Reevaluating the Etiology of Poliomyelitis

V. - 2023

What is polio?

The term poliomyelitis is a description of spinal pathology. The origin of the word comes from a composite of three Greek words; *polios* (gray), *myelos* (marrow), *itis* (inflammation), and denotes inflammation of the gray matter in the spinal cord.[1] The result of this inflammation is reflected by certain characteristic muscular symptoms, most notably paralysis.

The medical establishments definition of polio[2] refers to:

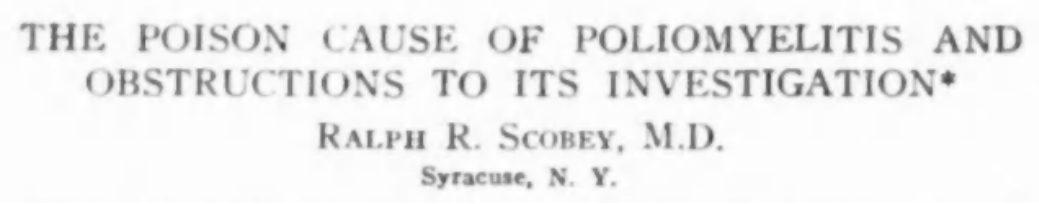
- Polio, or poliomyelitis, is a disabling and life-threatening disease caused by the **poliovirus**.
- The virus **spreads from person to person** and can infect a person's spinal cord, causing paralysis (can't move parts of the body).

It is important to distinguish between the symptoms allegedly caused by the 'poliovirus', and the disease referred to as poliomyelitis, since polio is considered a paralytic disease. The CDC[3] states:

> Note that "poliomyelitis" (or "polio" for short) is defined as the paralytic disease. So only people with the paralytic infection are considered to have the disease.

Paralysis is not a new phenomena

In 1952, Dr. Ralph R. Scobey MD, professor of clinical pediatrics and president of the Poliomyelitis Research Institute, Syracuse, N.Y. prepared a statement for the US Congress, entitled *The Poison Cause of Poliomyelitis and Obstructions To Its Investigation*.[4]



THE POISON CAUSE OF POLIOMYELITIS AND OBSTRUCTIONS TO ITS INVESTIGATION*

RALPH R. SCOBEY, M.D.

Syracuse, N. Y.

He states that the disease that we now know as poliomyelitis was not designated as such until about the middle of the 1800s. Prior to that, it was designated by many different names at various times and in different localities. The simple designations, paralysis, palsy and apoplexy, were some of the earliest names applied to what is now called poliomyelitis.

Scobey notes:

Paralysis, resulting from poisoning, has probably been known since the time of Hippocrates (460-437 B.C.). Boerhaave,[3] Ger-

Throughout the medical literature, we find that certain substances can cause paralysis, documented and collected in Dr. Scobey's statement.

In 1765 for example, a physician named Dr. Herman Boerhaave,[5] wrote:

frequently produces a palſey. We frequently find perſons rendered paralytic, by expoſing themſelves imprudently, to quickſilver diſperſed into vapours by the fire, as gilders, chemiſts, miners, &c. and perhaps there are other poiſons, which may produce the ſame diſeaſe, even externally applied. I ſhall only

In 1824, the English physician John Cooke[6] made similar observations:

Among the exciting causes of partial palsies we may reckon the poison of certain mineral substances, particularly of quicksilver, arsenic, and lead. The fumes of these metals, or the reception of them in solution into the stomach, have often produced paralytic affections.

Quicksilver is another word for mercury.

Scobey reviewed the literature and found many instances of poliomyelitis resulting from poisoning:

The foregoing reports indicate that poisons can cause poliomyelitis. It would appear that not any one poison in particular would be responsible for all cases of poliomyelitis but the effect of any one of several could produce the same ultimate result. When

He also noted a relationship of harvest to polio:

The material presented above appears clearly to indicate that there is a correlation between the harvesting of fruits and vegetables and epidemics of poliomyelitis. Epidemics of this disease

Why did epidemics of paralysis begin to rage in the late 1800s?

Sporadic cases of polio-like illnesses have been reported since the end of the 1700s. Late in the 1800s, larger and more frequent outbreaks of paralysis began to occur in industrialized nations.

This phenomenon could be attributed to the fact that arsenic-based pesticides were becoming increasingly popular in the late 1800s.

Figure 3. Spraying apple trees with lead arsenate

According to The Agency for Toxic Substances and Disease Registry,[7] the adverse effects of arsenic exposure may be that of:

> Sensory effects, particularly painful dysesthesia, occur earlier and may predominate in moderate poisoning, whereas ascending weakness and paralysis may be evident in more severe poisoning.

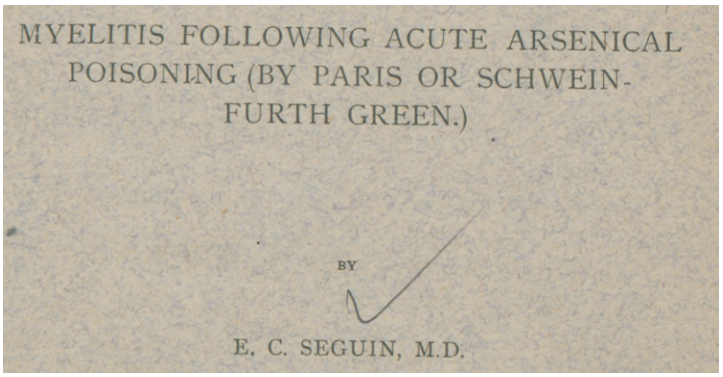
It therefore makes particular sense to take poisoning by industrial and agricultural pollution into consideration, to explain why this nervous disease began to appear in the late 1800s, in the course of industrialization.

Paris green

Paris green (copper acetoarsenite), also known as Schweinfurt green, is an arsenic based organic pigment that was invented in the early 1800s, and was mainly used in manufacturing and household products. Paris green became a popular pesticide as well. In 1867, an American farmer sprayed his potato bushes with the green compound in an attempt to protect them from the devastating Colorado potato beetle. News of Paris green's resounding success in controlling the beetle quickly traveled far and wide, and American farmers began applying it to other crops as well.

Myelitis Following Acute Arsenical Poisoning

The connection between pesticides and poliomyelitis was already noted in 1882, by a prominent neurologist, E. C. Seguin. He presented his research entitled *Myelitis Following Acute Arsenical Poisoning (by Paris or Schweinfurth green)*[8] at the New York Academy of Medicine.


MYELITIS FOLLOWING ACUTE ARSENICAL POISONING (BY PARIS OR SCHWEINFURTH GREEN.)

BY

E. C. SEGUIN, M.D.

Seguin describes the work of Popow, of St. Petersburg, who in 1881 published an essay upon the pathological anatomy of arsenical paralysis as produced artificially in animals. Popow concludes that arsenic, even in a few hours after its ingestion, may cause acute central myelitis or acute poliomyelitis.

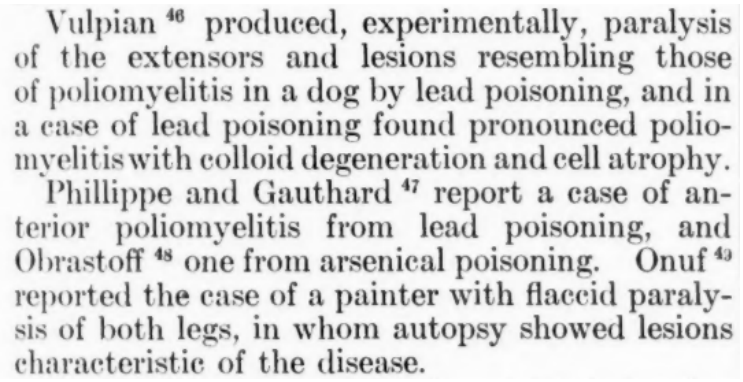
> 1. Arsenic, even in a few hours after its ingestion, may cause distinct lesions of the spinal cord, of the type known as acute central myelitis, or acute poliomyelitis.

Seguin gives condensed accounts of a few of the cases reported in the literature of the subject, and also reports three cases of his own, all poisoning with the arsenical insecticide Paris green.

His conclusions are practically the same as those of Popow:

> If we compare the symptoms present in the various human cases related and quoted, and the pathological appearances found by Popow in his animals, it is, it seems to me, legitimate to reach the following conclusions:
>
> 1. Arsenical paralysis is the expression of a myelitis.
>
> 2. This myelitis approximates the type known as poliomyelitis in so far as the symptoms are chiefly motor; that the paralyzed muscles undergo some atrophy, and exhibit the degeneration reactions to electrical currents; that the bladder is never palsied; and that in animals the ganglion cells of the anterior horns are extensively diseased.

