FORESTRY COMMISSION

BULLETIN No. 33

The Status and Development of ELM DISEASE in Britain

By T. R. PEACE, M.A.

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LONDON: HER MAJESTY'S STATIONERY OFFICE

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FOREWORD

In 1927 there appeared in Eastern England a new disease affecting elm trees, which has since spread extensively and caused conspicuous, if rather local, damage.

The elms are important timber-producing trees, and although they are usually grown along hedgerows rather than in woodlands, their economic importance is such that the Forestry Commission decided, in 1928, that investigations were desirable into the characteristics of this new disease.

These studies have been continued ever since by Mr. T. R. Peace, now the Commission's Chief Research Officer, and this Bulletin presents the results of his work.

FORESTRY COMMISSION,
25, Savile Row,
London, W.1.
January, 1960

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THE STATUS AND DEVELOPMENT OF ELM DISEASE IN BRITAIN

INTRODUCTION

Few tree diseases can have received more attention than the disease of elms caused by the fungus *Ceratostomella ulmi* (Schwarz) Buis. Both in Europe and in America it has been the subject of many varied investigations and experiments. Much work has been done on the causal fungus, and its relation to the bark beetles, which are its chief vectors. Efforts have been made to control the disease by felling infected trees, by insecticidal spraying, and by systemic fungicides.

Surveys have given a great deal of information on the spread of the disease. Work on selection and breeding of resistant elms has proceeded to the point where a number of clones are available for field testing. Viewing the plethora of literature already accumulated, one might question the possibility of adding anything worthwhile to our knowledge of the disease.

However, the discovery of the disease in Britain in 1927, and its persistence here until now (1959), have provided certain unique opportunities for its study. It is the results of this, with certain necessary references to European and American work, that are presented here.

There are two factors which specially favoured the investigation of the disease in Britain. Firstly it was decided very early in 1928 that eradication was impossible, and from then on no further large-scale phytosanitary measures were taken. Appeals to owners to remove seriously affected and dead trees met with little response, so that the disease has run a more or less uninterrupted course, subject only to the normal interferences common to any trees growing in a highly populated land. Secondly, the author's first assignment on joining the staff of the Imperial Forestry Institute in July 1928 was a survey of the disease on behalf of the Forestry Commission. Although after 1928 work on it was restricted, annual surveys were continued with some wartime interruptions until 1949, and a repeat survey was made in 1955. All the surveys were carried out by the author, so that the disease has been under the eye of a single observer for thirty-one years. These two factors have thus permitted the collection of comparative data on the behaviour of the disease, both in individual trees and in the elm population as a whole.

Apart from the survey work a few other aspects of the disease were investigated. These are discussed briefly at appropriate points in the text. From 1935 to 1938 the author was fortunate to have with him in Britain two American workers, D. E. Parker of the U.S. Bureau of Entomology and Plant Quarantine, and J. M. Walter of the Division of Forest Pathology of the U.S. Bureau of Plant Industry. Their main assignment in England was the study of the disease under more natural conditions than then existed in the United States, where attempts at eradication were in progress. They worked in close collaboration with the author, and with their kind consent some of their results and observations are quoted later in this Bulletin.

Acknowledgments are due to the United States Department of Agriculture for permission to publish some of the information collected by Mr. Parker and Dr. Walter, while they were working in Great Britain. Thanks are also due to Mr. W. R. Day and Mr. W. H. Guillebaud, whose advice and encouragement were available throughout the earlier surveys, to Dr. Walter and Mr. Parker for their collaboration during their stay in this country and for their willingness to allow their results to be quoted, and to numerous landowners, in particular Lt.-Col. E. R. Pratt, of Ryston Hall, Downham Market, Norfolk, for permission to work on their estates. The manuscript has been read by Miss Went, Mr. H. M. Heybroek, and Mr. J. S. Murray, all of whom made valuable corrections and suggestions.

The work carried out in the different years is summarised in Table 1 below. Except in 1928, when the whole summer from mid-July onwards was devoted to the disease, the surveys usually occupied 8 to 16 days. They were seldom started before mid-August and were usually completed by the third week in September.

WORK CARRIED OUT ON ELM DISEASE, 1928–1957

TABLE 1

Year	*Survey*	*Miscellaneous Observations*	*Special Projects*
1928	Detailed survey in the neighbourhood of the first recorded outbreak at Totteridge in Hertfordshire; carried out by James Macdonald. General sampling survey of most of the southern half of England, and parts of North Wales.	Observations started on individual trees.	—
1929	General sampling survey.	Observations continued on individual trees. Railway journeys first used as a subsidiary method of survey.	—
1930	General sampling survey. Detailed observations started in selected diseased areas.	Observations continued on individual trees.	—
1931	General sampling survey. Number of detailed observational areas increased.	,, ,, ,, ,, ,,	—
1932	General sampling survey. Continuation of detailed observations.	,, ,, ,, ,, ,,	—
1933	General sampling survey. Continuation of detailed observations.	,, ,, ,, ,, ,,	—
1934	General sampling survey. Continuation of detailed observations.	,, ,, ,, ,, ,,	—
1935	General sampling survey. Continuation of detailed observations.	Intensification of observations on individual trees.	Commencement of collaboration with American workers.
1936	General sampling survey. Number of detailed observational areas again increased.	Observations continued on individual trees.	Continuation of collaboration with American workers.
1937	General sampling survey. Continuation of detailed observations.	Disease first recorded in north-west England. Observations continued on individual trees.	Inoculation experiments in collaboration with American workers.
1938	General sampling survey. Number of detailed observational areas again increased.	Disease first recorded in Scotland. Observations continued on individual trees.	Inoculation of possibly resistant trees *in situ*, in collaboration with American workers.
1939	Restricted sampling survey. Continuation of most of the detailed observations.	Observations continued on individual trees.	Further inoculation experiments.
1940	Restricted sampling survey. Continuation of most of the detailed observations.	,, ,, ,, ,, ,,	,, ,,
1941	Restricted sampling survey. Continuation of most of the detailed observations.	,, ,, ,, ,, ,,	—
1942	No survey. Continuation of some of the detailed observations.	Only restricted observations on individual trees.	—

WORK CARRIED OUT ON ELM DISEASE, 1928-1957 (*continued*)

TABLE 1 (*continued*)

Year	*Survey*	*Miscellaneous Observations*	*Special Projects*
1943	Restricted sampling survey. Continuation of most of the detailed observations.	Observations continued on individual trees.	—
1944	Restricted sampling survey. Continuation of most of the detailed observations.	,, ,, ,, ,, ,,	—
1945	Sampling survey of a rather wider scope than in 1939-1944. Continuation of most of the detailed observations.	,, ,, ,, ,, ,,	—
1946	Sampling survey with particular reference to the north of England and South Scotland, which were not visited during the war years. Continuation of some of the detailed observations.	,, ,, ,, ,, ,,	—
1947	New method of survey introduced to give a better picture of the general situation. Detailed observations reduced to one area.	Observations on individual trees restricted to one area.	—
1948	New survey repeated and extended. Detailed observations continued in some areas.	Resumption of observations on some individual trees.	Resumption of inoculation experiments. Spraying experiments against *Scolytus* beetles at Folkestone.
1949	New survey repeated. Detailed observations continued in some areas.	Observations continued on some individual trees.	Spraying experiments against *Scolytus* beetles at Folkestone and Aldenham, Herts.
1950	No survey.	Casual observations in the course of other work.	Spraying experiments against *Scolytus* beetles at Aldenham
1951	No survey.	,, ,, ,, ,,	—
1952	No survey.	,, ,, ,, ,,	—
1953	No survey.	,, ,, ,, ,,	—
1954	No survey.	,, ,, ,, ,,	—
1955	New survey repeated. Detailed observations continued in some areas.	Observations continued on some individual trees.	—
1956	No survey.	Casual observations in the course of other work.	—
1957	No survey.	,, ,, ,, ,,	—

Chapter 1

THE APPEARANCE AND BEHAVIOUR OF THE DISEASE

Elm Disease and its causal fungus have been so well and frequently described, that details are unnecessary here. Full illustrated descriptions can be found in Forestry Commission Leaflet 19 (Anon. 1958), in a U.S. Department of Agriculture Circular (Walter, May & Collins 1943), and in a Canadian Department of Agriculture publication (McCallum and Stewart 1951).

The disease is a vascular wilt caused by the fungus *Ceratostomella ulmi* (Schwarz) Buis. This fungus has recently been transferred to the genus *Ceratocystis* by Moreau (1952) under the name *Ceratocystis ulmi* (Schwarz) Moreau, but the older better-known name will still be used here. It has also been known as *Ophiostoma ulmi* (Schwarz) Nannf. The conidial stage, which was discovered first, is *Graphium ulmi* Schwarz.

The fungus exists in the vessels of the wood of elms mainly in a yeast-like form, which can increase by budding, and which is distributed rapidly by the movements of the sap stream. The fungus also makes mycelial growth through dead wood, but appears to have great difficulty in growing through live summer wood from one annual ring to the next, a point of great importance in its behaviour and effect on the tree, and one which will be dealt with more fully later.

The fungus produces a toxin in the vessels, which stimulates neighbouring parenchyma cells to form tyloses and gum, which partially block the vessels. The toxin also acts directly on the leaves causing some wilting (Dimond 1947; Feldmann, Caroselli & Howard 1950). The effect of the fungus is thus a combination of poisoning and water restriction. Typically it causes wilting of the foliage on affected branches, followed by yellowing and browning of the leaves, and often, though not always, by dieback of the twigs and branches. It is sometimes difficult to tell from the appearance of a tree in late summer, when the external symptoms are fully developed, how much permanent damage has been done. The fungus is capable, though this rarely happens, of killing a small or moderate sized tree in one season, so that the amount of injury can vary from death to complete lack of permanent damage. In fact the fungus has been isolated from the current year's wood of trees, which showed no external symptoms at all. It is probable that the more severe injuries are the result of multiple infections, the tree being infected in many places in the same year.

The disorganized cells can be seen in section as a ring of dark brown spots in the spring wood, or longitudinally as discontinuous brown streaks. These markings are of considerable diagnostic value.

They normally appear only in those years when the tree was infected, though occasionally in years following infection, if the fungus succeeds in crossing from one annual ring to the next. They are not produced in rings which were formed prior to infection. Therefore, subject to the proviso that actively infected trees may sometimes fail to show external symptoms, they do indicate years when the tree was actively diseased. *Ceratostomella* can be cultured for several years after infection from rings showing markings, and often after the tree has ceased to show external signs of disease.

The fructifications are of two kinds, black flask-shaped perithecia (the perfect stage) and thin black, sheaf-like coremia (the conidial stage); both are about 1mm. high and bear minute spores at the top in round drops of mucilage. Both forms are produced only in sheltered moist situations and particularly on the surface of the wood under dead bark when it is starting to lift, and in galleries of elm bark beetles. In Britain Walter (1939b) found that both types of fructifications were produced abundantly in suitable places on the tree, but that coremia usually appeared on newly dead material a few weeks before perithecia. He also found that whereas coremia were produced all through the year, except in intensely cold periods, perithecial production ceased completely for 4 to 5 months during the winter. Thus there is always an abundant supply of spores, but they are produced in a position where they cannot readily reach the open and become airborne. In any case, since wounds are necessary for successful infection (Smucker 1937), the chances of infection by a sparse emission of airborne spores are relatively low. Experiments have proved that infection by airborne spores is possible (Smucker 1935), but it is certainly not the normal method, nor is it likely to have much influence on the spread-behaviour of the disease.

Transmission by root-grafts, or through a common

root system, has been recorded several times in the field (Verrall and Graham 1935). A very clear case was observed in England by Walter. This occurred in an area where experimental inoculations were being carried out. Three weeks after wilt symptoms had developed on an inoculated tree *A*, similar symptoms appeared on two neighbouring small trees X and *Y*, which were eight and nine feet respectively away from *A*. Excavation revealed that all three trees had a common root system. Typical discolorations were found to be continuous all along the connecting roots between *A* and X and *Y*. Culturing from any position yielded a brown type of *Ceratostomella ulmi*, which was the one used in the original inoculation, and which had not been isolated from any natural cases in the area in question. This method of transmission does of course necessitate that the fungus shall have reached the base of the tree first attacked.

Though it is not suggested that transmission through the roots is an important factor in the spread of the fungus from tree to tree, it certainly does occur in Britain, where many lines of elms in hedges or small copses have undoubtedly arisen as suckers on a common root system. This method of local spread may sometimes have been responsible for the rather spectacular collapse of groups of elms, that often took place in the earlier years, but it has probably had little effect on the wider spread of the disease.

There is no doubt that the major agents of spread in Britain are the two Elm Bark Beetles, *Scolytus scolytus F.* and *Scolytus multistriatus* Ratz. Both these insects breed freely in the bark of elms weakened by any cause. Although drought, flooding, windfall, etc., often make elms suitable for beetle breeding, trees weakened by Elm Disease have certainly for many years provided the beetles with the bulk of their breeding sites, so that the disease has probably had a major effect on the bark beetle population.

The two beetles have been fully described in Forestry Commission Bulletin No. 8 (Munro 1926), and *Scolytus scolytus* also by Fisher (1931, 1937). Their behaviour in relation to Elm Disease has been dealt with in many papers, but particularly by Fransen (1939a). In America transmission is mainly by *Scolytus multistriatus*, which was inadvertently imported early in this century, and by a native bark beetle *Hylurgopinus rufipes* Eichh. The relationship of these insects to the fungus has been dealt with by Parker *et al.* (1947). It is known that the fungus frequently fructifies in the larval galleries in infected elms, and that 20 to 70 per cent of the beetles emerging from such trees may carry spores. It is the habit of the beetles to feed for a short period before breeding in the twig crotches of healthy or relatively healthy elms, and it is at this time that infection takes place. It has been suggested that feeding beetles tend to fly high and that therefore feeding and infection is commoner in tall trees (Fransen 1939a). Certainly it is the case that infection of elm hedgerows and very small trees is relatively less common. Experiments and observations, too numerous to mention, have proved that *Scolytus* beetles are not only capable of carrying the fungus from infected to uninfected trees, but that they are in practice the principal means of spread.

Violent local outbreaks of the disease can often be traced to sources of large numbers of infected beetles (Liming, Rex and Layton 1951). Such sources may be a standing infected tree, a felled log, or a pile of firewood (Plate 33). Occasionally such material may not have been infected with the fungus at the time of felling, but may have reached this condition later owing to the breeding activities of infected beetles. Numerous instances of spread of this nature from an infection centre have been recorded in Great Britain by Parker, Walter and the author.

It has often been suggested, but definite proof is lacking, that the beetles prefer fast, succulent growth for their feeding. Certainly their feeding attack is not confined to healthy vigorous trees. The observed fact that vigorously growing trees tend to be more seriously affected by the fungus than slow growing ones, is probably not correlated with the degree of infection by feeding beetles, but to the effect of the toxin and sap flow interruption on shoots with a high water demand (Parker, Tyler, Welch and Pope 1947).

The generations of the beetles are complex and vary from year to year. Some overwinter in various stages of development in the bark, and emerge the following spring. Beetles from this emergence, which takes place from May to July, breed to produce not only some individuals which pupate and emerge later the same year, but others which overwinter. The beetles of the late summer emergence, which is at its height in August, produce only an overwintering brood. Local variations in climate, even on two sides of the same log, may cause big differences in the rate of development of the beetles, and it is really only possible to say that they may emerge at any time from May to October, and that the earlier emergences are mostly of overwintering beetles, while the later ones are mostly of beetles which have hatched and developed during the same summer. High temperatures in the spring or summer hasten emergence and stimulate breeding. An increase in beetle population therefore follows any hot period. Conversely low spring or summer temperatures delay emergence and restrict breeding, and thus have a depressing effect on the beetle population.

Dutch work on inoculation of elms has proved that infection is most effective in mid-summer

(Westerdijk & Buisman 1929). This, of course, suggests that the early emerging beetles are probably more important as carriers of the fungus, than those appearing later in the year. Even if infection were equally possible at all seasons, the earlier infections would naturally be more serious, since they have longer to develop. There is no evidence that the fungus continues to spread in live wood during the winter.

The fact that external symptoms do not usually appear till June is probably a reflection of the time needed for the fungus to develop, following its inoculation into the tree by infected beetles. Wilting observed by Walter on May 23rd in 1936, may have resulted from the successful recrudescence of an infection of the previous year, not from a fresh beetle infection. Fransen (1935) also noted the appearance of the disease in the absence of beetles. Active external symptoms were recorded by the author on 3rd June, 1939; but more typically they first appear about mid-June.

