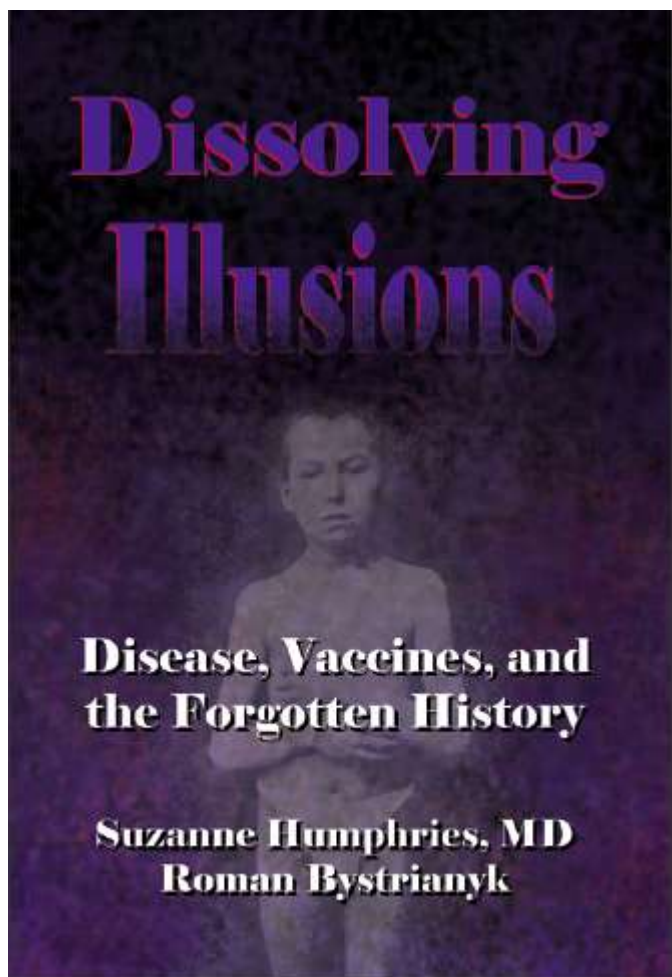


Dissolving Illusions

**Disease, Vaccines, and
the Forgotten History**

**Suzanne Humphries, MD
Roman Bystranyk**



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Rally of the Anti-Vaccination League of Canada, Old City Hall
November 13, 1919
Photographer: William James
Thanks to the City of Toronto Archives

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ISBN: 1480216895

ISBN-13: 978-1480216891

The “Dissappearance” of Polio

~ 12 ~

THE “DISAPPEARANCE” OF POLIO

I also looked at their children and wondered why they got so sick. This time the answer came rather quickly and from the mouth of an Aboriginal woman: “Before the white man came, we had good health and no sickness.”

–Dr. Archie Kalokorinos

Morris Beale, who for years edited his informative publication, Capsule News Digest, from Capitol Hill, offered a standing reward during the years from 1954 to 1960 of \$30,000, which he would pay to anyone who could prove that the polio vaccine was not a killer and a fraud. There were no takers.

– Eustace Mullins (1923–2010), *Murder by Injection*

Live virus vaccines against paralytic poliomyelitis, for example, may in each instance produce the disease it is intended to prevent; the live virus vaccines against measles and mumps may produce such side effects as encephalitis. Both of these problems are [due to the inherent difficulty of controlling live viruses in vivo [once they are placed in a live person].

– Jonas and Darrell Salk, *Science*, March 4, 1977

The polio story is a haunting one: long, complicated, and ugly. It’s not a story you will have read or that the medical profession will be able to tell. Beyond the smoke and mirrors lie sketchy statistics, renaming of diseases, and vaccine-induced paralytic polio caused by both the

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Salk,^{1,2} and the Sabin vaccines. Dr. Albert Sabin’s oral polio vaccine continues to cause paralysis in vaccine recipients today.^{3,4}

Despite many well-documented historical problems, polio and smallpox vaccines serve as the anchor for vaccination faith today. The subject stirs passion in those who believe their ancestors were affected by the dreaded virus or their children could be crippled by it today.

Many believe that a disease called polio has been eradicated in the Western hemisphere. Most everyone thinks that polio was eliminated by vaccination. But to fully understand where polio went, one must understand what polio was. Then, it becomes clear that it is impossible to eradicate it with a vaccine. However, the vaccine did lend itself to many well-documented—although not well-known—problems.

The term *poliomyelitis* is a description of spinal pathology. The meaning of the word comes from Greek *Polios* (gray), *myelos* (marrow), *itis* (inflammation) and denotes inflammation of the gray matter of the spinal cord.

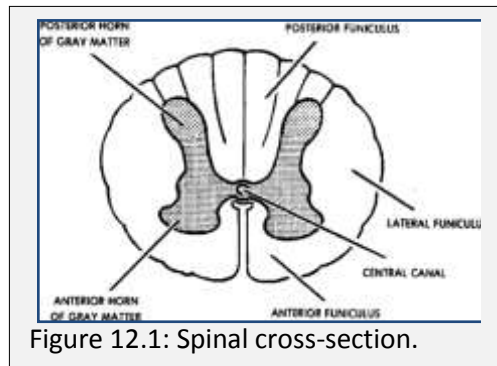


Figure 12.1: Spinal cross-section.

¹ A. Langmuir, *The Wyeth Problem: An Epidemiological Analysis of the Occurrence of Poliomyelitis in Association with Certain Lots of Wyeth Vaccine*, Polio Surveillance Unit, Epidemiology Branch, Communicable Disease Center, Department of Health, Education, and Welfare, September 6, 1955, p. 19.

² N. Nathanson and A. Langmuir, “The Cutter Incident,” *American Journal of Hygiene*, 1963, vol. 78, pp. 29–60.

³ J. F. Modlin, “The Bumpy Road to Polio Eradication,” *New England Journal of Medicine*, vol. 362, June 24, 2010, pp. 2346–2349.

⁴ A. Shahzad, “Time for a Worldwide Shift from Oral Polio Vaccine to Inactivated Vaccine,” *Clinical Infectious Diseases*, vol. 49, October 15, 2009, pp. 1287–1288.

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The gray matter is labeled here on the crosssection of the spinal cord. Poliomyelitis can occur in the brainstem and the spinal cord.

The result of this inflammation, whether chemical or viral, is reflected by certain characteristic muscular symptoms that have been conditioned into the minds of several generations to look like the boy in the picture to the right. The most visible aspects of polio were the braced limbs, iron lungs, deformities of hips and legs, clubbed feet, and scoliosis.

A small number of polio victims were placed on what is locked into our collective memory: iron lung machines. Those images are perhaps the most terrifying because they represent the most serious form of polio called *bulbar poliomyelitis*, where the brainstem is involved and the death rate is highest.

Poliomyelitis was widely believed to be caused by a virus that infects the intestinal tract and moves into the body.



Photo 12.1: George Clark walks on crutches and heavy braces. He had polio attack last April. (1956)

Prevalence of polio, 1912–1969

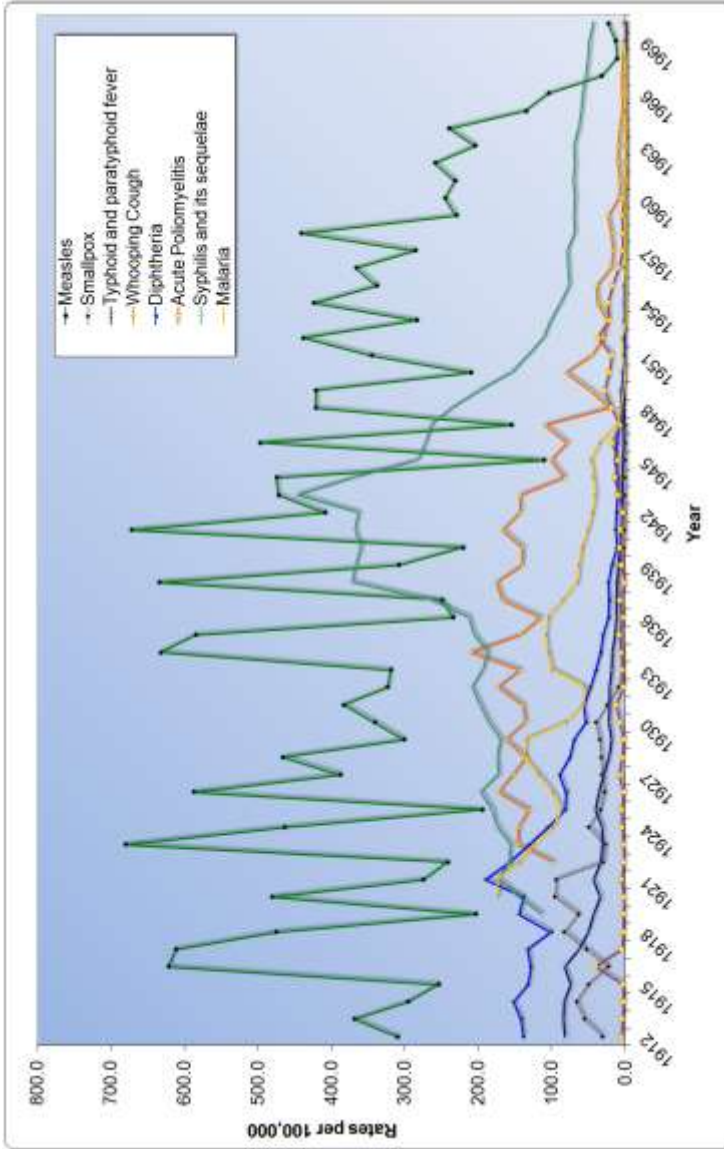
Since the early 1900s, we have been indoctrinated to believe that polio was a highly prevalent and contagious disease. Graph 12.1 depicts the incidence of various diseases in the United States between 1912 and 1970. Poliomyelitis is the line (with square points) at the bottom and reveals that the incidence was very low when compared to that of other infectious diseases. Polio has also been portrayed as a viciouscrippler in the early and mid-1900s when it was habitually diagnosed by doctorswho used a very loose

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definition of the disease. This graph denotes rates of clinical disease, most of which resolved and left no residual paralysis at all.

Given what a low-incidence disease it was, how did polio come to be perceived as such an infamous monster? This is a question worthy of consideration, especially in light of the fact that the rate was far less than other common diseases, some of which declined in incidence to nearly zero with no vaccine at all. Those who still embody a fear of polio may argue that polio was a monster because it crippled people, especially children. But it was later revealed, after a vaccine was lauded for the eradication of polio, that much of the crippling was related to factors other than poliovirus, and those factors could not possibly have been affected by any vaccine.

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Graph 12.1: United States disease incidence 1912-1970.

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Natural (wild) poliovirus

It is easy to assume that poliovirus appeared abruptly or somehow changed in the 1900s to become as problematic as it was alleged to be. Naturally existing poliovirus was a common bowel inhabitant for millennia, always there, continuously circulating through humans, but never causing paralysis until later when something changed. The key question is: What opportunities could have arisen that afforded poliovirus the ability to cause epidemics in the early 20th century?

Under healthy environmental and dietary conditions, certain populations had antibodies to all three types of the virus and did not develop paralysis or have significant symptoms after infection. The remote Brazilian Xavante tribe serves as an example. This tribe was relatively untouched by modern man because they fought encroachments by slaughtering anyone who got close. There was a brief period of time in the 1700s when some of the natives lived among the white men until they realized that doing so brought waves of disease and death upon them. Those who survived fled and moved farther west in an attempt to isolate themselves. Around the 1950s, a few Indian health service members managed to get some cooperation for a study that evaluated the resistance to disease and the immune status of those native people. The results of the pilot study were published in 1964.

Isolated native tribes seem to have had no problem with the infections that were plaguing white men, even though blood results showed that the natives were indeed exposed and infected by many of those very same germs. Dr. Neel found that:

The paradox of a virtual absence of paralytic poliomyelitis among such heavily infected groups as this [referring to the Xavante Indians], despite high antibody titers, is well

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*known, but the interpretation of the observation remains under discussion.*⁵

These isolated people, who had not adopted any of the habits or medical interventions that are now known to increase susceptibility to poliomyelitis, were fully infected and immune! Native Indian populations had evidence of infection with all three strains of poliovirus, but developed no poliomyelitis whatsoever.

*. . . studies of antibody avidity according to the techniques of Sabin (1957) were made on randomly selected specimens. All specimens were positive for antibodies to all three types of poliomyelitis, providing additional confirmation of the validity of the findings [that the Indians were all immune and none of them paralyzed]. . . . The percentage of positive reactors is striking.*⁶

These people had true herd immunity:

*. . . all of the 60 persons tested with both techniques have antibodies to type I, 59 [had antibody] (98.3 ± 1.7 per cent) to type II, and 56 [had antibody] (93.3 ± 3.2 per cent) to type III.*⁷

Within Neel’s paper, there is ample documentation and reference to the fact that native populations, when left to their natural diet and habitat, can become infected with influenza, salmonella, and measles. But the diseases do not spread clinically within the tribe, and mortality is nonexistent.

*. . . in this instance, under-reporting hardly can be invoked as an explanation, **one must conclude that in the Peruvian altiplano most infections are subclinical or give rise to***

⁵ J. V. Neel et al., “Studies on the Xavante Indians of the Brazilian Mato Grosso,” *American Journal of Human Genetics*, vol. 15, March 1964, pp. 52–140.

⁶ *Ibid.*

⁷ *Ibid.*

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*only trivial illness. . . . The demographic data make it clear that the eight persons positive for influenza did experience their disease while in contact with other members of the tribe. Why did the disease not spread?*⁸

Dr. Albert Sabin, inventor of the oral polio vaccine, also noted in 1947 that native peoples were infected by poliovirus before the age of five, and yet there was no paralysis among them. There was, however, a significant rate of paralytic poliomyelitis in American servicemen in the same areas. Paralytic disease was common in the colonizing communities but not in natives.

*. . . the most important question: why did paralytic poliomyelitis become an epidemic disease only a little more than fifty years ago, and as such why does it seem to be affecting more and more the countries in which sanitation and hygiene, along with the general standard of living, are presumably making the greatest advances, while other large parts of the world, regardless of latitude, are still relatively unaffected?*⁹

Sabin said that the virus was present all over the world and that asymptomatic infection was widespread, even in regions where epidemics were unknown. The incorrect assumption by Dr. Sabin was that polio had anything to do with wealth per se. It probably had more to do with the subtle deterioration of innate immunity due to what wealthy people and American servicemen were spraying in the environment, what doctors were doing to them, and other lifestyle habits.

⁸ J. V. Neel et al., “Studies on the Xavante Indians of the Brazilian Mato Grosso,” *American Journal of Human Genetics*, vol. 15, March 1964, pp. 52–140.

⁹ A. Sabin, “The Epidemiology of Poliomyelitis: Problems at Home and Among the Armed Forces Abroad,” *Journal of the American Medical Association*, vol. 134, June 28, 1947, pp.749–756.

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Dr. Archie Kalokerinos, a medical doctor who spent his career tending to the aboriginal people of Australia, was told repeatedly by the elders that the tribes had no disease until the white men arrived and that they didn't even have names for these diseases.¹⁰

Although Dr. Sabin was simply puzzled about the clean and advanced parts of the world getting sick, he failed to connect the rate of paralysis to easily identifiable factors. Examining what changed in the environment and the human diet and how that clearly affected the susceptibility to paralysis in developed areas is key to understanding polio.

The white man's diet of refined and processed foods with the resultant lack of vitamins, the environmental and agricultural poisons, and specific invasive medical procedures, all contributed to the rise in susceptibility of people living in industrialized parts of the world. But by the time those connections were made, disease-causing food was well ingrained in the modern palate. Medical advances were met with gratitude even though many of them were dangerous and overused. **Refined sugar, white flour, alcohol, tobacco, tonsillectomies, vaccines, antibiotics, DDT, and arsenic had become financial golden calves that led humanity blindly down a spiral of disease and misery.** Unfortunately, the paralysis was uniformly attributed to poliovirus infections which thus justified and prioritized vaccine research at all costs. **Many thousands of people were needlessly paralyzed because the medical system refused to look at the consequences of these golden calves, gave only lip service to the success of the Sister Kenny treatment of paralysis (discussed later in this chapter), and concentrated solely on vaccine research.**

¹⁰ A. Kalokerinos, *Shaken Baby Syndrome: An Abusive Diagnosis*, April 12, 2008, March 2012, Copyright 2012, Robert Reisinger Memorial Trust. E-book available on beyondconformity.co.nz and vaccinationcouncil.org websites.

