Pesticides and Polio: A Critique of Scientific Literature

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The following statement appeared in the *Handbook of Pesticide Toxicology*, 1991, edited by Wayland J. Hayes and Edward R. Laws: “It has been alleged that DDT causes or contributes to a wide variety of diseases of humans and animals not previously recognized as associated with any chemical. Such diseases included. . . poliomyelitis, . . . such irresponsible claims could produce great harm and, if taken seriously, even interfere with scientific search for true causes. . .”[1]

Hayes and Laws were informing their readers about the heretic, Dr. Morton S. Biskind. In 1953, when Biskind’s writings were published, the United States had just endured its greatest polio epidemic. The entire public was steeped in dramatic images–a predatory poliovirus, nearly a million dead and paralyzed children, iron lungs, struggling doctors and dedicated nurses. The late president Franklin D. Roosevelt had been memorialized as a polio victim who was infected with the deadly poliovirus near the beautiful and remote island of Campobello. The media was saturated with positive images of scientific progress and the marvels of DDT to kill disease-carrying mosquitos. Jonas Salk was in the wings, preparing to be moved center stage.

Through this intellectually paralyzing atmosphere, Dr. Biskind had the composure to argue what he thought was the most obvious explanation for the polio epidemic: **Central nervous system diseases (CNS) such as polio are actually the physiological and symptomatic manifestations of the ongoing government- and industry-sponsored inundation of the world’s populace with central nervous system poisons.**

Today, few remember this poignant writer who struggled with the issues of pesticides, issues that Rachel Carson would be allowed to politely bring to public awareness nine years later, as the lead story in *The New Yorker* magazine and then as a national best seller, by limiting her focus to the environment and wildlife. Biskind had the audacity to write about human damage.

I found “M.S. Biskind” in the endnotes to Hayes’ and Laws’ diatribe. What could possibly have motivated Hayes’ and Laws’ biased genuflection towards germ theory? Such offerings, commonly written into the final paragraphs of scientific articles, are usually done with an appearance of impartiality. With great anticipation, I went to a medical library and found Biskind’s 10-page 1953 article in the *American Journal of Digestive Diseases*.[2] Presented below are excerpts regarding polio from the article.

Biskind’s Warnings

“In 1945, against the advice of investigators who had studied the pharmacology of the compound and found it dangerous for all forms of life, DDT (chlorophenoethane, dichloro-diphenyl-trichloroethane) was released in the United States and other countries for general use by the public as an insecticide. . . .

“Since the last war there have been a number of curious changes in the incidence of certain ailments and the development of new syndromes never before observed. A most significant feature of this situation is that both man and all his domestic animals have simultaneously been affected. In man, the incidence of poliomyelitis has risen sharply. . . .

“It was even known by 1945 that DDT is stored in the body fat of mammals and appears in the milk. 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. . . .

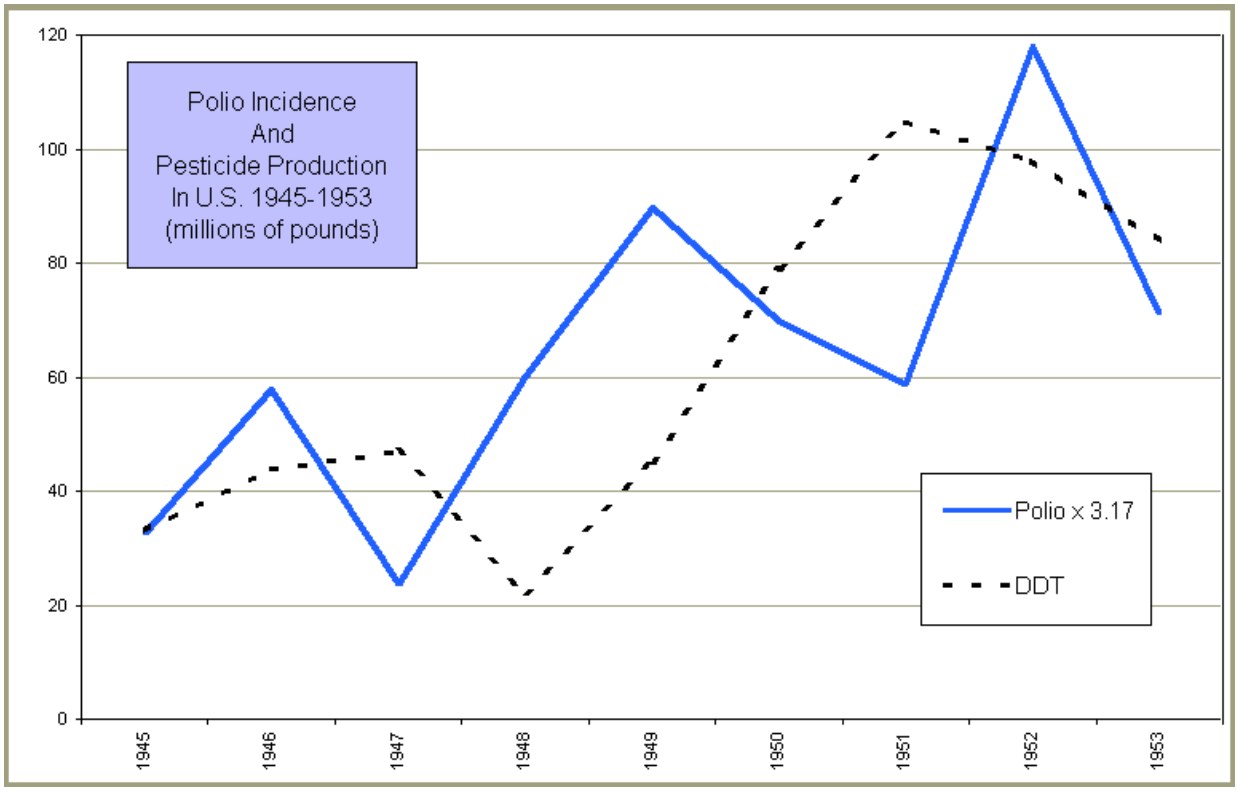
“Early in 1949, as a result of studies during the previous year, the author published reports implicating DDT preparations in the syndrome widely attributed to a ‘virus-X’ in man, in ‘X-disease’ in cattle and in often fatal syndromes in dogs and cats. The relationship was promptly denied by government officials, who provided no evidence to contest the author’s observations but relied solely on the prestige of government authority and sheer numbers of experts to bolster their position. . . .