Chapter 2

THE HISTORY AND DISTRIBUTION OF THE DISEASE

History

The first record of the disease was made in Picardy in France in 1918 (Guyot 1921). It was attributed to toxic gas or to some effect of the 1914–1918 war, but the description of the symptoms makes it quite clear that Elm Disease was involved. The first record for Holland (Spierenburg 1921) was made in 1919, but markings were visible in the 1917 ring. In Belgium (Anon. 1925) it was said to have been under investigation since 1919. In Germany (Anon. 1922) it was possibly recorded in 1920, though a suggestion was made at the time that the lack of foliage was due to over-blossoming; a more definite record was given for 1921 (Pape 1924).

Certainly the disease seems to have appeared very generally in north-west Europe at the end of the first world war. In view of the wide range of the early discoveries it is likely that its real introduction to Europe was at a considerably earlier date. Whence it came is still a matter of surmise. The most likely explanation is that it is endemic somewhere in eastern Asia on one of the resistant, but not immune, species which are native to that region, and that, imported to Europe, by what means is unknown, it found far more susceptible host elms and possibly more efficient vectors, than had hitherto been available to it.

At the outset there was considerable argument as to the cause of the disease. The rather frequent occurrence of bacteria in dead and dying elm wood led to the suggestion that they were responsible and Brussoff (1925) claimed to have performed successful inoculation experiments with his so-called *Micrococcus ulmi*. However, *Graphium* (*Ceratostomella*) *ulmi* had already been isolated (Schwarz 1922). The first really satisfactory inoculation experiments were reported from Germany in 1927 (Wollenweber 1927). A full description of the early history of the disease is given in one of the first American papers (Clinton and McCormick 1936).

Such was the position when the disease was first recorded in Great Britain in 1927. The symptoms had been well described, and the cause was known with reasonable certainty, though the bacterial explanation of Brussoff had not been entirely disproved and there remained the possibility that more than one disease was concerned. It had been observed that the disease sometimes killed trees very rapidly. It was generally assumed that trees which had not yet succumbed would do so later. At that time there was no real information on the fate awaiting attacked trees, nor was it possible to foretell the rate of the spread of the disease, since new records were generally more likely to be discoveries of areas already infected, but not previously visited, than evidences of spread. Nothing was then known about the means of transmission of the fungus. Evidence had been produced that some species of elm were more resistant than others, but the information was very vague.

However, within a very few years, more definite information became available. The bacterial concept was given up as a result of isolation of *Graphium ulmi* from Brussoff's material (Stapp 1928). The fungus was found fruiting in *Scolytus* galleries and the suggestion was immediately made that bark beetles might be vectors (Marchal 1928). A year later the fungus was isolated on and in beetles (Betrem 1929), and by 1931 the whole story of the connection between the fungus and these insects was more or less established (Fransen 1931). A year later the perfect form of the fungus, *Ceratostomella ulmi*, was produced in mixed cultures, and by inoculations with two heterothallic strains of *Graphium ulmi*

(Buisman 1932). Thus while the author was discovering the best methods for the long term study of Elm Disease in the field, European workers were producing the remainder of the basic knowledge, on which further study could be founded. This coincidence of timing provided a fortunate start, to what would otherwise have been a very difficult assignment.

To study all the further work on the disease is beyond the scope of this Bulletin. Only such references to later work will be made as are needed to give the necessary background to the surveys carried out in Britain.

The disease was first recorded in Britain at Totteridge in Hertfordshire in 1927. In 1926 a row of about fifteen elms along a road there had died suddenly and had been removed. Defoliation was said to have proceeded very rapidly, so that some of the trees became leafless in a week. In the following summer a large elm in a nearby garden died, showing the same external symptoms. Because of the possibility of Elm Disease, this tree was also felled. On examination it showed definite internal symptoms of Elm Disease. Another tree in the same neighbourhood showed yellowing of the crown, and was also found to have the typical brown markings of the disease, from which the fungus was successfully cultured; so that this tree, situated on Totteridge Golf Course (Plate 32) must be regarded as the first definitely proven case in Britain (Wilson and Wilson 1928). When the author first visited the area in 1928 there was still evidence to show that some trees other than elms, as well as shrubs, were included in the widespread deaths, which had taken place along the road in 1926. Subsequent experience in other areas has suggested that this rapid and more or less simultaneous death of a whole row of trees and shrubs, not all elms, was more likely due to a leak of illuminating gas than to Elm Disease. Thus it may merely have been coincidence that the attention which was drawn to Totteridge by the death of a whole row of trees resulted in the discovery of genuine Elm Disease in two other elms, one in a garden and the other on the golf course. It is quite possible that the high population of beetles bred from the gas-killed trees may have assisted in the development of the local outbreak of Elm Disease which subsequently occurred.

It was observed at this time that the golf course tree showed markings in the 1926, as well as in the 1927, annual ring of its wood. It is interesting that suckers from this tree were 15–20 ft. high by 1945 and perfectly healthy.

These trees, however, were certainly not the first cases. There is strong evidence that the disease was present in the country well before 1927. In 1928 several outbreaks were noted, which were said locally to have been active for 3 to 5 years. In one case, at Cowes in the Isle of Wight, diseased trees had been present for 15 years. This implies the existence of Elm Disease in 1913, but there was an unusually heavy *Scolytus* attack, and it is possible that the earlier cases may have been due only to *Scolytus* beetles killing trees weakened by some other cause. Both in Essex and in the Isle of Wight dead elms, some of which had been dead at least two years judging by twig deterioration, were present immediately adjacent to diseased ones. Assuming that they also died of the disease, this would mean that the fungus had been present for at least four or five years prior to 1928. In a few cases trees were found with typical markings in rings much earlier than 1927. Evidence will be given, when the question of recovery is under discussion, which strongly supports the suggestion that markings are formed only in years when the tree is actively attacked; though such an attack may not necessarily produce any external symptoms. Unfortunately no special study was made of rings with markings, nor were proper records kept of all the cases which were by chance observed in the course of other work. But among those that were recorded the most remarkable was the early discovery of a tree at Kettering, which showed clear markings in the 1912, 1913 and 1914 rings. In trees from near Newmarket the fungus was isolated from the 1923 ring, and in one tree from Totteridge, where the disease was first recorded in 1927, typical markings showed in the 1918 ring, but failed to yield the fungus. These observations suggest that the disease may have taken a long time to build up to a stage where it attracted public attention. In this connection it must be remembered that the Continental reports were with one exception records of actual outbreaks, not of the position of the markings, so it is probable that there also the presence of the disease greatly antedated its discovery. Indeed, markings in years earlier than 1918 have been recorded by Liese (1952).

In the early summer of 1928, the Forestry Commission started a survey which was centred on Totteridge and conducted by Mr. James Macdonald. This led to the discovery of several other cases in the vicinity. This stage had been reached by the middle of July, at which time the author joined the staff of the Imperial Forestry Institute at Oxford, and started to work on Elm Disease for the Forestry Commission. A few reports of suspected Elm Disease had already been received from other parts of England, and the scope of the survey was immediately widened to cover these areas. The discovery of the disease in places as far apart as Littlehampton in Sussex, Kidderminster in Worcestershire, and Swaffham in Norfolk obviously rendered detailed local distribution surveys of less value, and from then on the

surveys were conducted on a much wider basis.

The data collected on these surveys provide the main basis for this Bulletin. A summary of the data collected up to 1932 has already been published (Peace 1932), and up to 1948 a fairly wide distribution was given to a series of mimeographed annual reports on the disease; but no general review of the surveys as a whole has previously been made.

At no stage have the surveys been sufficiently detailed to give an exact picture of disease distribution in Britain. Only by the comparison of nation-wide periodic surveys would it have been possible to speak with confidence of the rate of spread or geographical progress of the disease. Certainly it was much less common in most areas in 1928, than it is now (1959). But it would be rash to say that it has made any wide extensions of its range since 1928. It may have done so, but equally its apparent first appearance in a new area may merely indicate that an attack, which was already there, had become sufficiently violent and widespread to attract attention. It is the author's opinion that the disease started somewhere in the south-east and spread north and west, and that this spread was not completed when the survey first started in 1928. That is an assumption for which there can now be no proof.

Now (1959) the disease is generally present all over England south of a line from Chester to Hull, and over the whole of Wales, wherever elm occurs. As far as England and Wales are concerned, therefore, all the major elm areas are infected. In the northern part of England elm is of much more sporadic occurrence, and, no doubt partially as a result of this, the disease is much less common. It has been found in a number of areas, including one a few miles south of Carlisle, and another in the extreme north of Northumberland. The last mentioned outbreak extends over the border and forms part of what was a quite serious outbreak in the Coldstream-Kelso-Jedburgh area. Northwards from this outbreak the disease can be found very sporadically as far as the outskirts of Edinburgh, but it has never been observed in Scotland west of a line from Edinburgh via Peebles and Galashiels to Hawick. In 1955 a single case, confirmed by culturing, was found in an avenue of elms between Inverkeithing and Cowdenbeath just north of the Firth of Forth. As far as could be ascertained *Scolytus* beetles had never been recorded in Scotland, until the appearance of *S. destructor* in conjunction with the Elm Disease outbreak in the Coldstream-Kelso area, where it was abundantly present. *S. destructor* was also found associated with most of the outbreaks in the north of England. The more northerly cases in Scotland have not been in a suitable condition for beetle breeding, and rather cursory examination has yielded no evidence of feeding galleries, though they may well have been present. It seems possible, indeed probable, that the distribution of the vector beetle is the main factor limiting the spread of the disease into Scotland, where there are certainly enough elms in the lowland regions to sustain an outbreak. The elms in this area are predominantly *Ulmus glabra* (wych elm), which is generally considered to be highly susceptible; so that the lack of progress of the disease cannot be attributed to the resistance of the elm species involved. The distribution of the disease in the north of England and in Scotland is shown in Fig. 1.

There is no published record of the disease in Eire and Northern Ireland, but Heybroek (personal communication) has recently observed the disease in many places in Eire. In early September 1952 the author made several long tours in Northern Ireland, but failed to see any symptoms at a time of year when they would have been most readily detected. Material showing clear markings was received from Guernsey in 1929. A few active cases were observed by the author in Jersey in 1954.

Nearly all the survey figures, which appear later in this paper, refer to the main area of attack, the southern half of England. Over the rest of the infected area elm is only locally a tree of importance.

In northern England, Scotland and Wales the disease has done a little damage to a genus, which in any case plays a very small part in the general treescape. Only on the eastern Borders, where England marches with Scotland, had the disease behaved in at all the same catastrophic fashion, as it did in so many areas in the south of England.

Over the period during which the disease has been under observation in England, it has spread widely, or possibly been discovered widely, in Europe, and it is now known over the whole of the Continent with the exception of Norway, Finland and possibly Greece (Anon. 1947). Its latest extension appears to have been into southern Sweden (Mathieson 1950) and into Denmark (Gram 1955). The extent of its spread into western Asia is not known. It was first recorded in the United States in 1930 (May 1930) and in Canada 1944 (Pomerleau 1946). Since then the disease has spread over nearly all the north-eastern States as far west as Illinois and as far south as Tennessee, with an isolated centre in Colorado (Swingle, Whitten and Young, 1949; Holmes 1956). In Canada, the disease, which originated as a separate outbreak from those in the United States, is limited to the provinces of Quebec and Ontario in the valleys of the St. Lawrence and Ottawa Rivers and sporadically to the north of Lakes Erie and Ontario (McCallum and Stewart 1951; Holmes 1956). Certain aspects of its transmission to and spread within these countries will be considered later in connection with its behaviour in Britain.

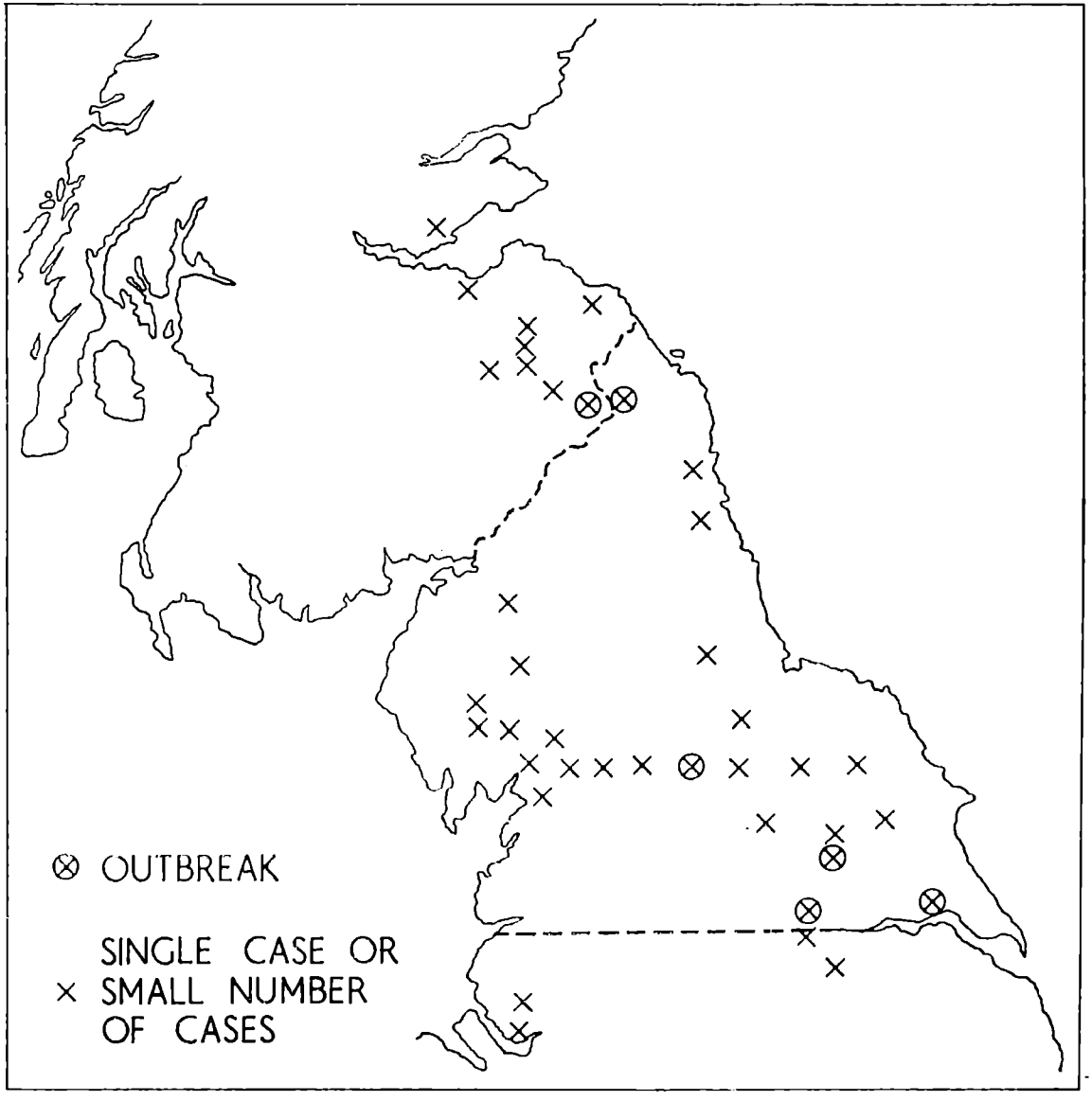

FIGURE 1. Map showing the known distribution of Elm Disease in the North of England and South Scotland, 1959.

Chapter 3

OTHER DISEASES OF ELM

It is no part of this Bulletin to give a general review of diseases of elm. There are, however, a number of organisms attacking elms and non-living agencies affecting them, the symptoms of which are not unlike those of Elm Disease due to *Ceratostomella ulmi*. These are briefly described below.

Verticillium Wilt

The fungus *Verticillium albo-atrum* Reinke & Barth. is a common cause of vascular wilt in broad-leaved trees, particularly maples. *Verticillium dahliae* Kleb. may also sometimes be involved. *Verticillium* is commonest on young plants in the nursery, particularly after transplanting. It is quite often fatal. It can attack older trees to which it usually does much less damage. It causes brown streaking in the wood, but in elm the markings are much paler and less well-defined than those caused by *Ceratostomella ulmi*. The external effect on the tree, however, is very similar to that caused by *C. ulmi*, though it is normally less violent.