Some other noteworthy examples can be found in a 1908 issue of the Boston Medical and Surgical Journal:[9]



Vulpian [46] produced, experimentally, paralysis of the extensors and lesions resembling those of poliomyelitis in a dog by lead poisoning, and in a case of lead poisoning found pronounced poliomyelitis with colloid degeneration and cell atrophy.

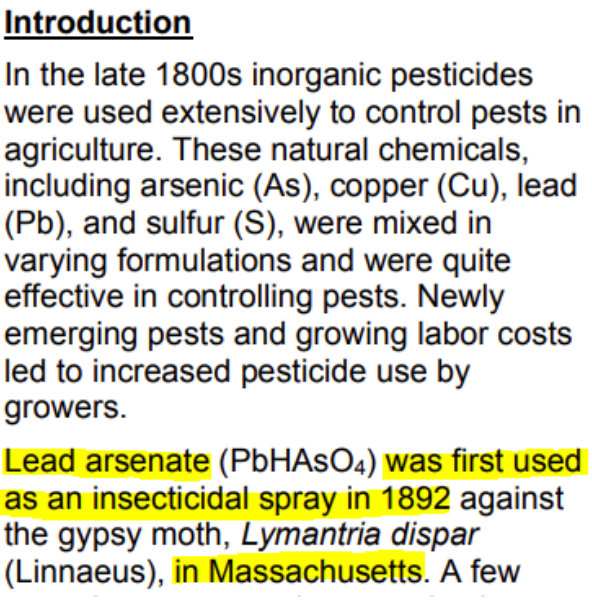
Phillippe and Gauthard [47] report a case of anterior poliomyelitis from lead poisoning, and Obrastoff [48] one from arsenical poisoning. Onuf [49] reported the case of a painter with flaccid paralysis of both legs, in whom autopsy showed lesions characteristic of the disease.

Massachusetts 1893

Paris green maintained its lead in the pesticide market until the 1890s, when it was dethroned by lead arsenate, a compound made from lead and arsenic, as the name implies. Lead arsenate was a new-and-improved insecticide invented to fight infestations of moths.

Lead arsenate became the most important pesticide in the industrialized world, since the main pesticide that had been used, Paris green, wouldn't stick to fruits and vegetables very well. Lead arsenate on the other hand was much more adhesive, contributing to its efficacy, popularity and danger.

In a paper entitled *The History of Lead Arsenate Use in Apple Production*[10] the following information can be found:

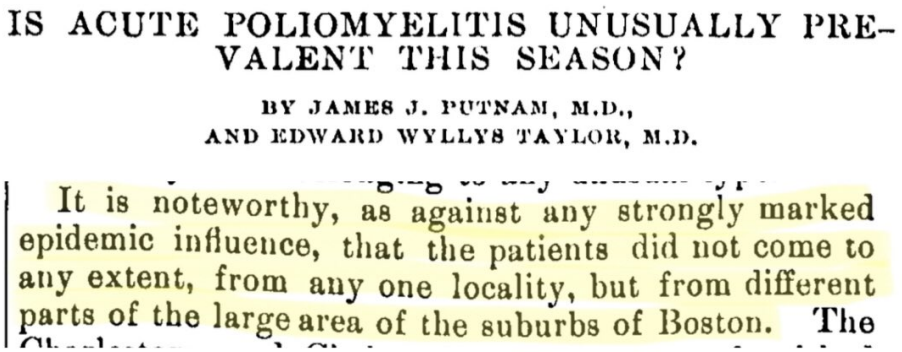


Introduction

In the late 1800s inorganic pesticides were used extensively to control pests in agriculture. These natural chemicals, including arsenic (As), copper (Cu), lead (Pb), and sulfur (S), were mixed in varying formulations and were quite effective in controlling pests. Newly emerging pests and growing labor costs led to increased pesticide use by growers.

Lead arsenate ($PbHAsO_4$) was first used as an insecticidal spray in 1892 against the gypsy moth, *Lymantria dispar* (Linnaeus), in Massachusetts. A few

The introduction of lead arsenate was accompanied by an increase in the incidence of poliomyelitis, as the following year an article entitled *Is Acute Poliomyelitis Unusually Prevalent This Season?*[11] appeared in a November 1893 issue of the widely circulated Boston Medical and Surgical Journal. Contrary to what is expected from an 'infectious' and 'contagious' disease, most of the polio victims came from the rural areas surrounding Boston, and not from the city itself.



IS ACUTE POLIOMYELITIS UNUSUALLY PREVALENT THIS SEASON?

BY JAMES J. PUTNAM, M.D.,
AND EDWARD WYLLYS TAYLOR, M.D.

It is noteworthy, as against any strongly marked epidemic influence, that the patients did not come to any extent, from any one locality, but from different parts of the large area of the suburbs of Boston. The

As seen on the chart below,[12] shortly after the introduction of the insecticide lead arsenate in Massachusetts, there was a large increase of polio cases, around the time of harvest. The first major U.S. polio epidemic occurred the following year in the neighboring state Vermont, which will be examined in the next section.

Table 1.

Recorded Epidemics of Poliomyelitis in the Conterminous U.S. Prior to 1910.

Year	Location	Cases	Deaths	Case fatality rate (percent)
1841	Feliciana, Louisiana	10	—	—
1893	Boston, Massachusetts	26	—	—
1894	Rutland and Proctor, Vermont	132	18	13
1894	North Adams, Massachusetts	10	—	—

Vermont 1894

A significant outbreak, with 132 recognized cases, erupted in the U.S. state of Vermont in 1894. After the outbreak in Vermont started, a physician named Dr. Caverly wrote up a number of reports which can be found in a 1924 book dedicated to him called *Infantile Paralysis in Vermont*.[13] In it, he made a number of interesting observations.

For one, he notes:

> The epidemic was one of an acute nervous disease whose chief distinguishing characteristic was motor paralysis, more or less complete, of one or more members or groups of muscles, and which prevailed in the State of Vermont, chiefly in a single valley, during the summer of 1894. The

He also states that:

> mentioned. There was a general absence of infectious disease as an etiologic factor in this epidemic. The element of contagium does not enter into the etiology either. I find but a single instance in which more than one member of a family had the disease, and as it usually occurred in families of more than one child, and as no efforts were made at isolation, it is very certain that it was non-contagious.

The toxic metal-based pesticides that were employed could be considered a likely causative agent. To make matters worse, common medical practices of the day included the administration of strong purgatives, often in the form of mercurial powders. Arsenical tonics such as Fowler's Solution might also be employed. Because of this, when someone of that time period presented with an illness, and a few days later showed signs of paralysis, one must consider that the medical treatments they might have received could have been a part of the problem.

Dr. Caverly also observed that animals appeared to be affected. However, this is a contradiction, since the polio 'virus' allegedly doesn't spread to animals[14] – which apparently is one reason why 'eradicating' it, is a possibility.

> .I might state further that there have been many deaths among horses, attended with symptoms of paralysis, and in which at least one veterinarian tells me he found meningitis. There have been, too, some deaths with similar symptoms among dogs and fowls.

People are the only reservoir

Hundreds of diseases can be transmitted between insects, animals and humans. One of the things that makes polio eradicable is the fact that humans are the only reservoir. No poliovirus has been found to exist and spread among animals despite repeated attempts to document this.

Lead arsenate

In an excerpt from the book *The Moth in the Iron Lung*:[15]

> Much of the early testing of lead arsenate toxicity was conducted on apples—the most popular fruit at the time. Apples had a smooth skin that allowed lead arsenate to be washed off more readily, despite its adhesive quality. As researchers began to test other produce, an alarming detail emerged: Unlike the apple, other fruits such as strawberries and blackberries had irregular, pitted surfaces. Coupled with the remarkable sticky properties of lead arsenate, these summer-time delicacies soaked up lead arsenate and would not let go.

A study entitled *Arsenical residues after spraying*[16] was carried out in 1917 in order to determine the amount of arsenical residue left of fruits and vegetables. The results showed that lethal amounts of arsenic may be present on crops after spraying:

> Spraying experiments were carried out with strawberries, currants and blackberries, including two lots of each picked before rain and two lots after rain.....
> With strawberries the residue recovered ranged from 8.6 to 34.2 milligrams AS_2O_3 per quart; with currants 6.8 to 10.2 milligrams per quart; with blackberries 3.8 to 11.2 milligrams per quart.....
> Experiments were carried out with cabbages and lettuce, including two plots of each before rain and two after rain.....
> Results with cabbages showed 43.5 to 51.4 milligrams per head. With lettuce 1.6 to 10.6 milligrams per head. Outer leaves were included.....

Organochlorines introduced

The use of synthetic pesticides became widespread after the end of World War II, following the discovery of the effectiveness of organochlorine chemicals, such as DDT and lindane .

Thus, the metal-based pesticides were by and large replaced by other synthetic pesticides, mainly DDT.

