., "Vehicular Traffic as a Determinant of Blood-lead Levels in Children: A Pilot Study in Mexico City," *Archives of Environmental Health: An International Journal* 47, no. 4 (1992): 246–249; Joel Schwartz and Hugh Pitcher, "The Relationship Between Gasoline Lead and Blood Lead in the United States," *Journal of Official Statistics* 5, no. 4 (1989): 421–431; Laidlaw et al., "Seasonality and Children's Blood Lead Levels."

this no, satisfactory explanation is available."[135] As formulated by researchers like Millican, Lourie, Chisolm and many other researchers, lead poisoning should have been greater in the winter when children were indoors with more lead paint and were more confined, exacerbating their pica. But as many studies since the 1970s have established, summer heat and winds produce considerable dust that can account for the upswing in cases in the summer. For this reason, the average wind data for Baltimore above was taken for the summer months (May through October).

In summary, the pica-centered explanation can account for some of the patterns in lead poisoning and exposure in this period, but there are significant patterns it cannot account for. Alternative exposure routes, through both dust and inhaled vehicle pollution, can account for some of these patterns in populations, time, and space. These sources can account for the children at the lower end of the high PbB spectrum (which is a very large group), and may account better for overall variation in blood lead than pica (which does so very poorly). They can account better for seasonal fluctuations, and can account for at least some of the increase in lead cases in the 1950s. These cases may have been more related to heavy traffic areas, for example. Moreover, all sources of lead exposure were cumulative, including exposure through pica. Thus the mere presence of pica in a child with lead poisoning did not mean that pica was the only important exposure contributing to poisoning.

Conclusion

The great increases in the amounts and types of exposure to lead in Baltimore in the first half of the twentieth century had, and would have, large effects on individuals, families, communities and the city as a whole. Even by the late 1930s, when Huntington Williams carried out the first study to try to get a sense of what average blood lead levels were, the penetration of lead into Baltimore was striking. Although his full study is not available, he found an average blood lead level of 30 micrograms, a level that would be rare and troubling to find in a child today. Bradley's study 15 years later revealed even higher average blood lead levels, about 40 micrograms, though his study was carried out with a less representative population. Moreover, only a handful of children in his study of hundreds were even close to what we might consider a safe range today. What this means is that, by the end of the 1930s, the cognitive development of most children in Baltimore was being seriously and negatively affected and this almost certainly got worse in the 1940s and 1950s as exposures intensified. The effects would not be easily recognized and they would take time to come to full fruition as children developed into adults, but they would nevertheless be profound, as I argue in Chapter X.

In the immediate situation of the 1950s, however, many children and their families and their communities suffered. Parents, families and communities struggled to deal with developmentally delayed and sometimes emotionally disturbed children. In the worst cases, children were institutionalized or died. These tragedies peaked in the late 1940s and 1950s – the Golden Age of America – and a few of them resulted from burning battery cases, leading to what had once been called the "Depression disease." For thousands of adults and children in Baltimore, the post war period was less of a Golden Age than an Age of Lead.

[135] Williams et al., "Lead Poisoning in Young Children."