As a cause of disease in elms *Verticillium* has attracted some attention both on the Continent (Meer 1926; Goidànich 1935) and in the United States, where it has done appreciable damage to quite large trees (May & Gravatt 1951). Even there it is far less important than *Ceratostomella* Elm Disease.

In Britain *Verticillium* has been recorded definitely on elm on only one or two occasions, and ever then it was associated with only very slight damage. It is possible that it may also occur in trees showing no external symptoms, as it certainly does in some species of maples of the genus *Acer*.

Cephalosporium Wilt

Another wilt disease of elms, which is confined to the United States, is caused by a fungus now called *Dothiorella ulmi*, but originally identified as a *Cephalosporium*, because it produced in culture spores typical of that genus. The wilt symptoms are essentially similar to those of true Elm Disease, but the streaking in the wood is much more diffuse and watery. At one time this disease attracted considerable attention (Goss & Frink 1934; Creager 1935; McKenzie & Johnson 1939; May & Gravatt 1951), but it was never regarded as approaching the *Ceratostomella* disease in importance. It was, however, considered to be more serious than *Verticillium* wilt (Johnson 1937). But of recent years it has attracted no attention in the United States, and it has never been found in Britain.

Phloem Necrosis

In the United States a virus disease, known as Phloem Necrosis, is regarded as equally if not more important than *Ceratostomella* Elm Disease. Its distribution there is generally more westerly than that of Elm Disease, but the two do overlap. It takes the form of a gradual decline in the health of the tree, but in large trees part of the crown may be affected before the remainder. The foliage at the tips of the highest shoots is first affected, the leaves drooping because of induced curvature of the petioles, and the leaf-blades, which often become stiff and brittle, turning up at the margins. The leaves then wither and fall, possibly partly as a result of the death of the roots. The phloem of the larger roots and near the base of the trunk turns yellow and then brown. Infected trees invariably die. The virus is carried by insects, so that preventative spraying to stop insect feeding is a possible, though expensive, method of control. Full descriptions of the disease have been given by Swingle (1942) and by Swingle, Whitten & Young (1949).

It occurs on the native American species *Ulmus americana* and *U. alata*. Fortunately it has not reached Europe, and it is not known whether European elms would prove susceptible.

Bacterial Infections

The comparatively short, dark, isolated streaks in elm twigs, which often arise in connection with dead side twigs, or broken twig stubs, usually yield bacteria on culturing. Indeed in some trees bacterial streaking, not apparently associated with any external symptoms, can be quite extensive (Westerdijk & Buisman 1929). Elms in this condition have occasionally been found in Britain. Bacteria are quite commonly found in a proportion of the cultures made from trees infected by *Ceratostomella*, and it was this no doubt that led Brussoff (1925) to believe that the disease was bacterial in origin. There is in fact no evidence that these undoubtedly common bacterial infections have any pathological significance.

Nectria Dieback

Sporadic twig or even branch dieback associated with the fungus *Nectria cinnabarina* (Tode) Fr. is fairly common but never serious in Britain. The fungus forms cankers on twigs and on branches occasionally up to arm thickness, quite often girdling them. There are seldom more than two or three girdled twigs on an individual tree so that the damage, which first shows as wilting and withering of the foliage, is not very striking. It can be mistaken for a localized infection by *Ceratostomella;* but even if there is occasionally confusion, *Nectria* dieback is too uncommon for this to produce any serious error when large numbers of trees are under investigation. More careful examination of individual trees will always show whether *Nectria cinnabarina* is present. The salmon pink, pin-head fruit bodies, which are quickly produced in great quantities on the dead bark, are very easy to detect.

This fungus is nowhere serious as a cause of disease on elms, but unfortunately the first of the Dutch selections resistant to *Ceratostomella ulmi*, *Ulmus* "Christine Buisman", turned out to be abnormally susceptible to *Nectria*, and has been more or less discarded on this account. The damage on this variety is sufficiently serious to cause obvious disfigurement and very unshapely growth. Testing for resistance to *Nectria cinnabarina* is now part of the Dutch elm breeding programme. The Siberian elm, *U. pumila* and its variety *U. pumila pinnato-ramosa*, both of which have the advantage of being resistant to *Ceratostomella ulmi*, have also been found to be abnormally susceptible to *Nectria cinnabarina* (Buisman 1935; Jacques 1944). These varieties are also subject to frost injury, and there may well be some connection between this and *Nectria* attack.

Dieback of Elms in Northern England and in Scotland

As soon as the surveys were extended northwards, elms, usually *Ulmus glabra*, were often found showing sparse, unhealthy-looking foliage and generalized dieback. It was clear that these were not cases of infection by *Ceratostomella*, though that fungus does occasionally cause very similar symptoms. No other pathogenic fungi or bacteria could be isolated and the cause of the disease remains a mystery. The decline of affected trees is usually very slow indeed, so that they may remain many years in an obviously sickly condition. This persistence of affected trees probably makes the disease appear more serious than it actually is. In the regions where it occurs it certainly adds considerably to the difficulties of Elm Disease survey, and probably impairs its accuracy.

Drought

Drought, causing premature yellowing and shedding of leaves, was a serious confusing factor in the surveys in some years. This was particularly the case in 1933, 1934, 1938, 1940 and 1947.

Autumn Coloration

Experience suggested that the surveys ought not to be started till mid-August, by which time most cases had developed visible symptoms. It was usually possible to complete the surveys by the third week in September. After that time there was sometimes difficulty, particularly on large old trees, in distinguishing Elm Disease from premature autumn colouring, which, however, often took place on branches that were already slightly affected by the disease, and occasionally on branches partially girdled by *Nectria*.

Chapter 4

VARIABILITY IN PATHOGEN AND HOST

Before dealing with the progress of the disease in individual trees and in elm populations, it is desirable to consider how far this may be affected by variations in pathogenicity of the fungus and in the susceptibility of the host.

Variations in Ceratostomella ulmi

Culturing the fungus from a large number of diseased elms clearly showed it to be very variable in appearance. Variants also appeared as mutant sectors in existing cultures (Walter 1937). Walter & May (1937) recorded a strikingly different form with brown mycelium, isolated both in England and in the United States. In two English trees the brown markings in the wood were accompanied by less well-defined streaks of faint crimson. Isolations from these trees yielded an unusually yellow and rapidly growing form of *C. ulmi*, with more than usually abundant production of yeast-like bodies in culture.

Tyler and Parker (1945) reported considerable differences in pathogenicity, which ranged from moderately virulent to virulent, among variants of

the fungus. One exceptional strain was practically non-pathogenic. Frederick and Howard (1951) also found variation in pathogenicity, as well as in physiological behaviour, between different isolates of the fungus. Swingle (1950) found a strain that was highly virulent to the normally resistant elm "Christine Buisman". However, Walter (unpubl.), working in England, found no significant variation among the isolates he tested, with the exception of one virtually non-pathogenic strain.

In view of this somewhat conflicting evidence, it is almost impossible to estimate what influence, if any, the variability of the fungus has on its local or general behaviour.

Variations in Host Resistance

Differences between Elm Species

Surprisingly little exact information exists on the relative susceptibility to *Ceratostomella ulmi* of European elm species. In most countries the obvious presence of the disease on all the species and varieties commonly occurring or planted suggested that relative resistance was hardly a matter of practical importance, and from an early stage selection and breeding for resistance was based more on a search for resistant individuals among generally susceptible species and on the use of resistant Asian elms, than on such variations in specific resistance as exist between European species.

In south-east England, where the earlier disease surveys were particularly concentrated, the elm population is a complex mixture of several species and their hybrids (Melville 1944; Richens 1955). Early difficulties in identification led the author, perhaps unfortunately, to abandon practically all effort to identify diseased elms, so that during the long period covered by the surveys very little information was collected on varietal susceptibility. Such information as was collected, together with that from other observers and other countries, is set out below. Some of this is observational; but of those quoted below, Westerdijk, Ledeboer and Went (1931), Wollenweber (1931), Went (1938), Walter (1939a) and Smucker (1941) based their results on inoculations, though of course susceptibility to inoculation may differ from that to natural infection. Walter's inoculations were carried out at Oxford in 1937. Reinoculation of the surviving trees by the author in 1939 substantially supported Walter's results. The British elms are dealt with below on the basis of Melville's classification, as adapted by Clapham, Tutin and Warburg (1952).

Ulmus glabra Huds., Wych Elm

This species, which is the common elm of the west and north, is certainly frequently infected. Melville (1944) regarded it as the most susceptible of the British elms. Mańka 1(953) found it the most susceptible species in Poland. Westerdijk *et al.* (1931), Wollenweber (1931) and Walter (1939a) all recorded it as very susceptible on inoculation. The fastigiate variety, var. "fastigiata" Loud., which is very occasionally planted, does sometimes display a quite high degree of resistance (Westerdijk *et al.* 1931; Noell 1935), but three young trees inoculated in Britain proved to be highly susceptible. A golden-leaved, fastigiate variety seems to be distinctly more resistant. But these fastigiate forms are slow growing and of no value for general planting. The attractive weeping form, var. "pendula" (Loud.) Rehd. is certainly highly susceptible, being commonly and often severely attacked.

X Ulmus hollandica Mill.

This elm is very variable, but all its forms are presumed to be hybrids between *U. glabra* and *U. carpinifolia*. *U. hollandica belgica* Rehd., which constituted quite a large proportion of the roadside elm plantings in Holland, proved unfortunately to be very susceptible (Buisman (1935). Westerdijk *et al.* (1931), Wollenweber (1931) and Walter (1939a) all found it moderately susceptible on inoculation.

The Huntingdon elm, *U. hollandica* var. *vegeta* (Loud.) Rehd., on the other hand, has always had a reputation for resistance. Wollenweber (1931) found it highly resistant on inoculation, while Walter (unpubl.) considered it the most resistant elm occurring commonly in Britain. Noell (1935), however, found that its resistance was not maintained, and the author has come across a considerable number of infected trees, some of which were quite badly damaged. About 60 trees of this variety were planted at Schiedam in Holland in 1917. Only 10 remain, most of the others having succumbed to the disease. The losses among *U. hollandica belgica* in the same place over the same period were, however, appreciably heavier (Heybroek, personal communication). The author also carried out inoculations on a few trees of this variety, which resulted in considerable damage to them. Its resistance is therefore not sufficient to recommend it for general use.

Ulmus procera Salisb., English elm

Melville (1944) regarded this species as relatively resistant, but Wollenweber (1931) found it very susceptible to inoculation. Westerdijk *et al.* (1931) considered that it showed more individual variation in resistance than most other species. Smucker (1941) found it less susceptible than *U. americana*. It is certainly quite commonly infected in nature in Britain.

Ulmus stricta Lindl., Cornish elm

This elm and its varieties, in particular the Jersey elm var. *sarniensis* (Loud.) Moss, have been widely

planted in Britain as street and park trees, mainly on account of their attractive short-branched habit. They have generally been regarded as relatively resistant (Melville 1944). Walter (1939a) found the Jersey elm the most resistant of the limited range of species and varieties he tested by inoculation. In the field in Britain there are many of these elms still healthy, some having recovered from earlier attacks. Nevertheless numerous cases of severe infection are known, and this elm is even more suspect than the Huntingdon elm for general use.

Ulmus coritana Melv.

This species has been distinguished only relatively recently, and no information is available on its relative resistance.

Ulmus carpinifolia Gled., Smooth-leaved elm

This species, once known as *U. nitens*, is certainly quite frequently attacked. Westerdijk *et al.* (1931) found some forms of it very susceptible. Smucker (1941) classed it with *U. americana* as highly susceptible. On the other hand, Went (1938) found that two of its varieties, var. *dampieri* Rehd, and var. *wredei* Rehd. were appreciably resistant.

Ulmus plotii Druce

This is another elm which has been clearly distinguished only comparatively recently. Melville (1944) regards it as fairly resistant; but inoculation experiments on a very small scale by the author indicated a moderate degree of susceptibility.

Ulmus diversifolia Melv., Small-leaved elm

This species, sometimes known as *U. minor*, is certainly often infected. Walter (1939a) included a mixed population of seedling elms, generally strongly characteristic of this species, in his inoculation experiments. Their susceptibility was very variable, but many trees were badly injured.

Foreign Elms and Related Trees

Among other species, *Ulmus americana* L., the most important elm in America, is probably more susceptible than any of the European species (Wollenweber 1931; Walter 1939a). Most Asian species, in particular *U. pumila* L., *U. pumila pinnato-ramosa* Henry and *U. wallichiana* Planch., are highly resistant (Westerdijk *et al.* 1931; Sibilia 1933; Buisman 1935; Went 1938). Inoculations of a few trees of *U. pumila* and *U. pumila pinnato-ramosa* in Britain caused no external symptoms, though extensive streaking occurred in the wood, and there seems to be no doubt that they really are highly resistant. Unfortunately their growth in our climate is slow, and they are subject to considerable winter dieback. They seem much more suited to countries with a hotter summer climate, such as Italy.

Wollenweber (1931) found *Zelkova serrata* Mak. susceptible to inoculation with *C. ulmi*. *Zelkova* is a genus very closely related to *Ulmus*. Although several species, in particular *Z. carpinifolia* K. Koch, have been planted quite widely in parks and gardens in Britain, no natural infections have been recorded. However, *Z. carpinifolia* is probably resistant (Heybroek, personal communication).

A specimen of *Z. serrata* in a collection of elms, most of which developed Elm Disease, remained quite unharmed. It is possible of course that it is unattractive to *Scolytus* beetles, and was thus never infected. Smucker (1941) found the Water Elm, *Planera aquatica* Walt., an American relative of *Ulmus* very rarely planted in Britain, also susceptible to *Ceratostomella*. The Hackberries, *Celtis* spp. are probably resistant (Heybroek, personal communication).

The limited evidence available suggests that *Scolytus* beetles may have some preferences among elm species, though they are probably capable of feeding on all of them (Fransen 1939b). In nature such preferences might well render the course of the disease very different from its theoretical behaviour based on reactions to inoculation.

It is clear that none of the elm species or hybrids occurring naturally, or commonly planted in Britain, is really resistant to *Ceratostomella*. If any of them are planted, some losses from the disease are a definite possibility. If elms are being planted, and if resistant selections are not available, it is probably better to choose the species on the basis of growth and form, rather than on the relatively slight differences in resistance that do exist.

The Possibility of Natural Resistance.

When the disease was at its height in the early nineteen-thirties, it was obvious that, even in areas where over 50 per cent of the elms had been killed, a small number of trees remained completely unaffected. It was thought that these might be naturally resistant individuals, and in 1938 a number were selected by Dr. Walter and the author for testing. Very heavy inoculations of spore suspensions into chisel cuts round the stem, made in the summer of 1939, produced in most cases only slight foliage yellowing. This appeared to support the idea that the trees were resistant, and in 1939 more trees were selected. Inoculations in that year, repeated in 1940, showed clearly that many of the trees were in fact susceptible. The project was abandoned after 1940 because of war-time difficulties, though vegetatively propagated stocks of some of the more promising trees were carried on for further testing. Details of these trees and of their reaction to inoculation are given in Table 2. Where several trees appear under

INOCULATION OF LARGE ELMS

TABLE 2

Location	*Key Letters*	*No. of Trees*	*Description of Trees and Site*	*Effect of Inoculation*			*Condition in 1944 or 1945*
				1938	1939	1940	
Silsoe, Bedford	W.P.	2	Vase-shaped habit. In a wood devastated by Elm Disease.	—	Severe attack	Moderate attack	One dead, the other nearly so.
Witham, Essex	H.F.	4	Trees of moderately good habit. In an area where the disease was very bad between 1928 and 1932.	—	None-moderate attack	—	One dead, two slightly diseased, one healthy.
Quendon, Essex	R.T.	2	Vase-shaped trees of particularly fine growth probably *Ulmus glabra* x *coritana* hybrids (Plate 1). In a heavily infected area.	Very slight yellowing	Slight yellowing	—	One healthy, one blown down.
Stansted, Essex	E.B.	4	Trees of good growth and form. Quite healthy, despite dead and diseased trees all around.	Very slight yellowing	None-moderate attack	None	All dead (Plate 30).
Ugley, Essex	U.W.	3	Trees of rather poor habit, but in a very heavily infected area.	—	None-slight attack	None-slight attack	All perfectly healthy.
Elsenham, Essex	N.N.	4	Large-leaved suckers in a hedge.	—	Slight-moderate attack	—	Not visited.
	M.B.	1	A specimen of *U. minor*, in a very heavily infected area.	Moderate	—	—	Not visited.
	F.M.	2	Upright trees.	—	None-slight attack	None-moderate attack	One still showed dead wood, the other healthy.
Studley, Oxford	M.H.	1	Probably a *U. glabra* hybrid. An attractive tree (Plate 31).	Slight	Very slight attack	—	Perfectly healthy, though still surrounded by diseased trees.
Hindlip, Worcestershire	H.H.	4	Trees of moderately good habit.	—	None-severe attack	Very slight-severe attack	One healthy, three felled.
Ryston, Norfolk	—	6	Six different varieties in an arboretum.	—	Slight-severe attack	Slight-dead	Three felled, one nearly dead, one, a form of *U. carpinifolia*, still healthy.

the same key letter there was usually good evidence, except at Ryston, that they were all of the same clone, though in fact separate records were kept for each of them.