In the early 1950s, physician Morton S. Biskind testified before the US Congress. Dr. Biskind argued that central nervous system diseases such as polio were actually the physiological and symptomatic manifestations of the ongoing government and industry sponsored inundation of the world's populace with central nervous system poisons.

Public health aspects of the new insecticides:[17]

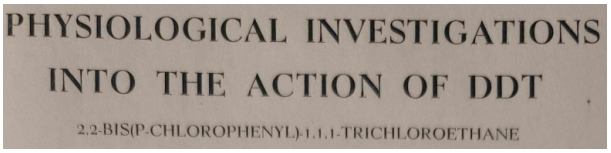
> When the population is exposed to a chemical agent known to produce in animals lesions in the spinal cord resembling those in human polio, and thereafter the latter disease increases sharply in incidence and maintains its epidemic character year after year, is it unreasonable to suspect an etiologic relationship?
>
> It was even known by 1945 that DDT is stored in the body fat of mammals and appears in the milk (106, 118). With this foreknowledge the series of catastrophic events that followed the most intensive campaign of mass poisoning in known human history, should not have surprised the experts. Yet, far from admitting a causal relationship so obvious that in any other field of biology it would be instantly accepted, virtually the entire apparatus of communication, lay and scientific alike, has been devoted to denying, concealing, suppressing, distorting and attempts to convert into its opposite, the overwhelming evidence. Libel, slander and economic boycott have not been overlooked in this campaign (21).

DDT toxicology

There is a wide variation in individual susceptibility in DDT poisoning according to the Council on Pharmacy and Chemistry of the American Medical Association:[18]

> and the period of exposure. As in acute poisoning, there are wide individual variations in susceptibility regardless of the route of administration; this makes it difficult to establish safe tolerance levels applicable to all individuals.
>
> vomiting. Exact determination of the acute oral toxic or lethal dose is difficult because the degree of gastrointestinal absorption of the material depends on the presence of fat or solvent.

Physiological evidence:
A 1949 study entitled *Physiological Investigations Into The Action Of DDT*[19] indicated that DDT poisoning may cause polio-like physiology; it was found that the cerebellum and the spinal cord are affected by DDT.



PHYSIOLOGICAL INVESTIGATIONS
INTO THE ACTION OF DDT
2,2-BIS(P-CHLOROPHENYL)-1,1,1-TRICHLOROETHANE

DANIËL DRESDEN

Conspicuous histological degeneration was, however, often found in the central nervous system. The most striking ones were found in the cerebellum, mainly in the *nucleus dentatus* and the cortex cells. Among other things an increase of the neuroglia and a necrotic degeneration and resorption of ganglionic cells was found. The Purkinje cells were less seriously affected than the other neurons [7, 11, 125]. Also in the spinal cord abnormalities of a degenerative nature were found [7, 27, 120].

So we find that especially the cerebellum and the spinal cord are histologically affected by DDT. Later we shall consider in how far

Biskind describes physiological evidence of DDT poisoning that resembles polio physiology:[20]

In 1951, the United States Public Health Service (49) pointed out:

"DDT is a delayed-action poison. Due to the fact that it accumulates in the body tissues, especially in females, the repeated inhalation or ingestion of DDT constitutes a distinct health hazard. The deleterious effects are manifested principally in the liver, spleen, kidneys and spinal cord.

"DDT is excreted in the milk of cows and of nursing mothers after exposure to DDT sprays and after consuming food contaminated with this poison. Children and infants especially are much more susceptible to poisoning than adults."

Particularly relevant to recent aspects of this problem are neglected studies by Lillie and his collaborators (74, 75) of the National Institutes of Health, published in 1944 and 1947 respectively, which showed that DDT may produce degeneration of the anterior horn cells of the spinal cord in animals.

Study 1
cited by Biskind:[21]

PATHOLOGY OF EXPERIMENTAL POISONING IN CATS, RABBITS, AND RATS WITH 2, 2 BIS-PARACHLORPHENYL-1, 1, 1 TRICHLORETHANE[1]

In the cats pronounced tremors, spasticity, and terminal extensor rigidity were observed. Spinal cord was examined in cat 6 which died 5 days after a single 300 mg. per kg. dose and in cat 7 which died on the twelfth day after 11 daily doses of 50 mg. per kg. fed in meat, not all of which was consumed. The spinal cord in cat 6 presented partial tigrolysis of anterior horn cells with pericellular vacuolation. A fat stain was negative. In cat 7 anterior horn cells lacked Nissl bodies in their reticulated lightly basophil cytoplasm and pericellular and paranuclear vacuoles were present. The other cats

Study 2 cited by Biskind:[22]

PATHOLOGIC ACTION OF DDT AND CERTAIN OF ITS ANALOGS AND DERIVATIVES

The brain and the spinal cord presented pericellular vacuolation about anterior horn cells and sometimes also in motor nuclei of the medulla and higher areas of the brain stem in the 3 cats studied. In one of these, fine fat droplets appeared in many anterior horn cells, and there was partial tigrolysis. This animal had had tremors and progressive paralysis for six days before it was killed.

The Council on Pharmacy and Chemistry of the American Medical Association outlined the pathologic effects of DDT in 1951:[23]

PHARMACOLOGIC AND TOXICOLOGIC ASPECTS OF DDT (CHLOROPHENOTHANE U. S. P.)

necrosis of the cardiac and voluntary muscles and central nervous system changes, involving vacuolization around the large nerve cells in the spinal cord and cerebral motor nuclei, and lesions in the roof and dentate nuclei of the cerebellum. In

Degeneration of the anterior horn in the spinal cord is classical polio physiology.

Symptomatic evidence:
Biskind outlines (in the previously cited statement) the symptoms of DDT poisoning after reviewing hundreds of instances of known exposure:

> The syndrome consists of a group of or all the following: Acute gastroenteritis occurs, with nausea, vomiting, abdominal pain, and diarrhea usually associated with extreme tenesmus. Coryza, 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. In some cases the hyoid bone becomes acutely painful to pressure for a few days. Pain in the joints, generalized muscle weakness and exhausting fatigue are usual; the latter are often so severe in the acute stage as to be described by some patients as "paralysis." Sometimes the initial attack is ushered in by vertigo
>
> exposure to DDT.) As already indicated, recurrent extreme fatigability is common. In acute exacerbations, mild clonic convulsions involving mainly the legs, have been observed. Several young children exposed to DDT developed a limp lasting from 2 or 3 days to a week or more.

The Council on Pharmacy and Chemistry of the AMA on the toxicologic effects of DDT, year 1951:[24]

> PHARMACOLOGIC AND TOXICOLOGIC ASPECTS OF DDT (CHLOROPHENOTHANE U. S. P.)
>
> DDT is a "cerebrospinal" poison which acts primarily on the central nervous system in man and higher animals as contrasted with its apparent peripheral action in insects. The principal systemic effects in higher animals are disturbances of the central nervous system characterized by hyperexcitability, generalized tremors, spastic or flaccid paralysis and convulsions.

A fact sheet from 1978 by the CDC[25] about the human health effects of DDT states that asymmetric weakness and paralysis have been reported in DDT poisoning:

> Hsieh 1954). Peripheral neuropathy has been occasionally ascribed to DDT, usually as a result of occupational exposure. One syndrome consists of numbness and paraesthesias, hypotonia, and asymmetric weakness or paralysis, with a slow spontaneous recovery when exposure is terminated (Jenkins and Toole 1964, Mackerras and West 1946, Onifer and Whisnant 1957).

The fact sheet describes an incident of 28 men who got poisoned by DDT and developed paralysis of extremities proportional to amount of DDT ingested:

> Onset in 30-60 min in those most severely affected; men first seen 2-3 hr after ingestion; in spite of severe early vomiting that reduced the effective dose, severity of illness and especially intensity of numbness and paralysis of extremities proportional to amount of DDT ingested; recovery in all but 8 men in 48 hr; 5 others fully recovered in 2 wk, but some weakness and ataxia of the hands in 3 5 weeks after ingestion

The Human Toxome Project states:[26]

