

A Level of Concern

Lead paint was known to be toxic in the early 1900s, but it wasn't banned in the United States until 1978 — a delay with grave consequences

BY JESSIE ROMERO

More than two years after testing first revealed elevated lead levels in the water in Flint, Mich., the city's residents — the majority of whom are black, and 40 percent of whom live below the federal poverty line — still can't drink their tap water without a special filter. By most accounts, the crisis began in April 2014, when the city began using highly corrosive water from the Flint River instead of from Lake Huron, part of an effort to reduce a multimillion-dollar budget deficit. But the problem actually dates back to the city's early days, when the water distribution system was built with lead pipes. Today, Flint is trying to come up with the \$80 million that engineers estimate it will cost to replace the city's pipes.

Lead is highly toxic; exposure can cause sterility, miscarriages, joint and muscle pain, and memory loss, among other symptoms. Children are especially susceptible to lead's effects and can suffer comas, convulsions, or death at high levels of concentration in their blood. In recent decades, researchers have linked even low blood levels to long-term cognitive and behavioral problems and health problems later in life. Both the Environmental Protection Agency (EPA) and the Centers for Disease Control (CDC) state that there is no known safe level of lead in a child's blood.

At the same time homes were being built with lead pipes behind the walls, those walls were being covered with lead paint, which would turn out to be another potent source of childhood lead poisoning. More than a dozen countries banned lead paint in the early 1900s, but it wasn't until 1978 that the United States followed suit. Throughout lead paint's history, children of lower socioeconomic status have been at greater risk of poisoning — and are still at greater risk today, nearly 40 years after lead paint was banned.

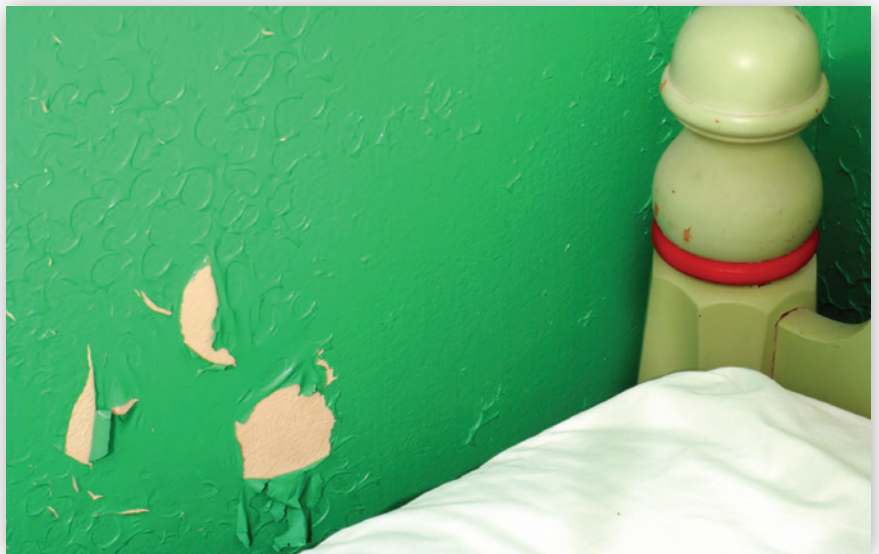
Living in a Lead World

Lead was one of the first metals used by humans. The element is relatively easy to mine and extract from ore, and it's also highly malleable and resistant to corrosion. This makes lead and its various compounds useful in a variety of applications; the ancient Romans used lead for everything from building aqueducts to sweetening wine.

In the United States, the increase in lead production and use coincided with the country's industrialization and urbanization in the second half of the 19th century and the early 20th century. "Lead was pulled out of the ground at the very same time we were building large urban areas, putting in huge water systems, and painting homes by the millions," says historian David Rosner, co-director of Columbia University's Center for the History and Ethics of Public Health. By the 1920s, lead was found in everything from makeup to bathtubs to canned goods to gasoline.

"A child lives in a lead world," wrote physician John Ruddock in a 1924 article in the *Journal of the American Medical Association*.

Lead paint became a desirable wall covering in homes. White lead, a powder created by corroding lead with acid, created a bright white paint that was highly opaque and water resistant, and that could be easily tinted other colors. Brightly painted walls were part of a "tremendous reaction against the dark, Victorian-era houses with a lot of wallpaper," says Gerald Markowitz, a historian at John Jay College and the Graduate Center at the City University of New York. And in an era where a flu pandemic had just killed an estimated 675,000 people in the United States, many people perceived them as more hygienic because they could be wiped down; doctors warned against the dust that collected on unpainted walls.