For many years the elms at Stansted formed a very striking group of apparently resistant trees amid the devastation surrounding them. They were in an area where more than 50 per cent of the elm population had been killed; in their immediate vicinity the death rate was even higher. They survived inoculation without developing significant symptoms. They were in fact used as an illustration of natural resistance in an U.S. Department of Agriculture Circular (Walter, May & Collins 1943). There is no doubt, however, that they died of Elm Disease during the war years, probably as a result of fresh natural infection.

A few of the most attractive clones were propagated vegetatively for further testing. A number of young plants, mostly on their own roots, were treated in 1940; further inoculations on much older and larger trees, established in the field, were carried out in 1948 and 1949, The results of these experiments are given in Table 3. The figures in brackets indicate the number of trees used in each case.

In view of these results the project was finally abandoned.

Around 1949–50 the disease did considerable damage in the Isle of Sheppey in Kent. Indeed this appears to have been its last recorded "flare-up". A very attractive vase-shaped tree, probably a form of *U. glabra*, in Eastchurch churchyard was found to be quite free from the disease, and was propagated for testing. It proved on inoculation to be highly susceptible.

There is thus considerable doubt whether the survival of some elms completely unharmed in areas once heavily attacked has anything to do with their resistance to the fungus. Indeed, the fact that some trees have been severely attacked, have recovered and have then remained healthy, points in the same, somewhat negative, direction. Possibly further investigation of the feeding behaviour of *Scolytus* beetles might throw more light on this matter. For the moment it remains quite unexplained.

Selection and Breeding for Resistance

The very rapid progress of the disease in north-west Europe, particularly in Holland, and later the very general high susceptibility of *Ulmus americana* in the United States, quickly drew attention to the need for resistant forms. In America, where work started in any case much later than in Holland, the high and uniform susceptibility of the American elm delayed progress, but the Dutch, working on a much wider selection range, have achieved promising results which have been reviewed by Went (1954) and later by Heybroek (1957).

The first successful result of the Dutch work was represented by *Ulmus* "Christine Buisman", an elm of high resistance to *Ceratostomella*, and of quite attractive, though rather floppy habit, but which unfortunately proved very prone to attack by *Nectria cinnabarina*. This susceptibility is found also in Britain; some of the relatively large trees maintained in the nursery for propagation were very badly attacked by this fungus. Nevertheless some of the trees introduced into Britain just before the war, and planted in various parts of the country, have made quite satisfactory, though not particularly rapid, growth when site conditions were good. In one case only has infection by *Ceratostomella* been found on this elm in Britain. This was in a plant cut back for layering; many of its shoots were dying back, and typical markings were abundant.

The second Dutch introduction, *Ulmus* "Bea Schwarz", a clone of *U. carpinifolia*, was tested for resistance to *Nectria* as well as to *Ceratostomella*. It was introduced into Britain after the war, and a number were planted in an area where the disease was still rife. They are still in the course of establishment. Unfortunately this clone has a poor shape, with

TABLE 3

INOCULATIONS OF VEGETATIVE PROGENY OF NATURAL SELECTIONS

Location	*Key Letters*	*Effect of Inoculation*		
		1940	1948	1949
Stansted, Essex	E.B.	None to moderate (5) attack	—	—
Elsenham, Essex ..	M.B.	Moderate (1) attack	—	—
Studley, Oxford ..	M.H.	Slight to moderate (3) attack	None to slight (5) attack	Slight to moderate (4) attack
Quendon, Essex ..	R.T.	None to moderate (7) attack	None to severe (5) attack	—

Note: Figures in brackets indicate number of trees used.

a pronounced tendency to produce large, unbalanced branches, which tend to break in high winds or under the weight of snow. For this reason it can hardly be considered for widespread planting, however good its resistance.

It has now been realized that, provided resistance reaches a reasonable level, other characters such as habit, leaf size (the Dutch favour large-leaved elms), and resistance to exposure should also be taken into account. Tests are now being carried out on selections and hybrids which are well above average in resistance, but which do not all reach the high level demanded earlier. Eleven of these selections have recently been introduced into Britain, and are being tested under conditions of heavy natural infection, in so far as these can now be provided. Many of these selections are highly vigorous and of good habit. They include:

U. hollandica vegeta x *carpinifolia* (3 clones). One of these is now being propagated in Holland under the name "Commelin".
U. pumila pinnato-ramosa x *hollandica vegeta* (1 clone).
U. carpinifolia, selfed (2 clones, in one of these the parent tree was *U.* "Bea Schwarz")
U. carpinifolia x *carpinifolia* "Bea Schwarz" (2 clones).
U. "Christine Buisman" open pollinated (1 clone).
U. wallichiana x *carpinifolia* (1 clone).
U. wallichiana x *U. glabra* "fastigiata" (1 clone).

Apart from the investigation of supposed naturally occurring resistance, described above, very little work has been done in Britain on selection for resistance, and none on breeding. Walter selected 21 individuals showing resistance above average from the 320 seedlings showing predominantly *Ulmus diversifolia* characters, which he inoculated in 1937. These were tested further by the author, but in none was resistance sustained and the last was finally abandoned in 1953.

Between 1951 and 1953 four triploid clones of *U. glabra*, raised in Sweden, were inoculated in Britain to test their susceptibility to *Ceratostomella*. All four proved susceptible.

Discussion

It is clear therefore that there is variation both in the pathogenicity of the fungus and in the susceptibility of the tree, though the comparatively slow progress of selection and breeding arises largely from the extremely low percentage of resistance in most of the elm populations initially available. Though no experiments have been designed expressly to throw light on this point, there is at present no evidence that different elms vary in resistance to different strains of the fungus. If this were the case it would obviously greatly complicate the whole matter of resistance testing, and might render suspect many of the results already achieved. Although most workers have used mixed sources of inoculum they have certainly never tried to include, even if it were possible, all the variants of the fungus.

Involved as we are with a fungus known to produce variants of differing pathogenicity, we must be prepared for apparent breakdowns in resistance in clones, which otherwise appeared to have been adequately tested. This alone gives us a good reason for abandoning the idea of planting a limited number of clones of apparently very high resistance, in favour of the use of a large number of clones, the resistance of which may be of a lesser degree. The whole question of planting elms, whether old varieties or new selections, viewed in the light of the present comparatively low level of attack, will be discussed later.

Chapter 5

THE PROGRESS OF THE DISEASE

Progress in the Individual Tree

Recovery and its Basis

For some years after the disease was discovered, it was assumed that all trees attacked would sooner or later die. The extreme virulence of the disease in its earlier manifestations certainly gave support to this view. In Britain, however, it was suspected in 1929, following the second survey, that trees were "recovering" from the disease, though the reasons for such recovery were quite unexplained.

In Germany recovery was reported at quite an early stage by Wollenweber and Richter (1930), though they regarded it as confined to young trees. They noted that elms which had "recovered" could be reinfected by inoculation.

In Holland, where the disease received more intensive study than in other European countries, recoveries were not easily detected because of the

prompt removal of diseased trees as part of a declared policy of phytosanitary eradication, and possibly also because of the high, uniform susceptibility of some of the clones that had been used there for roadside planting.

For the same reasons the possibility of recovery received scant attention in the United States, where in any case the high susceptibility of the American elm rendered it less likely to recover. Nevertheless, one case of recovery was noted by May (1931) soon after the disease reached America, while Smucker (1940) observed 97 per cent recovery in inoculated American elms. Dimond (1948), writing about chemotherapy, stated that recovery was infrequent, but commoner in treated trees.

Both in the United States and in Holland emphasis on the possibility of recovery would have weakened the case for the destruction of diseased trees, whereas in Britain such a possibility supported the official policy of *laissez faire*, and a very varied elm population made its occurrence likely.

The reasons for the phenomenon of recovery only gradually became apparent. In 1932 the author cultured the fungus from two trees that had shown no active external symptoms since 1928. In 1935 he found that successful symptom-producing inoculations could be made using the fungus isolated from trees which no longer showed external evidence. Fransen (1935, 1937) confirmed that the fungus could remain dormant but alive in the tree for periods of up to nine years without causing any external symptoms. The same phenomenon was observed at about the same time in the United States (Anon. 1938), and it was pointed out that in an eradication programme this would throw doubt on the value of scouting for external symptoms only. Smucker (1938), inoculating elms under greenhouse conditions, found that only 2.6 per cent of those showing active external symptoms, as a result of inoculation in one year, redeveloped symptoms the following year, apparent evidence that the fungus had difficulty in moving from one annual ring to the next. Later he confirmed that successful inoculations could be made with the fungus isolated from the annual rings of earlier years in which it had been lying dormant (Smucker 1940). His results were supported by Walter (unpubl.), who found that approximately 7 per cent. of very heavily infected young American elms developed symptoms the second year. With *U. stricta* the percentage of recurrence was nil.

Inoculations clearly show that the discoloration in the wood is normally limited to the year in which the fungus was introduced. Walter (unpubl.) inserted inoculum on wooden pegs into holes bored in the stem, so that earlier rings were infected, and found that the spread which did take place in the older rings was very limited, and had no pathological significance. One exceptional case was recorded as a result of the heavy inoculation of the large elm tree "R.T." at Quendon. This inoculation was made in 1938, but the markings extended 30 ft. up the tree in the 1937 ring, though they were much more extensive in the 1938 ring. However, dissection of a large number of trees by Walter and Parker gave more and more evidence that normally only those years when the disease was active in the tree gave ring markings, and that the markings were usually, though not always, associated with external symptoms of injury, sometimes very mild. Recurrence of the disease in a tree must therefore involve either a fresh infection or the outward movement of the fungus from one ring to the next. The low percentage of recurrence found by Walter and Smucker suggested that crossing-over of this nature did not often take place. Walter (unpubl.) carefully examined 16 American elms in which the disease had redeveloped. In five there may have been fresh infections; in six the cross-over had been aided by insect wounds; only in the remaining five did it appear that the fungus had crossed unaided.

These results were later elaborated by Banfield (1947), who found that recurrence of the disease depended mainly on the fungus having reached the roots, where crossing-over was relatively easy. Where infection was limited to the crown, he found no cross-over at all, whereas about 50 per cent of trees infected down to the roots redeveloped symptoms. It was found also that the probability of crossing-over increased with tree size. This may partially account for the very low percentages recorded by Smucker (1938) and by Walter (unpubl.), both of whom were working on nursery trees. Crossing-over was found to occur in all cases of trees over 15 inches in diameter (Banfield, Rex and May 1947), but they were of course dealing with highly susceptible American elms. Buchanan and Smucker (1942) examined the possibility that the fungus could reach the current ring by means of the feeding scars of *Scolytus* beetles, but were unable to obtain any clear evidence that this occurred. Field evidence, collected in Britain, and discussed fully below, appears to indicate that even in large trees the percentage of redevelopment in this country is low.

Further evidence on this matter was provided by experiments on spraying elms with insecticides to protect them from *Scolytus* feeding, and consequently from infection by *Ceratostomella ulmi* (Peace 1954). These were carried out at Folkestone and Aldenham during the years 1948–50. The results of all the experiments are summarised in Table 4.

The fact that in both sprayed and unsprayed trees the percentage of symptom development is higher in those trees that did *not* have symptoms the previous year than in those that *did*, hardly suggests a high percentage of cross-over. Spraying clearly reduced

EFFECT OF SPRAYING ELMS WITH INSECTICIDES ON SYMPTOM RECURRENCE

TABLE 4

Type of Tree	*Treatment*	*Total No. of Trees*	*Trees showing active symptoms in year of treatment*	
			No.	*%*
Trees showing symptoms in previous year	Unsprayed control	56	25	45
" " " " " "	Sprayed	53	13	25
Trees not showing symptoms in previous year	Unsprayed control	59	33	56
" " " " " " "	Sprayed	116	33	28

the amount of disease, which it could only do by preventing fresh infection; but again the similarity of the figures in the second and fourth lines of the table suggests a very low rate of cross-over. These experiments were carried out on street and park trees 20–30 ft. high, in which, according to American experience (Banfield, Rex, and May 1947), there ought to have been a fairly high rate of recurrence.

Case Histories of Individual Trees

A selection of case histories is set out in Table 5. Some of these are illustrated by serial photographs, always taken from the same direction. Records and photographs were all made in August or September of the year named. It will be quickly appreciated from the table that there is no standard pattern of development and therefore no way of summarizing the behaviour of elms as regards recovery and disease redevelopment. Many more case histories were collected, but even if they were all included in the table, no general pattern would emerge, for too many variables are involved in the development of the disease. We shall consider later the average behaviour of small and large populations of elms. Group behaviour does show certain general trends, larger numbers infected in one year, more severe symptoms on the average in another and so on, but these results are still based on the highly erratic individual behaviour of the trees making up the population.

In all the surveys and observations a purely visual method has been used to arrive at the various severity gradings for current year's symptoms. These gradings are not amenable to exact description, but nevertheless an effort has been made below to define them.

Very slight: Only small twigs affected, over not more than one-fifth of the whole crown. No possibility of permanent dieback.

Slight: Only small twigs affected or infection limited to one or two small branches. Twig injuries may be scattered over most of the crown. Unlikely that permanent dieback will occur.

Moderate: Small twig injuries scattered over the whole crown, or more commonly several minor or one or two large branches attacked. Almost certain that some dieback will ensue.

Severe: More than one-third of the crown attacked, and at least three large branches involved. Quite certain that dieback will follow.

In practice assessment depended much more on a remembered mental picture, than on any question of definition. It must be borne in mind that virtually all the severity ratings recorded in this paper were made by the author.

Assessment for current year's symptoms was confined to that part of the crown that came into leaf at the beginning of the year. It was perfectly possible therefore for a tree to be recorded as slightly, very slightly or even not attacked when it was carrying a great deal of dead wood resulting from the activities of the fungus in previous years. The dead wood only came into the assessment of the later surveys, and then it was considered separately from the current year's symptoms.

Many records were collected of trees which showed active disease for one or two years and thereafter remained quite free. In Table 5, elm No. 6 at Foulden, the elm at Stowmarket, and the Cottage elm at Kennington are examples of this. There were numerous cases of a constant alternation of active disease and recovery; tree No. 23 at Ryston (Plates 58–70) is a notable example. In some trees, such as Trowbridge No. 3 (Plates 41-50), the years of active disease preponderate, whereas in others, such as Bridge End No. 45, they are separated by long periods of freedom.