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What polio was and where it is now

Before the vaccine was in widespread use, many distinct diseases were naively grouped under the umbrella of “polio.” Only after the vaccine was widely accepted was there an effort to distinguish poliovirus from other types of paralytic disease. The following list represents a few that could have been categorized and documented as polio prior to 1958.

- Enteroviruses such as Coxsackie and ECHO
- Undiagnosed congenital syphilis
- Arsenic and DDT toxicity
- Transverse myelitis
- Guillain-Barré syndrome
- Provocation of limb paralysis by intramuscular injections of many types, including a variety of vaccines
- Hand, foot, and mouth disease¹¹
- Lead poisoning¹²

These are all conditions that still exist today and that a polio vaccine could not prevent.

The face of polio may have changed, but it was mostly due to the power of the pen, advances in diagnostic and life-support technology, removal of certain toxic influences, and advancements in physical therapy.

Specific polio diagnosis was not pursued with laboratory testing before 1958. The diagnostic criteria for polio were very loose prior to the field trials for the vaccine in 1954. Before the vaccine was

¹¹ W. Xu, C. F. Liu, L. Yan, J. J. Li, L. J. Wang, et al., “Distribution of Enteroviruses in Hospitalized Children with Hand, Foot and Mouth Disease and Relationship Between Pathogens and Nervous System Complications,” *Virology Journal*, vol. 9, January 9, 2012, p. 8.

¹² A. F. Braff, D. O. Lynn, and O. A. Wurl, “Fatal Lead Poisoning Simulating Poliomyelitis,” *US Armed Forces Medical Journal*, vol. 3, no. 9, September 1952, pp. 1353–1357.

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deployed, health-care professionals were vigilantly programmed to be on the lookout for polio. After the trials, they were vigilantly noting who developed polio—vaccinated or unvaccinated—and made every effort to diagnose a non-polio illness in a vaccinated person. Dr. Bernard Greenberg, head of the department of biostatistics of the University of North Carolina School of Public Health and chairman of the Committee on Evaluation and Standards of the American Public Health Association, stated in 1960:

Prior to 1954 any physician who reported paralytic poliomyelitis was doing his patient a service by way of subsidizing the cost of hospitalization and was being community-minded in reporting a communicable disease. The criterion of diagnosis at that time in most health departments followed the World Health Organization definition: “Spinal paralytic poliomyelitis: signs and symptoms of nonparalytic poliomyelitis with the addition of partial or complete paralysis of one or more muscle groups, detected on two examinations at least 24 hours apart.” Note that “two examinations at least 24 hours apart” was all that was required. . . . Laboratory confirmation and presence of residual [longer than 24 hours] was not required.¹³

The practice among doctors before 1954 was to diagnose all patients who experienced even short-term paralysis (24 hours) with “polio.” In 1955, the year the Salk vaccine was released, the diagnostic criteria became much more stringent. If there was no residual paralysis 60 days after onset, the disease was not considered to be paralytic polio. This change made a huge difference in the documented prevalence of paralytic polio because most

¹³ H. Ratner et al., “The Present Status of Polio Vaccines,” *Illinois Medical Journal*, vol. 118, nos. 2, 3, pp. 84–93, 160–68. Edited from a transcript of a panel discussion presented before the Section on Preventive Medicine and Public Health at the 120th annual meeting of the Illinois State Medical Society in Chicago, May 26, 1969.

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people who experience paralysis recover prior to 60 days. Dr. Greenberg said:

The change in 1955 meant that we were reporting a new disease, namely, paralytic poliomyelitis with a longer-lasting paralysis. Furthermore diagnostic procedures have continued to be refined. Coxsackie virus and aseptic meningitis have been distinguished from paralytic poliomyelitis. Prior to 1954 large numbers of these cases were mislabeled as paralytic poliomyelitis. Thus, simply by changes in diagnostic criteria, the number of paralytic cases was predetermined to decrease in 1955-1957, whether or not any vaccine was used.¹⁴

As a caseinpoint on how much paralytic disease thought to be polio was not at all associated with polioviruses, consider the well-documented Michigan epidemic of 1958. This epidemic occurred four years into the Salk vaccine campaign. An in-depth analysis of the diagnosed cases revealed that more than half of them were not poliovirusassociated at all(Figure 12.2 and Figure 12.3). There were several other causes of “polio” besides poliovirus.

During an epidemic of poliomyelitis in Michigan in 1958, virological and serologic studies were carried out with specimens from 1,060 patients. Fecal specimens from 869 patients yielded no virus in 401 cases, poliovirus in 292, ECHO (enteric cytopathogenic human orphan) virus in 100, Coxsackie virus in 73, and unidentified virus in 3 cases. Serums from 191 patients from whom no fecal specimens were obtainable showed no antibody changes in 123 cases but did show changes diagnostic for poliovirus in 48, ECHO viruses in

¹⁴ H. Ratner et al., “The Present Status of Polio Vaccines,” *Illinois Medical Journal*, vol. 118, nos. 2, 3, pp. 84–93,160–68. Edited from a transcript of a panel discussion presented before the Section on Preventive Medicine and Public Health at the 120th annual meeting of the Illinois State Medical Society in Chicago, May 26, 1969.

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14, and Coxsackie virus in 6. In a large number of paralytic as well as nonparalytic patients poliovirus was not the cause. Frequency studies showed that there were no obvious clinical differences among infections with Coxsackie, ECHO, and poliomyelitis viruses. Coxsackie and ECHO viruses were responsible for more cases of “nonparalytic poliomyelitis” and “aseptic meningitis” than was poliovirus itself.¹⁵

After the vaccine, there was a concerted effort to distinguish cases with poliovirus from cases without it. This was not a concern prior to 1958 when many diseases common today hid behind the name *poliomyelitis*. Transverse myelitis, viral or aseptic meningitis, Guillain-Barré syndrome (GBS), chronic fatigue syndrome, spinal meningitis, post-polio syndrome, acute flaccid paralysis (AFP), enteroviral encephalopathy, traumatic neuritis, Reye’s syndrome, etc., all could have been diagnosed as polio prior to 1958.

A modern scientific publication has even cast strong doubt on President Franklin Roosevelt’s well-publicized polio diagnosis. The conclusion of a team of modern researchers is that he actually had GBS and not polio as was originally believed.¹⁶

¹⁵ G. C. Brown, “Laboratory Data on the Detroit Poliomyelitis Epidemic 1958,” *Journal of the American Medical Association*, vol. 172, February 20, 1960, pp. 807–812.

¹⁶ Goldman et al., “What Was the Cause of Franklin Delano Roosevelt’s Paralytic Illness?” *Journal of Medical Biography*, vol. 11, 2003, pp. 233–240.

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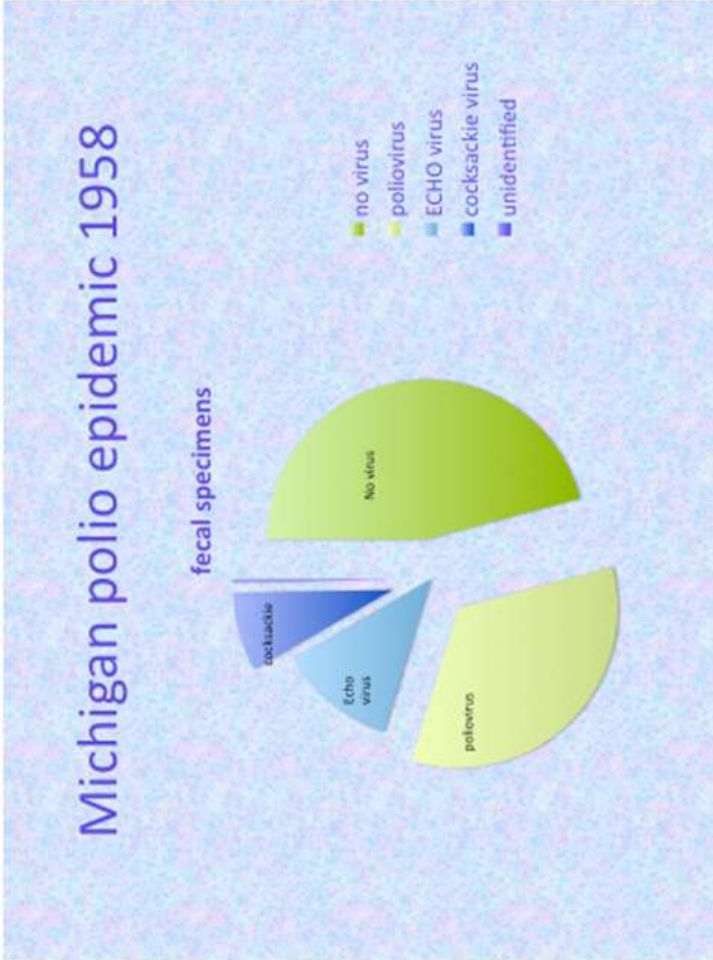


Figure 12.2: Michigan polio 1958 - epidemic virus identification via fecal analysis.

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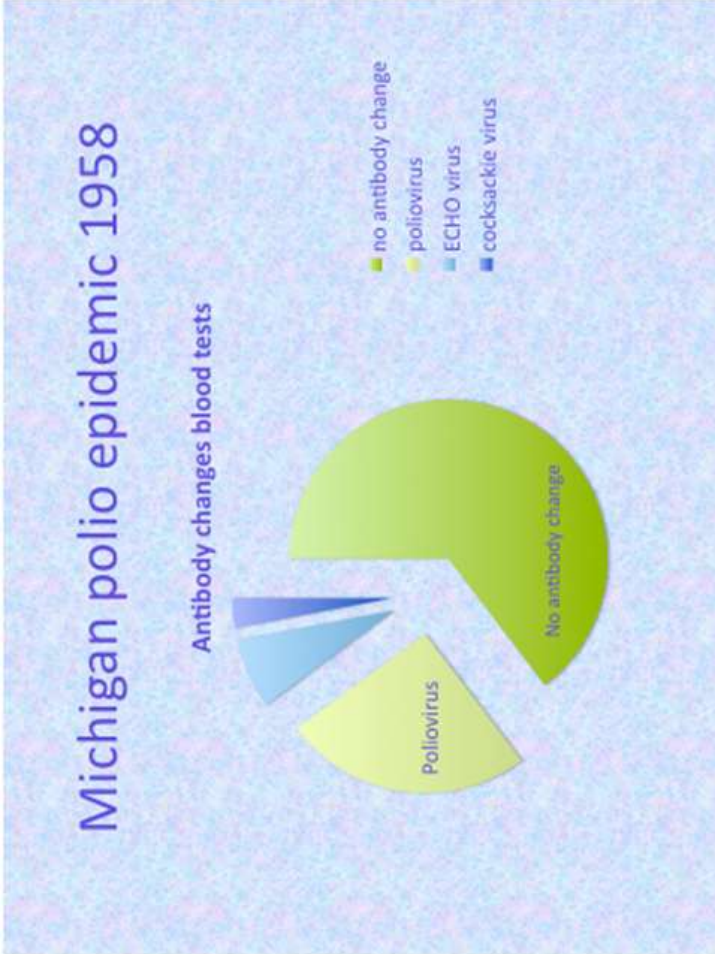


Figure 12.3: Michigan polio 1958 - epidemic viral antibody changes.

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If polio is still here, why don't we see it?

Wild polio virus was never the big killer or paralyzer the public was led to believe it was through the many frightening images shown repeatedly in the 1950s. Dr. Lennette, a well-respected virologist and pioneer of diagnostic virology with the California Department of Health said in reflection on September 1987:

*Actually, economically the disease wasn't very important. Secondly, not many cases were seen in this country. There weren't too many people paralyzed from polio in any one neighborhood, so it never made much of an impact.*¹⁷(See also Graph 12.1.)

The pictographic and cinematic images of polio that were used to rally the public toward vaccine development and acceptance dropped away after the vaccine campaign began. The public gratefully embraced the vaccine that was believed to have removed the frightful disease. In order to maintain public belief in the vaccine, especially in light of several serious instances of vaccine-induced paralytic polio, the images of polio in the new, highly vaccinated population had to be deleted. Optimism regarding the vaccine prevailed. The March of Dimes campaigns that were once designed to impact human fear and emotion transitioned into what we see today as advertising for “working together for stronger, healthier babies”—funding vaccines for infants and pregnant mothers.

In the 1940s, physical therapy and mobilization were ultimately recognized and developed as an important early intervention for paralysis victims. The cruel and barbaric treatments mentioned in

¹⁷ Edwin H. Lennette, *Pioneer of Diagnostic Virology with the California Department of Public Health: an oral history conducted in 1982, 1983, and 1986 by Sally Hughes, Regional Oral History Office, the Bancroft Library, University of California, Berkeley, 1988.*

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Dr. J.R. Paul’s book *A History of Poliomyelitis*,¹⁸ which included tendon cutting and transplantation and other such “salvage operations,” early and prolonged splinting, surgical straightening, and painful but ineffective electrical treatments, were abandoned.

As a result, images of crying children in plaster casts and splints were not as prevalent. Outcomes in paralysis and deformities improved simply because the disease began to be treated better from the outset. But this change did not happen overnight. It took Sister Elizabeth Kenny, a pioneer of what is now known as physical therapy, 30 years to get the orthodox medical community to accept that they had been incorrectly treating polio and were thus responsible for much of the residual paralysis, deformities, and lingering stiffness.

Dr. John Pohl, one of Sister Kenny’s strongest American supporters, reflected upon the misery that polio victims endured before the Kenny technique was used in Minnesota, circa 1940:

The more she talked, the more it seemed she made sense. Before she came, our city hospital was just crowded with polio. And treatment, in plain language, was just no damned good. If you could have visited the hospital, you would have seen little kids lying stiff and rigid, crying with pain, even though—as she saw—they were not necessarily



Photo 12.2: Sister Kenny encourages a polio patient to stand for the first time. (1946)

¹⁸ J. R. Paul, *A History of Poliomyelitis*, Yale University Press, New Haven, Connecticut, 1971, pp. 335–339.

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paralyzed. We'd take the children to the operating room in those days, straighten them out under anesthetic, and put them in plaster casts. When they woke up, they screamed. The next day they still cried from the pain. That was the accepted and universal treatment virtually all over the world. I saw it in Boston and New York City and London. She said, "That's all wrong."¹⁹

Splinting and casting of paralyzed limbs was the primary form of treatment in the first half of the 20th century. Affected limbs were routinely immobilized in casts for three to six months and often as long as two years.

This is a very important link to the story of polio. The manner in which stiff, painful, numb muscles were handled by doctors had a lot to do with the early face of polio and why it looks so different today in countries where paralytic poliomyelitis is treated differently.

The improper treatment of poliomyelitis led to the dysfunction of limbs, regardless of whether the virus was present or not. Dr. Donald Young Solandt and his associates at the University of Toronto reported that completely immobilizing an animal's limb produced similar muscle changes as nerve cutting or nerve removal.²⁰ Solandt's research demonstrated that immobilization alone was enough to induce flaccidity and apparent paralysis even with completely intact motor and sensory nerve pathways. Later writing by Mead also described how polio victims were treated in hospitals.

Orthopedists . . . believed in the “extreme fragility” of poliomyelitic muscle. Many victims of this disease were cast in plaster for 6 months or so, and their deformities were operated

¹⁹ Victor Cohn, *Sister Kenny: The Woman Who Challenged the Doctors*, University of Minnesota Press, 1975, p. 5. With reference to interview with Dr. John Pohl.

