DUTCH ELM DISEASE

DEPARTMENT OF FORESTRY AND RURAL DEVELOPMENT

CANADA

Published under the authority of
The Honourable Maurice Sauvé, P.C., M.P.,
Minister of Forestry and Rural Development
Ottawa, 1967.

ROGER DUHAMEL, F.R.S.C.
QUEEN'S PRINTER AND CONTROLLER OF STATIONERY
OTTAWA, 1967

Cat. No.: Fo 23-1067

DUTCH ELM DISEASE

Compiled and edited

by

A.G. Davidson

Reprinted from Bi-Monthly Progress Report
Vol. 20 No. 4 (1964)

DUTCH ELM DISEASE

FOREWORD

This report consists of a series of articles on the Dutch elm disease prepared by officers of the federal Department of Forestry and Rural Development and the federal Department of Agriculture. It has been prepared primarily to meet demands for an up-to-date review of the disease and, in addition, to indicate the direction in which current research is proceeding.

The Dutch elm disease, so-named because of the early and excellent studies by pathologists in the Netherlands, is caused by a fungus, Ceratocystis ulmi (Buism.) C. Moreau, that is transmitted by bark beetles. It was accidentally introduced into Canada and is currently causing extensive mortality of white elms in Quebec, Ontario, and New Brunswick.

At present, it is practically impossible to save an infected tree by pruning the diseased branches, spraying, or by any method known. Control of the disease is based, therefore, on prevention rather than cure. The role of bark beetles in the transmission of the causal fungus provides two ways by which infections can be prevented or reduced. Because of the costs involved, these measures can be applied only in urban areas for the protection of high value trees. The first prevents or greatly lessens feeding by bark beetles through the use of insecticides; the second reduces the bark beetle population by destroying elm material used as breeding sites by the beetles, that is, by sanitation. There has been some difference of opinion as to the relative effectiveness of the two methods but for best control, both should be used as one complements the other. Although the most carefully devised and executed program will not eradicate the disease, losses can be kept within reasonable limits. - A.G. Davidson.

HISTORY AND DISTRIBUTION

The Dutch elm disease was first observed in the Netherlands and northern France in 1919 (66). The origin, date, and mode of introduction of the causal fungus were not determined but circumstantial evidence indicated Asiatic origin. The disease rapidly invaded most European countries and spread into central Asia (64).

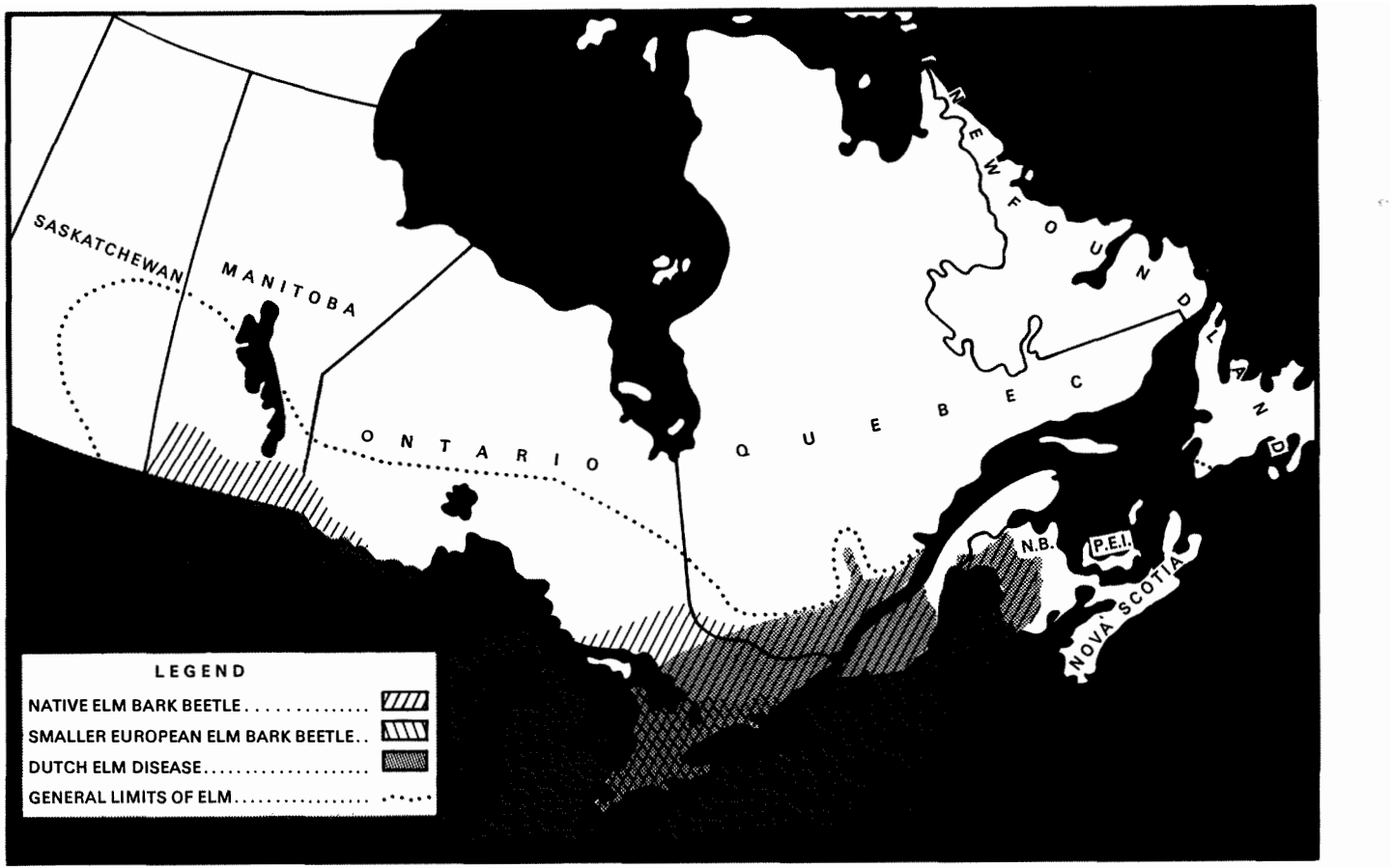

Figure 1. The known distribution of the Dutch elm disease and vectors in Canada, 1963.

The first cases of the disease in North America were discovered and identified in Ohio during 1930 and in northern New Jersey in 1932 (15), the causal fungus having been introduced in elm burl logs imported for the manufacture of veneer (6). By 1961, the disease occurred over some 600,000 square miles in the United States, a large part of the elm range north of Maryland and east of Montana, Wyoming, and Oklahoma (39).