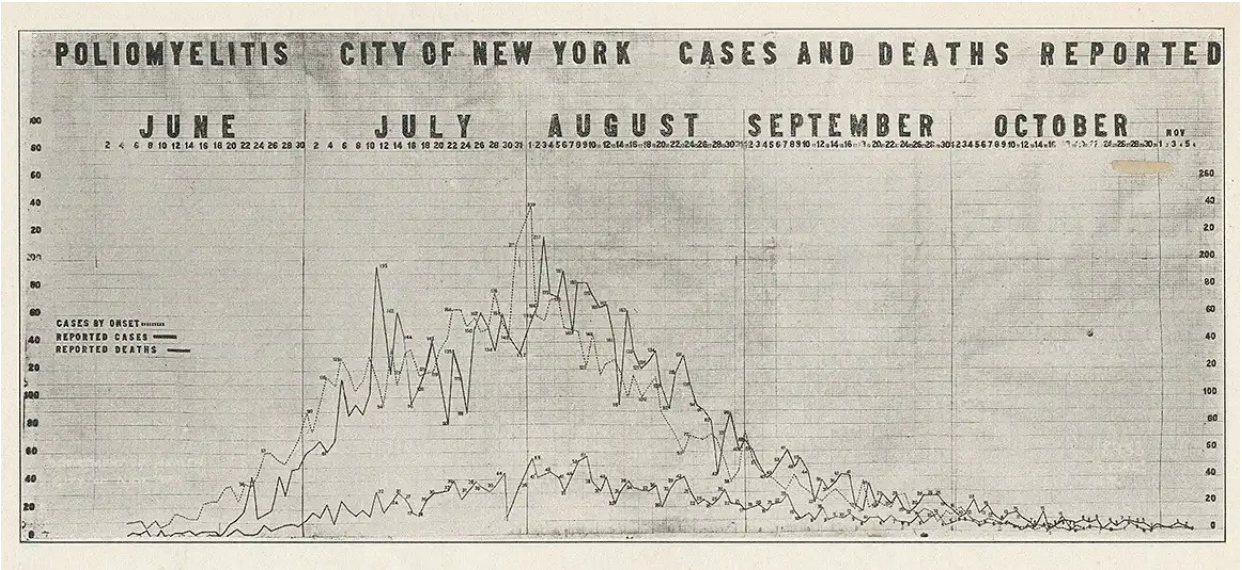
> Neurotoxic effects of DDT include tremors, convulsion, paralysis, decreased central nervous system lipid, phospholipid

Polio peaked during harvest season

The incidence of polio always peaked during the summer and early autumn, as indicated by an article entitled *Remembering the dreaded summers of polio*:[27]

> Remembering the dreaded summers of polio
>
> A Pulitzer Prize-winning historian writes about the epidemic – and two fiercely competitive NYU medical students who went on to develop polio vaccines
>
> By David Oshinsky, PhD Posted on 22 October 2014
>
> Every summer, a tremendous fear would descend.
>
> Tens of thousands of children in the United States would fall ill with paralytic poliomyelitis, also known as infantile paralysis. The major outbreaks would start around Memorial Day and become more and more prevalent, spiking in August before essentially ending for the year around Labor Day.

A graph depicting polio cases and deaths in New York City 1916:[28]



Late summer and early autumn was the time when pesticide exposure would have been the greatest.

Interesting observations

- **1.** In 1949, R. deRohan Barondes, M.D. points out that a study of the epidemiology of poliomyelitis shows a definite correlation with the harvesting of fruit and vegetable crops.[29] He notes that the harvesting of such fruits as cherries, grapes, berries, apricots, etc. and the edible vegetables, such as lettuce, radish, cucumbers, etc. usually from June to September, corresponds with the period of poliomyelitis epidemics.

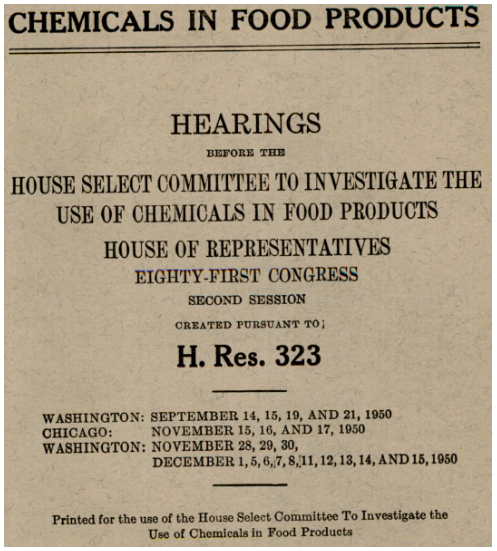
EXTRAHUMAN SOURCES OF POLIOVIRUS: NEW CONCEPT ON THE PATHOGENETICS OF THE VIRUSES

By R. deROHAN BARONDES, M.D.

A study of the epidemiology of poliomyelitis shows a definite correlation with the harvesting of the fruit and vegetable crops;

A source of infection apparently overlooked in the above survey is the contamination of fruit, as cherries, grapes, berries, apricots, etc., and the edible vegetables, as lettuce, radish, cucumber, etc.,

- **2.** Around the end of World War II, US troops in the Philippines had sprayed masses of DDT daily to wipe out insects. Polio was among the leading causes of death among soldiers. Meantime, populations in neighboring areas, where the poison had not been sprayed, experienced no problems with polio.[30]



CHEMICALS IN FOOD PRODUCTS

HEARINGS

BEFORE THE

HOUSE SELECT COMMITTEE TO INVESTIGATE THE USE OF CHEMICALS IN FOOD PRODUCTS

HOUSE OF REPRESENTATIVES

EIGHTY-FIRST CONGRESS

SECOND SESSION

CREATED PURSUANT TO

H. Res. 323

WASHINGTON: SEPTEMBER 14, 15, 19, AND 21, 1950
CHICAGO: NOVEMBER 15, 16, AND 17, 1950
WASHINGTON: NOVEMBER 28, 29, 30,
DECEMBER 1, 5, 6, 7, 8, 11, 12, 13, 14, AND 15, 1950

Printed for the use of the House Select Committee To Investigate the Use of Chemicals in Food Products

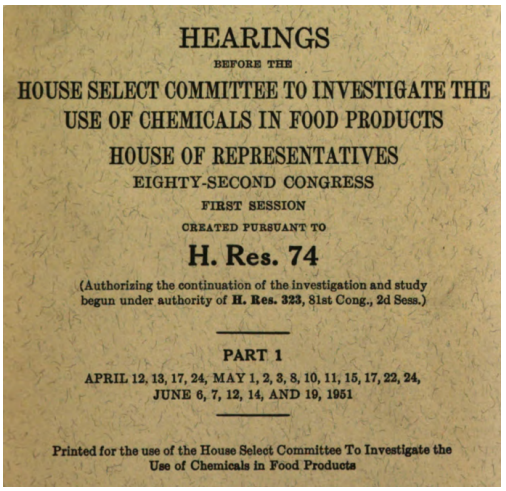
Following a recent extensive trip through the South, Dr. Mobbs informed me that wherever DDT had been used intensively against polio, not only was there an epidemic of the syndrome I have described but the incidence of polio continued to rise and in fact appeared where it had not been before. This is not surprising since it is known that not only can DDT poisoning produce a condition that may easily be mistaken for polio in an epidemic, but also being a nerve poison itself may damage cells in the spinal cord and thus increase the susceptibility to the virus.

In this connection, an observation reported by Dr. Albert B. Sabin in the Journal of the American Medical Association in June 1947 is significant:

Since the end of combat in the Philippines, poliomyelitis has been among the leading causes of death in American troops. Even though only the paralytic cases are reported from there, the incidence of poliomyelitis in American troops in the Philippines has been at least 10 times as high as in the Army within the continental limits of the United States during the past 2 years. Actually, I believe that it is even higher because hundreds of cases which would have been diagnosed as nonparalytic poliomyelitis here are being diagnosed as dengue-like or sandfly-like fevers * * * under conditions which, in my opinion, would preclude the occurrence of both dengue and sandfly fever. And yet, checks of the surrounding native population revealed no outbreaks of poliomyelitis.

Can the assiduous protection with DDT afforded our troops in the Philippines have any connection with this remarkable phenomenon?

- **3.** In 1951, Louis Bromfield pointed out that polio might merely be manifestations of poisons. He noted the use of great quantities of DDT in dairy production. Bromfield took notice of the fact that polio peaked in summer when the use of pesticides would be the greatest.[31]



HEARINGS

BEFORE THE

HOUSE SELECT COMMITTEE TO INVESTIGATE THE USE OF CHEMICALS IN FOOD PRODUCTS

HOUSE OF REPRESENTATIVES

EIGHTY-SECOND CONGRESS

FIRST SESSION

CREATED PURSUANT TO

H. Res. 74

(Authorizing the continuation of the investigation and study begun under authority of H. Res. 323, 81st Cong., 2d Sess.)

PART 1

APRIL 12, 13, 17, 24, MAY 1, 2, 3, 8, 10, 11, 15, 17, 22, 24, JUNE 6, 7, 12, 14, AND 19, 1951

Printed for the use of the House Select Committee To Investigate the Use of Chemicals in Food Products

A survey and examination of the milk supply in a nearby city has revealed astonishing amounts of this poisonous chemical in the milk supply, owing to the fact that DDT has been used in great quantities not only in the spraying of animals but of dairy and processing premises, where it is inevitable that much of the stuff finds its way into the interior of bottles and other containers, into the pasteurizing machinery and elsewhere.