²⁰ D. Y. Solandt et al., “The Effect of Skeletal Fixation on Skeletal Muscle,” *Journal of Neurophysiology*, vol. 6, January 1, 1943, pp. 17–22.

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*on in due course. Not even massage—much less, vigorous exercise of the affected muscle was countenanced.*²¹

Thus the manner in which acutely affected muscles were treated had everything to do with outcome. The expectation among doctors and the public was that poliomyelitis meant a life of corrective surgery and lameness. Sister Kenny proved them wrong.

Today, in Gaza, India, and Nigeria, where poliomyelitis is prevalent and limbs are treated according to the old ways, outcomes are similar to the images of the 1930s and 1940s.²² Those images of crying children in plaster casts, used to influence the population to accept vaccination, were quite rare when the Kenny method was used.

Given the history of successful treatment of paralyzed limbs from poliomyelitis, it does seem strange to revert back to the damaging old ways. How welcome would polio vaccine campaigns be today, if Sister Kenny’s method was implemented in Gaza, India, and Nigeria and those unnecessarily deformed and atrophied limbs were nonexistent?

The iron lung and transverse myelitis

We no longer have iron lungs that look like miniature space rockets, the continuous images of which could instill morbid fear in any parent. Instead, we have small boxes with tubes going directly into the airway, called ventilators. So, when a child is admitted to the hospital with compromised respiratory muscles or brainstem afflictions, instead of being put into an iron lung, she is connected to

²¹ S. Mead, “A Century of the Abuse of Rest,” *Journal of the American Medical Association*, vol. 182, October 1962, pp. 344–345.

²² Referencing the situation in Nigeria. www.gettyimages.co.nz/detail/news-photo/child-cries-as-his-polio-stricken-legs-are-placed-in-news-photo/52622460. Similar images can be seen in Gaza.

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a ventilator. Although this is still frightening, it does not elicit the trepidation of the iron lung.



Photo 12.3: Iron lung encases 27-year-old Boyce Rash whose respiratory muscles have been paralyzed. Breathing function is so impaired that a mechanical apparatus is required to force air in and out of the patient's lungs. Seven iron lungs were shipped to Hickory, two of them from Boston. John Bryan, 8, uses oxygen inhalator. It feeds oxygen to nose of patient who has difficulty in breathing normally. Most severe cases involve paralysis of respiratory muscles. Tube extending from mouth collect saliva which boy cannot swallow because of paralyzed throat muscles. (1943)

Dr. Douglas Kerr from Johns Hopkins stated in his foreword to *The Autoimmune Epidemic* published in 2009:

*Infants as young as five months old can get transverse myelitis, and some are left permanently paralyzed and dependent upon a ventilator to breathe . . . my colleagues at the Johns Hopkins Hospital and I hear about or treat hundreds of new cases every year.*²³

²³ Donna Jackson Nakazawa, *The Autoimmune Epidemic: Bodies Gone Haywire in a World Out of Balance—and the Cutting-Edge Science That Promises Hope*, 2009, p. xv.

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Does the public have any idea that there are hundreds of cases of something that is now called transverse myelitis that would have historically been called polio and is now leaving children permanently dependent on a modern version of the iron lung?

Approximately 33,000 people are afflicted by transverse myelitis in the United States, with 1,400 new cases per year. The symptoms of this disease are described by the National Institutes of Health.

*. . . loss of spinal cord function over several hours to several weeks. What usually begins as a sudden onset of lower back pain, muscle weakness, or abnormal sensations in the toes and feet can rapidly progress to more severe symptoms, including paralysis, urinary retention, and loss of bowel control. Although some patients recover from transverse myelitis with minor or no residual problems, others suffer permanent impairments that affect their ability to perform ordinary tasks of daily living.*²⁴

This is but one disease that would have been called polio in the years

Photo 12.4: Knox Out DDT product advertisement. (1948)

²⁴ *Transverse Myelitis Fact Sheet*, National www.ninds.nih.gov/disorders/transversemyelitis/detail_transversemyelitis.htm.

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leading up to 1954. What causes transverse myelitis?

*Researchers are uncertain of the exact causes of transverse myelitis. The inflammation that causes such extensive damage to nerve fibers of the spinal cord may result from viral infections or abnormal immune reactions. Transverse myelitis also may occur as a complication of syphilis, measles, Lyme disease, and **vaccinations**. Cases in which a cause cannot be identified are called idiopathic.²⁵*

DDT poisoning: A cause of polio-like illness

Insects were not just the bane of cattlemen and farmers throughout the world. Flies, in particular, were believed to spread polio outdoors and in the home. In response, fearful parents sprayed DDT on all their windowsills and sprinkled it on sandwiches in their children’s lunchboxes. DDT in water was used to rinse clothes, bedding, and mattresses. It was thought to be a safe and effective insecticide—even safe enough to spray at public beaches and directly onto children in an effort to halt the spread of polio. (See DDT Advertisement on the previous page. “Only a little fly you say? Yes... but what a



Photo 12.5: Flying and Biting Bugs on Jones Beach Die in a Cloud of DDT, New Insecticide—A truck-mounted for generator squirts the poison, mixed with oil droplets, over a four-mile area of the New York City playground. DDT has a drawback—it kills many beneficial and harmless insects, but does not kill all insect pests. Birds and fish which eat large numbers of DDT-poisoned insects may be casualties too. (1945)

²⁵ *Transverse Myelitis Fact Sheet*, National Institutes of Health, www.ninds.nih.gov/disorders/transversemyelitis/detail_transversemyelitis.htm.

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dangerous monster! He can carry polio, and many other horrible disease germs, right into your home!”)

But science did not support such practices. Most people wrongly thought that DDT was not only nontoxic, but that it was actually good for them.

By the 1960s, there was convincing evidence that poliovirus could live quite happily in pesticide-treated cells, and moreover, that the pesticides led to increased susceptibility of viral invasion.²⁶ DDT was found to enhance the release and intracellular multiplication of poliovirus.²⁷ Thus, it likely contributed to creating a monster out of a normally benign gut virus. Unfortunately, this information was not published in the medical literature until a full decade after the polio vaccine was an accepted solution to poliomyelitis. Coincidentally, DDT was phased out of use in the United States and Canada beginning in the 1960s, right around the time that polio was disappearing.

During summer months at the beach, sugared foods were consumed in large volume. Together with DDT, sugar would have created the perfect storm to paralyze the immune system and create a toxic environment in the gut, giving way to serious poliovirus invasion. Diet—in particular, diets high in refined sugar and flour—has a known impact on susceptibility to severe poliovirus infection. The harsh chemicals used in cane sugar refining are thought by some scientists²⁸ to have contributed to the synergy between an otherwise

²⁶ J. Gabliks, “Responses of Cell Cultures to Insecticides: Altered Susceptibility to Poliovirus and Diphtheria Toxin,” *Proceedings of the Society for Experimental Biology and Medicine*, vol. 120, October 1965, pp. 172–175.

²⁷ J. Gabliks and L. Friedman, “Effects of Insecticides on Mammalian Cells and Virus Infections,” *Annals of the New York Academy of Sciences*, vol. 160, 1969, pp. 254–271.

²⁸ F. Van Meer, “Poliomyelitis: The Role of Diet in the Development of Disease,” *Medical Hypotheses*, vol. 3, March 1992, pp. 171–178.

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innocent virus and the sugar. In addition, as Dr. Sandler demonstrated,²⁹ sugar metabolism and post-prandial hypoglycemia increased cellular viral susceptibility.

In the fear-baked summers of polio, many parents were totally unaware that exposure to DDT alone induced symptoms that were completely indistinguishable from poliomyelitis—even in the absence of a virus.³⁰

*Acute gastroenteritis occurs, with nausea, vomiting, abdominal pain, and diarrhea usually associated with extreme tenesmus [the feeling of having to pass stool with inability to do so]. Coryza [head cold], cough and persistent sore throat are common, often followed by a persistent or recurrent feeling of constriction or a “lump” in the throat; occasionally the sensation of constriction extends substernally and to the back and may be associated with severe pain in either arm. **Pain in the joints, generalized muscle weakness, apprehension and exhausting fatigue are usual; the latter are often so severe in the acute stage as to be described by some patients as “paralysis.”***³¹

²⁹ B. Sandler, *Diet Prevents Polio*, Lee Foundation for Nutritional Research, 1951.

³⁰ M. Biskind, “DDT Poisoning and the Elusive ‘Virus X’: A New Cause for Gastroenteritis,” *American Journal of Digestive Diseases*, vol. 16, no. 3, 1949, pp. 79–84.

³¹ *Ibid.*

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GOOD FOR FRUITS—Bigger apples, juicier fruits that are free from unsightly worms ... all benefits resulting from DDT dusts and sprays.



GOOD FOR STEERS—Beef grows meatier now days ... for it's a scientific fact that—compared to untreated cattle—beefsteers gain up to 50 pounds extra when protected from horn flies and many other pests with DDT insecticides.



KNOX FOR THE HOME—helps **Knox** to make healthier, more comfortable homes ... protects your family from dangerous insect pests. Use **Knox-Out** DDT Powders and Sprays as directed ... then watch the bugs "bite the dust"!



KNOX FOR DAIRIES—Up to 20% more milk ... more butter ... more cheese ... tests prove greater milk production when dairy cows are protected from the annoyance of many insects with DDT insecticides like **Knox-Out** Stock and Barn Spray.

KILLING SALT

CHEMICALS

87 Years' Service to Industry • Farm • Home



GOOD FOR ROW CROPS—25 more barrels of potatoes per acre ... actual DDT tests have shown crop increases like this! DDT dusts and sprays help truck farmers pass these gains along to you.



KNOX FOR INDUSTRY—Food processing plants, laundries, dry cleaning plants, hotels ... dozens of industries gain effective bug control, more pleasant work conditions with **Pennsalt** DDT products.

Photo 12.6: "The great expectations held for DDT have been realized." Penn Salt chemicals advertisement. (1947)

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How could doctors possibly have distinguished a case that presented like DDT poisoning from poliomyelitis? They couldn't. After all, most people thought DDT was completely nontoxic and even healthy. These toxicity cases would have been diagnosed as polio and treated as such, often with a crippling outcome. It is not surprising that Dr. Fred Klenner was able to cure 60 out of 60 cases (100 percent) of polio (including bulbar polio) with the detoxifying agent, vitamin C, given in high intravenous doses.^{32,33} Doctors were on the lookout for polio but not DDT poisoning.

Despite the fact that DDT is a highly lethal poison for all species of animals, the myth has become prevalent among the general population that it is safe for man in virtually any quantity. Not only is it used in households with reckless abandon, so that sprays and aerosols are inhaled, the solutions are permitted to contaminate the skin. Bedding and other textiles are saturated. Food and food utensils are contaminated. DDT is also widely used in restaurants and food processing establishments and as an insecticide on crops. Cattle, sheep and other food animals are extensively dusted with it and large areas are indiscriminately sprayed from airplanes for mosquito control. DDT is difficult and usually completely impossible to remove from contaminated foods (it is not affected by cooking) and it accumulates in the fat and appears in the milk of animals who feed on sprayed pasture or on contaminated fodder or who lick the DDT from their hides. As DDT is a cumulative poison, it is inevitable that large-scale intoxication of the American population would occur. In 1944, Smith and Stohlman of the National Institutes of Health, after an extensive study of the cumulative toxicity of DDT, pointed out, “The toxicity of DDT combined with its cumulative action

³² Fred R. Klenner, MD, “The Treatment of Poliomyelitis and Other Virus Diseases with Vitamin C,” *Southern Medicine & Surgery*, vol. 111, July 1949, pp. 209–214.

³³ R. Landwehr, “The Origin of the 40-Year Stonewall of Vitamin C,” *Journal of Orthomolecular Medicine*, vol. 6, no. 2, 1991.

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and absorbability from the skin places a definite health hazard on its use.”³⁴

This following diagram reveals the parallel between polio epidemics in the United States and tonnage of pesticide (most of which was DDT) production from 1940 to 1970.

³⁴ M. Biskind, “DDT Poisoning and the Elusive ‘Virus X’: A New Cause for Gastroenteritis,” *American Journal of Digestive Diseases*, vol. 16, no. 3, 1949, pp. 79–84.

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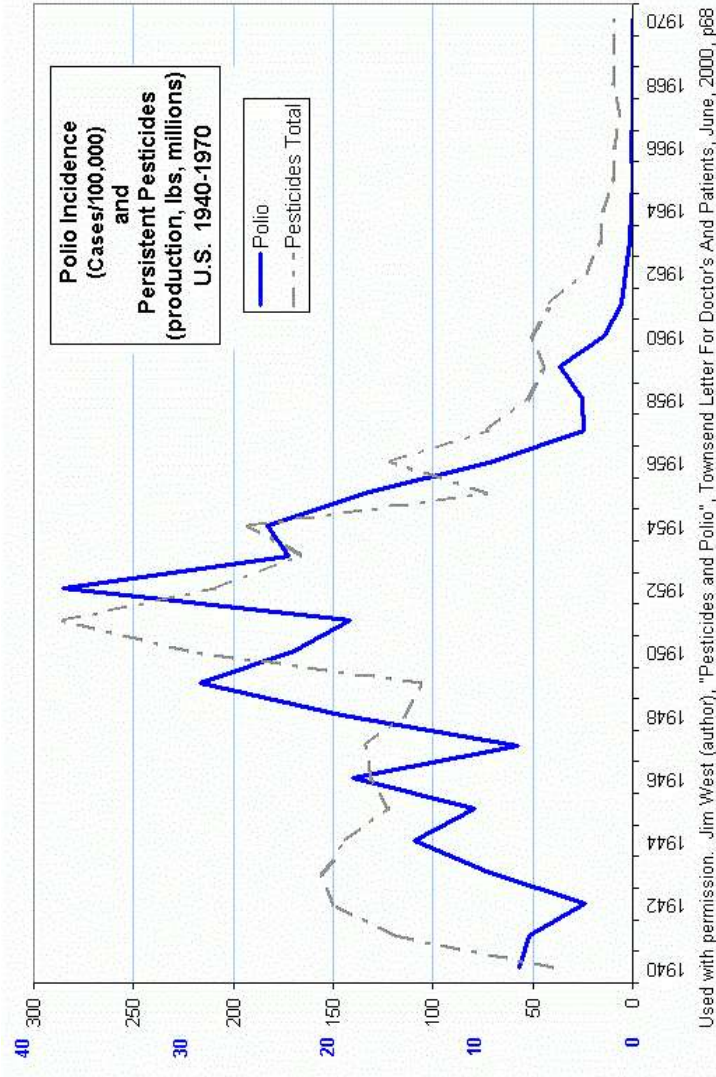


Figure 12.4: Polio incidence and persistent pesticide production.

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It’s no small wonder that polio appeared to be such a vicious entity from the late 1800s and up until DDT was phased out of use in the United States. But that didn’t happen until after a vaccine for polio was fully embraced as a savior of humanity in 1954. The United States was considered free of wild polio as of 1979.

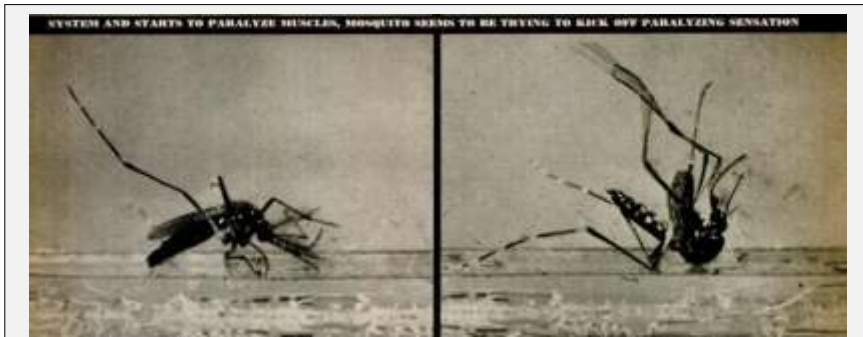


Photo 12.7: Speaking of Pictures . . . These Demonstrate How DDT Paralyzes a Mosquito—In glass case mosquito feels effects of DDT, gives frantic kick, leaps into air. **As DDT enters nervous system and starts to paralyze muscles, mosquito seems to be trying to kick off paralyzing sensation. Paralysis of the nervous system affects the mosquito legs.** The mosquito staggers, falls over, tries to push back onto its legs. It makes one last violent effort to rise but topples back onto its head. On its back and almost completely paralyzed, the mosquito continues to battle against DDT but only succeeds in wiggling convulsively. It took DDT 45 minutes to knock the mosquito out.