“[‘X-disease’] . . . studied by the author following known exposure to DDT and related compounds and over and over again in the same patients, each time following known exposure. We have described the syndrome as follows: . . . . 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. . . .

“Simultaneously with the occurrence of this disorder [X-disease], a number of related changes occurred in the incidence of known diseases. The most striking of these is poliomyelitis. In the United States the incidence of polio had been increasing prior to 1945 at a fairly constant rate, but its epidemiologic characteristics remained unchanged. Beginning in 1946, the rate of increase more than doubled. Since then remarkable changes in the character of the disease have been noted. Contrary to all past experience, the disease has remained epidemic year after year.”

DDT vs Polio

In the graph below, I provide confirmation of Biskind’s observations for 1945-1953, in terms of polio incidence and pesticide production. I have utilized pesticide data from Hayes and Laws which they had obtained from the US Transportation Board. Polio incidence data was gathered from *US Vital Statistics*.[3] Although I argue herein against Hayes’ characterization of Biskind’s work, credit goes to Hayes for publishing arcane pesticide data.



Physiological Evidence

Biskind also describes physiological evidence of DDT poisoning that resembles polio physiology: “Particularly relevant to recent aspects of this problem are neglected studies by Lillie and his collaborators 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. These changes do not occur regularly in exposed animals any more than they do in human beings, but they do appear often enough to be significant.”

He continues, bearing his exasperation in trying to make the obvious plain. “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?”

Before finding Biskind’s work, I had spent months engaged in a nearly futile search for the physiology of acute DDT poisoning. I began to sense that American DDT literature as a whole intends to convey that DDT is not dangerous except with regard to its general environmental effects due to persistent bioaccumulation, and that the physiology of acute DDT poisoning is therefore trivial. DDT literature uniformly jumps from descriptions of symptoms, over physiology, to the biochemistry of DDT-caused dysfunction in nerve tissue. It was as though detectives had come upon a mass-murder scene and immediately became obsessed with the biochemistry of dying cells around bullet holes, while ignoring the bullet holes.

Eventually, I did find one study, in a German publication, of the physiology of acute DDT poisoning. [4] The study confirmed that DDT poisoning often causes polio-like physiology. “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. Also in the spinal cord abnormalities of a degenerative nature were found. . . . such changes were not found invariably. . . there is neither an obvious relation between the size and spreading of the lesion and the quantity of DDT applied. . . . information of adequate precision about the nature of the anomalies is lacking.”

Thus we find that the cerebellum and the spinal cord are especially affected by DDT.

And more recently, in the works of Ralph Scobey, MD, [5] I found that from ancient times to the early 20th century, the symptoms and physiology of paralytic poliomyelitis were often described as the results of poisoning. It wasn’t until the mid-19th century that the word “poliomyelitis” became the designation for the paralytic effects of both severe poisoning and polio-like diseases assumed to be germ-caused.

Today, various other forms of the word “polio” are still used to describe the effects of neurotoxins, although usually with regard to paralysis in animals. (See below.)

In contemporary Britain, a farmer turned scientist, Mark Purdey, has found substantial evidence that mad cow disease, a form of polio-like encephalitis, was caused by a government mandated cattle treatment consisting of organophosphate pesticide and a compound similar to thalidomide.[6] Unlike most scientists, Mark Purdey became legally embroiled with the government during his research, and ” . . . was shot at, blockaded in his home to prevent him giving a lecture, and saw a new farmhouse go up in flames the day he was due to move in.”[7]

Morton S. Biskind had the courage to write about humans. His views fell into disfavor after the introduction of the polio vaccines, which was a grand act that proved in most people’s minds that polio was caused by a virus. By October, 1955, Biskind, whose works had been published in established medical journals and who testified before the Senate on the dangers of pesticides, was forced to self-publish his writings, one of which I found while browsing through an old card catalog. A scan of MEDLINE finds no other works by him except for a very tame article in 1972,