It is not impossible that some cases of sickness and partial paralysis diagnosed as poliomyletis or infantile paralysis are not the true disease at all, but merely manifestations of poisons used so commonly in and around dairies. Remember that this is the greatest milk-consuming Nation in the world, and that the drinking of milk in great quantities by children is virtually a fetish.

It is notable that polio is most prevalent in the southern and southwestern tier of States where insect attack both in degree and variety is greatest, and in which the use of every sort of poison for their control is the most common. It is always at a peak in the summer months when poisonous insecticides are used in a wholesale fashion. Polio is extremely difficult to diagnose accurately, even with laboratory tests of spinal fluid, and the disease manifests itself in a great variety of ways.

Virus causation

When you look into the history of polio, you will find that the 'discovery' of the polio 'virus' is fraught with insane leaps in logic and assumptions and is replete with grotesquely inhumane experiments.

The 'infectious' etiology of polio was first established in the mainstream mind by publications of an experiment by Landsteiner and Popper in the early 1900s.[32] Their method was to inject a purée of diseased brain tissue into the stomach of two monkeys. One monkey died and the other became paralyzed:

> The material employed for inoculation of two monkeys consisted of the emulsified spinal cord in salt solution obtained from a child nine years old, who died on the fourth day of attack from infantile paralysis. The emulsion was injected into the peritoneal cavity of the monkeys. One of the latter became severely sick on the sixth day and died on the eighth day after inoculation. The other monkey became paralyzed on the seventeenth day and was killed on the nineteenth day after inoculation.

A similar experiment was later conducted by Flexner & Lewis.[33] They injected a similar substance into the brains of monkeys:

> In order to favor the transmission of the disease to monkeys, we chose the brain as the site of inoculation, which was made under ether anesthesia, through a small trephine opening. After the operations, the animals were at once lively and normal. The injected material consisted, first, of emulsions in salt solution of the spinal cord from the children and, later, of emulsions and filtrates from the spinal cord and other organs from the monkeys developing paralysis.

These kinds of experiments do not provide 'evidence' of the infectious nature of any disease; these brutal and artificial methods bear absolutely no relationship whatsoever to the 'normal' transmission route of an alleged 'infection.'

Injecting noxious matter into a living animal can, and clearly does, produce paralysis, which is the only fact that has been 'proven' by these monkey experiments.

Not once did they question whether the action of injecting ground up spinal tissues of deceased children mixed with chemicals/additives, into the brains and guts of animals, was the cause of sickness/paralysis rather than any invisible 'virus' assumed to be present.
Not once did they perform proper controls to verify the accuracy of their findings. All of these factors make the experiments virtually worthless.

In 1951, Dr. Ralph R. Scobey stated:[34]

> Although poliomyelitis is legally a contagious disease, which implies that it is caused by a germ or virus, every attempt has failed conclusively to prove this mandatory requirement of the public health law. The manifest truth that we must take into consideration is that progress in poliomyelitis investigations has been impeded by this prematurely formulated public health law.

The virologists have provided no direct evidence of an alleged polio virus, and instead have resorted to indirect observations that are invalided due to the insufficient and uncontrolled nature of the methodologies. The alleged virus has never been physically isolated by purification and subsequently characterized, and hence remains entirely theoretical. There are claims of isolation/purification in the titles of scientific papers and mainstream headlines, however, these proclamations of isolation do not hold up when the evidence is examined, which has been thoroughly investigated by Mike Stone at his website viroliegy.com.[35]

Polio reclassifications: Was polio conquered?

The Pittsburgh Press

LATE NEWS

70 Pages—5 Cents

VOL. 71, No. 291 TUESDAY, APRIL 12, 1955

POLIO IS CONQUERED

Public Airing Urged on Transit Bill

3 County Assemblymen Back Hearings on Authority Plan

Feigel Supports Transit Bill

Thinks Politicians 'Unduly Alarmed'

Biggest Thrill Came 31 Months Ago

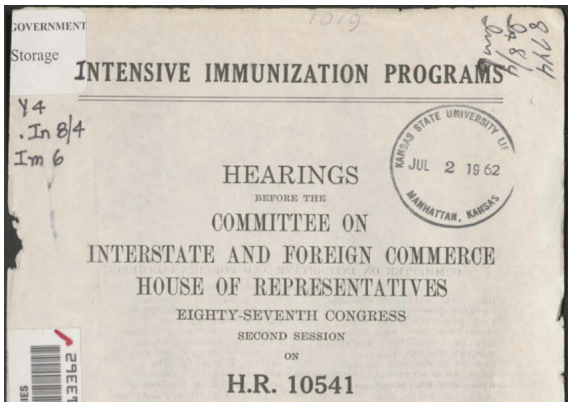
It's Anti-Climax for Dr. Salk

He Was Confident of Success After Blood Check in 1952

Salk Shots 80 to 90% Effective

After the introduction of the Salk vaccine in 1955, there were drastic changes in diagnostic methods and procedures. Because of these changes, the number of polio cases was therefore destined to fall.

All of the following information below is from the US Congress, year 1962:[36]



Polio reclassified #1

- **Redefinition of an epidemic**: More cases were required to refer to polio as epidemic after the introduction of the Salk vaccine (from 20 per 100,000 to 35 per 100,000 per year).

Dr. Herbert Ratner, M.D, director of public health, in Oak Park, and associate clinical professor of preventive medicine and public health, Stritch School of Medicine, Chicago:

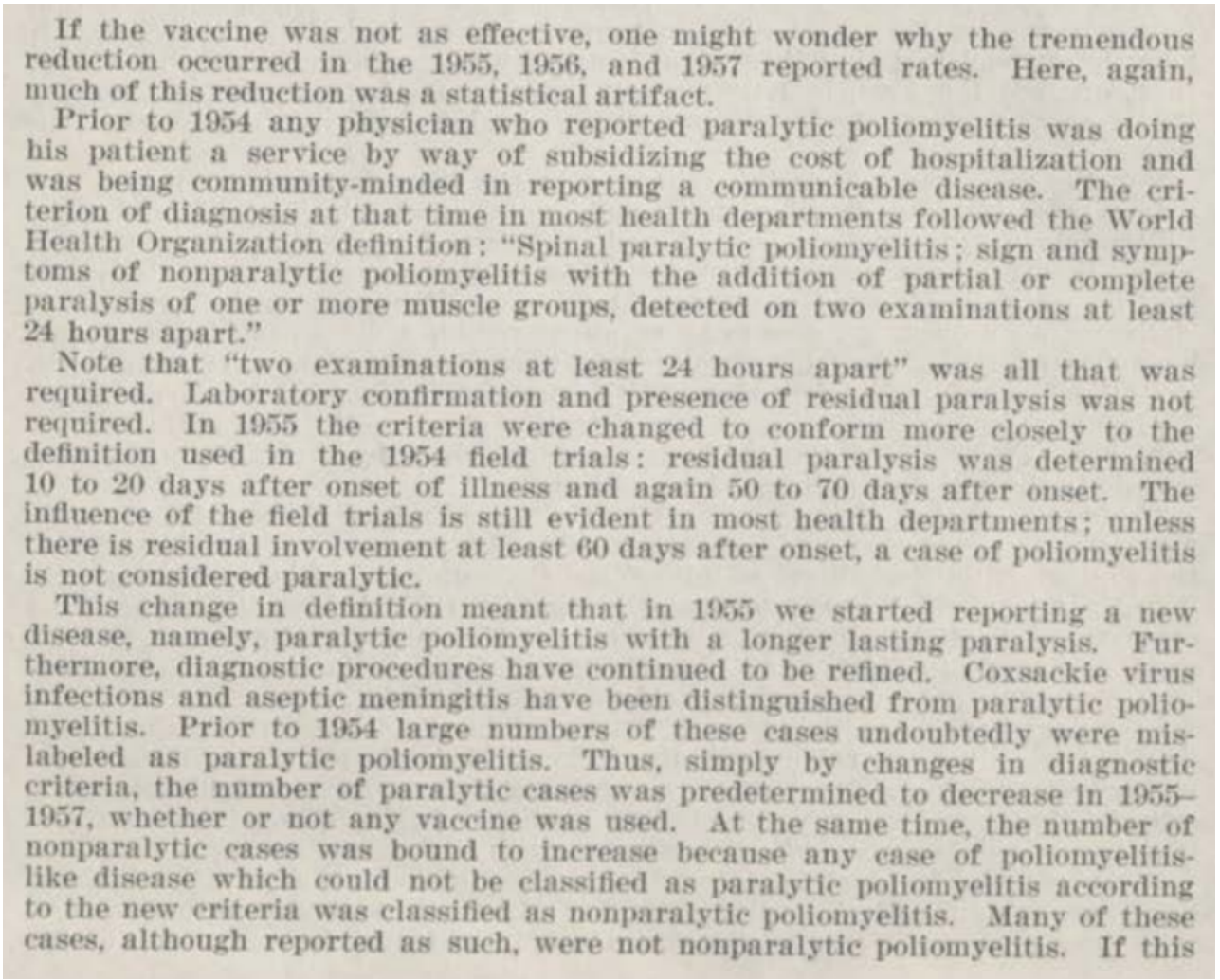
Dr. Ratner pointed out that "Prior to the introduction of the Salk vaccine, the National Foundation defined an epidemic as 20 or more cases of polio per year per 100,000 population. On this basis there were many epidemics throughout the United States yearly." After its introduction, a community was considered to have an epidemic when it had 35 cases of polio per year per 100,000 population. No reason is given for changing the rules. But in a community that before Salk vaccine release and by the old rules (of 20 per 100,000) would attract headline attention because of an "epidemic" could have the same number and more cases after 1955, and not a word woulld be printed. Indeed, there were less "epidemics" after the introduction of the Salk vaccine in 1955. But it was because they had changed the definition of an epidemic. It was not a real, but a semantic elimination of epidemics. It is no wonder that some physicians who remained skeptical about the original theories behind the vaccine, became increasingly bold in exposing the fallacies used in its evaluation.