Today in India, “polio” is a well-publicized problem, and DDT can be found on shelves just about anywhere. India, one of four countries that still manufactures DDT, remains the chemical’s largest consumer and producer.³⁵ China suffered an epidemic of polio in 2011 and is one of the four countries that has produced and

³⁵ *Report of the Expert Group on the Assessment of the Production and Use of DDT and Its Alternatives for Disease Vector Control*, Third Meeting Geneva, United Nations Environment Programme, November 10–12, 2010.

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continues to use DDT.³⁶ Although breast milk DDT levels in women in the United States is among the lowest in the world after decades of its banning, many other countries are still polluted with the chemical.

Polio by arsenic poisoning

Arsenicals, or compounds containing arsenic, are some of the oldest known causes of poliomyelitis. Yet old texts considered arsenic to be “potent,” “effective,” and “safe” and claimed that it “generally agrees very well” with children.³⁷ Doctors prescribed arsenic in cases of lung problems such as asthma, and it was added to tobacco for smoking. It was also used for cholera on the basis that a greater poison would destroy the lesser poison, and dentists used arsenous acid to kill nerve endings in decayed teeth.

Arsenic was used in wallpaper, paper, fabrics, paints, and dyes in the 1700s and 1800s until women’s groups responded to the poisonings by bringing in muted colors with vegetable dyes. Paris Green and Scheele’s Green were commonly used arsenic-based products that could result in polio symptoms.

After the removal of arsenic-containing pigments, arsenic poisoning resulted from medicines approved by the AMA in the form of supposedly therapeutic injections. Arsenic was used on fruits and vegetables in lead arsenate and calcium arsenate sprays, which resulted in human and animal ingestion. Washing or removing the outsidecontaminated layers of arsenic-treated produce was rarely recommended. Massive spray programs in the spring and at harvestare among the reasons why polio was once commonly

³⁶ Henk van den Berg, “Global Status of DDT and Its Alternatives for Use in Vector Control to Prevent Disease,” *Environmental Health Perspectives*, vol. 117, no. 11, November 2009, pp. 1656–1663.

³⁷ P. Bartrip, “A Pennurth of Arsenic for Rat Poison the Arsenic Act, 1851 and the Prevention of Secret Poisoning,” *Medical History*, vol. 36, January 1992, pp. 53–69. Page 55, second paragraph, Bartrip quotes several medical texts of the times.

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referred to as summer diarrhea. Later, after cold storage for produce was used to extend the shelf life, the programs extended into the winter.

Sister Elizabeth Kenny was a nurse from the Australian outback whose observations led her to treat polio with hot packs and physical therapy. When some of the first “infantile paralysis” epidemics were quietly beginning in remote areas of Australia, she was called to help.

The year was 1912, and she was 23 years old with rudimentary medical training under her belt. In the pages of her autobiography lies evidence that the poliomyelitis she treated was chemical in nature, although at the time she had no idea what might be causing it. Years later, she became quite famous throughout the world for reversing the deformed physical outcomes of polio, which were often caused by accepted orthopedic treatments at the time. In her autobiography, she commented on that fateful night in the Australian outback:

A very agitated father of seven children came to me with the appalling announcement that his ten-year old son and his four-year-old daughter had been taken with what he called the “cow disease” and neither of them could stand or walk. “They went lame yesterday, just like the cattle have been doing for the past two or three weeks,” he explained, “and today they can’t move.”³⁸

Cows are not clinically susceptible to poliovirus-induced poliomyelitis. But they were treated with arsenical dips to rid them of ticks, as noted in another part of Kenny’s autobiography.

On the range itself the cattle had to be moved from time to time for grazing purposes, and periodically “dipped” or run through a narrow canal of water treated with a chemical to

³⁸ E. Kenny, *And They Shall Walk*, Robert Hale Limited, 1951, p. 23.

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*kill the ticks which infect the herds with the disease known as “red water”—the arch enemy of the North Queensland cattlemen.*³⁹

Sister Kenny was naive to the significance of these events. But today we know that chemicals can and do produce symptoms of anterior horn spinal motor neuron disease that were, at the time, clinically and pathologically indistinguishable from viral polio and indistinguishable from what we think of as polio.^{40,41,42}

Not only could congenital syphilis be mistaken for polio, but the treatment of adult syphilis more than likely contributed to the statistical rise in pre-vaccine polio when copious amounts of arsenic-derived medications were prescribed by medical doctors.

In 1939 the AMA lent its Seal of Acceptance exclusively to drugs approved by Chair Morris Fishbein. One of the heavily endorsed products was the arsenical Tryparsamide, manufactured by Merck under license from the Rockefeller Institute for Medical Research. This drug was used with the hope of countering the symptoms of advanced syphilis, often giving more than 100 injections to a single patient.

Another patient who had previously received thirty-four injections of arsphenamine, twenty-three injections of bismuth

³⁹ E. Kenny, *And They Shall Walk*, Robert Hale Limited, 1951, p. 79.

⁴⁰ F. Burgess and G. R. Cameron, “The Toxicity of D.D.T.,” *British Medical Journal*, June 23, 1945, vol. 1, pp. 865–871.

⁴¹ M. Biskind, “DDT Poisoning and the Elusive ‘Virus X’: A New Cause for Gastroenteritis,” *American Journal of Digestive Diseases*, vol. 16, no. 3, 1949, pp. 79–84.

⁴² Ralph R. Scobey, MD, “The Poison Cause of Poliomyelitis and Obstructions to Its Investigation,” *Arch Pediatr*, vol. 69, April 1952, pp. 172–193. [PMID 14924801](#).

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*and seventy-six mercury rubs had a paretic type of serologic relapse after 104 injections of tryparsamide.*⁴³

It was widely known that any type of intramuscular injection could precipitate poliomyelitis, especially one with toxic chemicals and irritants.⁴⁴ Arsenic, even if swallowed, caused symptoms indistinguishable from poliomyelitis.

*Dr. Robert W. Lovett of the Massachusetts State Board of Health (1908), describing the epidemic of poliomyelitis in Massachusetts in 1907, and after reviewing the medical literature on experimental poliomyelitis, states: “The injection experiments prove that certain metallic poisons, bacteria and toxins have a selective action on the motor cells of the **anterior cornua** when present in the general circulation; that the paralysis of this type may be largely unilateral; that the posterior limbs are always more affected than the anterior; and that the lesions in the cord in such cases do not differ from those in anterior poliomyelitis.”. . . Popow **concluded that arsenic, even in a few hours after its ingestion, may cause acute central myelitis or acute poliomyelitis.***⁴⁵

Two other arsenic drugs, neoarsphenamine and neosalvarsan, were well known to cause polio-like syndrome, diagnosed as polio. Reports in Germany in 1914 and 1928 on provocation polio by arsenic injections must have been overlooked.⁴⁶ The AMA, Merck, and

⁴³ F. E. Cormia, “Tryparsamide in the Treatment of Syphilis of the Central Nervous System,” *British Journal of Venereal Diseases*, vol. 10, April 1934, pp. 99–116.

⁴⁴ M. Gromeier et al., “Mechanism of Injury-Provoked Poliomyelitis,” *Journal of Virology*, vol. 72, 1998, pp. 5056–5060.

⁴⁵ Ralph R. Scobey, MD, “The Poison Cause of Poliomyelitis and Obstructions to Its Investigation,” *Arch Pediatr*, vol. 69, April 1952, pp. 172–193.

⁴⁶ H. Kern, “Ueber eine anstaltsendemie von Heine-Medizinscher krankheit,” *Muen Med Wochen*, vol. 61, 1914, pp. 1053–1055; “Alterthum, Lues congenital and poliomyelitis,” *Deut Med Wochen*, vol. 54, 1928, pp. 522–523; H. Gougerot, “Eveil d’infection neurotrophe a virus filtrant a ls suite

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Rockefeller, despite warnings from the inventor of Tryparsamide regarding its danger, continued to distribute the drug,⁴⁷ and polio epidemics continued to rise.

Undiagnosed syphilis

Is it possible that some polio victims could have been undiagnosed syphilitics? (Graph 12.1) Tabes dorsalis, the slow deterioration of nerves and gray matter of the spinal column, is a crippling symptom of syphilis that also affects gray matter of the spinal column. At the time, syphilis was far more prevalent than polio. Infants infected with syphilis at birth may be asymptomatic and may not manifest signs commonly associated with congenital syphilis.

From a case report in 1988:

A 54 year old woman was referred for poor balance, leg weakness and pain, recurrent left knee effusions, and a previous history of “polio.”

Since her clinical and electrophysiological presentation was incompatible with previous poliomyelitis, we hypothesize that she acquired syphilis congenitally and experienced her first symptoms of tertiary disease at age 7 years.

Infants infected at birth may be asymptomatic and may not manifest signs commonly associated with congenital syphilis. Even though most features of tabes dorsalis do not develop until 10-25 years after primary infection, this latency may be as short as 5 years⁴⁸ in children.

d’arsenotherapie chez des syphilitiques,” *Bull Soc Derm Syph*, vol. 42, 1935, pp. 794–795.

⁴⁷ E. Mullins, *Murder by Injection*, National Council for Medical Research, 1988.

⁴⁸ H. H. Merritt, R. D. Adams, and H. C. Solomon, *Neurosyphilis*, Oxford University Press, New York, 1946.

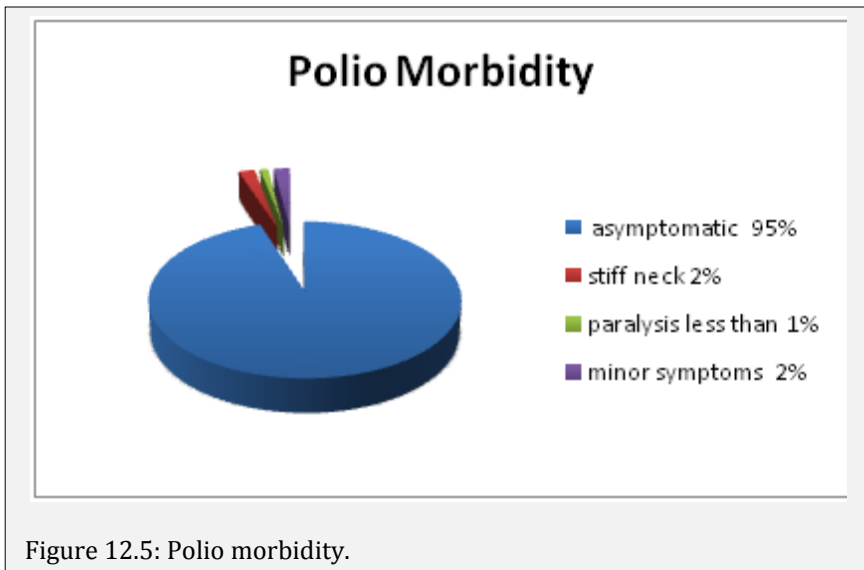
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Distal weakness and atrophy may be late manifestations of tabes dorsalis, attributed to extension of the syphilitic process to anterior horn cells or motor roots.⁴⁹

Although neurosyphilis usually affects the posterior horns of the spinal cord, here we see that anterior horns can also be affected, just like in poliomyelitis, when there is congenital syphilis. This case of syphilis exemplifies how congenital syphilis and polio could have been easily confused.

Morbidity of polio, then and now

The Centers for Disease Control (CDC) defines polio’s statistical paralytic rate and estimates that it is less than 1 in 100 for some sort of permanent paralytic syndrome.



Approximately 95% of persons infected with polio will have no symptoms. About 4-8% of infected persons have minor

⁴⁹ P. Donofrio et al., “Tabes Dorsalis: Electrodiagnostic Features,” *Journal of Neurology, Neurosurgery & Psychiatry*, vol. 51, 1988, pp.1097–1099.

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symptoms, such as fever, fatigue, nausea, headache, flu-like symptoms, stiffness in the neck and back, and pain in the limbs, which often resolve completely. Fewer than 1% of polio cases result in permanent paralysis of the limbs (usually the legs). Of those paralyzed, 5-10% [of that 1%] die when the paralysis strikes the respiratory muscles.⁵⁰

Prior to vaccination, Dr. Maurice Brodie reported that only 1 in 170 children with **no antibody** to polio became ill during epidemics. By these two drastically different risk estimations, you can see that statistics are not set in stone, nor are they necessarily a reliable indicator of risk. The CDC reports a 59 percent higher paralysis rate than was actually measured during a pre-vaccine epidemic.

It would seem that the lack of antibody is a factor predisposing to the disease inasmuch as over 85 per cent of those under 5, and over 70 per cent of the 6-10 year old group show no antibody or only a small amount of antibody. This does not explain why in an epidemic approximately only 1 of the 170 children under 5 showing no antibody, and about the same proportion of those under 10 develop the disease. This may be due to the individual non-specific variation in the susceptibility of the children . . .⁵¹

Dr. Brodie seemed clued in to the susceptibility factor, but he didn't proffer it any further—undoubtedly because he was also single-minded in the pursuit of a vaccine. Unfortunately for the dead and paralyzed recipients, Brodie's vaccine was not safe even though it was, in reality, no more dangerous than Dr. Jonas Salk's vaccine was

⁵⁰ “Polio Disease In-Short,” Centers for Disease Control and Prevention, Department of Health and Human Services, www.cdc.gov/vaccines/vpd-vac/polio/in-short-both.htm, accessed June 17, 2013.

⁵¹ M. Brodie and W. Park, “Active Immunization Against Poliomyelitis,” *American Journal of Public Health*, vol. 26, February 1936, pp. 119–125.

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in its 1955 production. Dr. Brodie allegedly committed suicide at the age of 36 in 1939.⁵²

The question to ask today is, how much poisoning by chemicals and infection with other viruses was counted as polio in the statistics? According to the CDC, less than 1 percent of infected people develop paralysis, and 5–10 percent of that 1 percent suffer respiratory death. Yet in several polio epidemics, far more than 1 percent were paralyzed and even died.

Sister Elizabeth Kenny in Australia reported that 6 of the 20 children in her district were afflicted by painful or paralytic polio. How could 6 out of 20 children in a thinly populated rural area be stricken with polio (infantile paralysis) if it is a viral illness supposedly asymptomatic in 95 percent of those infected? Was it because all were exposed to chemicals?

She went on to the house where the brother and sister were stricken. Their symptoms were the same. Within less than a week the inexperienced, self-appointed nurse found herself with a polio epidemic on her hands, affecting six of the twenty children in the thinly settled district.⁵³

Dr. Archie Kalokerinos was a doctor in a cotton-growing area of Australia. A prominent feature that he noted were all the drums of toxic cottonfield spray, which the children found marvellous to play on when full and in when empty.

Dr Kalokerinos rapidly became familiar with the paralytic disease called polio.

As far as I knew no epidemic of polio had been in progress. But the consultant was right – too right. It was the beginning of a

⁵² J. R. Paul, *A History of Poliomyelitis*, Yale University Press, New Haven, Connecticut, 1971, p. 261.

⁵³ Victor Cohn, *Sister Kenny: The Woman Who Challenged the Doctors*, University of Minnesota Press, 1975, p. 42.

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big epidemic. In a very short space of time I was to become the ‘expert’. I could almost smell polio from afar.

During emergencies I was sometimes covered with sputum, urine, and faeces. At one stage the domestic staff refused to clean my room. The fear of catching polio was understandable. But I came through it all without a scratch. I guess that God was looking after me.⁵⁴

Perhaps Dr. Kalokerinos and all the regular staff (except one junior surfer doctor who lived life in the fast lane) never caught polio, either because they were naturally immune to polio virus like most of the population, they were not directly exposed to the agrichemicals, or they were just lucky . . .

