

THE LONG BATTLE AGAINST DUTCH ELM DISEASE¹

by Charles L. Wilson

We are periodically reminded by newspaper headlines that a research battle is being waged against Dutch elm disease (DED). Few of us realize the extent of this battle or that many scientists have already dedicated their entire lives toward controlling this dread disease. There is also little awareness that DED was and is now a problem also in Europe, where it had destroyed elm populations before it was even known in this country.

The American people have been given repeated hope of possible control of DED. But meanwhile, the disease has been killing increasing numbers of our elms. In fact, so many false hopes have been raised that there is apathy in some quarters about the outcome of our battle against this disease.

My purpose in this article is to present a historical account of the research that has been done on DED and to assess the present status of the battle. There have been concrete developments in the control of DED in the past few years, and there is every reason to be optimistic. A full appreciation of what has been done can come only through knowledge of the legion of researchers that have and are still putting forward the good fight.

Discovered in Europe around 1919

In 1921 the first description of a dying elm was made by the Dutch scientist, Dr. Spierenburg, who had followed the disease since 1919. She was the first of a long line of women scientists in Holland who contributed to our understanding of DED. After it was recognized that there was an important killer of elms in Europe, a period of confusion as to its cause followed.

Dr. Spierenburg followed her original announcement of the disease with a description of

the bacteria and fungi associated with the diseased wood. Some weeks later, Dr. Beatrice Schwarz, in her doctoral dissertation, described in precise detail a new fungus associated with the disease, which she named *Graphium ulmi*, much later to be renamed *Ceratocystis ulmi*. Mrs. Schwarz inoculated trees with her new-found fungus, but was unable to bring about the disease. However, she still considered *Graphium ulmi* to be the causal agent. She inoculated her trees in the fall, when elms are not very susceptible as later research revealed. Had she inoculated trees in late spring or early summer, when trees are highly susceptible, she might have clearly shown *Graphium ulmi* to be the cause of the disease and prevented the confusion that followed.

Dr. Brussoff, in Aachen, Germany, published an article in 1924, in which he reported that the elm disease was caused by a bacterium, *Micrococcus ulmi*. Professor Johanna Westerdijk, head of the Phytopathologisch Laboratorium in Holland, made the following comments about Brussoff's work: "His inoculation experiments, which he considered positive, prove nothing: he inoculated with bacteria into the stems of large trees and later noticed that some branches on top died during the summer. If a tree treated in such a manner is not dissected after being cut down, and a connection between the place of inoculation and the diseased branches on top becomes evident, the experiments are not convincing."

In 1926, Brussoff published a series of articles that created considerable public concern. He indicated that the elm bacterium was also attacking many other species such as maple, linden, birch and poplar. Dr. Johanna Westerdijk, in 1929, stated that, "everyone becomes terrified

¹Information for this paper was obtained from unpublished records at the Agricultural Research Service Shade Tree and Ornamental Plants Laboratory, Delaware, Ohio.

when he reads in papers with wide circulation, such as *Umschau*, that the roots of nearly all species of our trees are infected with a bacterium."

As a result of all this confusion, the Biologische Reichsanstalt at Berlin-Dahlem urged Dr. Stapp, a bacteriologist, and Dr. Wollenweber, a mycologist, to investigate this problem.

In March, 1927, Stapp and Wollenweber in Germany and Westerdijk and Buisman in Holland began research projects that would define clearly the cause of DED. By October, 1927, Wollenweber had confirmed Schwarz's conclusion that *Graphium ulmi* was associated with Dutch elm disease. He observed that the fungus entered the wood after inoculation and noticed a withering and drying of seedlings of *Ulmus montana* five weeks after inoculation. Wollenweber was able to reisolate *G. ulmi* from the typically discolored streaks that developed upward and downward from the point of inoculation and thus satisfied Koch's rules of proof for a pathogen.