There are several instances of remarkable recovery from severe injury, These are best illustrated by the plates; Foulden No. 17 (Plates 34–40), Trowbridge No. 3 (Plates 41–50) and Ryston No. 23 (Plates 58–70) are good examples. There are instances of sudden, severe disease and even death following a long period of comparative freedom. Both Elm No. 4 at Totteridge (Plates 2–4) and the tree in the Botanic

CASE HISTORIES OF INDIVIDUAL TREES

TABLE 5

Year of Inspection: / *Tree No.*	1928	1929	1930	1931	1932	1933	1934	1935	1936	1937	1938	1939	1940	1941	1942	1943	1944	1945	1946	1947	1948	1949	1955
Casewick, Lincs. (No. 5)	—	—	—	—	—	—	—	—	—	—	s	R	S	M	—	—	—	R	R	—	—	vs	**R**
„ „ (No. 7)	—	—	—	—	—	—	—	—	—	—	**M**	**R**	s	R	—	—	—	**M**	R	—	—	R	**R**
Foulden, Norfolk (No. 17)	N	N	N	N	N	M	M	s	M	**M**	**M**	**R**	**M**	**R**	—	**R**	R	R	—	—	R	s	**R**
„ „ (No. 9)	N	N	I	—	R	R	R	R	R	R	R	S	M	s	—	R	R	s	—	—	R	R	—
„ „ (No. 6)	N	I	I	—	I	R+	R+	R	R	R	R	R	R	R	—	R	R	R	—	—	R	R	—
Kennington, Berks (Cottage)	N	N	I+	R	R	R	R	R	R	R	R	R	R	R	—	R	R	R	—	—	—	—	—
„ „ (School)	N	N	I+	R	R	R	R+	R+	R	R	s+ (1)	S	R	R	—	vs	R	R	—	—	—	—	—
Longstowe, Cambs.	s+	R	R	R	R+	R−	R	R	R	R	R	R	—	R	—	R	R	R	R	—	vs	—	R
Trowbridge, Wilts (No. 3)	—	—	—	—	—	—	**s**	s	**R**	**M**	**M**	S	**S**	**M**	**M**	s	**s**	M	S	R	M	**R**	R
„ „ (No. 1)	—	—	—	—	—	—	M	s	s	M	R	S	s	R	R	R	R	R	R	R	s	s	R
„ „ (No. 4)	—	—	—	—	—	—	M	**R**	**S**	**s**	**R**	R	s	**M**	R	R	R	R	R	R	s	**s**	R
Stowmarket, Suffolk	I+	—	R	—	R−	—	R−	R+ (2)	R	R	R	—	—	—	—	—	—	—	—	—	—	—	—
Cambridge (St. John's College)	—	—	—	—	—	—	—	—	I	M	M	R	—	M	—	M(3)	s(4)	R	R	—	D	—	—
„ (Botanic Garden)	s+	—	R	R	R	R	R	R	s	s	s	—	**R**	R	—	s	R	—	M	—	**M**	—	R
Bridge End, Bedford (No. 45)	M+	R	R	R+	R+	R+	R+	R	S	R	R	R	M	R	—	R	R	R	—	—	—	—	—
„ „ „ (No. 47)	—	—	—	—	—	—	—	—	N	N	N	N	N	**M**	—	**S**	**R**	M	—	—	**R**	—	**R**
Bedford (St. John's Road)	N	N	I	I	R−	R−	R+	s	s	R	R	—	—	—	—	—	—	—	—	—	—	—	—
Totteridge, Herts, (No. 4)	s+	R	R	R	s−	R+	R−	s+	vs	**M**	R	R	**M**	—	—	—	—	—	—	—	—	—	—
„ „ (No. 62)	s+	s	R	R	R	I	R+	R+	s	s	R	R	R	R	—	—	s	s	—	—	—	—	—
Ryston, Norfolk (No. 2)	—	—	—	I	I	I	R	**M**	**S**	**M**	**M**	**S**	D	—	—	—	—	—	—	—	—	—	—
„ „ (No. 23)	—	—	—	**M**	**R**	**M**	**S**	**R**	**R**	**R**	vs	**R**	**R**	**S**	—	—	**R**	—	—	—	**R**	—	—
Marston Thrussel, Leicester (No. 1)	—	—	—	—	—	—	—	—	—	**M**	s	s	s	**R**	—	—	—	M	R	—	**R**	—	R

N No sign of disease.
M Moderate active symptoms.
D Dead.
vs Very slight active symptoms.
S Severe active symptoms.
I Active symptoms, but severity not recorded.
s Slight active symptoms.
R No active symptoms, recovering.
— Tree not visited.

Heavy type indicates that a photograph is included among the plates.
+ indicates that cultures were made, which yielded *Ceratostomella ulmi*.
− indicates that cultures were made, which did not yield *C. ulmi*.

Notes: (1) Positive cultures from 1938 ring, negative from 1933 ring.
(2) Positive cultures from both 1928 and 1933 rings.
(3) Tree topped.
(4) Tree cut hard back.

Gardens at Cambridge (Plates 56–57) suddenly developed quite severe symptoms after long periods during which they had only sometimes shown very mild signs of the disease. Had it been possible to photograph a large number of healthy elms on the chance that they would later become infected, more examples would be available of sudden disease development. As it was, serial photographs were normally started only of trees which initially showed active symptoms. Two cases, the St. John's College tree at Cambridge and No. 2 at Ryston (Plates 51–55), of trees which showed rapid disease development followed by death are included in the table. Such behaviour was frequently observed in the nineteen-thirties when the disease was at its height, but thereafter it became less and less common.

In all these yearly comparisons it is necessary to remember that the severity of attack is certainly a reflection of the amount of beetle feeding with its consequent infection, and of the subsequent effect of the summer climate on the development of the disease, as well as of the susceptibility of the tree. In many cases it is probable that the reaction of the tree is the least important of these three factors.

In view of such variable behaviour it is obviously very difficult to prophesy the future of any particular infected elm. Once the tree is so weakened that *Scolytus* beetles start to breed in the trunk, it is normally beyond hope, but a few recoveries have been recorded even from this extreme condition. The possibility of pruning infected trees to remove diseased or dead wood and to promote recovery is discussed at a later stage.

Progress in Local Populations

In the early days areas were visited primarily because the disease had been reported from them, so that at the beginning notes tended to be made on the more virulent local outbreaks. As the surveys became more exploratory in character, they naturally extended to places where the disease was less virulent; but there was still a tendency to select for study areas where the disease had reached a moderate level. No disease-free localities were included. Thus all the records up to and including 1946 refer to localities deliberately selected because the disease was present there. They, therefore, show local trends of development, which to some extent may have a wider application. But they throw no light at all on the general level of the disease over the country as a whole, or for that matter over any large section of it.

For purposes of inter-annual comparison all the elms in a given area were classified as "disease-free" if they showed no active symptoms, "diseased" if they showed active symptoms of the current year, or "dead". In some of the earlier surveys, the trees were not plotted or marked in any way, so that the number observed tended to vary from year to year, largely according to visibility, while trees which were felled dropped from the count without record. For the sake of continuity it was necessary to carry on these surveys in the same way as they had been started, but they are obviously much less accurate than surveys of better-recorded groups.

To get more exact records, rows of elms or easily plotted groups were deliberately chosen, so that exactly the same trees were assessed each year, and record kept of those removed. Even by this method there was some variation in numbers. Trees were sometimes felled for reasons other than disease, and occasionally fresh trees were brought into the survey. In cases where individual records were kept for each tree, it was possible to add the category of "recoveries" to the three listed above.

The results of some of these surveys are set out in Figures 2–17. The (*a*) "dead", (*b*) "dead plus actively diseased", and in some cases (*c*) "dead plus actively diseased plus recoveries" are shown on the graphs as percentages of the total number of trees observed. From these graphs it is possible to follow the general trend of the disease in each of the selected areas. In the earlier, less accurate surveys the percentage of dead often falls with time, because dead trees that had been felled ceased to be counted. In the more accurate counts this is not the case, because dead trees remained on the records even after they had been felled. In areas where "recoveries" were separately noted, the total figure for dead, diseased and recoveries cannot fall, since once a tree has become diseased it is bound to be in one of these three categories. Only the percentage of "diseased" can vary both up and down. During and after the 1939-45 war visits were not made every year, so that there are some gaps in the records.

Some details of the localities subject to periodic survey and illustrated in the text figures are given in Table 6.

The behaviour of the disease in local populations, like that in individual trees, is extremely variable and cannot be fitted into any overall pattern. Nevertheless, various points, some of more than local interest, emerge and are discussed below.

The first three areas (Figs. 2-4) lay in the west of Essex, which was one of the worst affected areas in the early days of the disease. The recorded deaths were as high as 38 per cent, 42 per cent and 52 per cent respectively, and since these figures take no account of dead elms previously removed, the actual death rate was probably appreciably higher. Thus, about half the elms, in these areas where elm was the predominant tree, perished in the first few years of severe attack. In this part of the country disease reached its peak in 1931, a point which shows

PLATE 1. Large elm at Quendon, Essex, which escaped attack and was therefore tested for resistance. Elm R.T. 1944.

PLATE 2. Elm No. 4, Totteridge. 1936, very slight active symptoms.

PLATE 3. Elm No. 4, Totteridge. 1937, moderate active symptoms.

PLATE 4. Elm No. 4, Totteridge. 1940, moderate active symptoms.

(PLATES 2-4. Tree No. 4, Totteridge, Hertfordshire. A tree frequently showing slight chronic symptoms.)

PLATE 5. 1937, moderate active symptoms.

PLATE 6. 1938, slight active symptoms.

PLATE 7. 1941, no active symptoms, recovering.

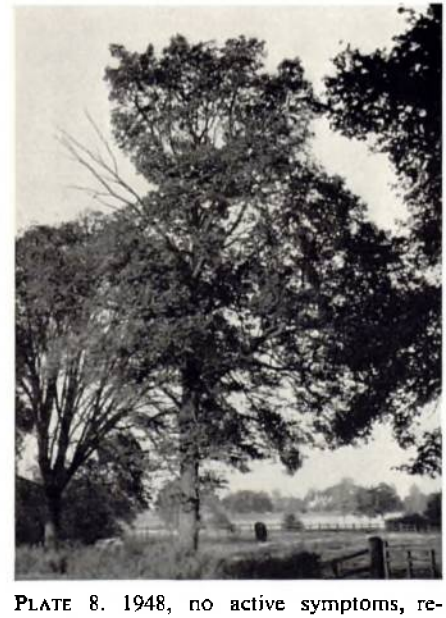

PLATE 8. 1948, no active symptoms, recovering but dead wood still shows.

PLATES 5-8. Elm No. 1, Marston Thrussel, Leicestershire.

PLATE 9. 1938, slight active symptoms.

PLATE 10. 1941, moderate active symptoms.

PLATE 11. 1949, very slight active symptoms.

PLATE 12. 1955, no active symptoms, a complete recovery.

PLATES 9-12. Elm No. 5, Casewick, Lincolnshire, A Jersey Elm.

PLATE 13. 1938, moderate active symptoms.

PLATE 14. 1939, no active symptoms, recovering.

PLATE 15. 1940, slight active symptoms. The thin foliage was nothing to do with the disease.

PLATE 16. 1945, moderate active symptoms.

PLATES 13-17. Elm No. 7, at Casewick, Lincolnshire. A tree with normally rather sparse foliage.

PLATE 17. Elm No. 7, Casewick. 1955, no active symptoms, recovered, but still rather thin.

PLATE 18. Elm No. 47, Bridge End. 1941, moderate active symptoms.

PLATE 19. Elm No. 47, Bridge End. 1943, severe active symptoms.

PLATE 20. Elm No. 47, Bridge End. 1944, no active symptoms.

PLATES 18-22. Elm No. 47, at Bridge End, near Bedford. A small tree beside a stream.

PLATE 21. Elm No. 47, Bridge End. 1948, no active symptoms, recovering.

PLATE 22. Elm No. 47, Bridge End. 1955, no active symptoms, almost completely recovered.

PLATES 21-22. Elm No. 47, Bridge End, Bedford.

PLATE 23. Elm No. 4, Trowbridge. 1935, no active symptoms, recovering from earlier attacks. It was severely pruned in 1933.

PLATE 24. Elm No. 4, Trowbridge. 1936, severe active symptoms.

PLATES 23-28. Elm No. 4, at Trowbridge, Wiltshire. A Jersey Elm in an avenue.

PLATE 25. Elm No. 4, Trowbridge, continued. 1937, slight active symptoms.

PLATE 26. 1938, no active symptoms, recovering.

PLATE 27. 1941, moderate active symptoms.

PLATE 28. 1949, slight active symptoms, but has recovered from earlier attacks.

PLATES 23-28, continued. Elm No. 4, at Trowbridge, Wiltshire. A Jersey Elm in an avenue.

PLATE 29. 1938. A severe outbreak at Holt in Cheshire, typical of the more active period of the disease. Despite their apparently dead condition several trees in this group made remarkable recoveries.

PLATE 30. 1943. Elms (E.B.) at Stansted, Essex, which were thought to have natural resistance, and which survived heavy inoculations unharmed They later succumbed to natural infection.

PLATE 31. Elm at Studley, Oxford, which escaped attack and was therefore tested for resistance. Elm M.H. 1939.

PLATE 32. Tree on the Golf Course at Totteridge, Hertfordshire. The first definite recorded case of the Elm Disease in Britain, 1928.

PLATE 33. 1945. An elm infected by beetles from a sawmill log pile.

PLATE 34. Elm No. 17, Foulden, Norfolk. 1937, moderate active symptoms, following several years of repeated attack.

(PLATE 34 is one of the series that continues overleaf.)

PLATE 35. 1938, moderate active symptoms again.

PLATE 36. 1939, no active symptoms, recovering.

PLATE 37. 1940, moderate active symptoms again.

PLATE 38. 1941, no active symptoms, recovering.

PLATES 34-40, continued. Elm No. 17, Foulden, Norfolk.

PLATE 39. Elm No. 17, Foulden, continued. 1943, no active symptoms, still recovering.

PLATE 40. Elm No. 17, Foulden. 1955, no active symptoms, a nearly complete recovery.

PLATES 34-40, continued. Elm No. 17, Foulden, Norfolk.

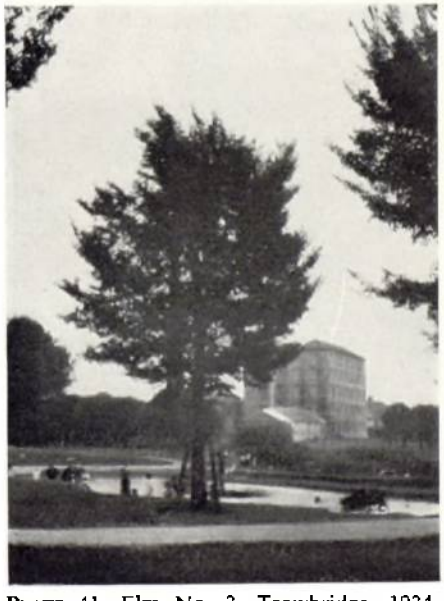

PLATE 41. Elm No. 3, Trowbridge. 1934, slight active symptoms.

PLATE 42. Elm No. 3, Trowbridge. 1936, no active symptoms, recovering.

PLATES 41-50. Elm No. 3, Trowbridge, Wiltshire. A Jersey Elm in an avenue.

PLATE 43. 1937, moderate active symptoms.

PLATE 44. 1938, moderate active symptoms again.

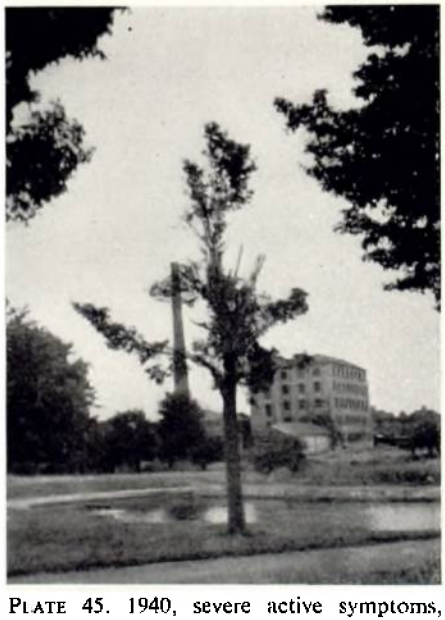

PLATE 45. 1940, severe active symptoms, heavily pruned as a result of previous attacks.

PLATE 46. 1941, moderate active symptoms, but has made considerable recovery growth since the previous year.

PLATES 41-50, continued. Elm No. 3, Trowbridge, Wiltshire. A Jersey Elm in an avenue.

PLATE 47. 1942, moderate active symptoms again.

PLATE 48. 1944, slight active symptoms.

PLATE 49. 1949, no active symptoms, recovering, but its top died in a severe attack in 1948.

PLATE 50. 1955, no active symptoms, a nearly complete recovery, but still a small tree compared with its neighbours.

PLATES 41-50, continued. Elm No. 3, Trowbridge, Wiltshire. A Jersey Elm in an avenue.

PLATE 51. 1935, moderate active symptoms.

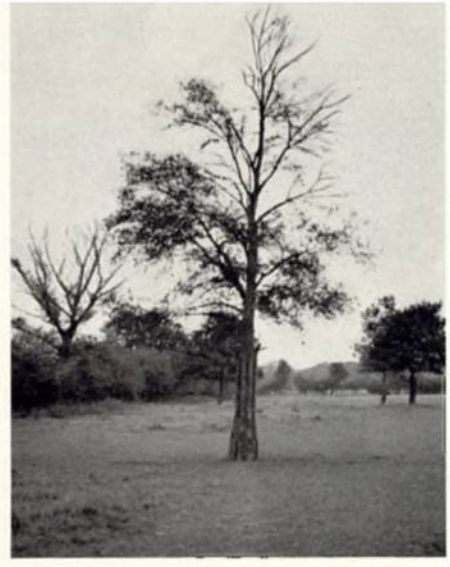

PLATE 52. 1936, severe active symptoms.