David Oshinsky’s book *Polio: An American Story* chronicles multiple incidents of more than one family member dying or becoming permanently paralyzed after supposed infection with poliovirus.

*Polio hit the Iowa farmbelt hard in 1952. They had tested the well water—it was fine—and **used extra DDT** to drive away flies. . . . Nine of the eleven children recovered, two were left paralyzed. . . . It was even worse for a family living near Milwaukee. Four of the six children came down with bulbar polio.⁵⁵*

All four children who were exposed to DDT died after being treated conventionally by medical doctors with oxygen, penicillin, and plasma.

Wonder drugs and iron lungs and round-the-clock attention had failed to keep these children alive. In an era without a vaccine, it was a terrifying thought.

⁵⁴ Personal correspondence in authors’ possession.

⁵⁵ David M. Oshinsky, *Polio: An American Story*, Oxford University Press, 2005, pp. 163–164.

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Indeed terrifying. Doctors certainly should have known that penicillin would do nothing for a virus. Furthermore, any injection could be a cause of paralyzing polio (provocation polio) if circulating poliovirus was a factor. Did those doctors unwittingly cause the deaths of the children by inducing bulbar polio—the most serious type of polio that affects the brain stem?

Were those children previously tonsillectomized, a well-documented underlying factor not just in bulbar polio but in poliomyelitis incidence? For poliovirus to cause damage requires access to the inside of the body through “peripheral nerve damage,” something which tonsillectomy provides in abundance. The invasive procedure of surgical tonsil removal raised the risk of bulbar polio, as revealed in numerous studies and reports.^{56,57,58,59}

Healthy tonsils were removed by surgeons for various financially rewarding but scientifically unsound reasons. Fifty to eighty percent of middle-class and upper-class children in the United States were needlessly subjected to tonsillectomies in the polio epidemic era. Anderson showed in his large group from a 1943 epidemic in Utah that poliomyelitis was more than 2.5 times more prevalent in tonsillectomized children than age-matched non-tonsillectomized children. The incidence of bulbar poliomyelitis was 16 times higher in tonsillectomized children than in the general child population.

⁵⁶ M. Siegel, M. Greenberg, and M. C. Magee, “Tonsillectomy and Poliomyelitis, II, Frequency of Bulbar Paralysis, 1944–1949,” *Journal of Pediatrics*, vol. 38, no. 5, May 1951, pp. 548–558.

⁵⁷ Francis Thomas, “Poliomyelitis Following Tonsillectomy in Five Members of a Family,” *Journal of the American Medical Association*, vol. 119, no. 17, 1942, pp. 1392–1396.

⁵⁸ J. A. Glover, “The Paediatric Approach to Tonsillectomy,” *Archives of Disease in Childhood*, vol. 23, 1948, pp. 1–6.

⁵⁹ R. V. Southcott, “Studies on a Long Range Association Between Bulbar Poliomyelitis and Previous Tonsillectomy,” *Medical Journal of Australia*, vol. 2, no. 8, August 1953, pp. 281–298.

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Forty-six percent of the bulbar polio cases had been preceded by recent tonsillectomy.⁶⁰

Cunning reported in his series of 0- to 10-year-old bulbar poliomyelitis cases that the ratio of tonsillectomized to non-tonsillectomized was 6 to 1.^{61,62} In 1971 Dr. Ogra reported in the *New England Journal of Medicine* that post-operatively, previously existing pharyngeal anti-polio antibody titers decreased sixfold to eightfold.⁶³

The issue with how doctors treated patients in the epidemic era does not end with what doctors did do, but with what they refused to do. Dr. Klenner had a nearly 100 percent success rate in curing dozens of cases of polio (even bulbar cases) with intravenous infusions of vitamin C. He presented this information at symposia and meetings. He was met mostly with disbelief and ignored. Nonetheless, he continued to cure case after case of polio with vitamin C and published extensively on the details of his experience.⁶⁴

In the poliomyelitis epidemic in North Carolina in 1948, 60 cases of this disease came under our care. . . . Two patients in this series of 60 regurgitated fluid through the nose. This was interpreted as representing the dangerous bulbar type. For a patient in this category postural drainage, oxygen administration, in some cases tracheotomy, needs to be instituted, until the vitamin C has had sufficient time to work—in our experience 36 hours. Failure to recognize this factor

⁶⁰ John Anderson, “Poliomyelitis and Recent Tonsillectomy,” *Journal of Pediatrics*, 1945, pp. 68–70.

⁶¹ D. S. Cunning, “Tonsillectomy-Poliomyelitis Survey, 1947,” *Laryngoscope*, vol. 58, no. 6, June 1948, pp. 503–513.

⁶² D. S. Cunning, “Tonsillectomy and Poliomyelitis,” *Archives of Otolaryngology*, vol. 46, no. 5, November 1947, pp. 575–583.

⁶³ P. L. Ogra, “Effect of Tonsillectomy and Adenoidectomy on Nasopharyngeal Antibody Response to Poliovirus,” *New England Journal of Medicine*, vol. 284, no. 2, January 14, 1971, pp. 59–64.

⁶⁴ Robert Landwehr, “The Origin of the 42-Year Stonewall of Vitamin C,” *Journal of Orthomolecular Medicine*, vol. 6, no. 2, 1991.

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*might sacrifice the chance of recovery. With these precautions taken, every patient of this series recovered uneventfully within three to five days.*⁶⁵

Dr. Klenner was not the only doctor to publish on the successful reversal of severe poliomyelitis cases with high-dose vitamin C.^{66,67}

Laboratory and vaccine sources of epidemics

Nowhere else in polio’s history was there more panic than during New York City’s 1916 epidemic. (Note the large 1916 peak on the polio curve in graph13.1.) Dr. H.V. Wyatt published a document in 2011 discussing the possibility that a highly virulent laboratory-engineered strain of poliovirus “escaped” from the Rockefeller laboratories, causing the largest epidemic of polio in US history. Just what exactly could have escaped from the lab is unknown.

The epidemic was thought to have affected 23,000 cases with 5,000 deaths through New England and the Middle Atlantic states, reaching Delaware, Maryland and the District of Columbia with a few cases in Vermont and Canada. It had no apparent connection to lesser epidemics in West Virginia and in Minnesota, Wisconsin and Michigan. These features were never experienced again. Three other aspects were not noted at the time: the number of children age 2 yr affected was the highest ever recorded; the case fatality rate of 25% was the highest ever recorded [certainly higher than natural wild type polio virus which is less than one percent]; the epidemic

⁶⁵ Fred R. Klenner, MD, “The Treatment of Poliomyelitis and Other Virus Diseases with Vitamin C,” *Southern Medicine & Surgery*, vol. 111, July 1949, pp. 209–214.

⁶⁶ E. Greer, “Vitamin C in Acute Poliomyelitis,” *Medical Times*, vol. 83, November 1955, pp. 1160–1161.

⁶⁷ Claus W. Jungleblut, “Further Observations of Vitamin C Therapy in Experimental Poliomyelitis,” *Journal of Experimental Medicine*, September 1937, pp. 470–471.

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*started in early May, well before the normal summer polio season.*⁶⁸

At the time, the epidemic was broadcast to the public as having been started by children who arrived from Italy. But the immigration data does not fit with that hypothesis. Official immigration books show that the epidemic began before those children arrived.

*The 1916 epidemic is featured in many accounts of polio, but details and emphases differ and many are incorrect. The early cases in May in Brooklyn had not been reported, but were found at a later date by the USPHS researchers.*⁶⁹

The epidemic was unique in that the virus was highly destructive to the nervous system, much like the Rockefeller labs cultivated “MV” strain.

Three miles from the epicentre of the outbreak, Simon Flexner and his associates at the Rockefeller Institute at 63rd Street and York Avenue, near Queensborough Bridge on Manhattan Island, had been passaging spinal cord tissue containing poliovirus, from one Rhesus monkey spinal cord to another. These experiments continued with the passage virus which at times was reinforced with newly acquired virus from patients. . . Those doctors had no awareness of what they were handling. . . . By 1916, mutants of the original Rockefeller virus had been selected for replication in monkey motor neurones, but were still capable of high levels of replication in other cells. . . . It is a remarkable coincidence that a unique neurotropic strain of poliovirus was developed a few miles from an epidemic caused by a uniquely pathogenic strain of the virus. . . . A few blocks from the Rockefeller Institute at Lexington Avenue and 63rd Street the 3rd Avenue elevated line

⁶⁸ H. V. Wyatt, “The 1916 New York City Epidemic of Poliomyelitis: Where Did the Virus Come From?” *The Open Vaccine Journal*, vol. 4, 2011, pp. 13–17.

⁶⁹ Ibid.

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*linked at Municipal Building station to the BRT line to Brooklyn over Brooklyn Bridge with a stop at 3rd Street and 5th Avenue where the first case lived. However, almost anywhere in New York was within a few streets of a rail link to the Rockefeller Institute.*⁷⁰

The significance of this epidemic is that it set the stage for the terror to come. Doctors and parents alike, after this aberrantly lethal polio epidemic, were perched for an ominous future and thus ready and willing to do whatever was necessary to eradicate polio.

Many doctors of the 1940s were aware that the pitchmen of the National Foundation for Infantile Paralysis (NFIP) and March of Dimes were responsible for the expanded terror that swept the nation.⁷¹ Few today are aware of the intimate relationship between the NFIP and the Rockefeller Institute. Nearly all the researchers for the polio vaccine were from Rockefeller. Dr. Thomas Rivers, virologist and director, was an “unpaid consultant” to NFIP and Basil O’Connor (NFIP’s founder) and also served as mentor and advisor to Albert Sabin and Thomas Francis.

Sabin developed the live vaccine that is now used in India, and Francis headed the largest public health experiment in history, the Salk vaccine trial of 1954. Rivers was the commandant of the plan to conquer polio in 1938.⁷² He is rumored to have had a serious distaste for Sister Kenny, as did AMA’s Morris Fishbein and NFIP’s Basil O’Connor. NFIP’s attempts to buy her and discredit her were, fortunately, futile.

Synthetic poliovirus

⁷⁰ H. V. Wyatt, “The 1916 New York City Epidemic of Poliomyelitis: Where Did the Virus Come From?” *The Open Vaccine Journal*, vol. 4, 2011, pp. 13–17.

⁷¹ Victor Cohn, *Sister Kenny: The Woman Who Challenged the Doctors*, University of Minnesota Press, 1975, p. 125.

⁷² David M. Oshinsky, *Polio: An American Story*, Oxford University Press, 2005, pp. 170, 60.

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Today, laboratory generation of infectious virus in the absence of a natural viral template has been accomplished by scientists. It was funded by the US Defense Advanced Research Project Agency (DARPA). Dr. Eckhard Wimmer, one of the scientists involved in the project, reported:

*The empirical formula of poliovirus is C332,652H492,388N98,-245O131,196P7,501S2,340 Placing the atoms in order, a particle of high symmetry emerges. . . . Our experiment has thus overthrown one axiom in biology—namely, that the proliferation of cells or, for that matter, viruses depends on the physical presence of a functional genome to instruct the replication process. It was believed that without parental genomes, no daughter cells or progeny viruses would arise. We have broken this fundamental law of biology by reducing poliovirus to a chemical entity, which can be synthesized on the basis of information stored in the public domain. . . . **Just like a common chemical, poliovirus has been synthesized in the test tube.**⁷³*

Dr. Wimmer also reports that neurovirulence can be manipulated readily in synthetic polioviruses, though he presumes that this capability will be used for attenuation rather than for raising more virulent species. Either one is equally possible.

The Cutter disaster and other vaccine blunders

Most people today don't know about the infamous Cutter disaster. This was a virus-related poliomyelitis epidemic that was initiated by the use of the Salk vaccines just after they were rapidly developed and fast-tracked into licensure by the US Department of Health,

⁷³ E. Wimmer, “The Test–Tube Synthesis of a Chemical Called Poliovirus: The Simple Synthesis of a Virus Has Far-Reaching Societal Implications,” *European Molecular Biology Organization Report*, vol. 7, no. SI, July 20, pp. S3–S9.

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Education, and Welfare. This record-breaking approval process took only two hours.⁷⁴

Because of outside pressure, the licensing committee in charge of approving the vaccine did so after deliberating but without first having read the full research, namely the Francis Report on which their approval was to have been based. Dr. Howard Shaughnessy, laboratory director, Illinois Department of Health, testified to this event:

Previously it [the vaccine] had been distributed as an experimental product, not a licensed product . . . the committee was asked to come to a decision very quickly . . . there was discussion of the report that Dr Francis had given, but we were not in a position to discuss it very intensively because we had not seen the report prior to this morning and the report was distributed to us after the presentation . . . we were pressured in the sense that we were told that speed was essential, and when we came up toward the 5:00 time, some of us felt we would like to discuss this matter more. We were told that to discuss the matter further it would have to go into the following week, and we would have to go to Washington or Bethesda and most of the members were unwilling to do so. We were in effect pressured into an earlier decision than we ordinarily would have made. . . . It was part of the pressure of events, put it that way.⁷⁵

Dr. Thomas Francis did not issue the final report of his evaluation of the 1954 field trials until April 1957, two years after the licensing of

⁷⁴ Richard Carter, *Breakthrough: The Saga of Jonas Salk*, Trident Press, New York, 1955, p. 282.

⁷⁵ Opening brief of Defendant and Appellant Cutter Laboratories Gottsdanker v. Cutter Laboratories (1960) 182 Cal. App.2d 602 pp. 31–33. Dr Shaughnessy was the Director of Laboratories and Head of Department of the Illinois Department of Public Health, University of Chicago, and member of the Ann Arbor Licensing Committee for the Salk vaccine.

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the vaccine.⁷⁶At the time, public health authorities decreed that physicians inject the fast-tracked vaccine before those doctors knew much about the science or the large Francis trial. The consequences of this impulsive action turned out to be significant.

The Salk invention was an injectable, supposedly formaldehyde-inactivated version of poliovirus vaccine. There were serious problems with the viral inactivation process that were known by insiders from the outset of the vaccine’s development. Any professional objection by scientists involved during the development of the vaccine was rapidly subdued.⁷⁷ Dr. Paul Meier attested to the practice of firing scientists who disagreed with the NFIP’s plans.

*Jonas Salk had a paper in which he argued that all the virus was inactivated, and that there was no live virus left. But, the sixth lot was not listed. And so I said that something was wrong. He cut out data in order not to show what happened to some lots. . . . Well, NFIP did form an advisory committee. And they reformed it five or six times. **Each time somebody didn’t agree, they dropped them and got somebody who might agree. By the time they were done forming the committee, everybody on it was distinguished, but very agreeable.**⁷⁸*

As a result of ignoring the warnings by highly qualified scientists who repeatedly and publicly explained why and how the inactivation process was flawed from the beginning, the vaccine virus needlessly infected, paralyzed, and killed children and their household contacts.

⁷⁶ T. Francis et al., “Evaluation of the 1954 Field Trial of Poliomyelitis Vaccine: Final Report,” Poliomyelitis Vaccine Evaluation Center, University of Michigan, Ann Arbor, April 1957.

⁷⁷ H. Eyer et al., *Social Medicine and Hygiene: An Evaluation of the Protective Immunization Against Poliomyelitis, Report of the Scientific Committee, 1956*. This 102-page document with 22 corresponding graphs is a translation of a larger 492-page German report from an article that appeared in the *Munch Med Wochenschr*, April 6, 1956. A copy of English translation is in the authors’ possession.

⁷⁸ H. A. Marks, “Conversation with Paul Meier, Interview by Harry M. Marks,” *Clinical Trials*, vol. 1, February 2004, pp. 131–138.