Polio reclassified #2

- **Redefinition of the disease**: In order to qualify for classification as paralytic poliomyelitis, the patient had to exhibit paralytic symptoms for at least 60 days after the onset of the disease. Prior to 1955 the patient had to exhibit paralytic symptoms for only 24 hours. Laboratory confirmation and the presence of residual paralysis was not required.

- **Polio-like diseases were branched off**: Polio-related diseases that had been diagnosed as paralytic polio were excluded from the definition of the disease.

Dr. Bernard Greenberg, head of the Department of Biostatistics of the University of North Carolina School of Public Health, and former chairman of the Committee on Evaluation and Standards of the American Public Health Association:



If the vaccine was not as effective, one might wonder why the tremendous reduction occurred in the 1955, 1956, and 1957 reported rates. Here, again, much of this reduction was a statistical artifact.

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; sign 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."

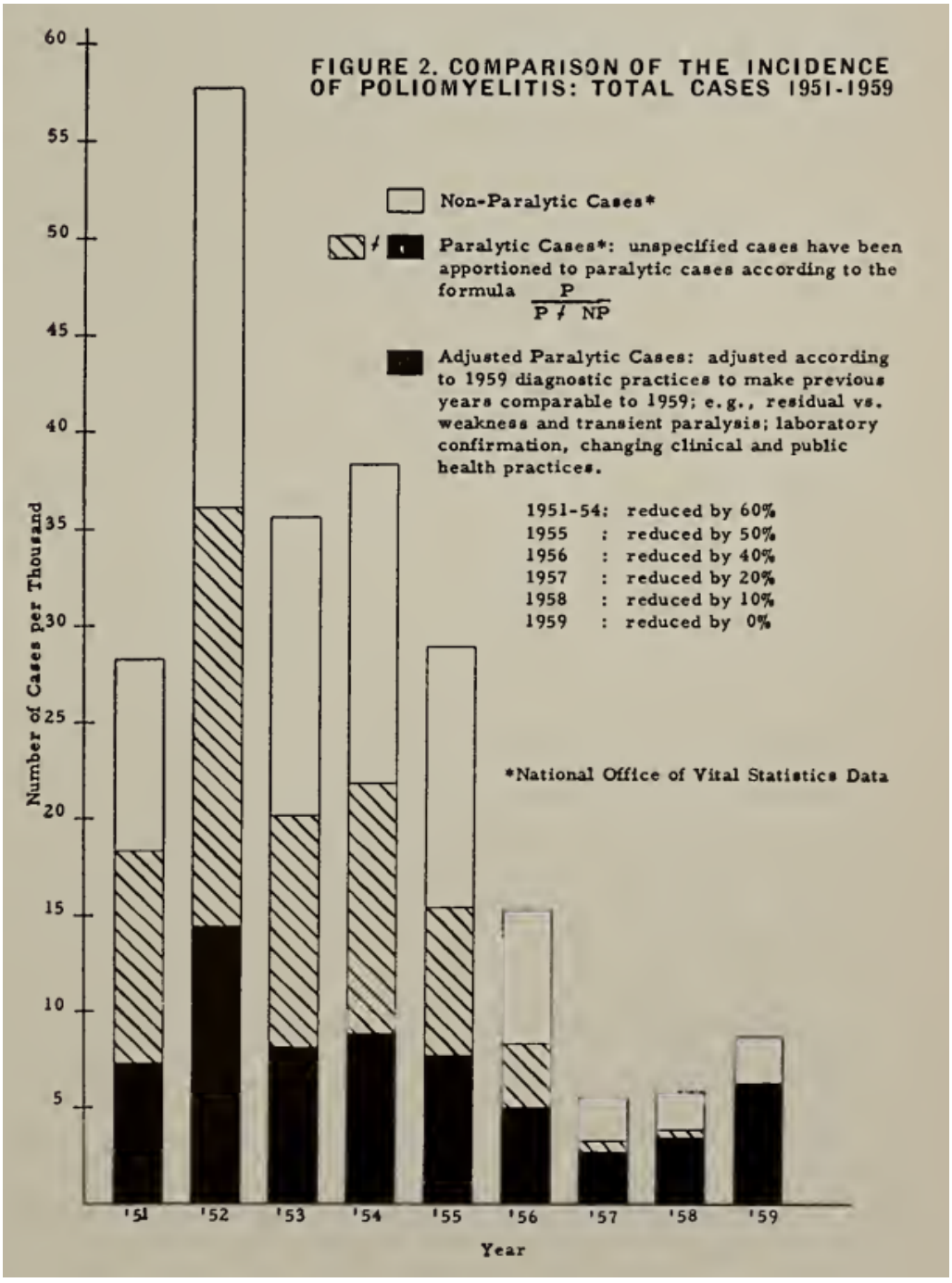
Note that "two examinations at least 24 hours apart" was all that was required. Laboratory confirmation and presence of residual paralysis was not required. In 1955 the criteria were changed to conform more closely to the definition used in the 1954 field trials: residual paralysis was determined 10 to 20 days after onset of illness and again 50 to 70 days after onset. The influence of the field trials is still evident in most health departments; unless there is residual involvement at least 60 days after onset, a case of poliomyelitis is not considered paralytic.

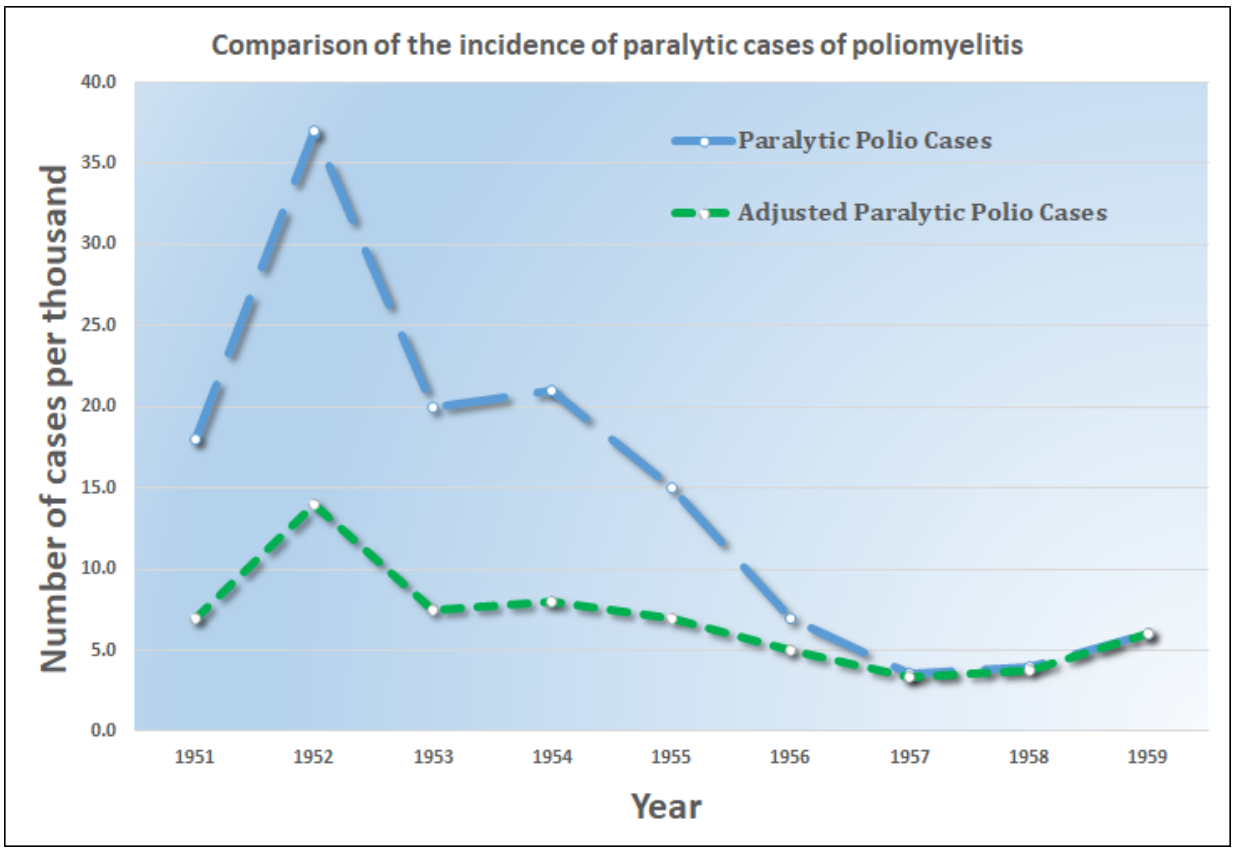
This change in definition meant that in 1955 we started reporting a new disease, namely, paralytic poliomyelitis with a longer lasting paralysis. Furthermore, diagnostic procedures have continued to be refined. Coxsackie virus infections and aseptic meningitis have been distinguished from paralytic poliomyelitis. Prior to 1954 large numbers of these cases undoubtedly 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. At the same time, the number of nonparalytic cases was bound to increase because any case of poliomyelitis-like disease which could not be classified as paralytic poliomyelitis according to the new criteria was classified as nonparalytic poliomyelitis. Many of these cases, although reported as such, were not nonparalytic poliomyelitis. If this