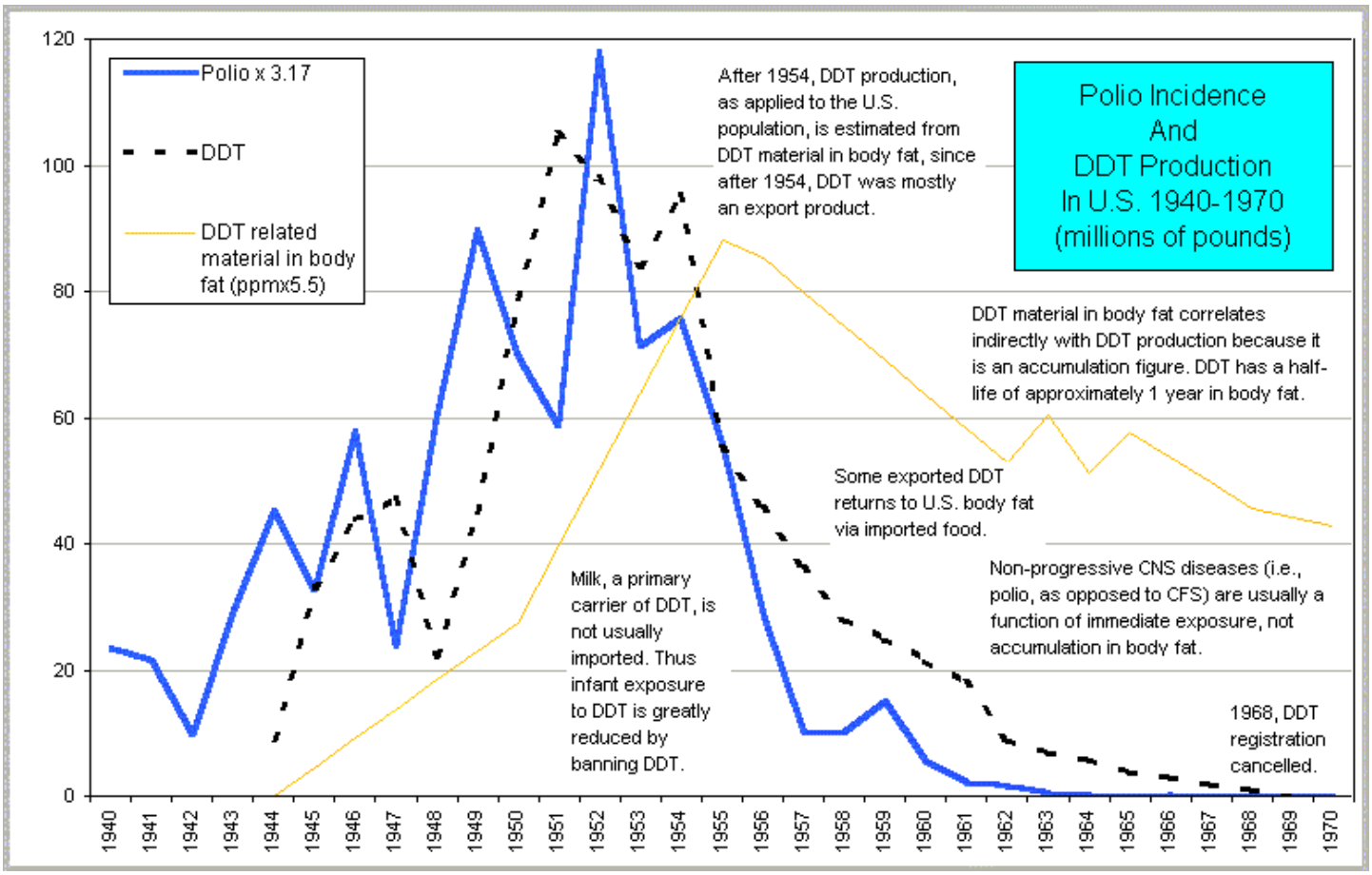
warning that diseases incurred during a patient's stay in a hospital are not necessarily due to microbes. He died not long thereafter, in his late 60s.

A Contemporary Study

I have compiled information that confirms Biskind's observations, utilizing data that extend far beyond his observations. These data are presented in the next three graphs.

Due to the paucity of data regarding pesticide exposure and locale, production data are given as an indication of exposure, keeping in mind the great changes in public awareness and legislation beginning in 1950. Again, this pesticide data comes from Hayes and Laws.

In the graph below, I did not include DDT data for the period of 1954 onward because, even though the US production of DDT skyrocketed, its distribution was then being shifted out of the US and into developing nations. Governmental hearings, including those with Biskind, Scobey and others, brought about greater awareness of DDT dangers, as well as better labeling and handling methods.[8] Due to public governmental debate in 1950-51 and numerous policy and legislative changes afterward,[8,9,10,11] DDT production figures after these dates do not correlate with US usage or exposure to DDT.



After 1950, DDT was continually incriminated until its registration cancellation in 1968 and ban in 1972. So 1950 marked a point of increased public awareness, changes in legislation and policy, voluntary phaseout, labeling requirements and discouragement from use in dairy farms. Much of the usage in the US may have moved over to forestry applications, placing less DDT directly into the food chain.