Concomitant with Wollenweber's work, Stapp obtained some of Brussoff's isolates, and was unable to produce a single infection. Commenting on Stapp's work, Johanna Westerdijk declared jubilantly that *Micrococcus ulmi* does no longer exist! Brussoff argued frantically against Stapp's findings. However, the decisive blow was delivered when Stapp was unable to isolate bacteria from diseased trees.

Westerdijk and Buisman in 1929 published their findings of the previous three years, which confirmed their original contentions and reinforced the work of Wollenweber and Stapp. Thus, at the end of 10 years' study and controversy, the cause of Dutch elm disease was clearly established. However, there was a period before public acceptance of these findings. Westerdijk and collaborators reflect on the public attitude toward their research in the following statement, "The Dutchman worries about his elms; he does not like to be told that the elm disease is of an infectious nature and is quite pleased when infection experiments are criticized; because now he may fall asleep under the impression that it means only temporary trouble for the elms." The trouble was not temporary, for DED was soon to

become a problem throughout Europe. In Holland alone, more than 50% of the elms would be killed by 1939.

Although the cause of DED was clearly established, other important questions remained unanswered. It was not known how the pathogen was spread or how it could be controlled. Johanna Westerdijk and Christine Buisman thought originally that the pathogen was probably airborne.

Dr. J. J. Fransen, for his doctor's dissertation, studied the vectors of DED under the direction of Professor Roepke at the Laboratorium voor Entomologie in Wageningen. He found that the DED fungus was closely associated with the galleries and pupal cells of two species of "elm bark beetles." He was able to get these insects to transmit the disease when they were artificially contaminated with the DED fungus. After this significant contribution, Professor Roepke decided to discontinue these studies because he felt they "offered little hope of anything practical for control."

Armed with this significant information on the means of spread of the DED fungus, scientists then had a basis from which to formulate possible control practices. A logical approach was to reduce the insect vector population by destroying breeding sites of the beetles. Since the beetle species that were vectors reproduced only in dead and dying elm wood, the battle line was considerably narrowed.

The Dutch, through a royal decree based on the Forest Law of 1921, set out to control DED. The royal decree gave authority to condemn such elms as were considered dangerous in the spreading process of the disease. The tree, once condemned, had to be removed and treated by the owner. Treatment consisted of the removal and destruction of the bark, or submergence of the log for three months. Scouts were sent all over Holland to mark dead or dying elm trees with one or two crosses. Those with two crosses were already infected with the beetles and were to be removed immediately. This plan was not expected to stop the disease but was devised as a holding action until resistant trees could be developed and planted.

The Dutch decided early in their battle against

DED to rely on the development of resistant trees as their main line of defense. This turned out to be a good tactic, as DED is no longer a major problem in Holland because of the extensive planting of resistant trees.

The Dutch breeding and selection program for elms was initiated in 1928. It was supervised by an Elm Disease Committee, which made decisions on the release of trees to the public. This Committee was under considerable pressure to release trees, and in 1936 it considered two clones for possible release. The Committee decided to issue clone 24 under the name 'Christine Buisman.' The 'Christine Buisman' elm began life the year that scientist Christine Buisman died. Unfortunately, its release was too hasty because this selection was not a suitable tree for the Dutch. Hans Heybroek, who presently heads the Dutch breeding program, states that this release "became a failure in our country and the few trees that remain, from the many planted in those years, provide a sorry sight today." In spite of their shaky beginning, the Dutch went on to develop many acceptable clones of elms resistant to DED.

Dutch elm disease was soon recognized throughout Europe. Control measures similar to those devised by the Dutch were tried in England, France, and Germany, but with no great impact in preventing the spread of the disease. The disease did not appear to be quite as severe in the other European countries as it was in Holland. This was explained by the fact that the Dutch had planted primarily one highly susceptible clone of elm, *Ulmus hollandica* var. *Belgica*, whereas there was a greater variety of elms in the other countries, with varying degrees of resistance.