Dr. Kleinman, M.D, epidemiologist from the Minnesota Department of Health:

I would also like to agree with Dr. Greenberg that the insistence upon a 60-day duration of paralysis for paralytic polio is absolutely silly. There isn't a doctor in this room who hasn't seen a case of frank paralytic polio which has not recovered within 60 days, or at least recovered sufficiently so that you could not estimate with clinical certainty that there was some residual paralysis.

A graph was provided which shows what the incidence of paralytic polio would have been from 1951 through 1959 if the figures were corrected for the radical changes in diagnostic criteria since the introduction of the Salk vaccine:



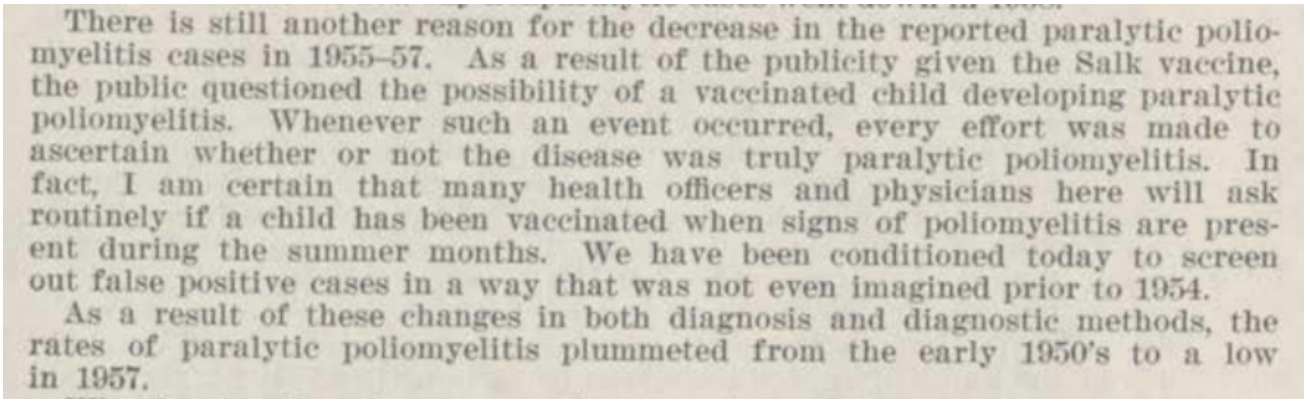


As seen on the graph[37] above, there was not a significant decrease of paralytic polio morbidity in the 1950s.

Polio reclassified #3

- **Restrictive diagnosing**: Doctors were more restrictive in diagnosing their patients with poliomyelitis.

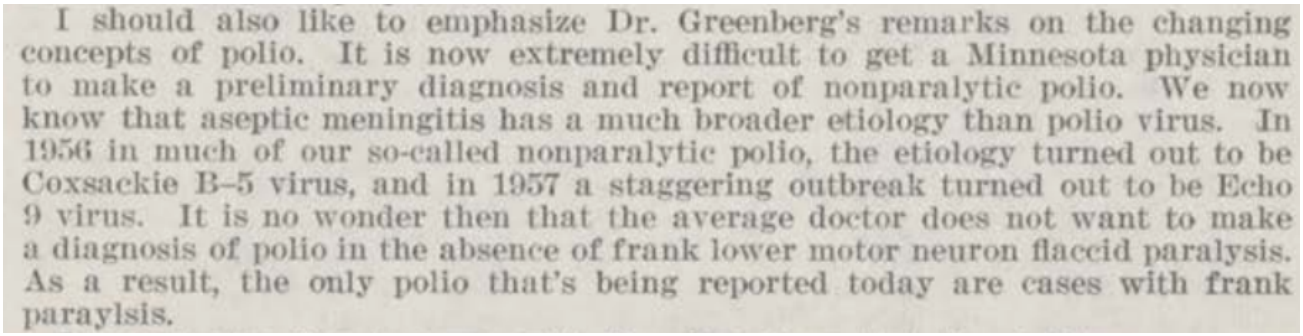
Dr. Bernard Greenberg:



There is still another reason for the decrease in the reported paralytic poliomyelitis cases in 1955–57. As a result of the publicity given the Salk vaccine, the public questioned the possibility of a vaccinated child developing paralytic poliomyelitis. Whenever such an event occurred, every effort was made to ascertain whether or not the disease was truly paralytic poliomyelitis. In fact, I am certain that many health officers and physicians here will ask routinely if a child has been vaccinated when signs of poliomyelitis are present during the summer months. We have been conditioned today to screen out false positive cases in a way that was not even imagined prior to 1954.

As a result of these changes in both diagnosis and diagnostic methods, the rates of paralytic poliomyelitis plummeted from the early 1950's to a low in 1957.

Dr. Kleinman:



I should also like to emphasize Dr. Greenberg's remarks on the changing concepts of polio. It is now extremely difficult to get a Minnesota physician to make a preliminary diagnosis and report of nonparalytic polio. We now know that aseptic meningitis has a much broader etiology than polio virus. In 1956 in much of our so-called nonparalytic polio, the etiology turned out to be Coxsackie B–5 virus, and in 1957 a staggering outbreak turned out to be Echo 9 virus. It is no wonder then that the average doctor does not want to make a diagnosis of polio in the absence of frank lower motor neuron flaccid paralysis. As a result, the only polio that's being reported today are cases with frank paraylsis.

The symptoms of polio are not non-specific, and can therefore be reclassified into other labels. Thus, case numbers can be manipulated by lumping pre-existing syndromes into different diagnoses.

DDT phase out

In 1950 Congress set up a special committee to "investigate the use of chemicals in food and cosmetics." The committee, chaired by Congressman James Delaney, summoned over 200 witnesses and devoted much time to examining the toxicity of pesticides. Its final report, published in 1952, contained a recommendation to Congress to "pass legislation to control the flow of chemical substances into the nation's food supply."[38]

Congress complied with the committee's recommendation, and relevant laws were indeed enacted; the Pesticide Residues Amendments of 1954 and the Food Additives Amendments of 1958.[39]

The PCA of 1954 was the first time Congress passed guidance regarding the establishment of safety limits for pesticide residues on food.[40]