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*Others Wendell Stanley, Sven Card, Enders, Herdis von Magnus and myself among others disagreed, convinced that the inactivation process did not follow a straight line and it was not permissible to extend the curve below the baseline. . . **And I remember Colin MacLeod raising the question whether this was really the way to go, but that's the way the matter stood, namely, "We'll go ahead and make the vaccine."** Well, the vaccine was made that way. Then Cutter made several batches of the vaccine, **which upon inoculation into man produced cases of poliomyelitis, some of them with severe paralysis.**⁷⁹*

Millionaire vaccine inventor Paul Offit, a supporter of mandatory vaccinations, wrote a book on the Cutter incident. In the book, even he admits:

*. . . the disease caused by Cutter's vaccine was worse than the disease caused by natural polio virus.*⁸⁰

History books credit Cutter Laboratories for the disaster. The official explanation of the problem was that the live virus particles clumped into cellular debris (monkey kidney tissue from the manufacture) and, as a result, formaldehyde could not penetrate the center of the clump. Although this clumping may have occurred, it was not the major reason for the presence of live virus in the 1955 vaccine.

There is a body of literature that speaks to the real cause of the problem, which was known from the outset of the development of Salk's vaccine.

⁷⁹ Edwin H. Lennette, *Pioneer of Diagnostic Virology with the California Department of Public Health*, an oral history conducted in 1982, 1983, and 1986 by Sally Hughes, Regional Oral History Office, the Bancroft Library, University of California, Berkeley, 1988.

⁸⁰ Paul Offit, MD, *The Cutter Incident*, Yale University Press, 2005, p. 86.

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Dr. Thomas Rivers, the mastermind of Rockefeller’s polio vaccine mission, hired all the chairmen of departments of virology. He had enormous clout, and nobody dared argue with him, lest their careers be ruined. Dr. Edwin Lennette had some interesting reflections in the 1980s about Rockefeller, Rivers, and the formalin inactivation curve:

Well, in those days, as I should point out perhaps, things were quite different from today because a professor in this country, just as in Germany, was a highly respected individual, and you didn't argue with him.⁸¹

Dr. Lennette, talking about a pre-vaccine trial meeting of the minds in New York City in 1953, said:

Tom Rivers was there, Tommy Francis, Joe Smadel, and Colin MacLeod, all of whom were deeply involved. These were people to whom you might apply the term "the establishment," . . . These were the "old graybeards" who had been through the mill of medical science . . . The question was raised as to whether the vaccine would be safe at the present level of inactivation with formaldehyde. And I remember distinctly Tom Rivers saying, "If you put any more formaldehyde in, you'll make it so damn safe it won't be any good." That's recorded somewhere in the minutes of that meeting.⁸²

Salk and the scientists who remained on the NFIP board interpreted the formaldehyde inactivation curve incorrectly. As a result, live virus remained. Stubbornly, they would not heed the warnings.

⁸¹ Edwin H. Lennette, “Pioneer of Diagnostic Virology with the California Department of Public Health,” an oral history conducted in 1982, 1983, and 1986 by Sally Hughes, Regional Oral History Office, the Bancroft Library, University of California, Berkeley, 1988.

⁸² Edwin H. Lennette, *Pioneer of Diagnostic Virology with the California Department of Public Health*, an oral history conducted in 1982, 1983, and 1986 by Sally Hughes, Regional Oral History Office, the Bancroft Library, University of California, Berkeley, 1988.

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*Salk’s basic hypothesis is false. As early as the poliomyelitis congress in Rome in September 1954, Swedish observations were put forward concerning virus inactivation with formaldehyde which showed that the inactivation curve is not a straight line but shows a continuous curvature. The phenomenon has nothing to do with the presence of aggregates; filtration does not in any way affect the shape of the curve.*⁸³

There was yet another factor in the virulence of the 1955 vaccine. The vaccine used in the 1954 trial contained Merthiolate, a mercury compound that had a virucidal (virus-killing action) effect. Because Jonas Salk was disappointed in the antibody-stimulating effect that the 1954 field trial demonstrated, the Merthiolate was removed in the 1955 vaccine to induce a faster antibody response in vaccine recipients. Not only was the 1955 vaccine not the same celebrated vaccine that was trialed in 1954, it was also riddled with live viruses of a highly neurovirulent nature—the Mahoney strain.

Between April 17 and June 30, 1955, 260 poliomyelitis cases were documented after inoculation of about 400,000 persons with the Cutter vaccine. Ninety-four cases were among vaccinees, 126 among family contacts, and 40 among community contacts. An estimate of the case-infection ratio is in the range of 1 case per 100 to 600 injected infections.⁸⁴

It is a documented fact that household adult contacts did contract polio—secondarily—from the vaccine,⁸⁵ and some became severely paralyzed. Thirteen household contacts required iron lungs, and five died. There were documented cases where infants received the

⁸³ Sven Gard, “Prophylactic Vaccination Against Poliomyelitis,” *Svenska Läkartidningen (Swedish Physician’s Journal)*, vol. 53, no. 121(nr3)a, January 1956 (3rd week). Translated from Swedish and distributed by the Oak Park Health Department, Oak Park, Illinois. Ref. p. 8.

⁸⁴ N. Nathanson and A. Langmuir, “The Cutter Incident,” *American Journal of Public Hygiene*, vol. 78, no. 1, 1963, pp. 29–60.

⁸⁵ *Ibid.*, pp. 16–81.

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vaccine injection, shed live virulent virus in the stool, and never got sick. But their mothers became very ill, and so did neighbors. A conservative report revealed that 39 friends and neighbors of children who received the Cutter vaccine were paralyzed. Many more were infected to lesser degrees.

The newly formed Polio Surveillance Unit (PSU) did not capture all the cases that developed from the domino effect of this grand mishap. The reason is that they had strict cutoff dates beyond which any reported polio was considered not to be from the vaccine.

Paul Offit summarized the estimate of known damage:

In the end, at least 220,000 people were infected with live polio virus contained in Cutter’s vaccine; 70,000 developed muscle weakness, 164 were severely paralyzed, 10 were killed. Seventy five percent of Cutter’s victims were paralyzed for the rest of their lives.⁸⁶

Anyone infected with live vaccine virus, whether symptomatic or not, was readily contagious and capable of spreading the dangerous Mahoney virus strain in their communities. It is evident that the viral ecosystem was forever altered by the introduction of polio vaccines.

Looking beyond Cutter

Here is some of what Paul Offit left out of his book. Even though Cutter Laboratories took the fall for the 1955 disaster, all manufacturers had difficulty killing the virus in their vaccines before and after the disaster.^{87,88} Cutter was not the only manufacturer documented to have produced live virus vaccine that was injected into children and caused paralysis. In 1990, after decades of

⁸⁶ Paul Offit, MD, *The Cutter Incident*, Yale University Press, 2005, p. 89.

⁸⁷ L. Scheele and J. Shannon, *Technical Report, Public Health Implications in a Program of Vaccination Against Poliomyelitis*, June 7, 1955, p. 7. Digital copy is in the authors’ possession.

⁸⁸ Richard Carter, *Breakthrough: The Saga of Jonas Salk*, Trident Press, New York, 1965, p. 324.

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information concealment, the Freedom of Information Act led to the release of documents that proved Wyeth also produced a paralyzing vaccine.^{89,90}

Wyeth and Cutter are thought today to have been the only companies that produced live virus vaccine; however, all the vaccine companies could have released active vaccine virus because the “minimum licensing requirements”⁹¹ set by the US Department of Health, Education, and Welfare were not met by any pharmaceutical company. The initial minimum licensing requirements established April 12, 1955, stated that “all virus infectivity is destroyed with certainty.”⁹² According to later documents and courtroom testimonies, this definition was not followed, and manufacturers were never held to such standards. In 1992 Dr. Neil Nathanson stated:

*Minimum requirements were meant to state the assurance that the final vaccine contained less than 5 tissue culture infectious doses per liter . . . in other words to assure that there would be less than one chance in 100,000 that the vaccine would contain one paralytogenic dose per 1,000 human doses of vaccine.*⁹³

Does this sound like insurance that “all infectivity was destroyed with certainty?” A Tissue Culture Infective Dose (TCID) is a mathematical calculation. According to late virologist Dr. Wendell

⁸⁹ H. Ratner, “An Untold Vaccine Story,” *Child and Family*, vol. 21, no. 3, 1993, pp. 253–263.

⁹⁰ A. Langmuir and N. Nathanson, “The Wyeth Problem,” Prepared by the Poliomyelitis Surveillance Unit, Epidemiology Branch of the Communicable Disease Center, Department of Health Education and Welfare, September 6, 1955.

⁹¹ Minimum requirements involved extra vaccine filtration steps and tests on cortisone-treated primates.

⁹² US Department of Health, Education, and Welfare, Public Health Service, Minimum requirements, first revision, April 12, 1955, p. 2.

⁹³ N. Nathanson, Declaration. *Mosley vs Health and Human Services*, p. 8.

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Stanley, a single TCID contained up to 30 poliovirus particles, and any one of them could have caused poliomyelitis.⁹⁴

There are a couple of problems associated with risk calculation using TCIDs. First, the cutoff choice of less than 5 TCIDs was arbitrary. Second, there is an assumption that all virions (complete, infectious virus particles) would be distributed evenly and necessarily be included in any test sample. Remember the problem with particulate clumping? According to statistician Dr. Paul Meier, if each virion injected did cause a case of paralytic poliomyelitis, the injection of 1 milliliter of vaccine where the batches contain 5 TCID per liter could cause up to 500 cases per 100,000 vaccinated.⁹⁵

The reason there was not much more paralysis among the vaccinated was because, as was already known, 80–90 percent of the childhood population at the time was already naturally immune to at least one strain of poliovirus.⁹⁶In his book, Dr. John Paul estimated that 80 percent would have had some pre-vaccine antibody to the polio-virus.⁹⁷ Anyone who was immune naturally would also, fortunately, have been immune to the corresponding vaccine virus.

You may be wondering how this information was concealed from the public for nearly fifty years. Congressman Percy Priest ordered and chaired a full investigation of the vaccine controversy. He admitted in 1956 that:

... in the previous year (1955) many responsible persons had felt that the public should be spared the ordeal of “knowledge about controversy.” If word ever got out that the Public

⁹⁴ *Gottsdanker v. Cutter Laboratories* (1960) 82 Cal. App. 2d 602 (2869:902870:3; 2871: 14-17) pp. 65–69.

⁹⁵ P. Meier, “Safety Testing of Poliomyelitis Vaccine,” *Science*, vol. 125, May 31, 1957, pp. 1067–1071.

⁹⁶ T. Francis et al., *Evaluation of the 1954 Field Trial of Poliomyelitis Vaccine: Final Report*, Poliomyelitis Vaccine Evaluation Center, University of Michigan, Ann Arbor, April 1957, p. 152.

⁹⁷ J. R. Paul, *A History of Poliomyelitis*, Yale University Press, New Haven, Connecticut, 1971, pp. 335–339, 427.

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Health Service had actually done something damaging to the health of the American people, the consequences would be terrible. . . . We felt that no lasting good could come to science or the public if the Public Health Services were discredited.⁹⁸

So much for evidence-based medicine and scientific truth. Instead of discrediting the PSU, the decision was made, after some deliberation, to leave Wyeth’s paralyzing vaccine on the market, place the whole blame on Cutter, and ignore the ongoing problem with live viruses in the vaccines that persisted even after the revisions for safer manufacture were carried out. Only Cutter’s vaccines were recalled. All other manufacturers’ vaccines released in the 1950s were sold and injected into America’s children. Millions of vaccines were also exported all around the world.

There were other more insidious and unaddressed problems with the Salk vaccine. Once a vaccine passed the minimum requirement tests showing that all the virus was theoretically killed, **the virus was found to have resurrected on the shelves weeks or months later**, even after the new safety standards were put in place in 1956.

*Dr. S Stephen Chapman . . . reported . . . he had centrifuged the vaccine and had obtained live virus, “more than we theoretically ever could have anticipated having . . . this brings up the problem of reactivation of the so called dead vaccine.”*⁹⁹

The most likely explanation for this apparent resurrection is that the safety testing didn’t detect small amounts of live virus, and without Merthiolate in the vaccine the virus was able to replicate. In 1954 Salk’s trial vaccine contained a mercury compound patented as Merthiolate, which was used to prevent mold from growing and to prolong the shelf life. When it was obvious that the vaccine was not

⁹⁸ Richard Carter, *Breakthrough: The Saga of Jonas Salk*, Trident Press, New York, 1965, pp. 318–319.

⁹⁹ Ratner, Herbert, “A Premature Salk Vaccine, April 19, 1956” *Child and Family*, vol. 20, 1988, pp. 255–263.

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as antigenic as hoped, a decision was made to remove the mercury compound in the 1955 manufacture. Salk never wanted the mercury in the first place and protested that it ruined the vaccine, making the Mahoney strain less antigenic.

Swedish scientists, after the Cutter disaster in 1955, began to test some of their vaccine that was waiting to be dispensed. They did this in response to the alarming newscoming from the United States about vaccine-induced paralysis. Tests were done on batches of vaccine that were previously shown to be free of active virus. Upon repeat testing, 30 percent of the vaccine samples showed the presence of active virus.¹⁰⁰

The fundamental problem was that, although required safety testing was done with the hope of releasing only safe vaccines, the foundational principles with which the vaccine was manufactured were highly flawed from the beginning. Salk’s hypothesis was false. This problem was never fully addressed. According to expert virologist Dr. Sven Gard, a fundamental property of the virus that had to do with its structure was overlooked.¹⁰¹ **Dr. Gard also stated that vaccination in the United States caused as many cases of poliomyelitis as it prevented in 1955.**¹⁰²

¹⁰⁰ H. Eyer et al., *An Evaluation of the Protective Immunization Against Poliomyelitis, Report of the Scientific Committee, Social Medicine and Hygeine*, 1956, p. 13. This 102-page document with 22 corresponding graphs is a translation of a larger 492-page German report from an article that appeared in the *Munch Med. Wochenschr* April 6, 1956. A copy of English translation is in the authors’ possession.

¹⁰¹ Sven Gard, “Prophylactic Vaccination Against Poliomyelitis, Translated for and distributed by the Oak Park Health Department, Oak Park, Illinois,” *Swedish Physician’s Journal*, January 1956. Ref. p. 8 of translation. Provided by Herbert Ratner, MD. Paper in authors’ possession.

¹⁰² Sven Gard, “Prophylactic Vaccination Against Poliomyelitis,” *Svenska Läkartidningen (Swedish Physician’s Journal)*, vol. 53, no. 121(nr3)a, January 1956 (3rd week), Translated from Swedish and distributed by the Oak Park Health Department, Oak Park, Illinois. Ref. p. 6 of translation. Courtesy of the estate of Herbert Ratner. Copy in authors’ possession.

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According to Dr. Wendell Stanley, the formaldehyde engendereda “tanning” effect upon the outer coating of the virus, but potentially left the infectious internal portion of the virus intact.

The outer protein portion of the virus is not infective, and yet it is this portion which produces antibodies. In making a vaccine, the effort is centered on trying to remove the virus activity contained in the nucleic acid core while at the same time keeping the protein unchanged so that it may produce antibody. This is complicated . . . This results in a “tanning” effect as leather is tanned, making it more resistant to anything attempting to pass through it . . . there is an intermediate stage [inthe inactivation] which is reversible, so that there is no viral activity shown by any of the safety tests and yet after further chemical treatment, activity can be gained from this same material. . . . Virus can be held for many days, and in fact may years and still be able to be reactivated at a subsequent time . . . formaldehyde comes off the protein. The partially tanned virus may be altered . . . will not give a positive test at the 14th day but would prove infectious at the end of three or four weeks. . . . In addition the virus in a vaccinal suspension is not homogenous but contains viruses which are slightly different in character and have different susceptibility and ability to resist activation.¹⁰³

Safety must be built into the method itself so that it automatically leads to a product of a well-defined quality. Instead of creating a reliably killed vaccine in the 1950s, companies had to rely upon post-manufacture safety tests alone for a vaccine that was known by all involved to consistently have some degree of live virus particles. Dr. Edwin Lennette, director of the California State Department of Healthstated that, in general, vaccines could test negative in the lab and in test animals, yet behave differently in humans:

¹⁰³ Gottsdanker v. Cutter Laboratories (1960) 82 Cal.App.2d 602 (2869:902870:3; 2871: 14–17).