Deteriorating lead paint is a serious health risk for children who may transfer the dust from hand to mouth or eat the sweet-tasting paint chips.

Lead paint manufacturers appealed to the desire for hygiene. “Painted walls are sanitary, cheerful, and bright,” stated a 1927 advertisement for Dutch Boy white-lead paint. “Cleanliness depends upon washability and consequent freedom from dirt and other impurities,” proclaimed other ads. These “results are best reached by the use of paint made with pure white-lead.”

Lead paint was advertised as especially appropriate for children’s rooms. Parents were advised it would make fingerprints and smudges easy to wipe up. Dutch Boy, the most popular brand, produced coloring books that depicted children repainting their rooms and furniture with lead paint to conquer “old man gloom” and “make this playroom fairly shine.”

The rooms might have shone, but they also were poisonous to the teething babies who chewed on lead-painted cribs and windowsills and to the toddlers who put lead-painted toys in their mouth or ate sweet-tasting paint chips that peeled off the walls. Even the dust created by opening a painted window frame could contain enough lead to make a child sick.

Young Minds Damaged

Although lead poisoning among factory workers and painters was well-documented in the late 18th century and early 1900s, physicians in the United States were slower to recognize the prevalence of lead poisoning in children. In part, that’s because the symptoms in children can resemble the symptoms of other diseases, and in part because testing was difficult and imprecise; it could take a lab worker two full days to analyze a urine specimen for elevated blood levels. Laws also restricted testing for lead poisoning to occupational cases.

The advent of an X-ray test around 1930 and wider availability of blood testing after 1940 helped doctors identify more cases of childhood lead poisoning. Between 1925 and 1945, children younger than 5 went from less than 5 percent of all reported lead poisoning deaths to nearly 30 percent. “Physicians have not been looking for lead poisoning with any vigorous search,” wrote Dr. Edward Vogt in a 1932 article in the *Journal of the American Medical Association*. “Now that they are suspecting it, they are finding three or four times as much lead poisoning as they found before.”

Doctors and public health officials in Baltimore were at the forefront of efforts to identify childhood lead poisoning. In 1914, Henry Thomas and Kenneth Blackfan of the Johns Hopkins Hospital were the first to publish an account of a child’s death from eating lead paint in the United States. (Researchers in Australia had documented childhood lead poisoning from paint as early as 1904.) In 1935, Baltimore’s health department started offering free laboratory tests to doctors who suspected their patients had lead poisoning, the first such program in the country.

City officials mounted a campaign to inform parents about the hazards of lead paint. One radio broadcast from

the mid-1930s warned that in addition to the risk of death, “lead poisoning leaves behind it a trail of eyes dimmed by blindness, legs and arms made useless by paralysis, and minds destroyed even to complete idiocy.” Despite the warnings, lead poisoning continued: Between 1931 and 1951, there were 293 recorded cases among Baltimore children, with 83 deaths.

During the 1940s and 1950s, it became clear that the problem was not confined to Baltimore. No national reporting system existed at this time, but there were some limited investigations. In 1952, an internal report of the Lead Industries Association (LIA), a trade group founded in 1928, counted 197 children poisoned by lead, including 40 deaths, in nine cities. A few years later, the *New York Times* reported on 165 poisonings and 94 deaths in New York, Chicago, Cincinnati, St. Louis, and Baltimore.

These reports identified only the most severe cases of lead poisoning; until the 1960s, children generally weren’t diagnosed until their blood lead level exceeded 60 or even 80 micrograms per deciliter ($\mu\text{g}/\text{dl}$), at which point they could be displaying acute symptoms such as convulsions or coma. Doctors also believed that once the acute symptoms were resolved, the danger had passed, assuming the child survived. But in 1943, Randolph Byers, a pediatric neurologist, and Elizabeth Lord, a psychologist, published the first study showing that children who had suffered acute lead poisoning remained intellectually and behaviorally impaired. And over the next few decades, evidence mounted that children could be harmed at levels well below what was generally considered the threshold for poisoning.

Lead Loses its Allure

By the mid-1930s, more than a dozen countries around the world had banned or restricted the use of white-lead interior paint, beginning with France, Belgium, and Austria in 1909.

The United States was slower to take action. One factor was the relative weakness of the labor movement in the United States compared to other countries. “The impetus for banning lead in paint came from the labor movement in Europe and Latin America; it was really to protect painters,” says Markowitz. “Children were the beneficiaries eventually, but painters were the major force pushing legislation.”