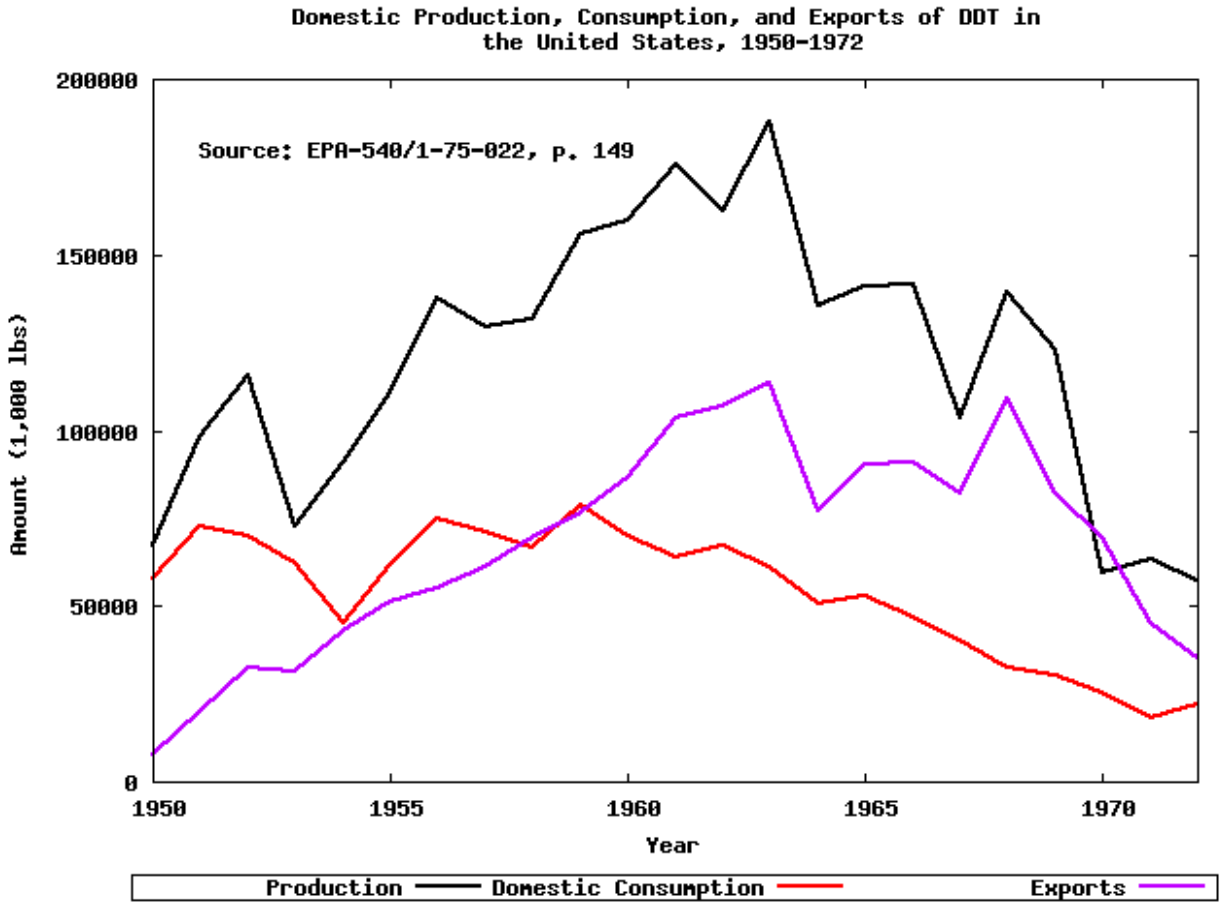
According to the Environmental Protection Agency, The Department of Agriculture began regulating DDT in the late 1950s, ending with the cancellation of the use of the pesticide in the US in 1972.[41]

Regulation Due to Health and Environmental Effects

The U.S. Department of Agriculture, the federal agency with responsibility for regulating pesticides before the formation of the U.S. Environmental Protection Agency in 1970, began regulatory actions in the late 1950s and 1960s to prohibit many of DDT's uses because of mounting evidence of the pesticide's declining benefits and environmental and toxicological effects. The publication in 1962 of Rachel Carson's *Silent Spring* stimulated widespread public concern over the dangers of improper pesticide use and the need for better pesticide controls.

In 1972, EPA issued a cancellation order for DDT based on its adverse environmental effects, such as those to wildlife, as well as its potential human health risks. Since then, studies have continued, and a relationship between DDT exposure and reproductive effects in humans is suspected, based on studies in animals. In addition, some animals exposed to DDT in studies developed liver tumors. As a result, today, DDT is classified as a probable human carcinogen by U.S. and international authorities.

In the late 1950s, DDT usage began to decline greatly, as seen on the graph below:[42]



DDT was largely phased out of the food chain. Before its cancellation in the early 1970s, 80% of it was applied on cotton, with the remainder being used predominantly on peanut and soybean crops.[43]

Rachel's Carlson's book *Silent Spring* published in 1962 stimulated widespread public awareness over the use of pesticide.

DDT Regulatory History: A Brief Survey (to 1975)

[EPA report, July 1975]

Background

DDT (Dichloro-diphenyl-trichloroethane), for many years one of the most widely used pesticidal chemicals in the United States, was first synthesized in 1874. Its effectiveness as an insecticide, however, was only discovered in 1939. Shortly thereafter, particularly during World War II, the U.S. began producing large quantities of DDT for control of vector-borne diseases such as typhus and malaria abroad.

After 1945, agricultural and commercial usage of DDT became widespread in the U.S. The early popularity of DDT, a member of the chlorinated hydrocarbon group, was due to its reasonable cost, effectiveness, persistence, and versatility. During the 30 years prior to its cancellation, a total of approximately 1,350,000,000 pounds of DDT was used domestically.

After 1959, DDT usage in the U.S. declined greatly, dropping from a peak of approximately 80 million pounds in that year to just under 12 million pounds in the early 1970s. Of the quantity of the pesticide used in 1970-72, over 80 percent was applied to cotton crops, with the remainder being used predominantly on peanut and soybean crops. The decline in DDT usage was the result of (1) increased insect resistance; (2) the development of more effective alternative pesticides; (3) growing public concern over adverse environmental side effects; and (4) increasing government restrictions on DDT use.

In addition to domestic consumption, large quantities of DDT have been purchased by the Agency for International Development and the United Nations and exported for malaria control. DDT exports increased from 12 percent of the total production in 1950 to 67 percent in 1969. However, exports have shown a marked decrease in recent years dropping from approximately 70 million pounds in 1970 to 35 million in 1972.

Public Concern

Certain characteristics of DDT which contributed to the early popularity of the chemical, particularly its persistence, later became the basis for public concern over possible hazards involved in the pesticide's use. Although warnings against such hazards were voiced by scientists as early as the mid-1940s, it was the publication of Rachel Carson's book *Silent Spring* in 1962 that stimulated widespread public concern over use of the chemical. After Carson's alert to the public concerning the dangers of improper pesticide use and the need for better pesticide controls, it was only natural that DDT, as one of the most widely used pesticides of the time, should come under intensive investigation.

Did polio vanish?

The medical establishment claims that polio is close to being eradicated due to the vaccination programme; as indicated by a 2016 CDC web page entitled *Updates on CDC's Polio Eradication Efforts*[44] that states:

> Polio incidence has dropped more than 99 percent since the launch of global polio eradication efforts in 1988.

This claim is highly questionable, because there are large incidences of paralysis in a number of countries around the world. One country in which this problem exists is India, where cases of paralysis are still high, despite the claim that the country is virtually 'polio-free.'

An article entitled *Polio programme: let us declare victory and move on*[45] describes:

> their scarce resources on an impossible dream over the last 10 years was unethical. Furthermore, while India has been polio-free for a year, there has been a huge increase in non-polio acute flaccid paralysis (NPAFP). In 2011, there were an extra 47,500 new cases of NPAFP. Clinically indistinguishable from polio paralysis but twice as deadly, the incidence of NPAFP was directly proportional to doses of

The situation in India is also explained by a 2013 article entitled *Polio free does not mean paralysis free*:[46]

> **Polio free does not mean paralysis free**
>
> No child in India has been diagnosed with polio for nearly two years now and all the indications are that the virus responsible for it is no longer circulating here. However, the country's polio surveillance system has indicated a sharp increase during recent years in the number of non-polio AFP cases.

These cases of paralysis do not however represent a new condition, especially as acute flaccid paralysis is described as clinically indistinguishable from polio. It would seem, therefore, that the 'new' condition referred to as acute flaccid paralysis is, in reality, purely a name change used for cases that would previously have been diagnosed as polio.

Symptoms previously associated with polio still exist today, but they have been reclassified under new labels, such as; acute flaccid paralysis, acute flaccid myelitis, meningitis, encephalitis, transverse myelitis, guillain-barré syndrome, myopathy, enteroviral myelitis, multiple sclerosis, enteroviral encephalopathy, traumatic neuritis, chronic fatigue syndrome, Reye's syndrome, etc.

Conclusion

The foregoing information has made it clear that there is a distinct connection between the use of certain substances known to be neurotoxic, and a diagnosis of poliomyelitis; this connection should have made it worth pursuing a toxicological investigation.

The change in polio incidence from a few cases a year to outbreaks of hundreds or more that began in the 1890s is consistent with the transition from the use of Paris green (copper acetoarsenite) to lead arsenate, a more adhesive pesticide, contributing to its efficacy and danger.

The sudden onset of polio epidemics in industrialized countries after World War II clearly coincides with the sudden explosion in DDT use. The phase out of DDT in the late 1950s corresponds with the reduction of polio morbidity.

Unfortunately, the medical establishment has ignored this connection between neurotoxins and paralysis and has only pursued the hypothesis that a 'virus' is the causal agent of polio. Astoundingly, instead of following up the obvious evidence, medical authorities viewed the pesticides as weapons in the 'battle' against polio.

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