

PUBLIC HEALTH Reports

Special Report on Lead Poisoning in Children

On April 20, 1904, the *Australasian Medical Gazette* published an article by J. Lockhart Gibson, MD, advancing his theory that lead in paint was the cause of lead poisoning in Queensland children. Before this, it was commonly held that a reason some children showed symptoms of lead poisoning while others in the same family did not was because the poisoned children's kidneys were slower to excrete the lead. Gibson found that many of the children he saw with lead poisoning lived in houses with painted walls and railings, and lead, at that time, was a common ingredient in paint. Removal of the children from their homes resulted in marked improvement in their condition.

In recognition of the importance of this discovery, *Public Health Reports* presents this special selection of articles on lead, including a reprint of Gibson's original article with a commentary by David Rosner. Christian Warren has written an account of the history of attempts by some to dominate research and suppress results that showed the harm done to



Dr. Herbert Needleman



J. Lockhart Gibson, MD

industry workers and to the public. We also have an interview with Dr. Herbert Needleman, in which he tells of the lead industry's attempts to discredit his research about the harmful effects of exposure to lead.

There are several articles about research being conducted today. Bruce Lanphear and colleagues demonstrate the need for screening of housing for lead contamination even in the present day to protect children from the harmful effects of lead poisoning. Richard Maas et al. discuss the issue of lead-contaminated drinking water and the regulatory steps that have been taken in the United States since 1986. Matthew Stefanak and his co-authors present data they've collected on both the immediate and long-term cost of lead poisoning to taxpayers.



SPECIAL REPORT ON LEAD POISONING IN CHILDREN

J. Lockhart Gibson and the Discovery of the Impact of Lead Pigments on Children's Health: A Review of a Century of Knowledge 296
 DAVID ROSNER, GERALD MARKOWITZ, BRUCE LANPHEAR

A Plea for Painted Railings and Painted Walls of Rooms as the Source of Lead Poisoning Amongst Queensland Children 301
 J. LOCKHART GIBSON

Screening Housing to Prevent Lead Toxicity in Children 305
 BRUCE P. LANPHEAR, RICHARD HORNUNG, MONA HO

Cost of Child Lead Poisoning to Taxpayers in Mahoning County, Ohio 311
 MATTHEW STEFANAK, JOE DIORIO, LARRY FRISCH

Reducing Lead Exposure from Drinking Water: Recent History and Current Status 316
 RICHARD P. MAAS, STEVEN C. PATCH, DIANE M. MORGAN, TAMARA J. PANDOLFO

Little Pamphlets and Big Lies: Federal Authorities Respond to Childhood Lead Poisoning, 1935–2003 322
 CHRISTIAN WARREN

Standing Up to the Lead Industry: An Interview with Herbert Needleman 330
 DAVID ROSNER, GERALD MARKOWITZ



Photos: Nancy Barnett

J. Lockhart Gibson and the Discovery of the Impact of Lead Pigments on Children's Health: A Review of a Century of Knowledge

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One hundred years ago, J. Lockhart Gibson published a seminal article on childhood lead poisoning, "A Plea for Painted Railing and Painted Walls of Rooms as the Source of Lead Poisoning Amongst Queensland Children."¹ [The article is reprinted in its entirety beginning on page 301 of this issue of *Public Health Reports*.] This article marked the beginning of the worldwide uncovering of the role of paint pigments in creating an epidemic of lead poisoning that has damaged literally millions of children in the United States. In his brief review of the situation in early 20th century Australia, Gibson made a series of observations that hold true today as much as they did a hundred years ago. The article described many of the discoveries that scientists have confirmed time and again using more refined technology and epidemiologic methods: Gibson described the importance of lead paint and dust in endangering children who ingest them. Gibson and A. Jefferis Turner, his pediatric colleague who was also instrumental in elucidating childhood lead poisoning as a distinct entity,² described seasonal variation in childhood lead poisoning. Gibson used a wipe sampling method to measure lead-contaminated dust, and proposed ingestion as the primary route of exposure. Finally, four years after Gibson argued for education efforts to prevent lead poisoning,¹ Turner concluded, "Prevention is easy. Paint containing lead should never be employed . . . where children, especially young children, are accustomed to play."²