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*You just put in some formaldehyde or whatever and inactivate the virus, and you do a few tests, and if nothing happens in the animal, then you think, well, we've got a vaccine. But you put it into man, who is the ultimate susceptible animal, and then something else goes wrong, and you've got a problem.*¹⁰⁴

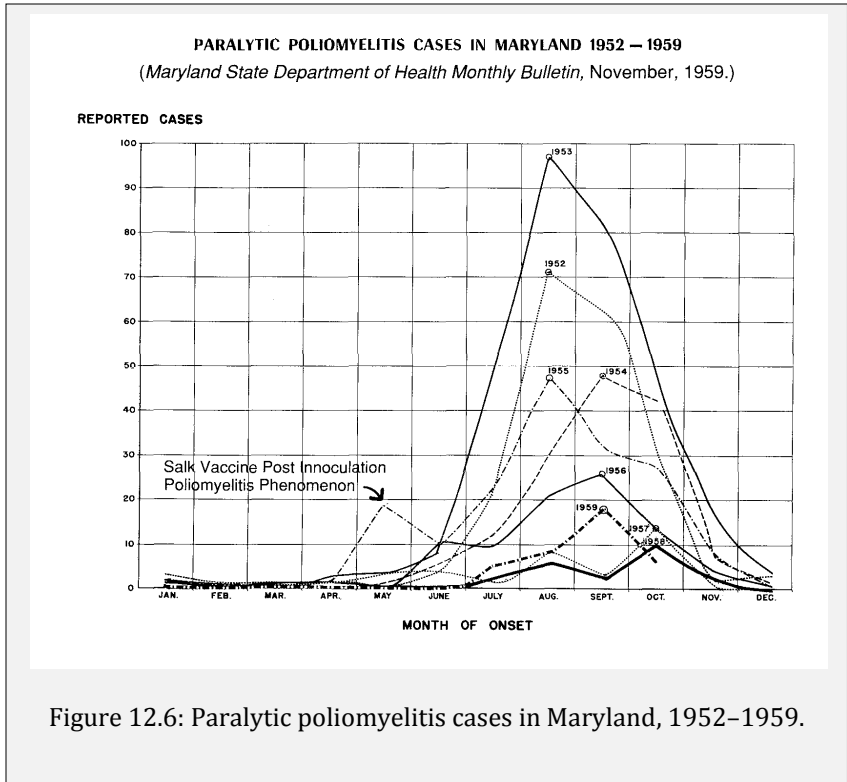
In subsequent years, instead of removing the dangerous Mahoney strain, American manufacturers continued releasing vaccines that were safer but far less antigenic. They tended to the problem, not by addressing the fundamental flaw, but by adding more filtrations of the vaccine. Dr. Gard said:

I am now quite confident that the whole philosophy behind the Salk vaccine . . . is wrong, indeed. When repeated filtrations are applied for removal of “aggregates” one is only hunting ghosts. The effect of filtration is nothing but a gradual removal of virus, live and dead alike. It could just as well be substituted by plain dilution of the vaccine.¹⁰⁵

¹⁰⁴ Edwin H. Lennette, *Pioneer of Diagnostic Virology with the California Department of Public Health*, an oral history conducted in 1982, 1983, and 1986 by Sally Hughes, Regional Oral History Office, the Bancroft Library, University of California, Berkeley, 1988.

¹⁰⁵ Herbert A. Ratner, “A Premature Salk Vaccine, April 19, 1956,” *Child and Family*, vol. 20, 1988, pp. 255–263. Referencing personal letter from Dr. Gard, a copy of which is in the authors’ possession.

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Even with such effective viral dilution of the vaccine and four revisions to the minimum requirements set forth by the government for producing safe vaccines in 1955, there was ongoing evidence that vaccine-induced infections continued. There were pre-season polio (as in vaccine-provoked polio) peaks that were not present before the vaccine years. As you can see in Figure 12.6, 1955 has the largest peak, but 1956–1959 also had pre-season increases that were not present in 1954 or earlier. As the years progressed, these peaks were smaller, due to filtering out both live and inactivated virus, so the vaccines had much lower viral levels.

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Alengthy German Scientific Committee report from 1956¹⁰⁶ contains graphs depicting the 1955 preseason peak in Wisconsin, Illinois, Massachusetts, Georgia, Pennsylvania, Colorado, Virginia, Louisiana, Nevada, Oregon, and Idaho.

Not all the vaccine-induced cases were accepted by the Polio Surveillance Unit. Many paralyzed recipients were denied validation and compensation for illness that occurred after the vaccine was given in 1955. The requirements for so-called accepted cases of vaccine-associated polio were more stringent than the requirements for reporting polio in nonvaccinated individuals.^{107,108}

For example, only cases that began in the inoculated limb were accepted by the PSU, and only within a very narrow timeframe. The PSU used norms that historically were not so restrictive. Thus, only first-generation infection cases were reported and only if they met the stringent laboratory validation criteria. This would have excluded chain reaction cases that broke out later.

The Salk vaccine was anything but a lifesaver. It was known from the start to be trouble, and trouble it was. Wild poliovirus was never a lone or major cause of poliomyelitis. But even if it was, the Salk vaccine could not possibly have been a solution to ridding the world of polio. Nonetheless, Jonas Salk and his vaccine have been forever cast into heroism in the archives of vaccine mythology.

¹⁰⁶ H. Eyer et al., *An Evaluation of the Protective Immunization Against Poliomyelitis*, Report of the Scientific Committee, Social Medicine and Hygiene, 1956. This 102-page document with 22 corresponding graphs is a translation of a larger 492-page German report from an article that appeared in the *Munch Med Wochenschr* April 6, 1956. A copy of English translation is in the authors' possession.

¹⁰⁷ *Poliomyelitis Trends, 1958*, Dominion Bureau of Statistics, Ottawa, Canada, June 29, 1959, p. 1m.

¹⁰⁸ Herbert Ratner, Declaration of Herbert Ratner, Diane Lynn Armbrust Mosley vs. Secretary of the Department of Health and Human Services, October 1, 1992.

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Monkey virus contamination

Vaccines manufactured using monkey kidneys up into the 1980s have been definitively noted¹⁰⁹ to contain a carcinogenic monkey virus that some medical researchers believe can result in cancer in a portion of the millions who were given them.¹¹⁰ Simian virus number 40 (SV40) is a monkey virus that has been found in several types of human cancers, including lung mesotheliomas, several types of brain tumors, and bone, breast, colon, and kidney tumors.¹¹¹ Unfortunately, the controversy over the percentage of tumor specimens containing SV40 DNA and proteins has paralyzed the research field. Because of financial and political conflicts of interest, the research necessary to firmly validate the vaccine-virus association will probably never be done.

*This controversy was magnified by the legal implications of associating the production and distribution of contaminated poliovaccines to the development of human mesotheliomas and brain tumors. Study sections reviewers have been unwilling to support SV40 research citing the need to first address the “controversy,” yet without funding it is impossible to conduct studies to address controversial findings.*¹¹²

SV40 is known to exist in cancerous tissue, but not in surrounding healthy tissue,¹¹³ to cause extensive genetic damage in vitro (cell

¹⁰⁹ Rochelle Cutrone, John Lednicky, Glynis Dunn, et al., “Some Oral Poliovirus Vaccines Were Contaminated with Infectious SV40 After 1961,” *Cancer Research*, vol. 65, no. 22, November 15, 2005, pp. 10273–10279.

¹¹⁰ Paola Rizzo, Ilaria Di Resta, Amy Powers, Herbert Ratner, and Michele Carbone, “Unique Strains of SV40 in Commercial Poliovaccines from 1955 Not Readily Identifiable with Current Testing for SV40 Infection,” *Cancer Research*, vol. 59, no. 24, December 15, 1999, pp. 6103–6108.

¹¹¹ F. Qi et al., “Simian Virus 40 Transformation, Malignant Mesothelioma and Brain Tumors,” *Expert Review of Respiratory Medicine*, vol. 5, October 2011, pp. 683–697.

¹¹² Ibid.

¹¹³ M. Carbone, R. A. Kratzke, and J. R. Testa, “The Pathogenesis of Mesothelioma,” *Seminars in Oncology*, vol. 29, February 2002, pp. 2–17.

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cultures) and to induce tumors when injected into volunteers¹¹⁴ and rodents. However, it is not considered scientifically valid to implicate the contaminating SV40 viruses with these human tumors.

*An association has been found between SV40 and certain types of cancer in humans. However, though the virus or its DNA have been found in certain types of cancer, it has not been determined that SV40 causes these cancers. Finding that two events are “associated” is not the same as establishing that one event caused the other.*¹¹⁵

Certain scientists who have had careers in polio and SV40 research know firsthand that inconvenient scientific truths can be abrogated by industry and politics. Two of the world’s most respected scientists in the SV40 realm, Dr. Harvey Pass and Dr. Michele Carbone, commented on how science was censored.

*I [Michele Carbone] wanted to have a press statement . . . and to be able to talk to the media if contacted by them. I also believe that the public and the media have the right to ask us any question they wish once our work has been accepted by a peer-review journal and that scientists should not decide what the media should or should not know . . . [Dr. Levine] told me that if I, or Harvey, talked to the press, against his wishes, we would be “punished.” . . . Pass was shocked at the uproar, particularly the threat. “I didn’t think you got punished for science.”*¹¹⁶

¹¹⁴ F. Jensen, H. Koprowski, J. S. Pagano, J. Ponten, and R. C. Ravdin, “Autologous and Homologous Implantation of Human Cells Transformed in Vitro by SV40,” *Journal of the National Cancer Institute*, vol. 32, 1964, pp. 917–932.

¹¹⁵ Centers for Disease Control and Prevention, *Vaccine Safety: Frequently Asked Questions About Cancer, Simian Virus 40 (SV40), and Polio Vaccine*, 2012.

¹¹⁶ D. Bookchin and J. Schumacher, *The Virus and the Vaccine*, St. Martin’s, Griffin, New York, 2004, p. 163.

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There are still the rare truth seekers, like attorney Stanley Kops, who continue to voice opposition to the claims that SV40 is no longer an issue with vaccines.

The news article by Nancy J. Nelson repeats the current scientific dogma that simian virus 40 (SV40) was removed from all oral polio vaccine sold and administered in the United States. In a recent article, however, I have challenged this accepted “fact” based on legal documents and the absence of test results from at least one of the principal vaccine manufacturers, Lederle. As noted in that article, internal Lederle documents indicate that the company has not been able to document that it tested all vaccine seeds to confirm the absence of SV40 contamination.

Every scientist who is attempting to determine the role of SV40 as a cause of cancer in humans and every news reporter who is interested in this issue should demand all of the records of both the government and the vaccine manufacturers so that there can be a full scientific and independent investigation as to whether there was full compliance with the removal of SV40 from all oral polio vaccine used in the United States from 1962 until 2000.¹¹⁷

How a virus dubbed “the perfect war machine”¹¹⁸ by Dr. Carbone because it affects at least four major cellular mechanisms that either promote cancer or interfere with cancer-fighting defenses, could be impacting countries that continue using oral polio vaccines by the ton today, is anyone’s guess. How much of the abrupt rise in human cancer rates since the introduction of monkey products into the human population is due to SV40 will also remain uncertain due to a lack of precise research.

¹¹⁷ S. Kops, “Re: Debate on the Link Between SV40 and Human Cancer Continues,” *Journal of the National Cancer Institute*, vol. 94, no. 3, February 6, 2002, pp. 229–230.

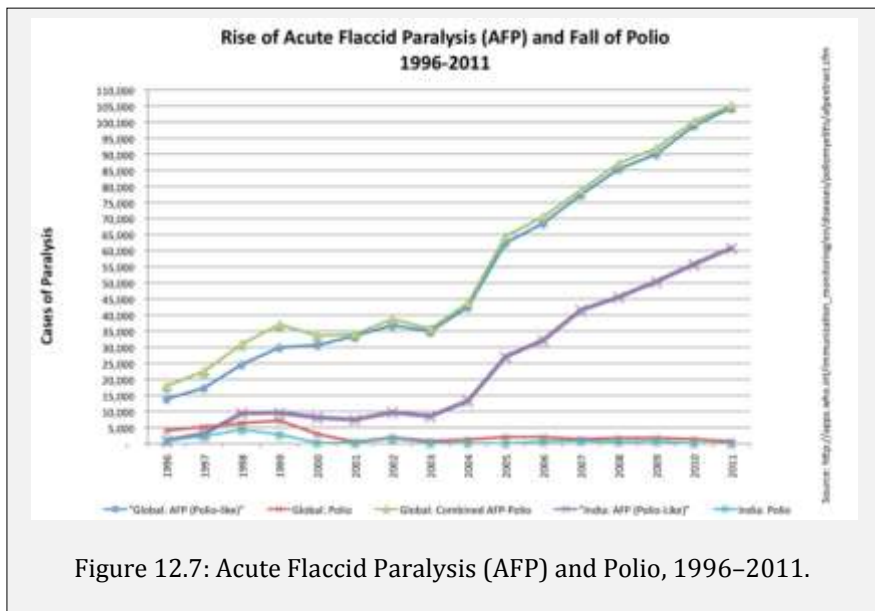
¹¹⁸ D. Bookchin and J. Schumacher, *The Virus and the Vaccine*, St. Martin’s, Griffin, New York, 2004.

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Monkeys are still used in polio vaccine production today. According to Stanley Kops’ allegations, SV40 was and still is a potential risk in both the OPV and IPV. The IPV used in the developed world is still treated with formaldehyde, but SV40 has been known since 1961 to survive formaldehyde beyond the usual 12-day minimum.¹¹⁹Vaccine manufacturers today cite a minimum of 12 days of formaldehyde treatment.¹²⁰

History repeats itself

In India today, as the World Health Organization (WHO) tracks polio during the vaccination campaigns, reports of paralytic cases associated with wild-type poliovirus have declined, and Acute Flaccid Paralysis (AFP) has increased annually, reaching 60,000 new cases in 2011.



¹¹⁹ P. Gerber, G. A. Hottle, and R. Grubbs, “Inactivation of Vacuolating Virus (SV40) by Formaldehyde,” *Proceedings of the Society for Experimental Biology and Medicine*, vol. 108, October 1961, pp. 205–209.

¹²⁰ Sanofi Pasteur, *Poliovirus Vaccine Inactivated, IPOL*, October 15, 2012, www.fda.gov/downloads/biologicsbloodvaccines/vaccines/approved/products/ucm133479, accessed June 17, 2013.

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The causes of AFP that have been identified are as follows:

*Poliomyelitis, non-polio enterovirus, vaccine-associated poliomyelitis (which can include polio vaccines), rabies virus, varicella zoster virus, Japanese encephalitis virus, Guillain-Barré syndrome, cytomegalovirus, sciatic neuritis from injection, transverse myelitis, epidural abscess, spinal cord compression, exotoxin of corynebacterium diphtheriae, toxin of clostridium botulinum, Karwinskia, tick bite paralysis, Lyme borreliosis, myasthenia gravis, polymyositis autoimmune, viral myositis, trichinosis, toxic myopathies among others.*¹²¹

In spite of (or perhaps because of) the aggressive Oral Polio Vaccine (OPV) campaigns in India, there has been a steepascent in Acute Flaccid Paralysis diagnoses. Nonetheless, the WHO and its sister organizations celebrate because the number of documented cases of *wild* poliovirus-associated paralysis has declined.

It just so happens that DDT is still heavily used in India. Despite the well-documented connection between poliomyelitis and DDT^{122,123} symptoms, including anterior horn spinal cord damage, respiratory paralysis, muscle spasm, and weakness, multi-billion dollar polio eradication campaigns march on. Often, an Indian child is vaccinated 15 times (or more) with live vaccine by age five.