In Canada, the first infected trees were found in St. Ours, Richelieu County, Quebec, during August 1944 (59). When the distribution of the disease in Quebec became known in 1945, it was apparent that the port of Sorel was the centre of the infection and subsequent analysis of early distribution records indicated that infectious material probably was introduced in that area before 1940 (58). Since the Quebec outbreak was separated from the known northern limits of the disease in the United States by more than 200 miles, it is reasonable to think that the pathogen was introduced by ship, probably from Europe, on crates made of diseased elm wood. These would have evaded quarantine regulations designed to prohibit the entry into Canada of elm and elm products from Europe and the United States that were placed in effect in 1928 and 1934, respectively. By 1959, the disease was prevalent in 55 Quebec counties and covered an area of about 24,800 square miles. Between 1954 and 1959 the disease spread at the rate of 1,200 square miles per year and caused the death of 600,000 to 700,000 elms (58). In 1962, infected trees were found in Temiscouata and Bonaventure counties along the New Brunswick border and removed from the main Quebec outbreak (51). This apparently represented a spread of the disease from New Brunswick. By 1963, the disease occurred over 46,000 square miles or 80 per cent of the range of elm in Quebec (Figure 1), the non-infected area including districts where elms are scarce and scattered.

The first infected tree in Ontario was found at St. Isidore, Prescott County, in 1946. This was a predictable extension of the Quebec outbreak. In 1950, the disease reached the Niagara Peninsula and Windsor from the United States. The disease now occurs in 46 counties south of a line between the northern part of Georgian Bay and Mattawa on the Ottawa River (18), an area of approximately 64,000 square miles (Figure 1). During its 17-year history in Ontario, the disease has spread at the rate of 3,700 square miles per year -- three times faster than in Quebec. This rapid rate of spread is attributed to the fact that the disease entered Ontario at three points in areas of high elm populations.

The first diseased tree in New Brunswick was found in November 1957 at Woodstock on the St. John River (4), the causal fungus apparently having been introduced from Maine. By 1963, diseased trees had been found in 9 of the 15 counties in New Brunswick (45) and the outbreak occurred in river valleys of the western half of the Province (Figure 1). This represents a spread of about 2,200

square miles per year, probably from a number of points along the Maine border.

The most important single factor affecting the progress of the disease appears to be the concentration of elm trees (58), and future spread is expected to occur mainly in areas of high elm concentrations. If the disease reaches other areas, it is unlikely to cause important damage because elm trees are scattered and the vectors are absent or occur in low numbers. Consequently, further spread in Quebec will be slow and restricted and according to Dance and Lynn (19), extensions of the disease in Ontario are likely to be confined to river valleys and to a narrow band bordering the northern shores of Lake Huron and Lake Superior. However, it is expected that the disease will spread to river valleys throughout much of New Brunswick and reach the other Maritime Provinces within the next few years.

Although naturally-occurring elm has a restricted distribution along lakes and in river valleys in Manitoba and Saskatchewan, elm is an important shade tree and has been planted in concentration in urban areas. Because of the sporadic distribution of elm throughout northern Ontario, Dance and Lynn (19) consider it unlikely that the disease will reach the western provinces by natural spread through Ontario. In the United States the disease occurs in the Minneapolis-St. Paul area of Minnesota approximately 250 miles from the Manitoba border (48). Elms occur in several river valleys between St. Paul and Winnipeg but the relative sparseness of these elms will undoubtedly hinder natural spread to Manitoba. As suggested by Hafstad (37), long distance spread of the fungus is possible by infected beetles transported on motor vehicles. Surveys to determine the presence or absence of the disease are being maintained in Manitoba and Saskatchewan by the federal Department of Forestry and Rural Development and the Plant Protection Division, Canada Department of Agriculture. - R. Pomerleau.

SYMPTOMS AND DIAGNOSIS

External symptoms usually are evident by late June, but may appear later if the season has been retarded (Figure 2). They become most pronounced in July and August, and usually are more acute in young, succulent, vigorously growing trees than in slow-growing or senile specimens (69). Initially there is sudden wilting of the leaves on one or more limbs in the upper crown. These leaves turn dull green, dry out, and fall, or they turn brown, curl, shrivel, become brittle and remain attached to twigs for many weeks. When the tree is dormant, tufts of such leaves are symptomatic of the disease (69). From midsummer onward symptoms consist mainly of the development of yellowish leaves in one part of the crown or on occasional twigs. This has been termed "flagging" (75). Following

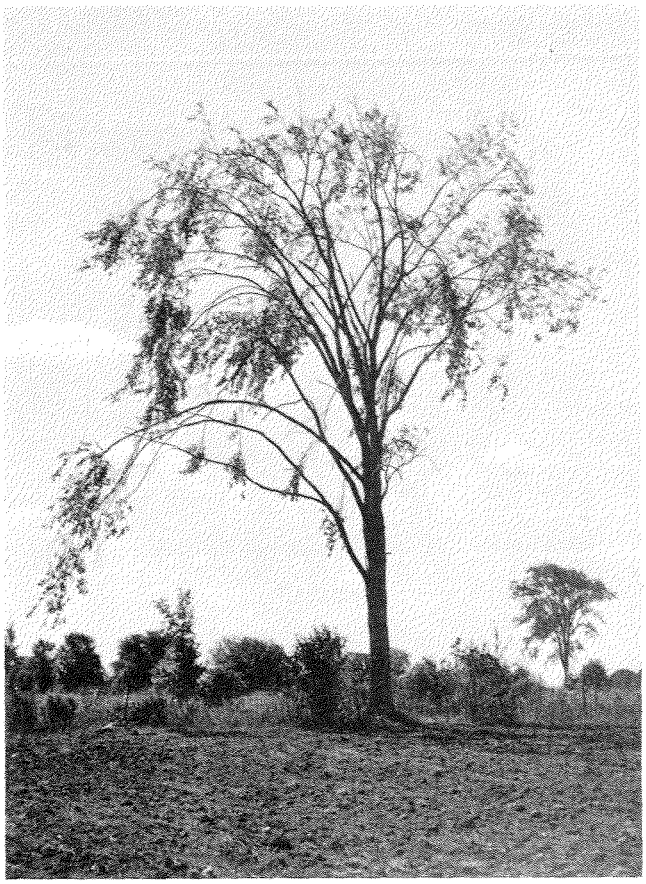

Figure 2. Diseased elm tree.

the development of foliar symptoms affected branches die and the condition extends to all other branches until the tree dies.

Wide variation occurs in the time required for the disease to kill individual trees. Small trees may die in a single season but in large trees the disease usually progresses more slowly and such trees may survive for a number of years (12). In the latter, the foliage is likely to be thin with undersized and yellowish leaves. At times, however, even large trees may be killed rapidly.

The internal symptoms of the disease occur in the outer sapwood (Figure 3) as long, discontinuous, brown streaks visible in longitudinal sections of infected branches and stems. In transverse sections the discoloration appears as dark spots, or a partial to complete ring. This staining results from the development of a dark, gummy substance (69), including spores (52), deposited in the large vessels formed in the spring.

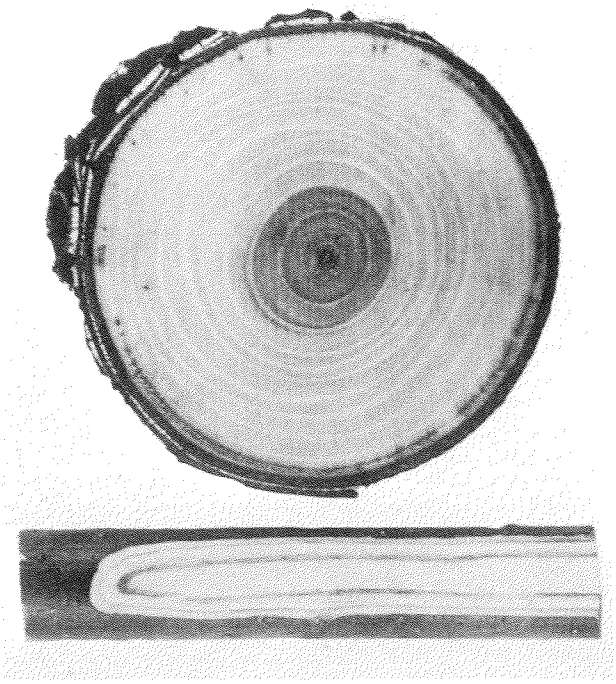

Figure 3. Diseased elm branch in cross and longitudinal sections, showing the brown ring and streaks.