In this commentary, we will discuss the social, cultural, and scientific context of Gibson's work and trace its ongoing legacy, showing how the questions and issues he raised became the basis for future scientific and medical discoveries. A century after Gibson first observed that lead in paint and the dust it produced were a major health threat to children, his work, unfortunately, remains relevant for scientists, clinicians, and historians.^{3,4}

THE EMERGENCE OF CONSCIOUSNESS ABOUT THE HEALTH OF CHILDREN

As lead became an integral part of new middle-class life in cities and suburbs in the late 19th century, changes were happening in the world of medicine and public health that would eventually lead to the discovery of lead's effect on children. Pediatrics was developing as a specialty. New technologies and skills dramatically improved the care of

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young patients: the 19th century saw the growth of children's hospitals where, among other medical advances, pediatric surgeons could reset the deformed bones of children afflicted with rickets. In the early 20th century, a network of infant and child welfare clinics was established. These hospitals and clinics became teaching centers for doctors who focused on the special problems of childhood.⁵

For much of American history, children had worked alongside their parents—planting, harvesting, and tending crops, and eventually toiling in factories and cities. During the early years of the 20th century, reformers began to view children's work in factories and on farms as victimization, an example of exploitation and regressive ideas about children's "use value." At the same time, the public health community began to focus on the broad array of childhood diseases. By World War I, when draft boards found they had to reject approximately 25% of draftees for physical and psychological problems, it became clear that America had been neglecting its children.

Better nutrition, housing reforms, the introduction of pure water supplies and sewerage systems, and street cleaning helped provide a more healthful urban environment for children. The horse, which deposited up to 25 pounds of manure and two quarts of fresh urine on city streets every day, was replaced by the electric streetcar and trolley in the 1890s and the automobile in the early 1900s, leaving city streets looking and smelling better. The numerous granaries needed for the maintenance of nearly 200,000 horses in New York City began to disappear, making it easier to control the huge rat and rodent problem that was linked to the spread of lice- and tick-borne diseases.⁶ Public health stations that provided pasteurized milk,⁷ settlement houses that provided emergency shelter, visiting nurses, and educational programs for mothers and their children also improved the chances of childhood survival. The development of maternity hospitals as well as pediatric and foundling hospitals further improved conditions for children.⁸

Public health and medical workers strove to improve sanitation and living conditions, to improve prenatal care, and to intervene medically to prevent deaths at birth and immediately afterward. As a result, there was an extraordinary decline in the number of infant and early childhood deaths between 1890 and 1920. Virtually every cause of death could be fitted into the bacteriological, social reform, and sanitary models that dominated the thinking of political progressives, settlement house social reformers, and public health and medical professionals of the period. Symptoms of lead poisoning, such as convulsions, could be explained as the result of physical trauma during birth or bacterial infections of the brain or central nervous system. Until the 1920s, except for a few extraordinary observations, few ever broke free from the prevailing paradigms to envision toxic exposures as a possible cause of diseases of infancy and childhood.

THE IDENTIFICATION OF LEAD POISONING IN CHILDREN

In the early 20th century, physicians, first in Australia and then in the United States, began diagnosing cases of childhood lead poisoning associated with lead in paint on toys,

cribs, and woodwork. Public health officials, preoccupied with infectious childhood diseases and the demands for better medical and prenatal services, slowly picked up on the cases of lead poisoning that were being reported. Ironically, the lead industry itself was quite attuned to the incidence of lead poisoning because it feared that attention in the media could devastate the expanding consumer lead market which, by the 1920s, included not simply lead paint, but lead pipes, lead car batteries, and lead in gasoline. Over the next 30 years, the industry embarked on a program to control and delimit the scientific study of childhood lead poisoning and to obscure the relationship between lead, paint, and children's illness and death.