In fact, at the end of 2005, children under 5 years old were reported to have received on average 15 doses of tOPV [trivalent OPV] in UP and Bihar, compared with 10 in the rest of

¹²¹ Marx et al., “Differential Diagnosis of Acute Flaccid Paralysis,” *Epidemiologic Reviews*, vol. 22, 2000, pp. 298–316.

¹²² F. Burgess and G. R. Cameron, “The Toxicity of D.D.T.,” *British Medical Journal*, June 23, 1945, vol. 1, pp. 865–871.

¹²³ M. Biskind, “DDT Poisoning and the Elusive ‘Virus X’: A New Cause for Gastroenteritis,” *American Journal of Digestive Diseases*, vol. 16, no. 3, 1949, pp. 79–84. Paper contains 13 references to the effect of DDT and poliomyelitis symptoms.

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*India, and only 4% of children were reported to have received fewer than 3 doses, of whom 90% were under 6 months old.*¹²⁴

Pulse Polio is an immunization campaign established by the government of India beginning in 1995 to eradicate poliomyelitis by vaccinating all children under the age of five against poliovirus. The initial goal for India to be free of polio by 2005 was not met. The Pulse program involves setting up vaccine booths in all parts of the country; arranging employees, volunteers, and vaccines; vaccinating children with OPV on National Immunization Days; and identifying children missing from the immunization process.

A major oversight on the part of the press and the medical establishment as they observe the WHO’s version of history is that massive “pulse” vaccination campaigns have done nothing to eliminate childhood paralysis and, in fact, there is strong evidence pointing to the likelihood that experimental polio vaccination is related to the sharp rise in AFP. It has been reported in the *Lancet*¹²⁵ that the incidence of AFP, especially non-polio **AFP, increased drastically in India after an experimental, high-potency polio vaccine was introduced**. Worse still is that children identified with non-polio AFP are at more than twice the risk of dying than those with wild polio infection.¹²⁶ Isn’t vaccination really about eliminating paralysis . . . or is it simply to replace wild virus with a vaccine virus regardless of the outcome?

Non-polio AFP rate increases in proportion to the number of polio vaccine doses received in each area. . . . Nationally, the non-polio AFP rate is now 12 times higher than expected. In the states of Uttar Pradesh (UP) and Bihar, which have pulse polio rounds nearly every month, the non-polio

¹²⁴ Grassly et al., “New Strategies for Elimination of Polio from India,” *Science*, vol. 314, November 17, 2006, pp. 1150–1153.

¹²⁵ J. Puliyeel, C. Sathyamala, and D. Banerji, “Protective Efficacy of a Mono-valent Oral Type 1 Poliovirus Vaccine,” *Lancet*, vol. 370, 2007, pp. 129–130.

¹²⁶ C. Sathyamala., “Polio Eradication Programme in India,” *The Indian Journal of Medical Research*, vol. 125, 2007, pp. 695–696.

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AFPrate is 25- and 35-fold higher than the international norms. . . . The non-polio AFP rate during the year best correlates to the cumulative doses received in the previous three years . . . Association of the non-polio AFP rate with OPV doses received in 2009 was 41.9%. Adding up doses received from 2007 increased the association ($R^2 = 55.6\%$ $p < 0.001$).¹²⁷

The WHO says that wild polio is declining in India, but will it really be eradicated? It could still circulate in the future, just as it could still be circulating in the United States today. In order to say it is eradicated, they would have to examine the stools of everyone more than just once. However, they only examine the stool of those who develop paralysis—most of whom have been vaccinated with live vaccine virus, which often displaces wild virus in the intestine. Could the increase in AFP in India be the result of the release of so much vaccine virus into the population? Are these people getting more polio paralysis as a result of natural recombination and mutation?

Wild polioviruses, vaccine polioviruses, and neurovirulent Coxsackie viruses can all interact, recombine, and evolve into seriously neurovirulent entities.^{128,129,130,131} Why would a vaccine virus be

¹²⁷ N. Vashisht and J. Puliyel, “Polio Programme: Let Us Declare Victory and Move On,” *Indian Journal of Medical Ethics*, vol. 9, April–June 2012, pp. 114–117.

¹²⁸ S. Jegouic et al., “Recombination Between Polioviruses and Co-Circulating Coxsackie A Viruses: A Role in the Emergence of Pathogenic Vaccine-Derived Polioviruses,” *PLoS Pathology*, vol. 5, no. 5, May 2009.

¹²⁹ R. Crainic et al., “Measles and Poliomyelitis: Vaccine, Immunization, and Control,” in *Natural Evolution of Oral Vaccine Poliovirus Strains*, pp. 371–390.

¹³⁰ S. Guillot et al., “Natural Genetic Exchanges Between Vaccine and Wild Poliovirus Strains in Humans,” *Journal of Virology*, vol. 74, no. 18, September 2000, pp. 8434–8438.

¹³¹ M. M. Georgescu, F. Delpyroux, and R. Crainic, “Tripartite Genome Organization of a Natural Type 2 Vaccine/Nonvaccine Recombinant Poliovirus,” *Journal of General Virology*, vol. 76, September 1995, pp. 2343–2348.

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stable and not follow the laws of nature, which involve the clear likelihood of recombination?

The response to the rise in AFP in India by the WHO and the Global Alliance for Vaccines and Immunisation¹³²(GAVI) has been to ramp up the oral polio vaccination campaigns in recent years. Now some children are reported to have received 32 vaccines by five years of age. In the past, there was never such an aggressive effort to inoculate children up to 30 times *for one disease* by their fifth birthday.

*At a vaccinators’ meeting in Sultangunj Referral Hospital held Tuesday, supervisors reported a “new” **resistance coming from the “educated middle class people”** who were getting tired of several rounds of immunisation: one family claimed that their **five year old child had received pulse polio vaccination 32 times.***¹³³

Just what are GAVI members trying to accomplish? Does it look like the sustainable health and betterment of India’s people are the main goals? Dr. V. I. Agol commented in *Nature* that vaccination against poliomyelitis might have to continue indefinitely.¹³⁴

¹³² GAVI members include WHO, the World Bank, Unicef, and the Bill and Melinda Gates Foundation, www.gavialliance.org.

¹³³ “Multiple Doses of Pulse Polio Vaccine Irritate People,” *Times of India*, August 25, 2002.

¹³⁴ V. I. Agol, “Don’t Drop Current Vaccine Until We Have New Ones,” *Nature*, June 16, 2005.

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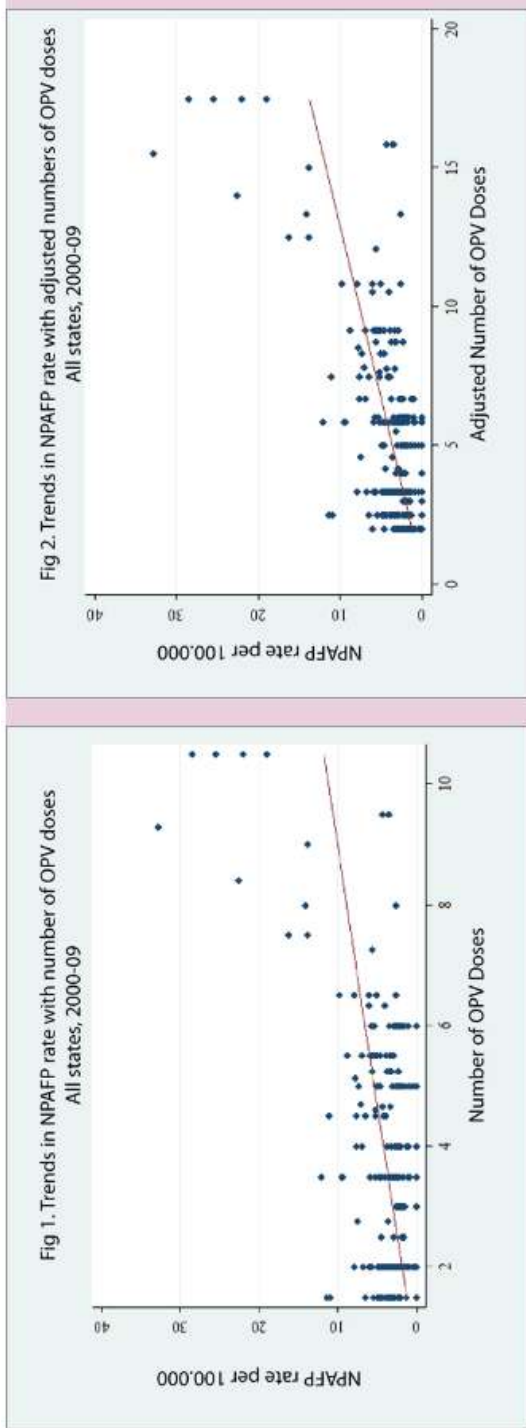


Figure 12.8: Non-Polio Acute Flaccid Paralysis (NPAFP) correlation to Oral Polio Vaccine (OPV).

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The charts (Figure 12.7) on the previous page unequivocally reveal how the rate of AFP has risen with the number of OPV doses. Given all the information available to scientists and politicians today and a century of polio literature to reflect upon, one must surely wonder . . . what in the world are they thinking? The rising numbers of paralyzed children in India deserve a better explanation than “It is for the greater good,” because clearly it is not.¹³⁵

Conclusion

By now it should be obvious that there was more to the “polio” story than a crippling virus and a world that was saved by a vaccine. Isn’t it strange that the reasoning behind polio epidemics in the United States in the 1940s was increased societal hygiene?^{136,137,138} Filth, back then, was thought to be protective against polio! The explanation given was that babies in areas with better hygiene (unlike the native people who were known to be immune without developing poliomyelitis) were not exposed to wild virus early enough due to societal cleanliness and therefore did not develop early natural immunity.

Today India is told that paralytic poliovirus infections are a result of poor societal hygiene. Such double speak demonstrates how the tenet changes to accommodate the vaccine agenda and deny the true causes of paralysis.

As of today, no programs have been funded to investigate or validate the scientific findings that implicate associations between chemicals like DDT and arsenic and the syndrome of poliomyelitis. Instead, the world is reliant upon blemished vintage research that was funded by the major medico-political powers of the first half of the 20th century.

The National Foundation for Infantile Paralysis was overseen by the major medical monopoly, the Rockefeller Institute. Vaccination continues as the sole intervention for the perceived problem of

¹³⁵ “National Polio Surveillance India Data 2000–2010, NPSF Polio Surveillance Data on Acute Flaccid Paralysis (AFP) and Non-Polio AFP and Demographic Data,” <http://jacob.puliyel.com/download.php?id=248>.

¹³⁶ Nidia H. De Jesus, “Epidemics to Eradication: The Modern History of Poliomyelitis,” *Virology Journal*, 2007, 470.

¹³⁷ “Polio,” Massachusetts Society for Medical Research, Inc., 2004.

¹³⁸ Albert B. Sabin, “Paralytic Consequences of Poliomyelitis Infection in Different Parts of the World and in Different Population Groups,” *American Journal of Public Health*, vol. 41, October 1951.

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poliomyelitis in India and other undeveloped countries, even in the face of vaccine-induced paralysis, vaccine virus mutations, and obvious failures. When vaccine programs don't live up to their promises, the blame is always placed on the unvaccinated, or a new angle is drawn to the tune of “five vaccines per child may not be enough.” By sleight of hand—changing the diagnosis of old-time polio to AFP—any ongoing paralysis will be covered while the dimes continue to roll in.

In addition to the rise in Acute Flaccid Paralysis that correlates with rising oral polio vaccine dosing in India, there are numerous reports of vaccine viruses mutating to virulence, causing polio outbreaks in China, Nigeria, and India. As always, the finger is pointed at under-vaccinated populations rather than at the vaccine itself or the myriad other causes of viral mutation.

*Apart from the resilience of circulating wild-type viruses, major problems have emerged as a result of intrinsic properties of the OPV. It has the propensity to escape its designated role as a protecting immunogen by circulating in poorly immunized populations, thereby evolving into highly neurovirulent poliovirus strains **after recombination with other enteroviruses** (Kew et al, 2005; P. Jiang, J.A.J. Faase, A.E. Gorbalenya and E. Wimmer, unpublished data). This independent occurrence in different parts of the world causes yearly outbreaks of poliomyelitis.¹³⁹*

We often hear that OPV circulating in poorly immunized populations is wonderful because the unvaccinated get the benefit. But OPV vaccines will always be able to recombine with enteroviruses no matter how highly vaccinated the population, and dangerous recombination viruses that cause paralysis will not be called “polio.” This is one way that a mountain of new AFP cases builds, while GAVI and WHO celebrate eradication of polio.

Today the GAVI deserves criticism and examination of its goals. This is a time when the developing world needs improved nutrition, clean and chemical-free water, sustainable farms with clean soil, and the luxury of being free from war, famine, and spiritual persecution. If philanthropists want to go down in history as truly making the world a better place, is \$10 billion best spent on vaccines?

¹³⁹ E. Wimmer, “The Test-Tube Synthesis of a Chemical Called Poliovirus: The Simple Synthesis of a Virus Has Far-Reaching Societal Implications,” *European Molecular Biology Organization Report*, vol. 7, no. SI, July 2006, pp. S3–S9.

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The Bill and Melinda Gates Foundation will donate \$10 billion over the next decade to research new vaccines and bring them to the world's poorest countries. . . they said the money will produce higher immunization rates and aims to make sure that 90 percent of children are immunized against dangerous diseases such as diarrhea and pneumonia in poorer nations. “We must make this the decade of vaccines,” Bill Gates said in a statement. “Innovation will make it possible to save more children than ever before.”¹⁴⁰

Perhaps a \$10 billion decade of sustainable farming, nutrition, and sanitation would have a long-lasting impact on saving the children under discussion.

The WHO’s current strategy calls for cessation of oral polio vaccination three years after the last report of wild poliovirus-induced poliomyelitis.¹⁴¹

It is ironic that the vaccine on which the world has depended for polio eradication will itself become a risk to eradication once the transmission of wild poliovirus has been interrupted.¹⁴²

If WHO’s plan succeeds, the artificially immune herd stands to become the completely non-immune herd, as new children are born who have not been infected with wild-type viruses or even exposed to vaccine poliovirus. This **condition has never existed in human history**. Under these conditions, any reintroduction of poliovirus could be disastrous to this newly virgin population. The people of India, Pakistan, and Nigeria stand to become more vulnerable to viral reintroduction than any population ever before.

During the United States epidemics, roughly 50–80 percent of the population was naturally immune to at least one type of poliovirus. Wild poliovirus alone in healthy people was never a major threat. Natural herd immunity has always been protective (recall Xavante natives). In due time, India’s people will have the lowest level of herd protection ever, and in the face of continued DDT use, intramuscular injections of antibiotics, diets high in sugar and low in essential

¹⁴⁰ A. Higgins, “Bill Gates Makes \$10 Billion Vaccines Pledge,” *Huffington Post*, January 29, 2010.

¹⁴¹ WHO, “Framework for National Policy Makers in OPV-Using Countries,” 2005, www.polioeradication.org/Posteradication.aspx, accessed June 17, 2013.

¹⁴² D. L. Heymann et al., “A Vision of a World Without Polio: The OPV Cessation Strategy,” *Biologicals*, vol. 34, June 2006, pp. 75–79.

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vitamins, and stress, their susceptibility to paralytic disease is enormous.

If poliovirus is reintroduced into the toxic, unhealthy, immunologically naive population—from residual samples stored in laboratories, some of which are highly neurovirulent (recall 1916 New York City); circulating vaccine-derived polioviruses; or poliovirus that is chemically synthesized—the potential outcome is unfathomable.

Today children are forced to submit to vaccines because the WHO and others are just targeting wild poliovirus and not the problem of paralysis. Once this very shortsighted goal is met, there will undoubtedly be future trouble. The WHO knows this and already has considered the steps necessary to deal with the immunologically naive population if viral reintroduction occurs.¹⁴³

History books of the future may reflect upon a disaster with this conclusion: Wild poliovirus should have been left alone and the real sources of paralysis pursued and addressed.

¹⁴³ WHO, “Framework for National Policy Makers in OPV-Using Countries,” 2005, pp. 6–12.