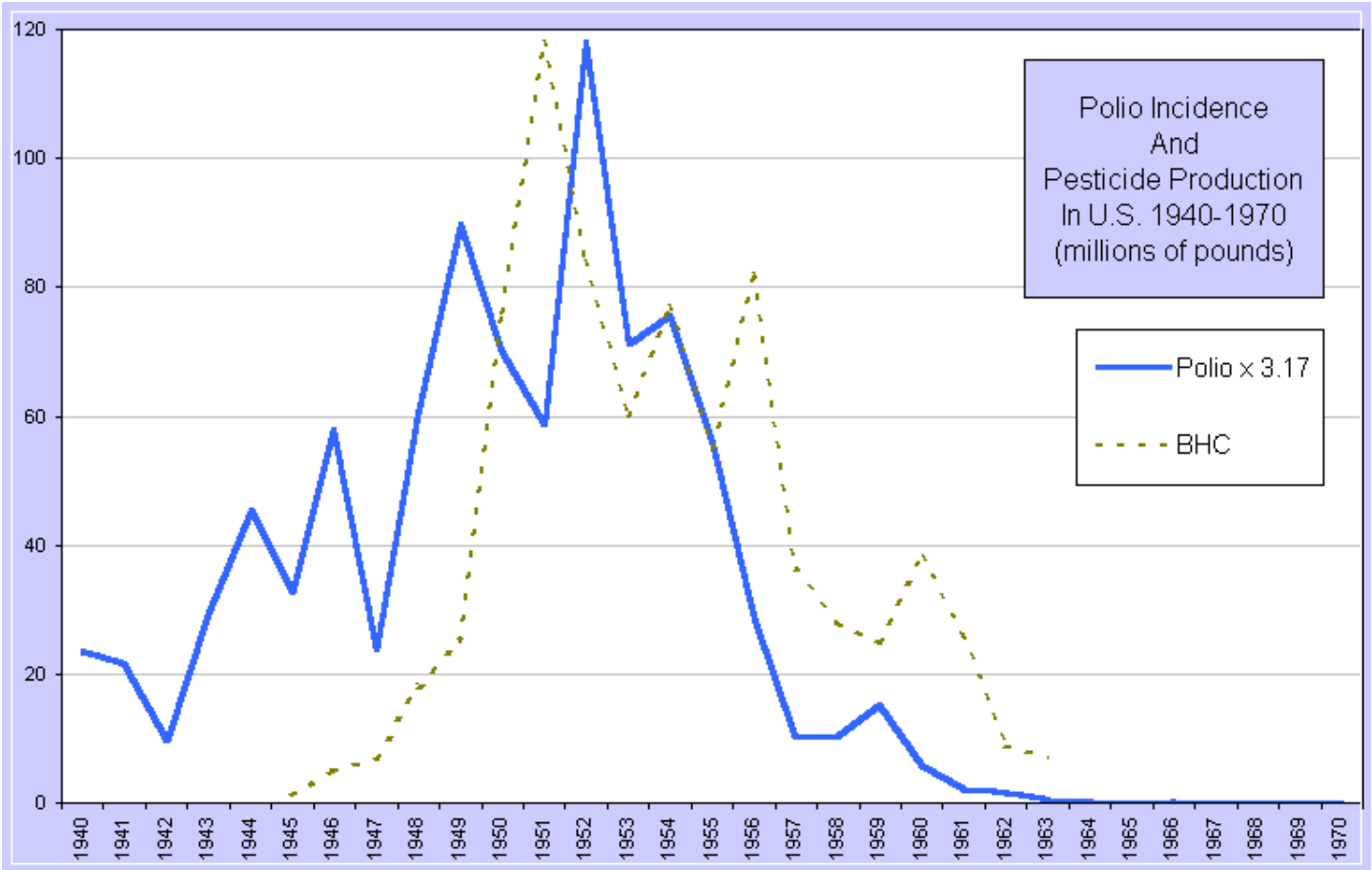
Therefore, DDT production, as an indicator of human exposure in the US, is estimated in the graph above by reviewing levels of DDT in adipose tissue (National Adipose Tissue Survey, and other studies)[12] and considering the

context of DDT in imported food. Levels of DDT in adipose tissue before 1955 were estimated by drawing a straight line from the low to the high levels of DDT in adipose tissue for that period. The estimate of DDT exposure is fairly accurate because DDT has a half-life of about one year. To achieve any downward trend in the DDT/adipose line, DDT exposure had to have decreased sharply. It is, however, not an assumption, but a fact, that the lowering of DDT levels in adipose tissue parallels the hyped advent of the Salk vaccination programs.

BHC vs Polio

BHC (benzene hexachloride), a persistent, organochlorine pesticide, is several times more lethal than DDT, in terms of LD50 (lethal dosage required to kill 50 percent of a test population).