The medical literature on lead poisoning in children can be traced back to the work of Tanquerel des Planches.⁹ In his 1848 treatise, he remarked on children placing lead-painted toys in their mouths and developing lead colic. As early as 1887, medical authorities in the United States noted cases of lead poisoning. David Stewart reported in *Medical News* that nine members of a single family developed lead poisoning from lead chromate used to dye bread yellow.¹⁰ (In the 19th century, lead chromate was often added to lead sulfate to form what was called "chrome yellow," a substance used by bakers and candy makers as a coloring agent.) In 1889, an article in *Science* reported on the deaths of two children from the ingestion of baked confectioners' products that contained chrome yellow.¹¹ In 1892, Gibson and Turner reported that many of Brisbane's lead-poisoned children were "remarkably fond of sweets and chewing things." One boy chewed the foil covering chocolates "to make pellets to pelt other boys." Others were so delighted by the taste of the foils that covered sweetmeats that they chewed the sweets with the foil still on them.¹² A U.S. physician, R. Abrahams, noted that toys were often made of lead and painted with lead paint and wondered how important it might be to guard against their use since "infants and older children, [and] especially young babes, refer all objects to the mouth."¹³ Abrahams reported on a 9-month-old baby poisoned by painted lead soldiers.¹³ But Australian physicians really opened the issue to the international medical community. In Australia, in 1897, Turner documented "Lead Poisoning Among Queensland Children."¹⁴ He listed a series of cases and noted that lead poisoning cases were widespread among children aged 3 to 12 years.

Gibson, a specialist in diseases of children, particularly diseases of the eye, practiced in Brisbane near the lead and silver mining region of Queensland. His 1904 article in the *Australasian Medical Gazette* was the first publication to directly link lead-based wall paint to disease in children.¹ The article, while written in a style foreign to modern clinicians, documented the cases of four children who suffered from "ocular neuritis," damage to the optic nerve and the motor nerves to the eye that interfered with the children's sight and eye motion. According to Gibson, all of these cases would have resulted in permanent blindness if the children had not been removed from their surroundings.¹

In the article, Gibson engaged in what at the time was a classic example of clinical deduction and "gumshoe" epidemiology in which he sought to understand the source of the lead poisoning that physicians had been identifying in children for about a decade. In earlier cases, he had observed

children who had gone blind, suffered convulsions, and even died from lead poisoning. He slowly eliminated a variety of sources of lead from consideration, including lead in drinking water, absorbed from the inside of storage tanks. He detailed the case of a two-year-old child who had ingested lead dust that had come off painted walls and floors of his house and accumulated on his fingers and hands, to be ingested when he put his fingers in his mouth. Two sources of lead paint dust were seen as especially threatening. First, Gibson argued that newly painted walls were dangerous to children because of the “stickiness of comparatively new paint.” Second, he argued that lead paint tended to deteriorate over time and to easily become “powdery or semi-powdery.” Dust that easily came off walls when touched or disturbed was a major threat to children as they crawled around the house. Gibson pointed out that painted surfaces that have “lost some of their oil and gloss, and which when rubbed yield a powdery substance to the touch and possibly distribute it to the dust of rooms” were dangerous. “Dust, of course, is capable of being both swallowed and inhaled.”¹¹

Lead-based paint remains the major source of lead intake for U.S. children.^{15–18} As anticipated by Gibson, lead-contaminated house dust is recognized as the major pathway by which children are exposed to lead from paint.^{19–21} In 1974, Charney, Sayre, and coworkers showed that normal mouthing behaviors lead to ingestion of lead-contaminated house dust.¹⁷ More recently, Shannon and Graef have shown that the primary source of lead for the majority of children with blood lead >25 $\mu\text{g}/\text{dl}$ is lead-based paint.²² Others studies have shown that, for children with blood lead >10 $\mu\text{g}/\text{dl}$ and ≤ 25 $\mu\text{g}/\text{dl}$, the primary source is house dust contaminated by lead-based paint that is damaged or in disrepair.^{20,21,23} Scraping, sanding, or construction during painting, renovation, and abatement also increases lead contamination of house dust.²⁴ Thus, over the past 100 years, we have built upon the evidence first articulated by Gibson.

Gibson observed that paint dust stuck to his fingers more readily when his hands were sweaty, leading him to wonder “whether cases of lead poisoning were more frequent in summer than in winter. . . . It was found that from 1898 to 1903, viz., six years, 85 cases of lead poisoning had been treated as inpatients at the hospital; that of these, 42 were admitted during the months of December, January, and February, our hottest months; that 28 were admitted during October, November, March, and April; and that 16 only were admitted during the five cooler months.”¹¹

Since this observation, numerous investigators have confirmed that lead poisoning peaks in the summer months. These investigators have proposed numerous hypotheses to explain seasonal variation, including solar radiation activation of vitamin D with increased absorption of both lead and calcium; greater likelihood of soil ingestion; greater likelihood of playing on porches, which often contain higher concentrations of lead than indoor surfaces and are exposed to weather; increased renovation or remodeling activity during summer months; and higher emissions of leaded gasoline.^{25–28} Although seasonal variation in childhood lead poisoning persists, it remains an enigma.