A few instances have been recorded where recovery was indicated by the disappearance of external symptoms. These cases have been attributed to the ability of certain trees to seal-off infections under layers of more resistant summer wood (69). However, these trees remain susceptible to reinfection.

Because there are two less virulent fungous diseases with similar sumptoms, Verticillium wilt caused by Verticillium albo-atrum Reinke and Berth. and Dothiorella or "Cephalosporium" wilt, (sometimes called dieback) caused by Dothiorella ulmi Verrall & May, Dutch elm disease cannot be safely diagnosed from symptoms alone. Therefore, accurate diagnosis of the disease requires laboratory examination to identify the causal fungus.

If an owner suspects that one or more of his elm trees have Dutch elm disease, samples should be collected and submitted for diagnosis. Take samples from elm branches that have wilted leaves and the discoloration in the outer sapwood described previously. Several samples from ½ to 1 inch in diameter and from 6 to 7 inches long should be taken, preferably from more than one branch. Wrap the samples in waxed paper to prevent drying and send them to the

laboratory with as little delay as possible. In the Maritime Provinces send samples to the Forest Research Laboratory, P.O. Box 4000, Fredericton, N.B.; in Quebec to the Forest Research Laboratory, P.O. Box 35, Sillery, Quebec; in Ontario to the Forest Research Laboratory, Southern Research Station, Maple, Ontario; and in Manitoba and Saskatchewan to the Forest Research Laboratory, 25 Dafoe Road, Fort Garry, Winnipeg 19, Manitoba. - B.W. Dance.

VECTORS OF THE DISEASE

Two bark beetles, the native elm bark beetle, Hylurgopinus rufipes (Eichh.), and the smaller European elm bark beetle, Scolytus multistriatus (Marsh.), are the primary vectors of the Dutch elm disease. Two weevils, Magdalis armicollis (Say) and M. barbita (Say) are capable of transmitting the disease (62), but neither species is considered an important vector (34). The Forest Insect and Disease Survey has conducted intensive surveys to determine the distribution of the bark beetle vectors in Canada and the results of these operations are shown in Figure 1.

Until recently it was assumed that the native elm bark beetle occurred throughout the range of white elm. However, it is now known that, although the beetle occurs from western Manitoba to central New Brunswick, there are extensive areas along the eastern, northern, and western limits of the host tree where this insect has never been recorded. This may be due largely to the rather low incidence of elm in these regions.

The European elm bark beetle was first reported in the United States at Boston in 1909 (14), and in Canada near Windsor in 1948 (71). The spread of this beetle in Ontario has been recorded annually by the Survey (72), and a study of these records has provided information on the direction and rate of dispersion. The beetle has never been recorded outside of Ontario and at present its distribution is limited to approximately 22,000 square miles in the southern part of that Province. Over the past 15 years, the European beetle has spread about 20 miles per year to the north and east across southern Ontario. This is equivalent to its westward spread in the United States from Massachusetts to Minnesota (approximately 1200 miles) in 54 years. In contrast, the beetle has failed to move any appreciable distance northward in some areas. Along the north shore of Lake Ontario, the northern limit of distribution has remained virtually static since 1959. The same may apply in the New England States, for it is 27 years since the beetle was reported in southeastern New Hampshire, only 160 miles from the Quebec border (16). Dispersal northward is probably regulated to some extent by low winter temperatures, as demonstrated for another introduced pest in Ontario, the European pine shoot moth, Rhyacionia buoliana (Schiff.) (35). Nevertheless, the distribution of the European elm bark beetle will be kept under close surveillance

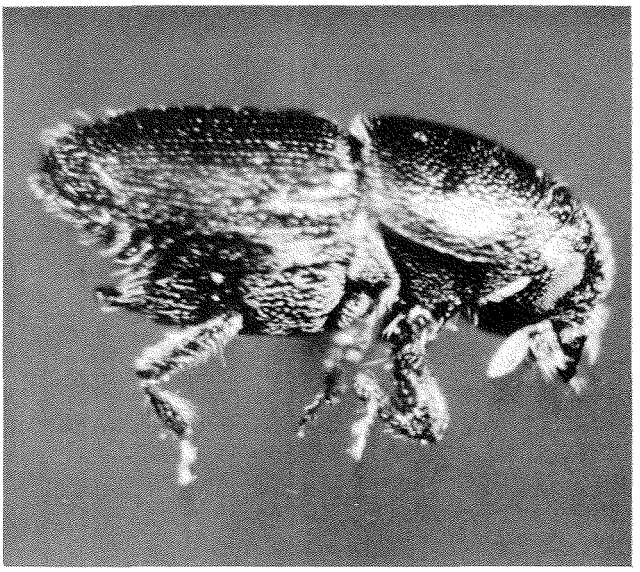

Figure 4. Adult of the smaller European elm bark beetle. (X24).

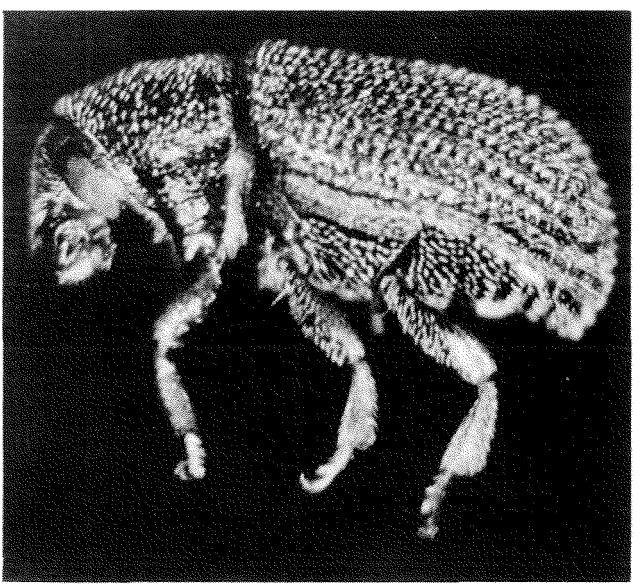

Figure 5. Adult of the native elm bark beetle. (X24).

particularly in the west where both the disease and the vector are known to occur in Minnesota about 250 miles to the south of the Manitoba boundary (48).