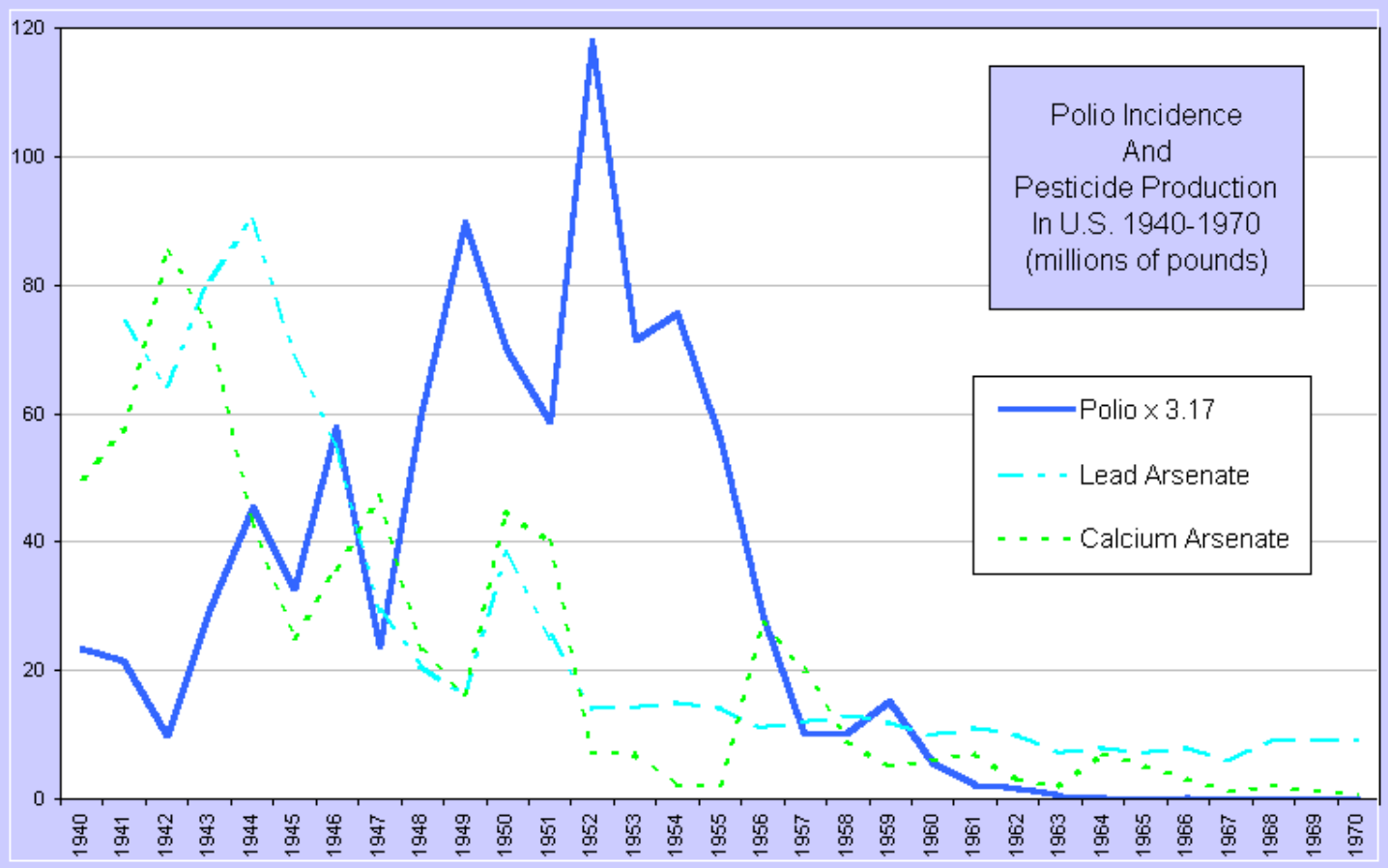
As shown in the graph below, BHC was produced in 1945-1954 at quantities similar to DDT. In spite of BHC's lethal quality, it has received much less publicity than DDT. While DDT was banned for such things as an association with the thinning of eagles' eggs, BHC was phased out of production because it was found, after 15 years, to impart a bad taste to food. It is still used in developing nations. One is tempted to ask whether the more controversial DDT, known to be dangerous, was "fronting" for the more dangerous BHC? BHC's correlation with polio incidence is astonishing.



Lead-Arsenic vs Polio

Note that the period 1940-46 is unaccounted for in terms of polio-pesticide correlation in the DDT and BHC graphs. The missing piece of the puzzle for this six-year period is supplied by the lead and arsenic compounds, shown in the graph below. These central nervous system (CNS) poisons were the major pesticides during the several centuries previous to the advent of the organochlorines in the early 1940s. For those who think that "organic" food was the norm before the release of DDT to the civilian sector in 1945, the immense production of lead-arsenic compounds seen in this graph proves otherwise. This data requires a reconsideration of statements regarding the "natural"

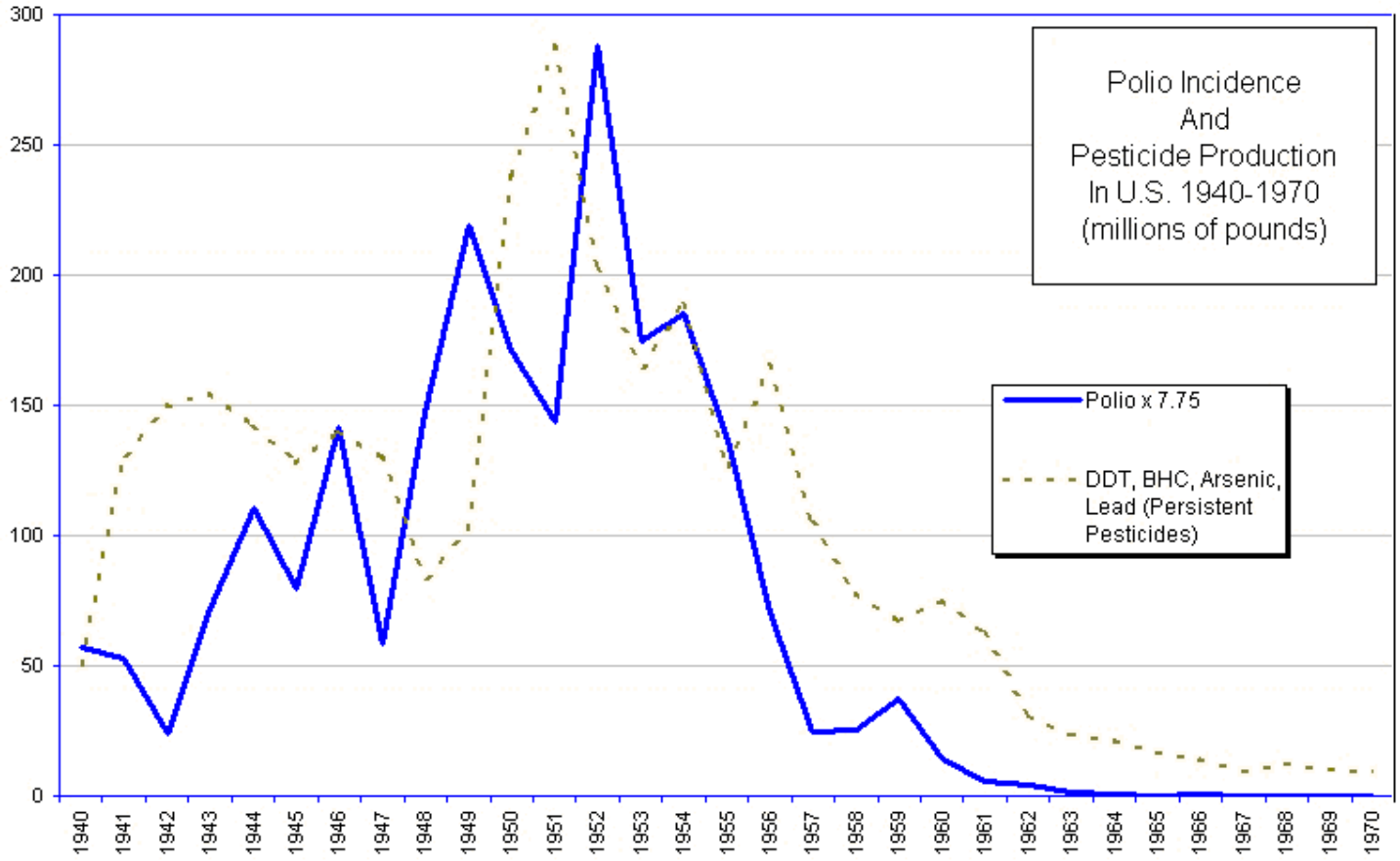
quantities of arsenic found in apple seeds, apricots, or almonds or “natural” chemotherapies derived from seeds where pesticides can accumulate in the ground.