Gibson’s observations led him to believe that there was little that the medical profession could do to adequately

address the problem unless children were removed from dwellings that contained lead. He described the treatment of children he had diagnosed in which he gave muscle relaxants, iodine, and other common treatments for eye disorders. Yet, he was “convinced that removal of the cases from their homes to the hospital is more important than anything.”¹¹

In an article published a year later, Gibson reported on more cases of ocular neuritis in children. He held that poisoning was due to paint powder that came off of walls and verandah railings and urged that “the use of lead paint within the reach of children should be prohibited by law.”²⁹ A series of Australian studies came from Queensland, where a group of physicians largely working out of the Brisbane Hospital for Sick Children observed symptoms in children that mimicked the symptoms of industrial workers. Christian Warren has hypothesized that these clinicians were attuned to the significance of these symptoms because of the importance of silver and lead mining in the local economy.⁴ For the Australian researchers, particularly in Queensland, the damage done to children led them to push for legislative bans on the use of lead as a pigment in paint.

AMERICAN PHYSICIANS DISCOVER AUSTRALIAN STUDIES

In 1907, American physicians could learn of the Queensland studies from David Edsall of Harvard, who noted their significance in a chapter he contributed to the textbook *Modern Medicine*.³⁰ The Australians continued to document the role of lead paint in the poisoning of children, publishing in medical journals in their own country and also in the prestigious *British Medical Journal*.³¹

This literature was picked up by U.S. researchers when they began to document cases of lead poisoning in the 1910s and 1920s. The first U.S. documentation of the case of childhood lead poisoning from paint came in 1914, when Henry Thomas and Kenneth Blackfan, physicians at the Harriet Lane Home, a children’s facility affiliated with Johns Hopkins Hospital, detailed the case of a Baltimore boy who died of lead poisoning from white lead paint bitten from the railing of his crib.³² Five days before admission, the child began to complain about “pain in his face and head, to be restless at night and to look ill.” He began vomiting and rapidly deteriorated. He then began to convulse and went into a coma, and when he entered the hospital he was comatose with his head thrust forward “and his arms and legs . . . extended and spastic.”³² In 1917, Blackfan published an article in which he reviewed the extensive English-language literature on lead poisoning in children.³³ In his own case histories, he described children poisoned by gnawing on lead, and he concluded his review with the recommendation that children should be prevented from eating or mouthing painted articles. He described children who first became “fretful, peevish and often very restless at night.” Their appetite was poor and their gums began to bleed, and soon pain shot up and down their legs. Their stomachs began to ache and they became constipated. Their muscles became “so painful as not to permit the weight of the bed-clothing.” They devel-

oped a waddling gait, only walking on the "outside of the feet." They dragged their toes and their legs swung out sideways as they walked. Soon, seizures occurred and some children died.³³

In the 1920s, American clinicians produced a number of articles linking lead-based paint to lead poisoning in children.³⁴⁻⁴³ These early casualties were signs of a much broader problem that was not being addressed. Isaac Abt argued in his standard text on pediatrics that childhood lead poisoning was "more common in children than generally supposed,"⁴⁴ a point that was echoed over and over in the coming years. In 1924, the *Journal of the American Medical Association* published an article by John Ruddock, which showed that the true extent of lead poisoning in children was understated because there were "many mild cases . . . manifested by spasms or colic, the true nature of which are never suspected."⁴⁵ In 1926, Charles F. McKhann, a Harvard physician, detailed 17 case studies, concluding that lead poisoning was "of relatively frequent occurrence in children" and was usually associated with the ingestion of lead paint.⁴⁶