The bark beetle vectors may be distinguished by the general appearance of the adults and by the pattern of feeding damage on the inner bark and outer surface of wood. The adults of both the native and European species are about 1/8 inch long and range in color from brown to black (Figures 4 and 5). The European species can be identified by its shiny surface, the concavity on the posterior ventral side of the abdomen, and the blunt spur extending back from the centre of the second ventral segment. Egg galleries of the European species are cut along a single line parallel to the grain of the wood (Figure 6), whereas the native species cuts two diverging egg galleries forming a broad "V" across the grain of the wood (Figure 7). The larvae of each species feed in galleries constructed at right angles to the egg gallery: the European species feeding across the grain and the native species feeding parallel to the grain of the wood. Larvae of both species are similar in appearance being white, wrinkled, legless grubs with brown heads.

Both of the vectors pass through one and a partial second generation a year in Canada but there are significant differences in the seasonal activity and occurrence of the various stages (31). The main population of H. rufipes overwinters in the adult stage and emerges in early May to feed and construct brood galleries. They breed and oviposit in May, June, and July. This gives rise to a new generation of adults which emerge in late summer and fall and feed in the bark of branches and stems of healthy trees until late fall. They overwinter in special hibernation tunnels. A very small proportion of the population of fall adults constructs brood galleries and oviposit to give rise to overwintering larvae which do not emerge as adults until the following June. S. multistriatus overwinters in the larval stage and the adult beetles emerge in June or July feeding in the bark of small branches and twigs. Most of the progeny of these adults emerge in August and September although some do not complete development and remain as larvae which form the bulk of the overwintering population. Only a few of the adults which emerge in late summer are able to breed, oviposit, and give rise to overwintering larvae. The remainder simply feed and die with the approach of cold weather without establishing a brood.

In Canada, the disease has spread and caused extensive losses in Quebec, New Brunswick, and parts of Ontario in the presence of only H. rufipes. Adults of this beetle are active nearly one month earlier than those of S. multistriatus. As will be shown in the following section, this is important from a pathological point of view since inoculations causing extensive infections of healthy trees occur almost completely in the spring and early summer. When

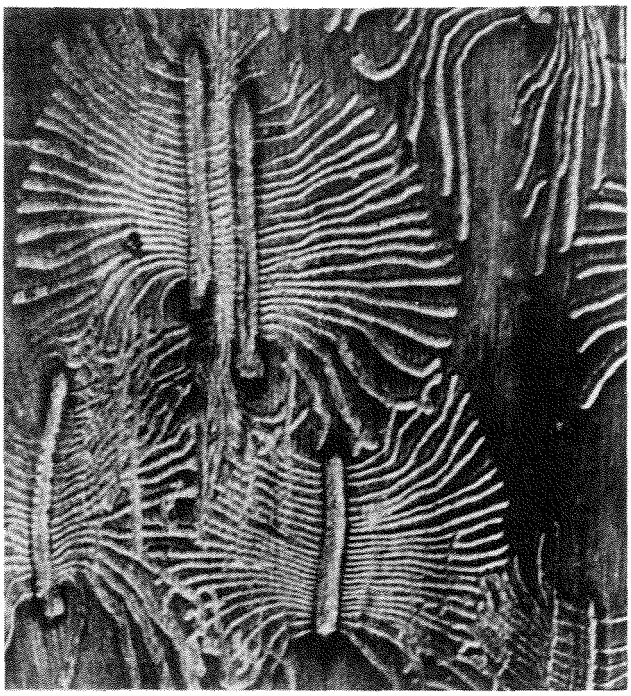

Figure 6. Galleries of the smaller European elm bark beetle on the outer wood surface of elm. About natural size.

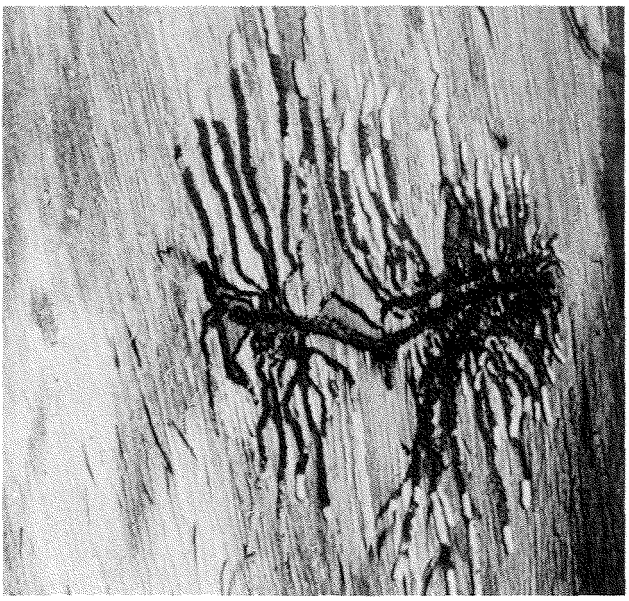

Figure 7. Galleries of the native elm bark beetle on the outer wood surface of elm. About natural size.

both species are present, S. multistriatus is considerably more aggressive than H. rufipes, invades available breeding material more readily, and limits the H. rufipes population to relatively small numbers. For this reason, as well as the fact that it feeds in parts of the tree with thinner bark, S. multistriatus is generally accepted in the United States as the most important vector of C. ulmi. - R.J. Finnegan and W.L. Sippell.

MECHANISM OF PATHOGENESIS

The most frequent means of inoculation of Ceratocystis ulmi into healthy trees is by the feeding of adult elm bark beetles. Feeding wounds must reach the xylem for inoculation to be successful but other factors are involved. Studies by Al-Azawi and Norris (2) indicate that feeding wounds of Scolytus multistriatus 3 mm. or longer are required for transmission of the fungus and infection of the tree through terminal twigs. Ouellette (53) observed that feeding wounds of this insect in twig crotches were less efficient infection courts than those extending to the sides of crotches and those made on the sides of branches. High relative humidity at wounds made by S. multistriatus was found by Kais, Smalley, and Riker (42) to be a prerequisite to penetration by the fungus.

The pathogen may also pass from diseased to healthy trees by means of root grafts (70). Himelick and Neely (38) have pointed out the importance of this mechanism of transmission in city-planted elm trees.

The fungus invades all types of xylem cells. It grows from cell to cell through pits and directly through cell walls. In the vessels it spreads by means of spores which are distributed by the sap stream to other parts of the tree (5). Microspores formed by the pathogen in culture (55) and observed in infected elms (52) may explain the rapidity with which the fungus is distributed. The rate and extent of spore distribution within the tree and of subsequent disease development are dependent on a number of factors, some of which are related, including: the amount of inoculum, season of inoculation, length of vessels into which the fungus is introduced, moisture conditions in the soil, and soil and air temperatures (5, 42). Extensive infection of living elms, however, depends upon the spores entering the long, functioning conductive vessels of the spring and early-summer wood. During the spring and early summer these vessels are close to the surface of the wood and the spores may be introduced by adult beetles feeding at this time. The vascular system produced after midsummer consists of much shorter and relatively compressed conducting vessels at the wood surface that restrict spore movement. Inoculation at this time usually results in a very localized and temporary infection (12, 56).

Kerling (43) observed that the first alteration in infected tissue is the coloration of vessel walls, followed by changes in the appearance of the contents of living cells which become darker, exudation of gum droplets through pits from parenchyma and ray cells, and the formation of tyloses in non-discolored parts of vessels. These tyloses later disintegrate (66). Histochemical tests performed by the author show that the changes in the appearance of living cells are due to the formation and oxidation of polyphenols. As the disease progresses, these phenolic compounds are extruded through pits into vessels and other xylem elements and are even deposited in tyloses when these have not disintegrated.