Pesticide Composite: Summary

Just over three billion pounds of persistent pesticides are represented in the graph below. Virtually all peaks and valleys correlate with a direct one-to-one relationship with each pesticide as it enters and leaves the US market. Generally, pesticide production precedes polio incidence by 1 to 2 years. I assume that this variation is due to variations in reporting methods and the time it takes to move pesticides from factory to warehouse, through distribution channels, onto the food crops and to the dinner table. A composite of the three previous graphs, of the persistent pesticides–lead, arsenic, and the dominant organochlorines (DDT and BHC)–is represented.

These four chemicals were not selected arbitrarily. These are representative of the major pesticides in use during the last major polio epidemic. They persist in the environment as neurotoxins that cause polio-like symptoms, polio-like physiology, and were dumped onto and into human food at dosage levels far above that approved by the FDA. They directly correlate with the incidence of various neurological diseases called “polio” before 1965. They were utilized, according to Biskind, in the “most intensive campaign of mass poisoning in known human history.”



Virus Causation

A clear, direct, one-to-one relationship between pesticides and polio over a period of 30 years, with pesticides preceding polio incidence in the context of the CNS-related physiology just described, leaves little room for complicated virus arguments, even as a cofactor, unless there exists a rigorous proof for virus causation. Polio shows no movement independent from pesticide movement, as one would expect if it were caused by a virus. Both the medical and popular imaginations are haunted by the image of a virus that invades (or infects) and begins replicating to the point of producing disease.

In the laboratory, however, poliovirus does not easily behave in such a predatory manner. Laboratory attempts to demonstrate causation are performed under conditions which are extremely artificial and aberrant.

Poliovirus causation was first established in the mainstream mind by publications of an experiment by Landsteiner and Popper in Germany, 1908-1909.[13] Their method was to inject a pulverized purée of diseased brain tissue into the brains of two monkeys. One monkey died and the other was sickened. Proof of poliovirus causation was headlined by orthodoxy. This, however, was an assumption–not a proof–of virus causation. The weakness of this method is obvious to everyone except certain viropathologists and has recently been criticized by the molecular biologist Peter Duesberg regarding a modern-day attempt to establish virus causation for kuru, another CNS disease.[14] Since 1908, the basic test has been repeated successfully many times using monkeys, dogs and genetically altered mice. The injected material has even been improved–scientists now use a saline solution containing purified poliovirus. However, a crucial weakness exists–polio epidemics do not occur via injections of poliovirus isolate into the brains of the victims through a hole drilled in their skull–except, of course, in laboratories and hospitals.

If injection into the brain is really a valid test for causation then it should serve especially well as a proof for pesticide causation. I propose that pesticides be injected directly into the brains of test animals. If paralysis and nerve

degeneration subsequently occur, we then would have proved that pesticides cause polio.

Going further, towards much higher standards of proof than those used to prove virus causation, pesticides could be fed to animals and found to cause CNS disease. This has already been done with DDT and the histology of the spine and brain was poliomyelitis. Virus proofs require injection, often intracranial, to get any reaction from the experimental animal. It is axiomatic that a theory is only as good as its ability to predict future events. I predict that such a test would prove pesticides to be the most reliable causative factor.

The injection of purée of diseased brain tissue into the brains of dogs was the method preferred by Louis Pasteur to establish virus causation with rabies, another CNS disease. A recent, definitive biography of Pasteur finds him to be a most important publicist for germ theory, a crucial promoter for the notion that rabies is caused by a virus. Unfortunately, his rabies experiments were biased and unsupported by independent studies.[15]

Therefore, in my opinion, even a cofactor theory, where pesticides catalyze predatory poliovirus activity, or where pesticides weaken the immune system to allow opportunistic predatory poliovirus activity, cannot stand up to simple, common sense explanations that include the concept of a symbiotic virus. Neurotoxins are enough of a cause for neurological disease.