In the late 1920's, DED somehow made it past the Alps into Italy. During the summers of 1930 and 1931, sporadic cases of DED occurred in the Emilia region. These occurrences did not attract much attention, because for two seasons it had been exceedingly dry, and elms were thought to be dying from the drought. After attacks in the Padana Valley, DED was soon found in Tuscany, Marches, Umbria and Lazio. It was not long before the disease had reached southern Italy.

Dutch elm disease had a greater impact in Italy than in many other European countries because, in addition to its forest and ornamental value, the elm was also used as a living support for vineyards. Also, elm foliage was harvested and fed to cattle in the summer when pastures were poor. In some of the districts of Emilia, the land was not valued according to acreage, but according to the number of elm trees on it.

The loss to the Italian grape industry because of DED was considerable. Arturo Ansaloni dramatically stated the situation when he said, "The vine wedded to the elm has become a widow."

Mr. Arturo Ansaloni, a former peasant who became a wealthy nurseryman, had set out when DED was first found in Italy to promote the use of the Siberian elm, which is resistant to DED. The highly susceptible *Ulmus foliacea-nitens* had been used almost exclusively. Mr. Ansaloni relied heavily on Professor Goidanich, who was Italy's early authority on DED. Professor Goidanich held reservations as to whether just one species should be relied on to replace the dying elms. Nevertheless, Mr. Ansaloni worked diligently to get growers to use Siberian elms. He, of course, was in the business of selling these trees.

Introduction into the United States

At the same time that the DED fungus was slithering over the Alps, it was taking a giant step across the Atlantic. Dr. Curtis May, a young research technician at the Ohio Agricultural Experiment Station at Wooster, found several cases of DED in 1930 in Ohio. Dr. May was fortunate in that Dr. Christine Buisman of Holland was visiting the Arnold Arboretum, in Massachusetts, and she was able to confirm his diagnosis. No one at that time knew what devastation DED was to cause in this country. These few diseased trees gave no indication that Chicago, Illinois, alone by 1970 would have to spend 5 million dollars just to remove trees killed by the DED fungus. Alarmed by the potential destructiveness of DED, researchers held a special conference on October 26, 1931 in Washington, D.C. A report in the *Forestry News Digest* gives the following details of that meeting:

The present status of the disease was reported. The infection in Ohio seems to be under control. The new outbreak found this year around New York City on Oct. 27 consisted of 628 trees in New Jersey, 48 in New York and one in Connecticut. A third independent infection has been found at Baltimore, Md.

During the summer it was discovered that the disease has been crossing the Atlantic Ocean and entering the United States in elm logs imported for cutting fancy veneer.

It is now possible to understand the present known distribution of the disease in this country. The Baltimore infection is not far from the piers where imported logs were unloaded; the Cincinnati tree is in a city where a veneer plant which has imported burl elm is located; the Cleveland trees are near a railroad which hauled imported logs; the New York City infected area surrounds the piers where several shipments have arrived and its most heavily infected section is penetrated by log transporting railways.

Primarily through the detective work of Drs. Beattie and Verrall, we now had an idea as to how DED was introduced into this country, but the big question was, "Could we realistically stop its spread?" The arm-chair quarterbacks for chestnut blight, which rapidly wiped out the American chestnut, had chided the plant pathologists for not starting soon enough to try to control this disease. Everyone was determined not to let this happen again.

A small group of plant pathologists and entomologists joined Dr. May in 1933 to set up a Federal laboratory as a command center for the war against DED. The laboratory that was formerly at Wooster, Ohio was moved in 1934 to Morristown, N.J., which was closer to known concentrations of the disease. Out of this laboratory, a control and research program was initiated. Notable among the early warriors at the Morristown laboratory were Drs. True, Swingle, Moses, Myers, Verrall, Banfield, Moak, Smith, Smucker, Wester, Ahrens, Clarke, Helmich, and Tharp. Drs. Parker and Walter were sent to England, where they set up studies at Oxford to monitor and experiment with DED under European conditions.