Most workers before the mid-forties attributed the rapid wilting and dying of infected elm trees to the plugging of the water-conducting vessels by gums, tyloses, fungus growth, or degradation products of parenchyma cells (10, 15, 60, 74).

In the late forties, a trend developed towards a toxin theory of disease causation. Much evidence in support of this theory is derived from the injection into healthy plants of cell-free filtrates of pathogen cultures. Broekhuizen (10) produced tyloses and Zentmeyer (76) a wilting of test plants, discoloration of cell walls, and formation of gum in vessels following injection with culture filtrates. Dimond (22) separated two fractions in the toxic filtrate; one caused the upcurling and marginal withering of the leaves, and the other caused severe interveinal necrosis when injected into elms. The work of Feldman *et al* (29) showed that the first of these fractions is of minor importance in the toxin complex, that the heat stability of the toxin is only partial (suggesting the presence of active enzymes in the toxin complex), and that the filtrate is almost completely inactive at pH 6.0 and above. Tests conducted by the author have shown that xylem discoloration may be obtained by injection of water adjusted to pH 5.4 and below. Consequently, the toxicity of culture filtrates, being active below pH 6.0, could be an effect of the low pH.

All evidence for the toxin theory of pathogenesis of the Dutch elm disease is based on the toxicity of culture filtrates, but no one has demonstrated that the toxic substances produced by the fungus in culture are also produced and active in diseased elm trees. However, if translocated, polyphenols liberated in diseased elms under the action of the fungus could conceivably produce toxic effects in distant cells, for example, in leaves.

In the early fifties, attention was drawn to the role of enzymes in the wilt diseases. The theory proposed by Ludwig (44) is a modification of the plugging theory in that it explains the origin of the gums from alteration of cell wall constituents, especially of pectins, through the action of hydrolytic enzymes. Although enzymes of this type have been demonstrated in culture filtrates of *C. ulmi* (9, 23, 40), Dimond and Husain (23) concluded

that their main function was the digestion of cell wall constituents to provide food for the fungus. However, alteration of cell walls may affect the flow of materials into the vessels from parenchyma and ray cells and contribute to their plugging.

Dimond (20) discussed the role of other enzymes in the formation of brown substances in wilt diseases. He postulated that these liberate and oxidize polyphenols which are condensed to produce pigmented, melanin-like compounds. There is a possibility that some of the polyphenols are liberated from lignin in the cell walls, as indicated by histochemical tests conducted by the author.

Recently, Ouellette (53, 54) suggested that acute symptoms of the disease are due to the complete plugging of the vessels of small branches by spores and mycelium of the pathogen, alone or in combination with cytoplasm and residues from adjoining cells. He stated also that gradual and partial plugging of vessels in stems and larger branches and disintegration of cell walls contribute to chronic symptoms of the disease.

In summary, the causal fungus of Dutch elm disease is well adapted for rapid spread in elm. Large numbers of microspores can attack many living cells of the xylem at many points along the vessels. This multisite infection of living cells, which results in the production of polyphenols and death of the cells, is of prime importance when considering the fact that living cells are probably necessary for the active transport of sap, as shown by recent workers (36, 61). In a more advanced stage of disease, enzymatic action of the fungus on cell walls would allow the contents of recently dead parenchyma and ray cells to leak into the vessels. These materials, which have become "gummy" may contribute to vessel plugging along with fungous spores and hyphae, or may be translocated and act as toxins in distant parts such as leaves. Further studies are in progress to determine the exact role of these substances in pathogenesis. - C. Gagnon.

METHODS OF CONTROL

Quarantine Regulations - When it became known that the Dutch elm disease occurred in several European countries, a regulation (No. 17 Foreign) under the Destructive Insect and Pest Act was passed, effective April 12, 1928, prohibiting the importation of elm and elm products from Europe. This regulation was amended May 9, 1934, extending the prohibition to all countries, and is still in effect.

A regulation (No. 12 Domestic), in accordance with the provisions of the Destructive Insect and Pest Act, effective April 24, 1945, was drawn up restricting the movement of elm and elm products to disease-free areas from certain counties in the Province of Quebec

where the disease had been found. This regulation was designed to prevent long-distance spread of infected elm material as well as the insect vectors.

Amendments were made to Regulation No. 12 (Domestic) in 1947, 1949, and 1955 as elms in additional territory in Quebec and Ontario were found to be infected. The present quarantine embraces the entire Province of Quebec and all of the Province of Ontario except the districts of Thunder Bay, Rainy River, Kenora, Patricia, Cochrane, and Algoma. This regulation is now being revised because of recent changes in the distribution of the disease. - L.L. Reed, Plant Protection Division, Canada Dept. of Agriculture, Ottawa.

Sanitation - Sanitation for the control of Dutch elm disease was recommended as early as 1936 in the United States when it was observed that the incidence of the disease was reduced by the rapid removal of infected trees around New York City (16). By 1940, research and experience had shown that other dead and dying elm material used as breeding sites by the bark beetles is also of importance in the spread of the disease (17). Since then, the prompt destruction (at least before the beetles emerge early in May) of diseased trees, all recently dead elm wood with the bark present, and dying trees and branches in the vicinity of healthy trees has been recommended in the United States and Canada. Several methods of destroying this material are currently in use: burning; debarking and burning or burying the bark in soil to a depth of 1 foot or more; burying; or spraying the material with a 1% DDT solution (12, 73). To prevent the spread of the disease through root grafts, Himelick and Neely (38) suggest that a trench 3½ feet deep be cut in the soil between non-infected and diseased trees within 40 feet of one another immediately after discovery of the diseased tree and before its removal. In addition to the above procedures, the use of measures that help to maintain tree vigour, such as fertilizing, watering, and the control of other insects and diseases, are also recommended because elms in good condition are less attractive to bark beetles (73). - R. Pomerleau.

Protection of Healthy Trees by Chemical Insecticides - There are two approaches to the protection of healthy trees from bark beetles and consequent infection by the fungus. The first, the conventional method, is to apply a stomach-contact insecticide to the trees to kill attacking beetles, and the second, which has attracted attention in recent years because of the undesirable toxicity of conventional insecticides to other forms of animal life, is to place a systemic insecticide into the tree to be carried in the sap stream to all parts and kill the insects before their feeding niches have reached a sufficient size to constitute infection courts.

DDT is the most commonly used conventional insecticide. If applied when the tree is dormant, it will remain effective throughout the spring and early summer when trees are most susceptible to

infection. DDT may be applied either as a 2% emulsion by hydraulic sprayer or as a 12% emulsion with a mist blower (12). A high volume of spray is necessary with a hydraulic sprayer and this leads to excessive run-off and contamination of public and private properties. The effect of run-off is minimized by the use of the mist blower since a smaller volume of spray is used.