PLATE 53. 1937, moderate active symptoms and a lot of dead wood.

PLATE 54. 1938, moderate active symptoms, top completely dead.

PLATES 51-55. Elm No.2, Ryston, Norfolk, A form of Ulmus carpinifolia.

(PLATE 55 appears opposite.)

PLATE 55. Elm No. 2, Ryston, continued. 1939, severe active symptoms, nearly dead; the tree was completely dead a year later.

PLATE 56. Tree in Botanic Gardens, Cambridge. 1940, no active symptoms, recovering from a series of very slight attacks.

PLATE 57. Botanic Gardens, Cambridge, 1948, moderate active symptoms, the top is dead.

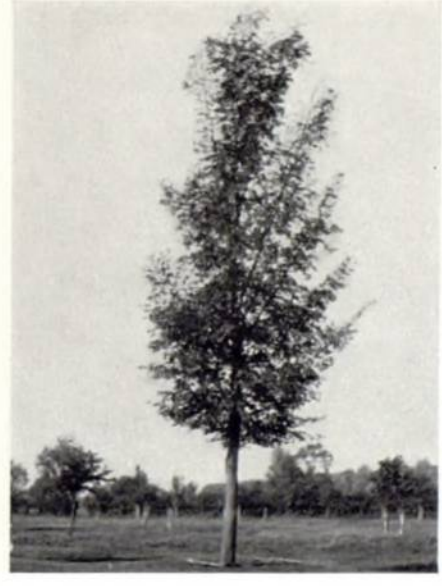

PLATE 58. Elm No. 23, Ryston, Norfolk, probably Ulmus hollandica vegeta, 1931, moderate active symptoms.

(PLATES 56-57. Tree in Botanic Gardens, Cambridge. A form of Ulmus carpinifolia. In most years, when it showed any active symptoms at all, this tree only displayed a scattering of yellow foliage.)

PLATE 59. 1932, no active symptoms, recovering.

PLATE 60. 1933, moderate active symptoms.

PLATE 61. 1934, severe active symptoms.

PLATE 62. 1935, no active symptoms, recovering.

PLATES 58-70, continued. Tree No. 23, Ryston, Norfolk, probably Ulmus hollandica vegeta.

(PLATE 58 appears on preceding page.)

PLATE 63. 1936, no active symptoms, still recovering.

PLATE 64. 1937, no active symptoms, recovery continuing, but dead top still shows.

PLATE 65. 1938, very slight active symptoms.

PLATE 66. 1939, no active symptoms, recovering.

PLATES 58-70, continued. Tree No. 23, Ryston, Norfolk, probably Ulmus hollandica vegeta.

PLATE 67. 1940, no active symptoms, sparse foliage is not due to disease.

PLATE 68. 1941, severe active symptoms.

PLATE 69. 1944, no active symptoms, but there has been a lot of dieback since 1941.

PLATE 70. 1948, no active symptoms, almost complete recovery, except that the tree has lost its crown, and is therefore quite a different shape.

PLATES 58-70, continued. Tree No. 23, Ryston, Norfolk, probably Ulmus hollandica vegeta.

PLATE 71. 1936, five years after the disease was first recorded in 1931.

PLATE 72. 1941, the trees have grown a lot despite the disease.

PLATE 73. 1955, one recent replacement can be seen just to the right of the perambulator. Most of the earlier replacements, several of which are included in the photograph, have nearly caught up with the original trees.

PLATES 71-73. War Memorial Avenue of Jersey Elms at Trowbridge, Wiltshire. This avenue was subject to periodic careful pruning of diseased limbs and removal of a few of the worst diseased trees. Every tree in the avenue has shown active symptoms at least once.

PLATE 74. 1938, left-centre tree shows severe active symptoms; right-centre tree shows no active symptoms.

PLATE 75. 1943, left-centre tree shows no active symptoms, still recovering; right-centre tree shows no active symptoms.

PLATE 76. 1944, left-centre tree has been felled; despite its recovery it was still unsightly; right-centre tree shows severe active symptoms.

PLATES 74-76. Elms beside the river Cam on Stourbridge Common, Cambridge. The tree on the left never developed the disease at all.

LOCALITIES SUBJECT TO DETAILED ANNUAL SURVEY

TABLE 6

Fig. No.	*Locality*	*Years covered*	*Number of Trees (Min. and Max.)*	*Type of Survey*	*Description*
2	Newport, Essex	1930–37	190–389	Less accurate	Scattered elms along and near a stretch of road.
3	Ugley, Essex	1930–45	56–145	,, ,,	,, ,, ,, ,, ,, ,, ,, ,, ,,
4	Stansted, Essex	1931–45	48–103	,, ,,	,, ,, ,, ,, ,, ,, ,, ,, ,,
5	Bridge End, Bedford ..	1936–45	36–60	,, ,,	Groups of suckers in a field.
6	Buxted, Sussex	1930–45	230–231	Accurate, recoveries not noted	Avenue of large elms in a park.
7	Trowbridge, Wilts ..	1930–55	80–98	,, ,, ,, ,,	Avenue of Jersey elms in a park.
8	Great Hinton, Wilts ..	1939–45	141–150	,, ,, ,, ,,	Medium-sized trees in hedges round a group of fields.
9	Cambridge	1939–46	59–61	,, ,, ,, ,,	Large trees along and near a road.
10	Longstowe, Cambs. ..	1941–55	37	,, ,, ,, ,,	Trees of varying size in and around fields.
11	Foulden, Norfolk	1928–49	92–95	Accurate, recoveries noted	Avenue of medium-sized trees along a road.
12	Wimpole, Cambs. ..	1936–55	274–283	,, ,, ,,	Double avenue of very large trees.
13	Casewick, Lincs.	1938–55	102	,, ,, ,,	Avenue of medium-sized Jersey elms.
14	,, ,, ...	1938–55	158–166	,, ,, ,,	Small elms along a road.
15	Wimpole, Cambs. ..	1938–55	59–60	,, ,, ,,	A collection of medium-sized elms of different varieties.
16	Lubenham, Leicester ..	1939–55	152–176	,, ,, ,,	Avenue of medium-sized to large trees along a road.
17	Ashorne, Warwick ..	1945–55	229–278	,, ,, ,,	Elms of all sizes along a road.

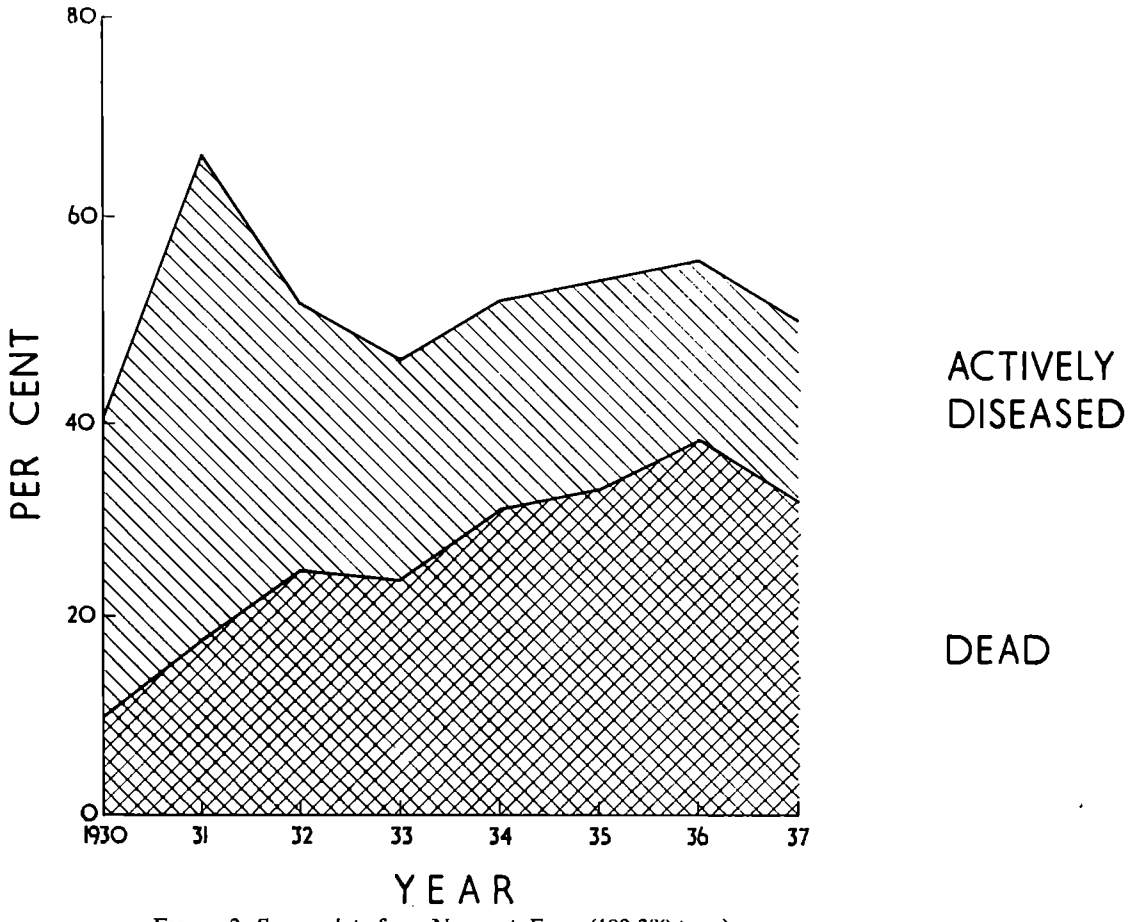

FIGURE 2. Survey data from Newport, Essex (190-389 trees).

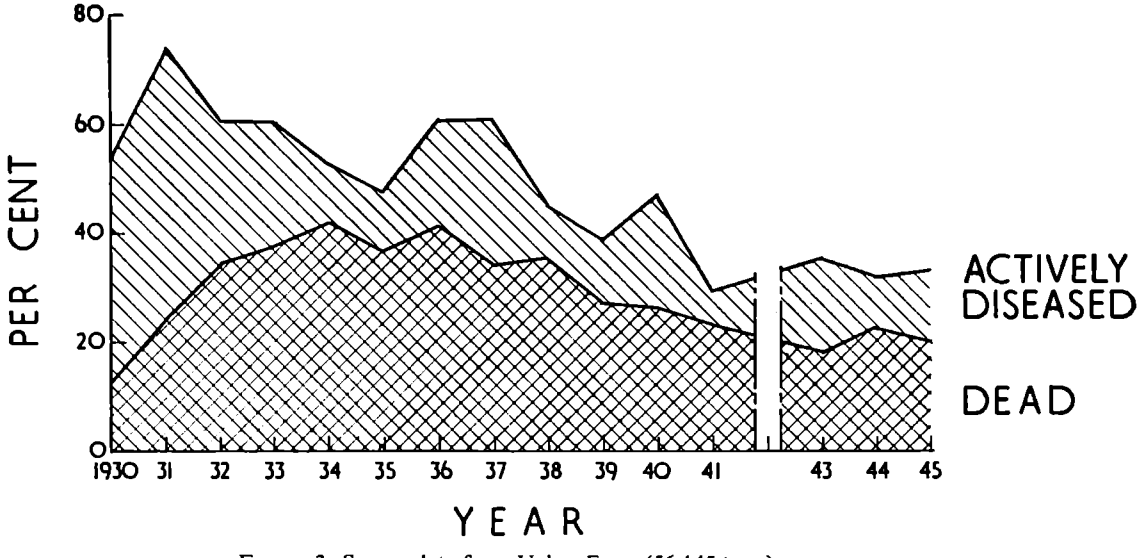

FIGURE 3. Survey data from Ugley, Essex (56-145 trees).

clearly on the graphs, and thereafter declined to a level where it was obviously present each year, but did comparatively little damage. It is tempting to suggest that the earlier attacks killed all the more susceptible elms, and that the disease is now attacking trees sufficiently resistant to recover from the less severe injuries to which they are periodically subjected; but it must be admitted that there is little supporting evidence for this supposition. Among these Essex areas, Newport shows a subsidiary peak in 1936, and Ugley in 1936 and 1937. These years were peak years in a number of other areas, 1936 at Bridge End (Fig. 5) and Foulden (Fig. 11) and 1937 at Trowbridge (Fig. 7). This phenomenon of an initially very severe attack, followed by a slow decline, was not confined to west Essex. It occurred

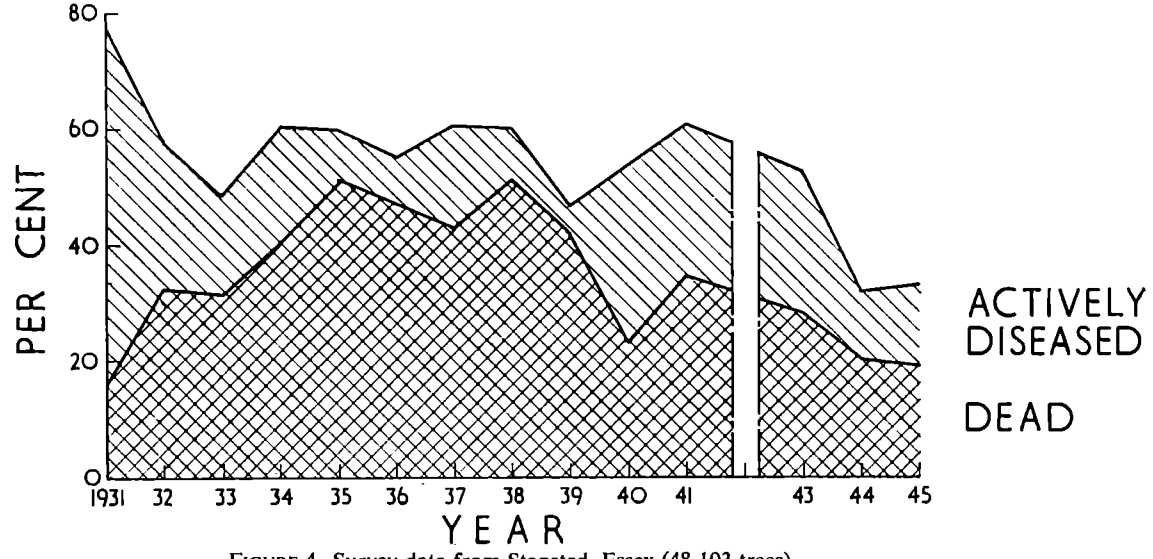

FIGURE 4. Survey data from Stansted, Essex (48-103 trees).

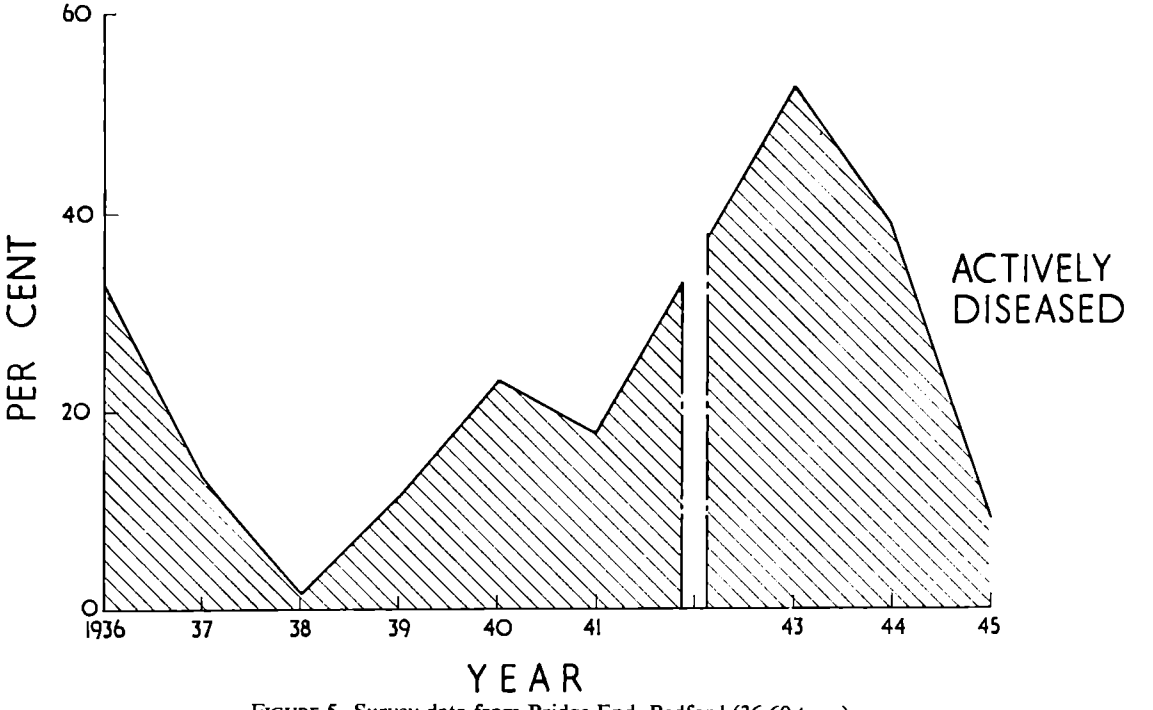

FIGURE 5. Survey data from Bridge End, Bedford (36-60 trees).

in several other areas where the attack developed rapidly in the early nineteen thirties. Unfortunately, few of these were the subject of surveys, but the count made in Cambridge (Fig. 9) is in fact in such an area. In this case, most of the damage had occurred before 1939, when the survey started, and is reflected in the high percentage (31 per cent) of dead trees. During the period of the survey, the disease tended to decline, and by 1946 only one tree (representing 2 per cent) was showing active symptoms.