The most obvious theory–pesticide causation–should be the dominant theory. But the opposite exists, a pervasive silence regarding pesticide causation juxtaposed against a steady stream of drama regarding virus causation. In light of the evidence presented herein, the silence could ultimately discredit mainstream medical science, institutions of the environmental movement, and the World Health Organization.

LEFT: Before 1950, DDT was hailed as a miracle of progress that was virtually nontoxic to humans, in spite of the FDA's attempts to keep it off the market. The photo above is one of several similar photos from *DDT: Killer of Killers*, 1946, by O. T. Zimmerman, PhD, and Irvin Lavine, PhD.

BELOW: Zimmerman and his colleagues advised that DDT be sprayed directly on dairy cows, their feed, bedding and water, in a 5 percent solution. Dairy products are ideal pesticide carriers because they are emulsions of fats and water. DDT is a waxy organochlorine with an affinity for fats, and is efficiently carried by dairy products through the digestive tract into the organs. In the mid-1940s the FDA advised against the use of DDT, especially on dairy farms. However, the pressure of the industry to promote DDT was overwhelming and the FDA's advice went unheeded.

Years later, the compelling nature of the evidence ensured the passage of stronger labeling laws and restrictions from use on dairy farms. In the early 1950s, scientists at the US Department of Agriculture found that although fodder treated with DDT caused no damage to the cows eating it, the health of their calves was severely impaired, sometimes with fatal results. The DDT was passed along from cow to calf via the milk (Van Nostrand's Encyclopedia of Science and Engineering, Van Nostrand Reinhold 1995, v 5, p1775). DDT is a neurotoxin and the calves developed something very much like infantile poliomyelitis. Calves weren't the only infants drinking cow milk during the early 1950s.

Virus Presence

When the symptoms of polio are recognized, there is often a claim of virus presence in the body of the polio victim. Sometimes a virus is found. Sometimes that virus is an enterovirus (a virus of the digestive tract). Sometimes that enterovirus is a poliovirus. During polio epidemics, orthodoxy blames the poliovirus, and therefore, in my argument for the innocence of the poliovirus, it is necessary to explain the claims of virus presence and the actual presence of the poliovirus.

First we must consider the economic motivation. During the great epidemic of 1942-1962 polio victims were diagnosed with poliovirus-caused polio, regardless of whether or not the poliovirus was found, because the NFIP (March of Dimes) funds paid only for this kind of polio. Therefore, if patients were going to spend time hospitalized, in iron lungs and undergoing therapy, it would have been economically imperative for the hospital to diagnose them in this way.[16] Thus, presence of poliovirus in poliomyelitis was rarely determined in order to arrive at a diagnosis of polio.

Even if one believes in virus culpability, other viruses are also claimed by orthodoxy to be the cause of polio-like CNS diseases that are "clinically indistinguishable" from polio. During the 1940-50s, relatively few polio victims were confirmed technically for presence of the poliovirus. In 1958, a laboratory analysis of 222 diagnosed polio victims of the Detroit epidemic found poliovirus in only 51 percent of the cases.[17] In other studies, lab tests for multiple

pathogens reveal that a mix of pathogens, multiple viruses, fungi, and bacteria can be associated with a single diagnosed case of polio.[18]

Coxsackievirus and echoviruses can cause paralytic syndromes that are clinically indistinguishable from paralytic poliomyelitis.[19] These “polio” cases are usually categorized as encephalitis or meningitis during a proclaimed polio epidemic.

Benign Virus?

The poliovirus is considered to have been endemic throughout the world going back to ancient times, yet this is not the case with paralytic polio. According to Arno Karlen, author of *Man and Microbes*, the “polio virus lives only in people; it probably adapted to the human small intestine countless millennia ago.” He continues, “. . . some historians have claimed that [paralytic] polio goes back to ancient Egypt; it may, but the evidence is thin.”[20]

Karlen makes a lot of sense here in view of the pesticide graphs, Biskind’s arguments and ancient statements regarding paralysis from inhalation of vaporized chemicals during blacksmithing operations. However, Karlen goes on to write that “the first undisputed case dates from the late eighteenth century.” This statement, however, must be invalid (in its attempt to establish polio images that have a basis in early history) because of Menkes’ statement (above) that other viruses can also be causative for polio symptoms and because common industrial poisons such as arsenic and lead compounds can cause polio-like symptoms. Poisoning, as a method of assassination has also been frequently employed. It is not unreasonable to assume that unsuccessful poisonings may have left their victims paralyzed. Thus, Karlen’s offer of an undisputed case as early as the late 18th century can be no more than a guess.

Orthodox medical literature can offer no evidence that the poliovirus was anything else than benign until the first polio epidemic, which occurred in Sweden in 1887. This small epidemic occurred 13 years after the invention of DDT in Germany, in 1874, and 14 years after the invention of the first mechanical pesticide crop sprayer, which was used to spray formulations of water, kerosene, soap and arsenic. The epidemic also occurred immediately following an unprecedented flurry of pesticide innovations. This is not to say that DDT was the actual cause of the first polio epidemic, as arsenic was then in widespread use and DDT is said to have been merely an academic exercise. However, DDT or any of several neurotoxic organochlorines already discovered could have caused the first polio epidemic if they had been used experimentally as a pesticide. DDT’s absence from early literature is little assurance that it was not used.

We need to remember that the poliovirus is an enterovirus. There are at least 72 known enteroviruses discovered to date. According to Duesberg, many enteroviruses are harmless “passenger viruses.”[21] In view of the material presented here, probably unknown to Duesberg, it is reasonable that we also view poliovirus as harmless outside of extreme laboratory conditions.