However, even if applied by mist blower, DDT is toxic to many forms of animal life, especially birds. This has been particularly true in the case of robins in areas where DDT has been used for several years (41). DDT contaminates the food of earthworms which are a staple diet of robins. Because of the additive residual toxicity of DDT, a great many chemicals including dieldrin, heptachlor, chlordane, toxaphene, lindane, methoxychlor, thiodan, zectran, malathion, and parathion (11, 26, 27, 46) have been tested in a search for a safer insecticide. Of these only two show promise. Methoxychlor, which has only 1/25 the toxicity of DDT to birds and is less persistent than DDT, provides almost as good protection as DDT and is now recommended for use in areas of high bird hazard. Lindane shows considerable effectiveness and warrants further investigation.

A number of systemic insecticides including demeton, phorate, dimefox, di-syston, phosphamidon, dimethoate, tetram, and bidrin (1, 2, 3, 49) have been tried but only two, tetram and bidrin, were effective in restricting beetle feeding. Tetram is unfortunately so toxic to mammals that it cannot be used safely, which leaves bidrin as the most promising systemic to date. It, however, has not been adequately tested. Many new systemics are in the experimental stage of development and may prove to be satisfactory. Because this class of chemicals is toxic to both plants and animals, their effects must be thoroughly investigated and safe dosages defined before any can be recommended for use. - J.J. Fettes.

Chemotherapy - Chemotherapy has been defined as the control of a plant disease by compounds that, through their effect on the host or the pathogen, reduce or nullify the effect of the pathogen after it has entered the plant (21). Many promising compounds have appeared in recent years but the striking feature of the work on chemotherapy is that, to date, no successful therapeutant is available for general use in the control of Dutch elm disease. A number of chemicals have been tested experimentally, but for one reason or another, they have failed to fulfill their early promise. However, in spite of these failures, the potential value of such treatments is so great that new groups of chemicals are continually being studied, and new techniques employed in the hope that the basic problems will eventually be overcome.

The chemicals currently under intensive investigation for the Dutch elm disease employ the principle of indirect action. Here a chemical is applied to, or injected into, a healthy tree to change

its physiological and anatomical state in such a way that it is immune or resistant to penetration of the causal organism, or failing this, to prevent the development of extensive damage after the causal organism has become established.

One group of chemicals being studied is the growth regulators and a number of investigators have studied the effect of some of these on the control of the disease. Beckman (8) obtained a reduction in symptom incidence in nursery elms implanted with dry sodium-4, 5-dimethyl-2-thiazolylmercaptoacetate prior to inoculation with the causal fungus. Disease inhibition was correlated with the prevention of normal sapwood development, for when trees treated with the chemical were re-inoculated after normal sapwood development had resumed (that is, when the effect of the chemical had worn off), the trees became infected.

Subsequently, Beckman (7) working with 2, 3, 5, 6-tetrachlorobenzoic acid, a chemical which penetrates the bark and is mobile in the host, obtained a significant decrease in the incidence of disease in trees treated prior to inoculation. However, the therapeutic level of the chemical was about the level at which it caused damage to the host trees.

Smalley (68) tested salts of 2, 3, 6-trichlorophenylacetic acid (TCPA) applied to large nursery elms in several concentrations and by several methods before inoculation. The results indicated a degree of control with bark applications and a high level of protection following injection. With the appropriate chemical concentration and application time, it was possible to obtain complete protection without phytotoxicity. Smalley's study offered some support to Beckman's (7, 8) suggestion that control results from inhibition of formation of large spring vessels, but also suggested that a second mechanism was involved. Smalley observed that TCPA treatments induced heavy tyloses development in large xylem vessels and felt that such vessel occlusion limited spread of infection and was at least partially responsible for the observed control of the disease. This is implied morphological resistance, but the morphological change believed to be imparting resistance to the host is strikingly similar to one of the morphological changes which occur when an untreated host is affected by the disease.

Edgington (28) obtained fewer systemic infections in young trees injected with various concentrations of aminotrichlorophenylacetic acid (HRS-399). Phytotoxicity occurred at the higher concentrations. Histological studies revealed that HRS-399 induced the elms to form dense, starch-filled summer wood immediately after treatment. This induced summer wood appeared to delimit the fungus, allowing the trees to lay down functional non-infected xylem during the growing season.

At the Maple laboratory, since the spring of 1962, TCPA and HRS-399 have been employed in a program designed to test their

efficacy to control Dutch elm disease. The techniques employed have been: (a) injection of the chemical directly into test trees by means of a bottle-siphon system (47), (b) bark sprays, (c) bark paints, and (d) soil injection. The test trees were inoculated before and/or after the various treatments. Trees employed were up to 45 feet in height under natural conditions, and 2- to 4-feet high in greenhouse experiments.

While it would be premature to present the results of these experiments at this time, indications are that direct injections of the chemical into host trees, at appropriate concentrations, will prevent establishment of Dutch elm disease. Furthermore, there are indications that if trees in a very early stage of disease development, and at the same time in a very early growth stage (about 1/3 full leaf), are treated with appropriate concentrations, disease development is arrested. - J. Reid.

Resistant Varieties - While the white elm is one of the most susceptible species, several elms are relatively resistant to Dutch elm disease, notably: European strains including the Christine Buisman and the Bea Schwarz elms, and the Asiatic Chinese and Siberian elms. However, these are susceptible to other diseases or storm damage, are not cold hardy, or are of poor form, and cannot be generally recommended as suitable replacements for the white elm in Canada. The most promising approach in this method of control at present appears to be the search for resistant strains of the native elm.

For this purpose, artificial inoculations have been carried out extensively at L'Assomption Experimental Station, P.Q., since 1950 on cuttings from 290 healthy trees occurring in heavily infected areas and on 178,000 seedlings from irradiated and non-irradiated seeds. None of the adult elms and the 32,000 seedlings from untreated seeds collected from 309 elms in 35 counties of Quebec were found to be resistant. This has confirmed the very high susceptibility of the white elm and the absence of resistant strains in nature.

The remaining 146,000 seedlings tested were from seeds treated with X-rays or thermal-neutrons in an attempt to induce artificial mutations promoting disease resistance. Only four of these seedlings were considered promising - two from seed treated with X-rays and two from seed treated with thermal-neutrons. Of the former, one appears to be immune and has not shown disease symptoms after 7 consecutive years of inoculation; the other showed light symptoms in 2 of the 7 years of inoculation. The latter were inoculated during 6 years and showed light symptoms in 1 and 2 years, respectively.

Testing is being continued on cuttings from the "immune" specimen and these have resisted inoculations for 2 years. - C.E. Ouellet, Plant Research Institute, Canada Dept. of Agriculture, Ottawa.

Biological Control - Although parasites, predators, and diseases of the bark beetle vectors occur in North America (13, 24, 25, 30, 57, 65), information collected to date indicates that they do not occur in sufficient numbers for effective control.

Three species of Hymenoptera have been observed to parasitize from 50 to 89 per cent of bark beetle populations in parts of Europe (32, 33, 63, 67) and at least two of these have been recorded in North America (13, 57). However, the possible transmission of the causal fungus of Dutch elm disease by these parasites lessens their potential in controlling the insect-disease complex and has discouraged extensive study in North America.