IMPLICATIONS FOR THE PRESENT

In the 1920s, the lead industry and its defenders argued that the real "culprit" where lead poisoning was concerned was the child. They were able to do so because in that era many viewed lead poisoning as the result of pathological behavior on the part of the child. Some physicians reporting cases of lead poisoning in children described the poisoning as a consequence of a condition known as pica, which was defined by these physicians as an abnormal craving for non-edible substances.^{45,47,48} To make such a diagnosis pathologized the child's behavior, given that pica was often associated with mental retardation. Others argued that the problem was not the child's behavior but the fact that there were many opportunities for children to put lead in their mouths. For these physicians, pica (if they used the term) was a normal habit, not a pathology. This distinction had enormous social and political implications for the lead industry: if the ingestion of lead was defined as due to the pathology of a small number of individual children, the lead industry could justify the continued use of lead. But, if this gnawing and mouthing were a normal habit in children, the number of potential victims of poisoning would be increased astronomically, and the industry's responsibility less easily skirted.

Although children with excessive mouthing behaviors are at particular risk for lead poisoning, contemporary researchers have confirmed that mouthing behaviors that put children at risk for lead ingestion are quite common. In one recent study, mouthing behaviors that put children at risk for lead ingestion were found to peak during the second year of life, when about 30% of children reportedly put soil or dirt in their mouths.²³

One indication of how important Gibson's work remains is the fact that the lead industry, currently under siege in a number of important lawsuits, has tried to denigrate Gibson's work and its impact on international researchers and physicians in the early decades of the 20th century. The industry has historically argued that the relationship of lead paint to

childhood poisoning went virtually unrecognized until the 1950s, at which time the industry removed lead from paint. To acknowledge the impact on U.S. researchers and clinicians of Gibson's work, and that of the other researchers to follow in the first half of the century, would undermine the industry's legal argument that it bears no responsibility for the damage done to children prior to the 1950s. In recent court proceedings, an historian employed by the lead industry argued that Gibson's work, even if it was accurate for Australia, had little bearing on the U.S.: "The ecological setting of tropical Queensland was so exotic and the symptoms that brought most victims to doctors so peculiar that American physicians—noting the differences—relegated the Queensland observations to the uniqueness of the tropics," argued Peter English, a pediatrician and historian at Duke University.⁴⁹ Unfortunately for the industry's legal argument, American physicians by the second decade of the century very clearly understood the impact of Gibson's work. Early U.S. articles on childhood lead poisoning acknowledged the importance and relevance of Gibson's work. In 1914, in the first published report of a U.S. case of childhood lead poisoning due to paint, Thomas and Blackfan noted that "J. Lockhart Gibson has for a number of years repeatedly called attention to a most remarkable group of cases seen in children of Queensland, Australia. The case which we have reported seems closely allied to these."³² Similarly, but even more prominently in the second case report in 1917, Blackfan began with an homage to Gibson and the Australian physicians whose pioneering work brought the dangers of lead paint to worldwide attention: "We are indebted to the Australian writers . . . Gibson, Love, Turner, Breinell & Young and others for much of the recent literature regarding lead poisoning in children."³³

While Americans were indebted to Gibson and the other Australians for highlighting the importance of lead paint as a source of childhood lead poisoning, there were no restraints placed on the use of lead paint on surfaces accessible to children for many decades. For much of our history, we have focused our preventive efforts on educational interventions, rather than primary prevention. Even Gibson recognized that educating parents about the risks of exposing children to lead paint was not enough. In the conclusion of his seminal article a century ago, Gibson remarked that he was "not adverse to leading a crusade against the semi-vandalism of covering the prettily grained pine linings of our houses with paint."

As foretold by Gibson and Turner, we are learning, as though for the first time, that education is, by itself, inadequate to prevent lead poisoning.^{1,2} In 2002, the Centers for Disease Control and Prevention (CDC) released a document that reviewed the evidence for the various educational efforts to prevent children's exposure to lead in and around their homes.⁵⁰ The CDC concluded that there was little evidence that the various educational efforts that were relied on throughout the 1980s and 1990s, which focused on factors such as frequent handwashing, calcium supplementation, and reducing children's mouthing behaviors, protected children from lead poisoning. Until we shift our efforts toward regulations that reduce lead hazards in and around children's homes, as forecast almost 100 years ago by the

Queensland physicians, we will continue to allow children to develop lead toxicity.

The authors would like to acknowledge the support of the Robert Wood Johnson Foundation's Investigator Awards Program in Health Policy Research.

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