The Symbiotic Poliovirus

Having now established the possibility of an innocent poliovirus, its presence in polio can be explained as follows: accelerated genetic recombination. Genetic recombination is accelerated whenever a biological system is threatened[22] and pesticides can be that threat. The proliferation of viruses can be part of the process of accelerated genetic recombination.

When a cell is critically threatened, accelerated genetic recombination (which may include virus proliferation) is just one of a set of events that may occur. This set of events is called the “SOS response,” which is known to be triggered by exposure to toxic chemicals or radiation.[23] Arnold Levine, writing in *Field’s Virology*, provides an example: “When lysogenic bacteria were lysed [split open] from without, no virus was detected. But from time to time a bacterium spontaneously lysed and produced many viruses. The influence of ultraviolet light in inducing the

release of these viruses was a key observation that began to outline this curious relation between a virus and its host."[24]

It is ironic that common medical procedures such as chemotherapy, radiation therapy, and the use of toxic pharmaceuticals accelerate genetic recombination and thus the potential for a necessary virus proliferation.

The SOS response is utilized in the Ames Assay Test, a standard test whereby chemical toxicity is determined. According to the procedure, bacteria are exposed to a chemical solution in question, and if a genetic recombination accelerates via the spontaneous proliferation of viruses from these bacteria, then the chemical is determined to be a poison. The phenomenon is analogous to a poker player with a bad hand who must request an exchange of cards and a reshuffled deck to improve the possibilities for survival. In the Ames Assay Test, bacteria are concerned with their genetic "hand" in order to improve their abilities to metabolize poisons, create utilizations for poisons, and shield against poisons. Thus they engage in this well-known phenomena of "gene shuffling," facilitated by virus proliferation.

Thus, I propose that the poliovirus is a symbiotic (and possibly a dormant) virus that behaves in a manner suggested by the phenomenon found in the Ames Assay Test, a test used to determine toxicity.

Conclusion

The word "virus" is ancient Latin, meaning "slime" or "poison." Mainstream science admits that most viruses are harmless, yet the word "virus" adds to a biased and highly promoted language of fear regarding nature. Definitions of viruses range from "pathogenic" to "not usually pathogenic." The more popular the media source, the more frightening the definition. Less fearful definitions would change the relationship between the medical industry and its "patients."
Paradoxically, early virus studies considered virus filtrates to be a poison, not a microbe, thus the name virus. Today, we know that viruses are information.

Now, nearly a half-century later, the validity of Dr. Biskind's work appears even more certain. Biskind's warning bears repeating:

> *"It was even known by 1945 that DDT is stored in the body fat of mammals and appears in the milk. 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."*

The unique correlations between CNS disease and CNS poisons present a variety of research opportunities not only in medical science, but political science, philosophy, media studies, psychology, and sociology.

Sidebars

Polio and Poison

A search of MEDLINE ("polio" and "poison") finds about 45 contemporary articles where polio-like disease is attributed to poison. This recognition of the relationship between polio and poison is restricted to the agriculture

industry–animals cannot hold industry liable. The terminology found includes the following:

polioencephalomalacia
poliomyelomalacia
polyradiculoneuritis
polioencephalomyelomalacia
lumbal poliomyelomalacia
multifocal-poliomyelomalacia
spinal poliomalacia
polio and high-sulfate diets
bovine polioencephalomalacia
neurological picture similar to that of poliomyelitis
cerebrocortical necrosis (polioencephalomalacia)
lead poisoning in grey-headed fruit bats (Pteropus poliocephalus)
atypical porcine enterovirus encephalomyelitis: possible interaction between enteroviruses and arsenicals
Polioencephalomalacia and photosensitization associated with Kochia scoparia consumption in range cattle

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Food for Thought

Polio outbreaks occurred most often during the summer and were blamed on viruses picked up in swimming pools. But summer was the time when DDT spraying was at its peak and exposure would have been greatest, either directly or through foods from animals eating sprayed crops. Summer foods like ice cream from DDT-sprayed dairy cows would have been a likely source.

In developing countries, polio is blamed on poor sanitation. But in the United States, polio was blamed on lack of immunity due to good sanitation!

Until the advent of AIDS, polio was the only disease whose cause was enshrined in legislation. US public health law stated that poliomyelitis was an “infectious contagious disease,” yet proof of poliovirus causation is astoundingly weak, and the obvious toxicology was entirely avoided.

The man most responsible for the view that poliomyelitis was contagious was Dr. Simon Flexner, author of the famous (or infamous) Flexner Report, which led the way to the closing of the naturopathic and homeopathic colleges in the United States. Said Flexner: “It was not easy to establish in an individual case precisely how the disease was acquired; it was difficult to bring evidence that was not at all convincing that this disease was contagious.” In discussing Flexner’s report, L. Emmett Holt stated: “Even five years ago, if anyone had suggested that the disease under discussion was an infectious or contagious one, it would have been looked upon as a joke (Scobey, *Archives of Pediatrics*, May 1951).

In 1953, Dr. Kumm was appointed Director of Research of the National Foundation for Infantile Paralysis (NFIP). The NFIP was funded by its “March of Dimes” program, and it sponsored the hasty development of the Salk vaccine in the early 1950s, at the height of the DDT/polio controversy. Dr. Kumm also “served as a civilian consultant to the Surgeon General . . . directing field studies of the use of DDT. . .” (*American Journal of Digestive Diseases*, 1953 20:330).

The World Health Organization directs both DDT application (for mosquito control) and polio vaccination worldwide.

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