All wars are expensive, and this one was to be no exception. Various forestry organizations, and the public in general, put pressure on the Federal government to do something. The Providence Journal in July 1935 carried the following headline: *ROOSEVELT ALLOTS \$2,500,000 FOR FIGHTING DUTCH ELM DISEASE.*

A small army had been assembled at Morris-

town, N.J., and funds were available to operate. But, how were they to meet the enemy? Based on the information that was available, a decision was made to try to eradicate DED by locating and destroying all infected trees. Willing workers were not hard to find, since those were depression days, and members of the CCC made natural recruits. Early in February, 1935, 1,287 men, divided into 120 crews, were at work felling and destroying diseased and suspected elm trees in New York, New Jersey and Connecticut.

Among the researchers at Morristown, there were doubts as to the feasibility of an eradication program, but no one dared say they should not try. There were still many gaps in the knowledge of DED that might undermine the biological soundness of this approach. For instance, could the DED fungus survive in places other than in elm trees? How long could the fungus survive in root systems of diseased trees? Were there symptomless carriers of the DED fungus? Experiments were designed to answer these questions. But funds for experimentation were limited since so much money was required for the eradication program.

By the end of February in 1934, it was reported that all trees definitely known to be infected with the Dutch elm disease had been destroyed. The big question was what was going to happen next spring. Those in the field knew what was going to happen. They knew that it was an impossible job to detect all diseased trees, even in the areas where they had concentrated in New Jersey and New York. How about all the unknown infection centers outside of these areas?

The spring of 1935 found DED still present. Eradication efforts continued, but the disease rapidly gained ground. During the first four and one-half years that the Federal Elm Disease Eradication Program was in effect, 28,319 diseased trees were "eradicated", but DED was not. By 1938 DED was known in New Jersey, New York, Connecticut, Pennsylvania, Indiana, Ohio and West Virginia. There are still some who argue that if ample support had been provided, we could have eradicated DED. But our inability to detect all trees having DED would seem to have made this task impossible. By the late 1930's, our preoccupation with Hitler's activities

in Europe caused a cessation in our war against DED.

The outbreak of the Second World War put the brakes on research on DED. The disease continued to spread during the war. Some felt that spread of the disease in Europe was accelerated by the destruction of elms by artillery fire. These dead and dying trees increased the vector population and thus the chances for spread. The greatest impact of the war on DED was the break in continuity of research programs. Ideas and resistant plant material were lost both here and in Europe.

The post-World War II era

The war also brought forward a chemical that has had, and continues to have, considerable public attention — DDT (1, 1, - trichloro-2, 2-bis (p-chlorophenyl) ethane). Until DDT came along, it was not feasible to consider controlling DED by controlling the insect vector. DDT was a highly effective, residual insecticide that would persist long enough to protect elms from vectors of DED. Early in 1944, small amounts of DDT were made available to R. R. Whitten at the Morristown, New Jersey laboratory. Preliminary tests showed that it was very effective in protecting elms from the feeding of the lesser European bark beetle, *Scolytus multistriatus*. Subsequent tests showed that DDT was effective in preventing the feeding of the other known vector of DED in the USA, the North American elm bark beetle, *Hylurgopinus rufipes*.

Our main approach to control up until recently was based on this information. This approach involved the destruction of all dead and dying elms (sanitation) and the protection of healthy elms with DDT. Such control programs have been adopted extensively on a community-wide basis across the United States. Their effectiveness is difficult to assess since all trees in the community are treated, and no untreated "checks" are left for comparison.

It is apparent that the sanitation - spray program has not stopped the spread of DED. It is now present throughout the natural range of the American elm. The disease has spread as far west as Idaho and Colorado and appears to be starting its climb over the Rockies.

After World War II, the Federal effort against DED continued. However, research suffered from poor funding and bureaucratic reshuffling. The laboratory in New Jersey was relocated in 1946 at Columbus, Ohio. This was to facilitate the investigation of another debilitating disease, elm phloem necrosis. The lessor of the facility at Columbus failed to renew the lease, so in 1949 it was necessary to move the lab and its nursery to Lockborne Air Force Base. Soon thereafter the air base was reactivated, and about a year later the lab was moved back to Columbus. In 1960, a Federal laboratory was built at Delaware, Ohio on 248 acres of land. This and the National Arboretum are the current locations of most of the Federal research on DED and other urban tree problems.