In 1930, an endoparasitic nematode was reported as sterilizing 39 per cent of a population of S. multistriatus in England (50). This and other nematodes are associated with S. multistriatus in the United States (65). Since nematodes are known to reduce populations of similar scolytids significantly, research was initiated by the Institute for Biological Control, Belleville, Ont., to determine the efficacy of parasitic nematodes to reduce bark beetle numbers in Canada. In 1963, hundreds of beetles collected throughout Ontario were dissected and found to be free of pathogenic nematodes. Attempts will be made within the next year to manipulate indigenous nematodes in an effort to have them attack the elm bark beetles. Contacts have also been made with scientists in other countries for the importation of nematodes for use against the beetles. The successful establishment of these natural enemies would reduce bark beetle numbers and provide a self-perpetuating method of control. - W.R. Nickle, Entomology Research Institute for Biological Control, Canada Dept. of Agriculture, Belleville, Ont.

REFERENCES

1. Al-Azawi, A.F. and J.E. Casida. The efficiencies of systemic insecticides in the control of the smaller European elm bark beetle. J. Econ. Ent. 51:789-790. 1958.

2. Al-Azawi, A.F. and D.M. Norris. Experimental prevention of bark beetle transmission of Ceratocystis ulmi (Buis.) C. Moreau with the systemic insecticide Chipman R-6199. J. Econ. Ent. 52:902-904. 1959.

3. Al-Azawi, A.F., D.M. Norris, and J.E. Casida. Hazards associated with the implantation of tetram into elm trees for Dutch elm disease control. J. Econ. Ent. 54:127-129. 1961.

4. Anonymous. Occurrence of Ceratocystis ulmi (Buism.) C. Moreau in New Brunswick. Can. Dept. For., For Ent. and Path. Branch, Bi-Mon. Prog. Rept. 13(6):1. 1957.

5. Banfield, W.M. Distribution by the sap stream of spores of three fungi that induce vascular wilt diseases of elm. J. Agr. Res. 62:637-681. 1941.

6. Beattie, R.K. How the Dutch elm disease reached America. Nat. Shade Tree Conf. Proc. 9:101-105. 1933.

7. Beckman, C.H. Dutch elm disease control with polychlorobenzoic acid. (Abstr.) Phytopath. 49:227. 1959.

8. Beckman, C.H. Growth inhibition as a mechanism in Dutch elm disease therapy. Phytopath. 48:172-176. 1958.

9. Beckman, C.H. Production of pectinase, cellulases, and growth-promoting substances by Ceratostomella ulmi. Phytopath. 46:605-609. 1956.

10. Broekhuizen, S. Wondreacties van hout; het ontstaan van thyllen en wondgom, in het bizonder in verband met de iepenziekte, 80 p. Leiden. 1929. Translation by E.W.J. Reyers (typescript), Pl. Path. Libr., Cornell Univ., Ithaca, N.Y.

11. Bromley, S.W. Tests on elm scolytus 1949. J. Econ. Ent. 43: 397-398. 1950.

12. Can. Dept. Agr., For Biol. Div., Publ. 1010. Dutch elm disease. 1958.

13. Chamberlin, W.J. The bark and timber beetles of North America. Oreg. State Coll. Coop. Assoc. 513p. 1939.

14. Chapman, J.W. The introduction of a European scolytid, the smaller elm bark beetle, Scolytus multistriatus (Marsh.), into Massachusetts. Psyche 7:63-68. 1910.

15. Clinton, G.P. and F.A. McCormick. Dutch elm disease, Graphium ulmi. Conn. Agr. Exp. Sta. Bull. 389:701-752. 1936.

16. Collins, C.W., W.D. Buchanan, R.R. Whitten, and C.H. Hoffman. Bark beetles and other possible insect vectors of the Dutch elm disease, Ceratostomella ulmi (Schwarz) Buisman. J. Econ. Ent. 29:169-176. 1936.

17. Collins, D. L., K.G. Parker, and H. Dietrich. Uninfected elm wood as a source of the bark beetle (Scolytus multistriatus Marsham) carrying the Dutch elm disease pathogen. Cornell Univ. Agr. Exp. Sta. Bull. 740:1-14. 1940.

18. Dance, B.W. and D.F. Lynn. Ontario, Forest Disease Conditions. In Annual Report of the Forest Insect and Disease Survey, 1962. Can. Dept. For., For. Ent. and Path. Branch, Ottawa. 1963.

19. Dance, B.W. and D.F. Lynn. Ontario, Forest Disease Conditions. In Annual Report of the Forest Insect and Disease Survey, 1963. Can. Dept. For., For Ent. and Path. Branch, Ottawa, 1964.

20. Dimond, A.E. Pathogenesis in the wilt disease. Ann. Rev. Plant Physiol. 6:329-350. 1955.

21. Dimond, A.E. Plant chemotherapy. In Plant Pathology, Problems and Progress: 1908-1958. Univ. of Wisc. Press. p.221-228. 1959.

22. Dimond, A.E. Symptoms of Dutch elm disease produced by Graphium ulmi in culture. Phytopath. 37:7. 1947.

23. Dimond, A.E. and A. Husain. Role of extracellular enzymes in the pathogenesis of Dutch elm disease. Science 127:1059. 1958.

24. Doane, C.C. Bacterial pathogens of Scolytus multistriatus Marsham, as related to crowding. J. Insect Path. 2:24-29. 1960.

25. Doane, C.C. Beaveria bassiana as a pathogen of Scolytus multistriatus. Ann. Ent. Soc. Amer. 52:109-111. 1959.

26. Doane, C.C. Evaluation of insecticides for control of the smaller European elm bark beetle. J. Econ. Ent. 55:414-415. 1962.

27. Doane, C.C. The residual toxicity of insecticides to Scolytus multistriatus. J. Econ. Ent. 51:256-257. 1958.

28. Edgington, L.V.A. A chemical that retards development of Dutch elm disease. (Abstr.) Phytopath. 53:349. 1963.

29. Feldman, A.W., N.E. Caroselli, and F.L. Howard. Physiology of toxin production by Ceratostomella ulmi. Phytopath. 40: 341-354. 1950.

30. Finnegan, R.J. Bionomics of the native elm bark beetle, Hylurgopinus rufipes (Eich.), in Quebec. M.Sc. Thesis, Univ. of New Brunswick, Fredericton, N.B. p. 62. 1950.

31. Finnegan, R.J. Elm bark beetles in southwestern Ontario. Can. Ent. 89:275-280. 1957.

32. Fransen, J.J. Report on the investigations on elm disease and elm bark beetles made during 1935 in the Laboratory of Entomology of the Agricultural College at Wageningen. Tijdschr. Plziekt. 43(9):195-217. 1937. (R.A.E. 25:796).

33. Fransen, J.J. Some data on the spread by the cambium beetles, Scolytus scolytus and S. multistriatus, of the Dutch elm disease due to G. ulmi in connection with the control of the disease. Tijdschr. Plziekt. 37(3):49-62. 1931. (R.A.E. 19:499).

34. Goeden, R.D. and D.M. Norris. The potential of Magdalis spp. in the transmission of Ceratocystis ulmi (Buis.) Moreau. J. Econ. Ent. 56:238-239. 1963.

35. Green, G.W. Low winter temperatures and the European pine shoot moth, Rhyacionia buoliana (Schiff.) in Canada. Can. Ent. 94:314-336. 1962.

36. Greenidge, K.N.H. Ascent of sap. Ann. Rev. Plant Physiol. 8:237-256. 1957.

37. Hafstad, G.E. Possible long distance spread of Dutch elm disease by transported beetles. Plant Disease Reptr. 42: 893-894. 1958.