The somewhat erratic graphs for these three Essex areas are to some extent a reflection of the inaccurate survey methods; it is quite certain, however, that more careful collection of results would still have shown the disease at its height in 1931, and thereafter declining. A different state of affairs existed at Buxted (Fig. 6), where right up to 1945, when the survey was abandoned, the disease was making steady and continuous progress, so that by then 66 per cent of the trees had been killed. This was a locally violent outbreak in a part of the country where elm is not a tree of primary importance. There were other places where the disease progressed in this steady and damaging manner, but such behaviour cannot be regarded as typical.

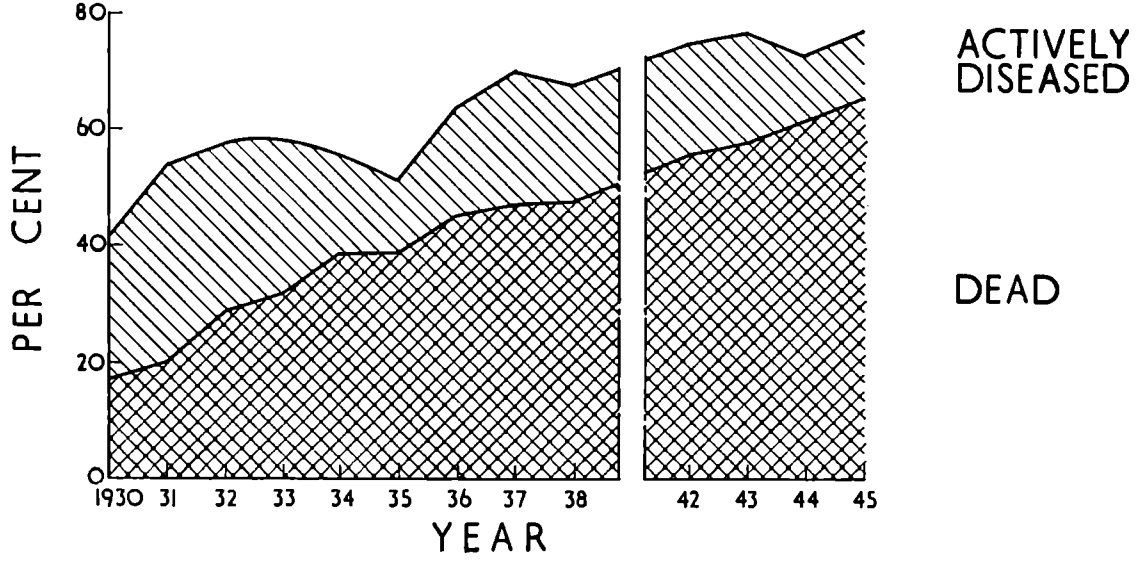

FIGURE 6. Survey data from Buxted, Sussex (230-231 trees).

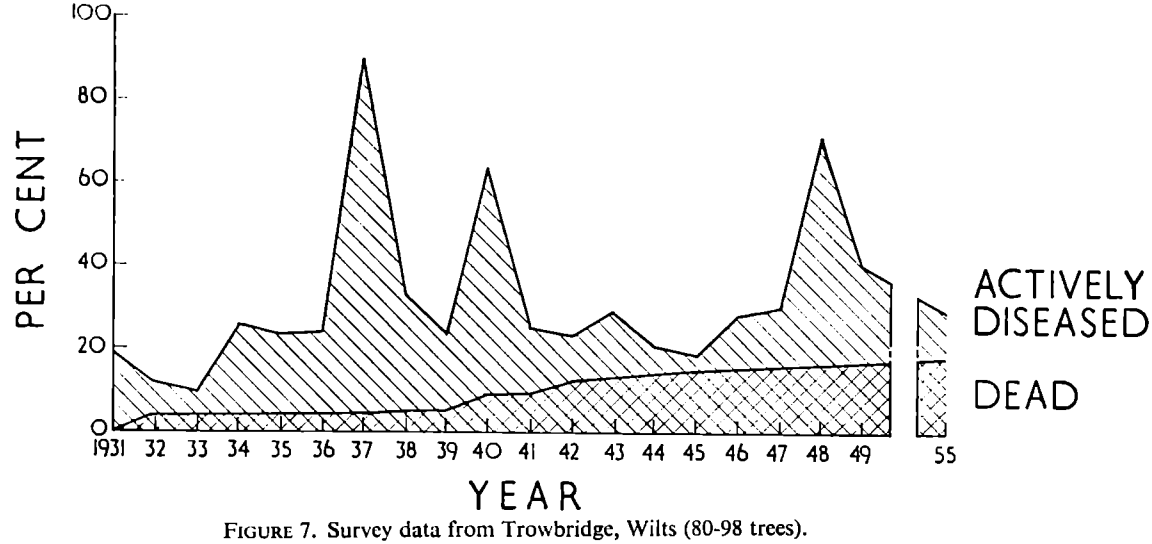

FIGURE 7. Survey data from Trowbridge, Wilts (80-98 trees).

In other areas the disease made slower, but nevertheless very definite, progress in the nineteen-thirties, but levelled off before it had become nearly so damaging as at Buxted. Foulden (Fig. 11) is a good example of this. Here the disease reached a peak in 1936, then a still higher level in 1940, and thereafter declined. This area provided a number of examples of quite spectacular recovery from severe injury. Indeed the rate of recovery was so high that in 1949, at the end of the survey, while 75 per cent of the elms had had the disease sometime during the preceding twenty-one years, only 14 per cent had succumbed to it. The 1940 peak which was prominent at Foulden also occurred in other areas, for instance Ugley (Fig. 3), Trowbridge (Fig. 7), and Casewick (Fig. 14).

At Foulden the highest percentage showing active disease was 46 per cent in 1940. At Trowbridge (Fig. 7) where there was an even stronger tendency to recovery, 90 per cent were actively diseased in 1937, 64 per cent in 1940, and 70 per cent in 1948, yet the death rate when the survey was closed in 1955 was only 18 per cent. The avenue of Jersey elms, here surveyed, was planted as a memorial after the first World War, and had 19 per cent diseased when it

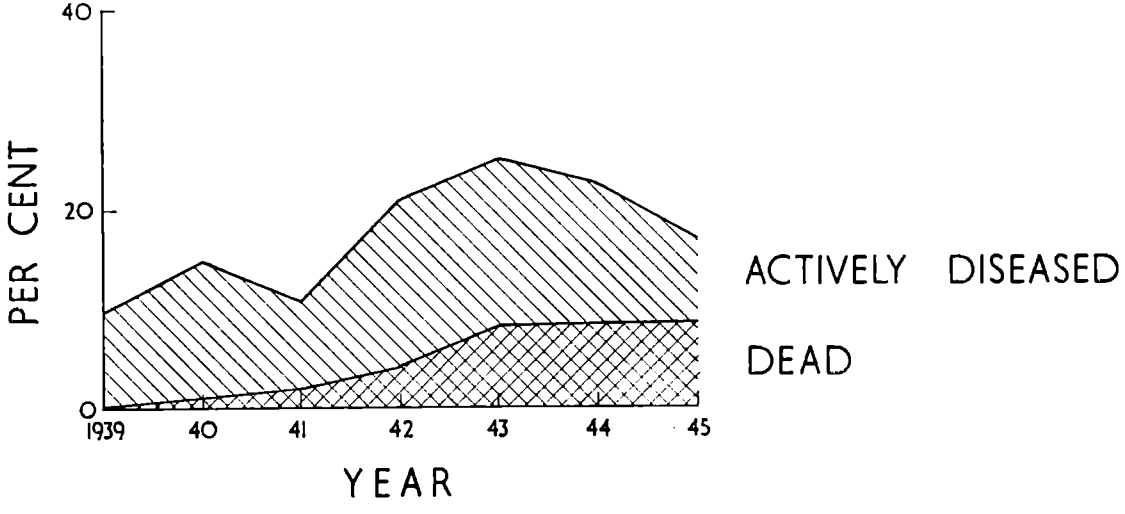

FIGURE 8. Survey data from Great Hinton, Wilts (141-150 trees).

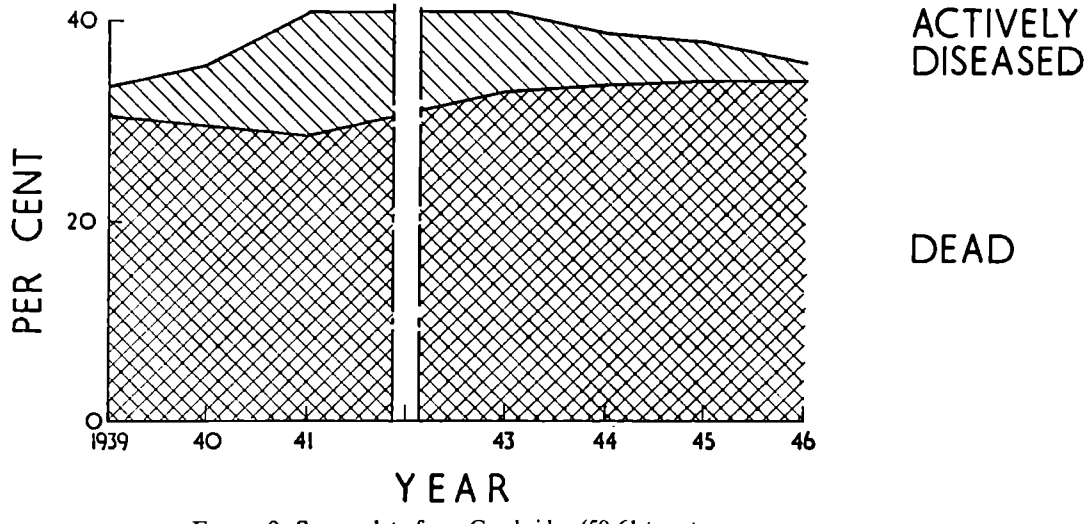

FIGURE 9. Survey data from Cambridge (59-61 trees).

was first inspected in 1931. A policy of pruning-out diseased wood each year and replacing only very severely attacked trees was adopted, so that in effect the trees listed as "dead" are really those removed because they were considered beyond hope of recovery. Inclusion of the replacement trees is responsible for the increase in the number of trees in the survey. In this case, it is known that every elm in the avenue has displayed active symptoms at least once during the twenty-four years of observations; most trees did so on a number of occasions. Yet the final effect on the avenue as a whole has proved surprisingly slight (Plates 71–73). The trees are probably smaller than they would have been without dieback due to the disease and the resultant pruning. The more recent replacements cause some unevenness, but the earlier ones have nearly caught up with their companions. The significance of the behaviour of the disease in this avenue will be considered further, in view of its bearing on questions of control.

Another area with a high recovery rate was Bridge End (Fig. 5), when a peak of 56 per cent showing active symptoms was reached in 1943; but there were no deaths, and when the survey was abandoned in 1945 only 8 per cent showed active symptoms. The 1943 peak noted here also occurred at Longstowe (Fig. 10) and Wimpole (Fig. 15).

Several of the surveys were conducted in areas where the disease never reached even a moderate level. Great Hinton (Fig. 9) is an example. The avenue of Jersey elms at Casewick (Fig. 13) is another. At Casewick, however, the elms did display the same characteristic of a high percentage infection over the period as a whole, accompanied by a high

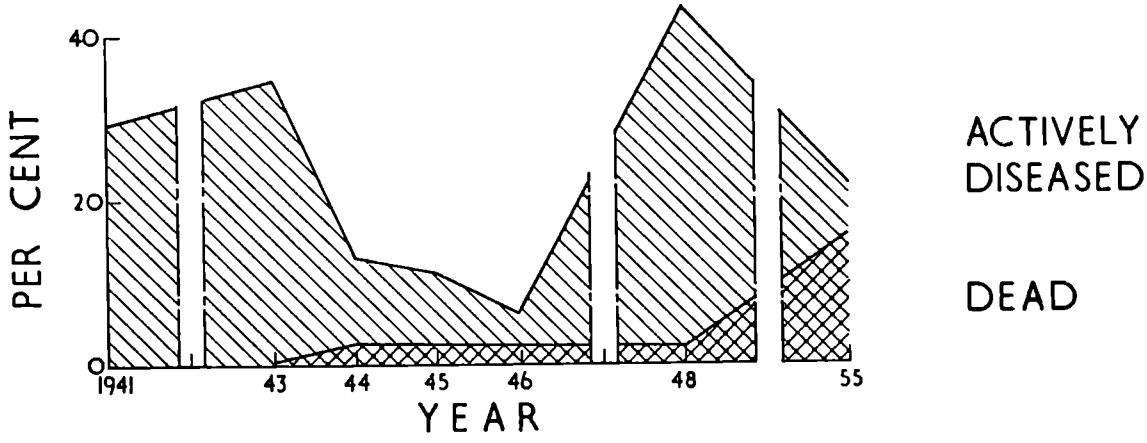

FIGURE 10. Survey data from Longstowe, Cambs. (37 trees).

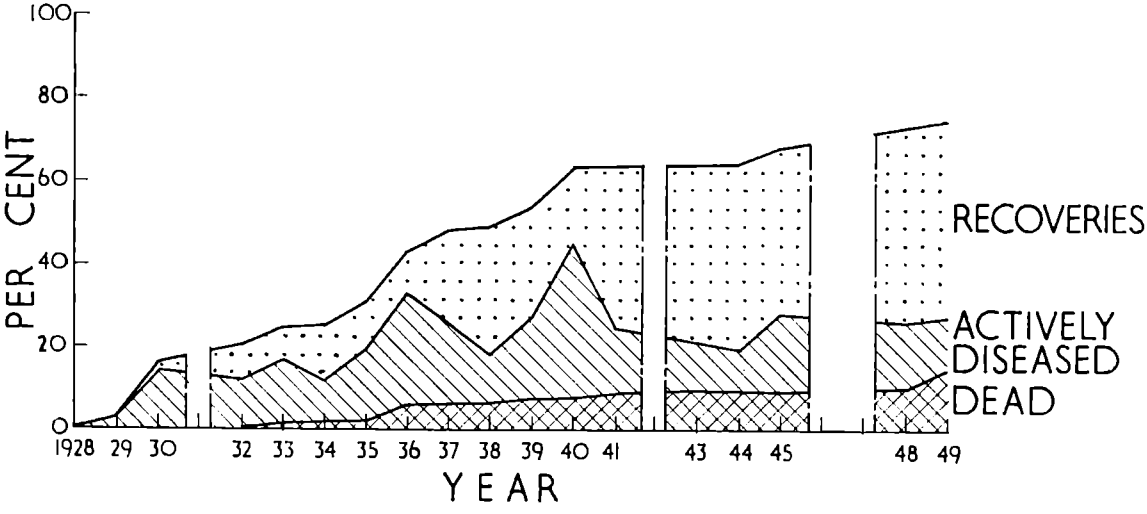

FIGURE 11. Survey data from Foulden, Norfolk (92-95 trees).