Current status of our knowledge

Through research at U.S. universities, Federal and state laboratories, many gaps in our knowledge of Dutch elm disease have been filled. Among the universities, research at the Connecticut Agricultural Experiment Station, University of Wisconsin, University of Massachusetts, Iowa State University, Cornell University, Illinois Natural History Survey, and Virginia Polytechnic Institute and State University is notable. Other research has been done in university and government laboratories in Canada.

We have learned much about the insects that carry the DED fungus and the activity of the fungus in diseased trees. Our knowledge has led primarily to one approach in control, which is control of the disease through control of the insect vector. Community-wide control programs have been recommended to control the beetle vectors of DED. In some areas these practices have reduced the incidence of DED within a community.

It also was discovered early that the DED fungus could move from diseased to healthy trees through underground root grafts. There have been a number of attempts to control spread by mechanically breaking or chemically inactivating functional root grafts between trees. None of the methods developed, so far, has been completely satisfactory.

Resistant Trees.—Programs to develop trees resistant to DED in the United States have had many false starts. In our fast-moving economy, it is difficult to sell programs that may not produce results for 20 years. The Christine Buisman elm was introduced early into this country from the Netherlands, but its limited adaptability restricted its use.

In 1965, the Dutch clone #296, called the Groeneveld (or Greenfield) Elm was released from its post-entry quarantine for U.S. use. It is more resistant to DED than the Buisman Elm, but has not yet been tried in all parts of our country. Fortunately, projects to develop resistant elms have now been funded by the U.S. government, certain universities and private foundations. The first hybrid elm resistant to DED developed in this country was released this year by the Agricultural Research Service of the USDA. American elm selections that are resistant to DED have also been identified in several state and Federal research programs, and it is hoped that these will be available to the public soon.

A fatalism about DED has retarded efforts to develop trees resistant to the disease. Some people have said, "Let the elm go, we have other trees." These people fail to realize the many attributes of the elm. The fact that elms have been planted so extensively attests to their desirability and adaptability. A solution to the DED problem would allow us to preserve our most useful shade tree. Surely it is worth the trouble.

Also, many fail to realize the versatility of the elm. Elms come in various sizes and shapes to fit most requirements for landscape plantings, and some elms have a relatively high degree of resistance to DED.

Control of the Insect Vectors.—Recent banning of DDT forced a look at other means of controlling the vectors of the DED pathogen. Some new approaches involve the introduction of parasites of the beetle-vectors and manipulation of the vectors through sex attractants and sterilants.

A fundamental question remains to be answered concerning control of DED by control of the vectors. This question is: "Just how much insect control is needed to have a major impact on DED losses?" Unfortunately, most of our data has shown what certain practices do to the vectors, and little attention has been given to what these practices do to the disease.

Systemic Fungicides.—Ideally, we would like to detect DED in early stages and arrest its development. It is only recently that any hope was held that this might be accomplished. The recent development of a number of systemic fungicides, which move effectively throughout plant cells, has increased prospects that we might actually "cure" trees infected by the DED agent.

A systemic fungicide called benomyl has shown the most promise so far. This compound moves readily in elm trees, and it is highly toxic to the DED fungus. Recently, scientists have developed a pressure-injection system that can be used to apply large volumes of a systemic fungicide into trees.

The battle against DED is not won. But, after 40 years of combat, there is considerable room for optimism. In the opinion of the author the most promising assaults on this disease have been the use of resistant trees and the development of systemic chemicals that will protect trees against the fungus. Research in progress may provide us with additional weapons. If only public trust and support can be kept until these thrusts begin to show their effects!

ARS, USDA, North Central Region, and
Ohio Agricultural Research and
Development Center,
Wooster, Ohio