38. Himelick, E.B. and D. Neely. Root grafting of city-planted American elms. Plant Disease Reptr. 46:86-87. 1962.

39. Holmes, F.W. Recorded Dutch elm disease distribution in North America as of 1961. Plant Disease Reptr. 46:715-718. 1962.

40. Holmes, F.W. The Dutch elm disease as investigated by the use of tissue cultures, antibiotics, and pectic enzymes. Ph.D. Thesis, Cornell Univ., Ithaca, N.Y. 143pp. 1954.

41. Hunt, L.B. Songbird breeding populations in DDT-sprayed Dutch elm disease communities. J. Wildl. Mgt. 24:139-146. 1960.

42. Kais, A.G., E.B. Smalley, and A.J. Riker. Environment and development of Dutch elm disease. Phytopath. 52:1191-1196. 1962.

43. Kerling, L.C.P. Reactions of elm wood to attack of Ophiostoma ulmi. Acta. Bot. Neerl. 4:398-403. 1955.

44. Ludwig, R.A. Studies on the physiology of hadromycotic wilting in the tomato plant. Macdonald College, McGill Univ., Bull. 20. p. 1-38. 1952.

45. Magasi, L.P. Maritime Provinces, Forest Disease Conditions. In Annual Report of the Forest Insect and Disease Survey, 1963. Can. Dept. For., For. Ent. and Path. Branch, Ottawa. 1964.

46. Matthysse, J.G., H.C. Miller, and H.E. Thompson. Insecticide deposits for control of elm bark beetles. J. Econ. Ent. 47:739-746. 1954.

47. Morris, R.F. Tree injection experiments in the study of birch dieback. For. Chron. 27:313-329. 1951.

48. Minn. Dept. Agr., Div. Plant Industry. Consolidated Insect Survey Report, Minnesota - 1962-1963.

49. Norris, D.M. Systemic insecticidal action in the cortical tissues of elm twigs. J. Econ. Ent. 53:1034-1036. 1960.

50. Oldham, J.N. On the infestation of elm bark beetles (Scolytidae) by the nematode, Parasitylenchus scolyti n. sp. J. Helminth. 8:239-248. 1930.

51. Ouellette, G.B. Quebec, Forest Disease Conditions. In Annual Report of the Forest Insect and Disease Survey, 1962. Can. Dept. For., For. Ent. and Path. Branch, Ottawa. 1963.

52. Ouellette, G.B. Morphological characteristics of Ceratocystis ulmi (Buism.) C. Moreau in American elm trees. Can. J. Bot. 40:1463-1466. 1962.

53. Ouellette, G.B. Studies of host and pathogen in relation to the infection process of the Dutch elm disease, caused by Ceratocystis ulmi (Buism.) C. Moreau. Ph.D. Thesis, Cornell Univ., Ithaca, N.Y. p. 123. 1960.

54. Ouellette, G.B. Studies on the infection process of Ceratocystis ulmi (Buism.) C. Moreau in American elm trees. Can. J. Bot. 40:1567-1575. 1962.

55. Ouellette, G.B. and C. Gagnon. Formation of microendosphores in Ceratocystis ulmi (Buism.) C. Moreau. Can. J. Bot. 38: 235-241. 1960.

56. Parker, K.G., D.L. Collins, L.J. Tyler, D.P. Connola, W.E. Ozard, and H. Dietrich. The Dutch elm disease: association of Ceratostomella ulmi with Scolytus multistriatus, its advance into new areas, method of determining its distribution and control of the disease. Cornell Univ. Agr. Exp. Mem. 275. 1947.

57. Peck, O. A catalogue of the nearctic Chalcidoidea (Insecta: Hymenoptera). Can. Ent. Suppl. 30. p. 1092. 1963.

58. Pomerleau, R. History of the Dutch elm disease in the Province of Quebec. For. Chron. 37:356-367. 1961.

59. Pomerleau, R. La maladie hollandaise de l'orme au Canada. Rev. Can. de Biol. 4:116-118. 1945.

60. Pope, S.E. Some studies on the Dutch elm disease and the causal organism. Ph.D. Thesis, Cornell Univ., Ithaca, N.Y. p. 66. 1943.

61. Postlethwait, S.N. and B. Rogers. Tracing the path of the transpiration stream in trees by the use of radioactive isotopes. Amer. J. Bot. 45:753-757. 1958.

62. Robert, A. Insect vectors of the Dutch elm disease in the Province of Quebec. Proc. Xth Intern. Congr. Ent. 4:433-436. 1958.

63. Russo, G. Contributo alla conoscenza dei coleotteri scolitidi fleotribo: Phloeotribus scarabaeoides (Bern.) Fauv. Portici Lab. Ent. Agr. Bol. 2:1-420. 1938.

64. Rovski, V.M. On a radical method of fighting the Dutch elm disease. Bot. Zurn. 41:1478-1481. 1956.

65. Saunders, J.L. and D.M. Norris. Nematode parasites and associates of the smaller European elm bark beetle, Scolytus multistriatus. Ann. Ent. Soc. Amer. 54:792-798. 1961.

66. Schwarz, M.B. Das Zweigsterben der Ulmen, Trauerweiden und Pfirsichbäume. Mededeel. Phytopath. Lab. "Willie Commelin Scholten" Baarn. 5:7-32. 1922. (Translated by Lilian D. Kelsey. In Bartlett Res. Lab. Bull. 1. p. 5-25. 1928).

67. Sitowski, L. Spostrzenzia nad pasorgytami kornikow (Ipidae). Polsk. Pismo Ent. 9:1-13. 1930. (R.A.E. 18:323).

68. Smalley, E.B. Prevention of Dutch elm disease by treatments with 2, 3, 6-trichlorophenyl acetic acid. Phytopath. 52: 1090-1091. 1962.

69. U.S. Dept. Agr. Circ. 677. Dutch elm disease and its control. 1943.

70. Verral, A.F. and T.W. Graham. The transmission of Ceratostomella ulmi through root grafts. Phytopath. 25:1039-1040. 1935.

71. Watson, E.B. The European elm bark beetle. Can. Dept. Agri., Div. Ent., Bi-Mon. Prog. Rept. 4(5):2. 1948.

72. Watson, W.Y. and W.L. Sippell. Scolytid vectors of the Dutch elm disease in Ontario. Can. Ent. 93:403-405. 1961.

73. Welch, D.S. and J.G. Matthysse. Control of the Dutch elm disease in New York State. Cornell Univ. Ext. Bull. 932. 1958.

74. Wollenweber, H.W. Das Ulmensterben und sein Erreger, Graphium ulmi, Schwarz. Sonderabsdruck aus dem Nachriechtenplatt für den deutschen Pflanzenschutzdienst. No. 10. 1927. (Translated by Lilian D. Kelsey. In Bartlett Res. Lab. Bull. 1:26-31. 1928)

75. White, L.T. Dutch elm disease and its control. Inf. Bull. Ont. Dept. Lands and Forests. 1961.

76. Zentmeyer, G.A. Toxin formation by Ceratostomella ulmi. Science 95:512-513. 1942.

1187-3-67-10M