Another factor might have been the trade group the LIA, which lobbied against lead paint bans and labeling laws that would have designated lead paint as poisonous. Still, as concerns about lead paint became more widespread, pigments made from zinc and titanium began to replace lead. In 1951, Baltimore issued the first U.S. ban on the use of lead paint on the interior of any dwelling. Several years later, the LIA, perhaps concerned about the swell of negative publicity and the potential for more stringent regulations, voluntarily worked with the American Standards Association to develop a standard limiting the amount of lead in paint to 1 percent — still enough to be toxic to children. (Historians, public health researchers, and

present-day lead industry executives continue to debate how much, and when, the industry knew about the health consequences of lead paint.)

By the 1970s, health authorities had acknowledged that children could be harmed at lower levels of exposure than previously thought. In 1970, the surgeon general recommended that children with blood lead levels above 40 µg/dl should be closely monitored, official recognition that children were at risk even if they weren't acutely symptomatic. The CDC lowered its "blood lead level of concern" to 30 µg/dl in 1975 and to 25 µg/dl in 1985. Six years later, the CDC lowered the level again, to 10 µg/dl. In 2012, the CDC replaced the "level of concern" with a "reference value" to reflect the belief that there is no known safe level of lead. This value is based on children aged 1 to 5 whose blood lead levels are in the highest 2.5 percent of children — that is, the roughly half a million children with the greatest exposure. Currently, the reference value that triggers continued testing and observation is 5 µg/dl.

The 1970s also saw the first federal legislation on lead paint. The Lead-Based Paint Poisoning Prevention Act, which took effect in 1971, prohibited lead paint in federal housing, on toys, and in cooking utensils. In 1978, all consumer uses of lead paint were effectively banned — although the Department of Housing and Urban Development (HUD) estimated in 2006 that more than 37 million U.S. homes still contain it.

The Basic Problem is Poverty

From the beginning, the poor were especially at risk for lead paint poisoning. "It was always the poorest people living in the most dilapidated housing, where absentee landlords let properties disintegrate, who were the most victimized," says Rosner. The link between poverty and lead paint was strengthened during the post-World War II era, when "white flight" to the suburbs and discriminatory housing practices led to a greater concentration of poor and minority residents in the inner cities. Their homes and apartments tended to be older and poorly maintained, increasing the chance that children were exposed to chipping and peeling paint.

Some lead industry advocates argued that the problem wasn't the paint itself, but rather parents who lacked the knowledge to adequately supervise their children. In a 1957 letter to toxicologist Robert Kehoe, for example, Manfred Bowditch, the LIA's health and safety director, wrote, "Childhood lead poisoning is essentially a problem of slum dwellings and relatively ignorant parents." In another letter, to the former head of the LIA, Bowditch expressed doubt those parents could ever be educated. Kehoe, whose research lab was funded in part by the Ethyl Corporation, a manufacturer of leaded gas additives, argued in a 1960 lecture that poor children living in "unsatisfactory" conditions

By the 1920s, lead was found in everything from makeup to bathtubs to canned goods to gasoline. "A child lives in a lead world," wrote a physician in 1924.

developed an "appetite" for lead paint that was not found among more affluent children.

Civil rights and community activists used the association with inner cities to pressure the government for increased lead

screening and treatment programs, and landlords for improvements to substandard housing. As New York housing activist Paul DuBrul wrote in 1968, "We have already been told by the Health Department that no money can be found for a testing program until the black community begins yelling 'Murder.'"

One group yelling "murder" was the Black Panthers. In publications from the early 1970s, the group railed against the "silent epidemic" of lead paint poisoning; it blamed the housing conditions created by slumlords and the medical profession's inattention to a problem of primarily poor, minority children. To help combat lead poisoning, the Black Panthers added a lead screening program to the free clinics they operated in several cities. They were joined by the Young Lords, a Puerto Rican activist group. In the late 1960s, the group went door to door in East Harlem testing children for lead exposure. When 30 to 40 percent of the children tested positive, the Young Lords held press conferences and staged a sit-in at the New York City Health Department.

In his 2000 book, *Brush with Death*, historian Christian Warren of Brooklyn College (part of the City University of New York) credited these and other community groups with helping to raise awareness about childhood lead poisoning among doctors, public health officials, and policymakers. "[T]he impetus for change ran from the community to the city and beyond," he wrote.

The CDC began monitoring blood lead levels in the population in 1976, as part of the National Health and Nutrition Examination Survey. The second wave of this survey, conducted between 1976 and 1980, confirmed that black and lower-income children had much higher blood lead levels than white and higher-income children. More than 12 percent of black children between the ages of 6 months and 5 years had blood lead levels above 30 µg/dl, the level of concern at the time, compared with 2 percent of white children. Children from households with an annual salary of less than \$6,000 (then the poverty line for a family of four) had an average blood lead level of 20 µg/dl, versus 14.1 µg/dl in children from families with an income greater than \$15,000. (Median household income was about \$13,000 in 1976).

Since the 1970s, when lead paint was banned and leaded gasoline began to be phased out, blood lead levels have fallen significantly across all socioeconomic groups. But lower-income children and black children have remained at greater risk. According to the American Healthy Homes Survey, conducted by HUD between 2005 and 2006, 29 percent of families earning less than \$30,000 per year

had a lead-based paint hazard in their home, versus 18 percent of those with higher incomes. Because cities and states vary in how they collect and report data on blood lead levels, it's difficult to calculate precisely how lead exposure varies with race and income. But a survey conducted by the CDC between 1999 and 2004 found that the average blood lead level among black children aged 1 to 5 was 2.8 µg/dl, versus 1.7 µg/dl among white children. Black children also were nearly three times more likely to have a blood lead level above 10 µg/dl. Nonwhite children also are less likely to receive follow-up testing after an initial screening test, which might increase the risk of permanent cognitive damage, according to researchers at the University of Michigan.

Weighing the Costs

Lead paint abatement is expensive. In 2000, HUD estimated that it would cost \$166 billion over 10 years to inspect and fully abate all the pre-1960 homes at risk of having a lead paint hazard, or about \$9,000 per housing unit. Over the years, some cities and the federal government have planned large-scale lead removal programs that were abandoned due to time and cost constraints. At present, HUD offers several grant programs. In 2016, the agency granted nearly \$100 million to 38 state and local governments for testing and abatement. The grants covered an estimated 6,000 housing units.

While 37 million U.S. homes contain lead paint, “not all of these houses have children living in them,” notes Ludovica Gazzo, a postdoctoral scholar at the University of Chicago who has studied the costs and benefits of lead-abatement programs. And not all of these homes pose an immediate hazard, so long as the paint is intact. “So it’s probably not efficient or cost-effective to abate all of them.”

One solution is to mandate that homes be tested for lead and abated only if children move in, or if a child living in the home is found to have an elevated blood lead level, as 19 states have done. But in a 2017 paper, Gazzo found these laws can have unintended consequences. While they do appear to result in lower blood lead levels, it’s not necessarily because landlords are abating lead paint; rather, it’s because many landlords with older homes discriminate against families with children,

leaving them with a smaller selection of housing. Those who don’t discriminate pass the costs of abatement on to their tenants in the form of higher rents. Overall, Gazzo found that the mandates increased rental costs for families with children by about \$400 per year for at least several years, and that lower-income families were disproportionately affected. “Given the distributional consequences,” Gazzo says, “we should also think about how to focus the mandates to ensure that the costs are not falling on those families that are already disadvantaged.”

Whoever bears the costs — landlords, tenants, or taxpayers — “there are potentially large benefits to society from introducing lead reduction regulations,” Gazzo notes. For example, childhood lead exposure is linked to problems with aggression and impulse control and thus with criminal behavior later in life. Many researchers have identified a strong correlation between the reduction in childhood lead levels that started in the 1970s and the drop in violent crime that began in the mid-1990s. Other research has linked childhood lead exposure to lower test scores, higher medical costs as an adult, and lower lifetime earnings, which leads to lower tax revenue. In another paper, for example, Gazzo found that preventing one microgram above 10 µg/dl in a child’s blood lead levels increased individual lifetime earnings by \$110,000 and tax revenue by more than \$16,000 per child. Lower blood lead levels also reduced state expenditures on special education by as much as \$111 million per cohort of children.

On Aug. 22, 1913, a 5-year-old boy was admitted to Johns Hopkins Hospital. Five days before he was admitted, he started having neck and face pain, became restless, and vomited repeatedly. He deteriorated rapidly, and “[o]n admission he was comatose,” wrote Johns Hopkins doctors Thomas and Blackfan. “His head was retracted, and his arms and legs were extended and spastic... There were recurrent, general convulsions.” A century later, lead poisoning as severe as that experienced by that little boy is rare. “It really was a tremendous public health victory that we got rid of lead in paint and in gasoline,” says Markowitz. “But there are still a lot of kids with blood lead levels high enough to cause damage.” Whether the benefits of preventing that damage outweigh the costs — and who should pay — is a question policymakers will continue to debate. **EF**

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