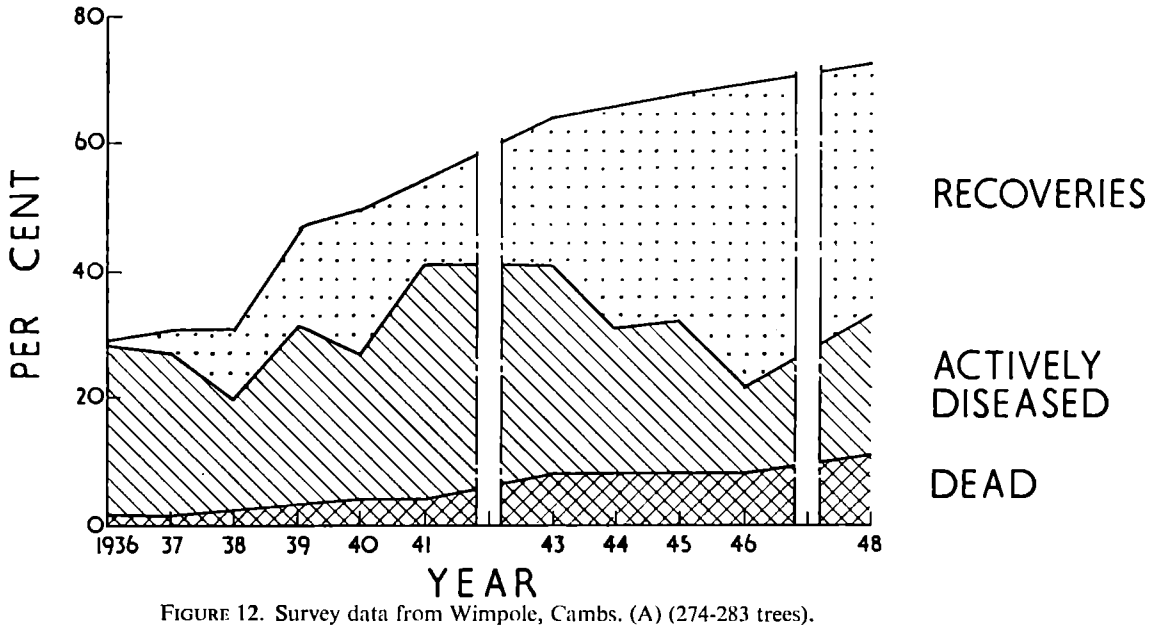

FIGURE 12. Survey data from Wimpole, Cambs. (A) (274-283 trees).

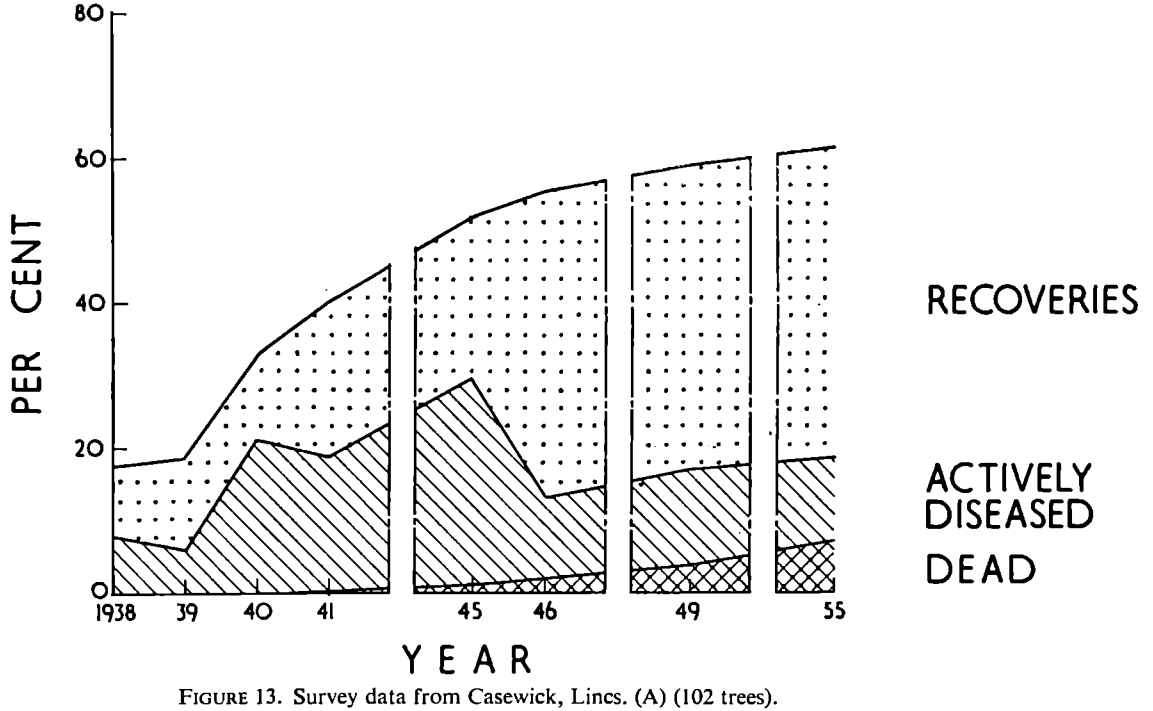

FIGURE 13. Survey data from Casewick, Lincs. (A) (102 trees).

rate of recovery, as did the same species at Trowbridge. At Casewick, although the percentage of active disease in any one year never rose above 30 per cent, the proportion that had the disease at least once reached the high figure of 61 per cent. Lubenham (Fig. 16) is another example of very slow progress. Over a period of 16 years the percentage of trees that had developed the disease rose only from 14 per cent to 29 per cent and the death rate from 2 per cent to 5 per cent. Ashorne (Fig. 17), which was surveyed for only a few years, showed very little advance, though the disease rose to a

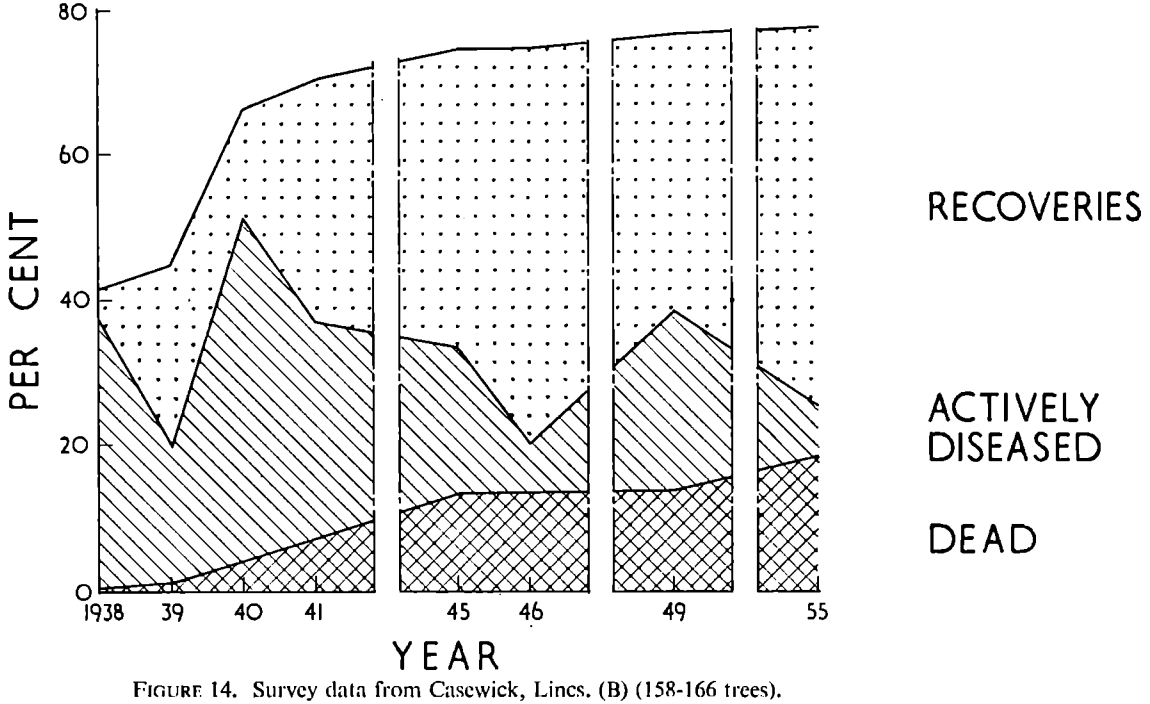

FIGURE 14. Survey data from Casewick, Lincs. (B) (158-166 trees).

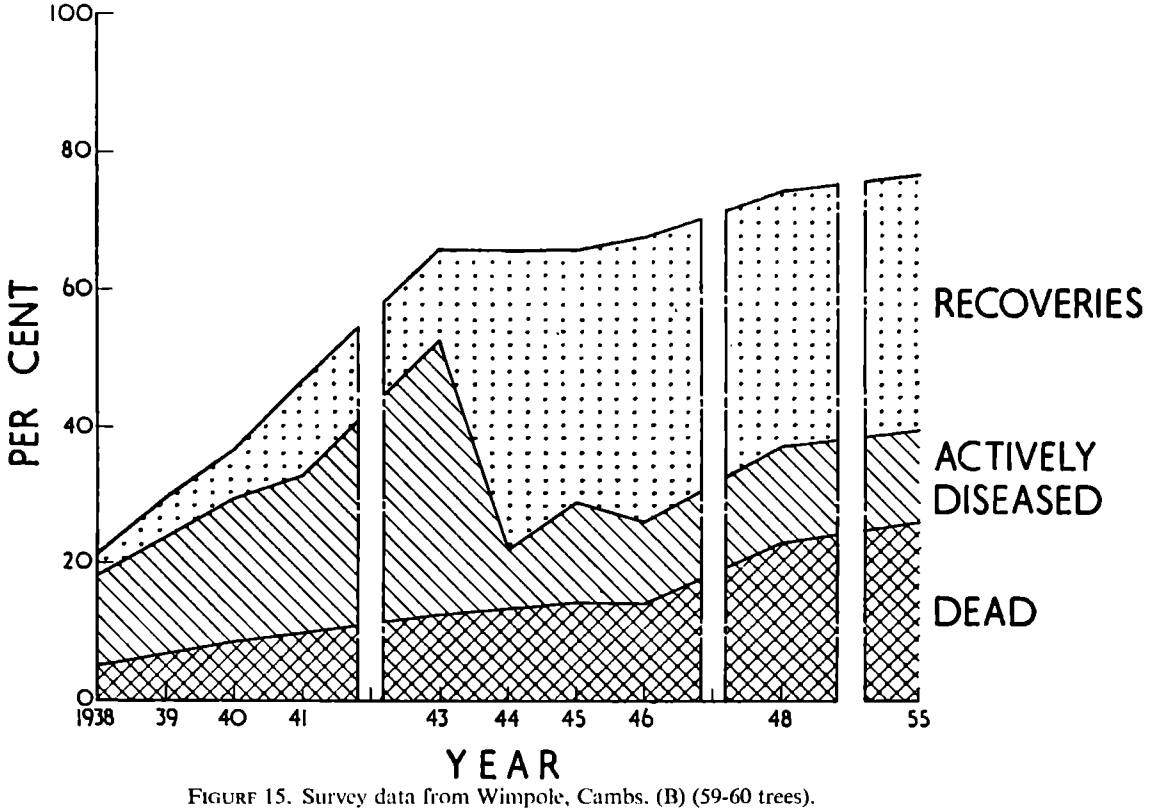

FIGURE 15. Survey data from Wimpole, Cambs. (B) (59-60 trees).

distinct peak in 1948. The trees were generally in good health when the survey started, so that there was no suggestion that they were survivors from an earlier attack. The 1948 peak, here displayed, also occurred at Trowbridge (Fig. 7), and Longstowe (Fig. 10).

There is no doubt that a more random method of selection of survey areas would have brought in far more places where the disease progressed very slowly at a low intensity, and where the final damage was very slight. In 1937, feeling quite correctly that the author's survey areas were too small to give an absolutely typical picture of disease behaviour, Walter and Parker surveyed the extensive elm avenues in Blenheim Park near Oxford, There are probably few places in Britain where one could find nearly 2,600 elms so arranged that they could be easily mapped. The survey actually included 2,590 elms. It certainly provided a very clear example of a low intensity of attack. In 1937, 97.5 per cent were healthy, 1.7 per cent showed active symptoms and 0.8 per cent were dead; by 1946 the proportion free had only fallen to 94.7 per cent, 3.6 per cent were

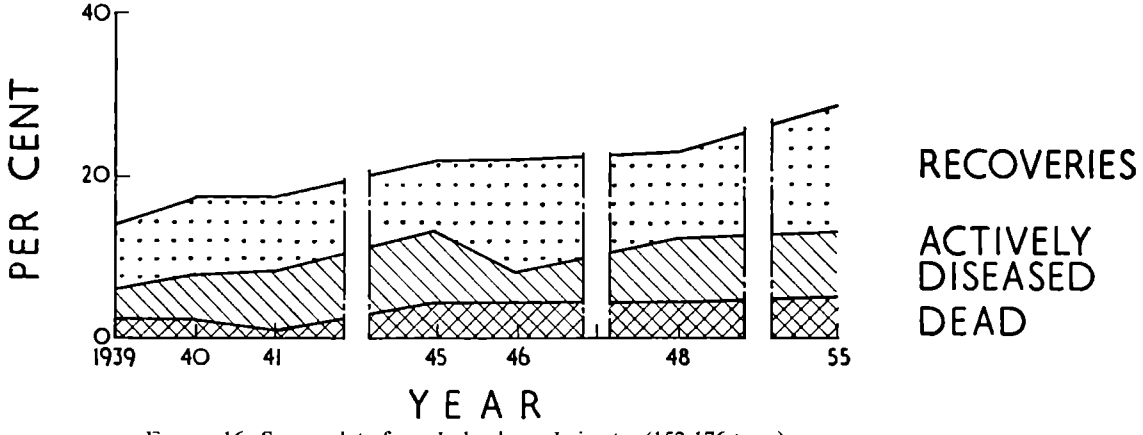

FIGURE 16. Survey data from Lubenham, Leicester (152-176 trees).

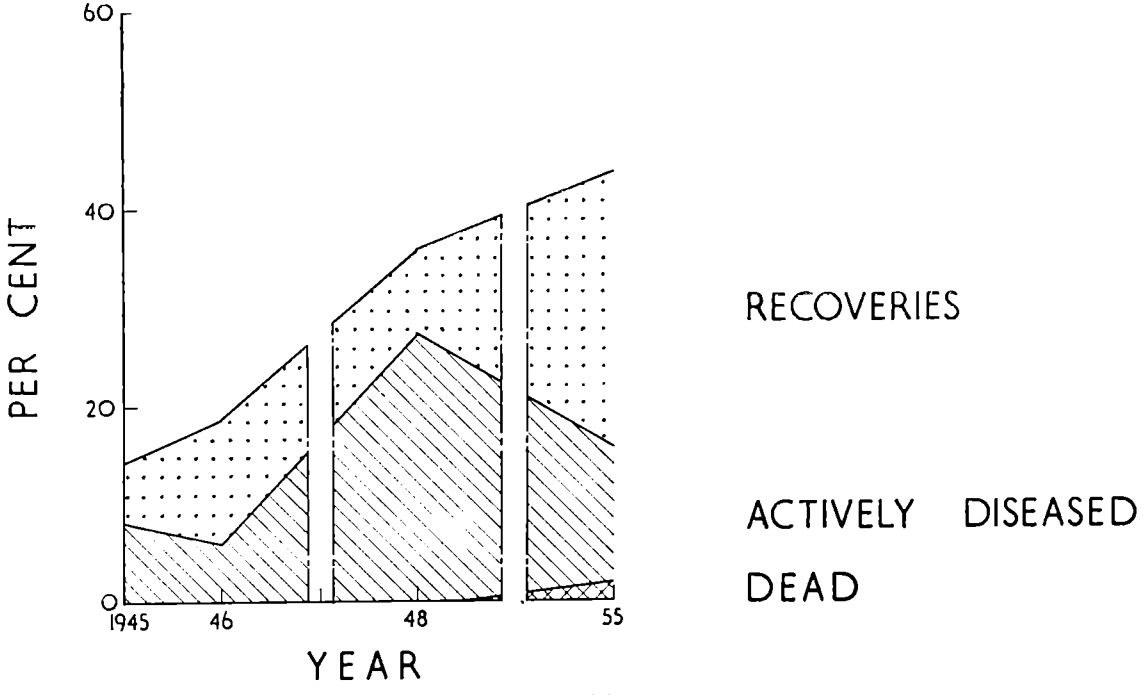

FIGURE 17. Survey data from Ashorne, Warwick (229-278 trees).

actively diseased, and 1.7 per cent dead.

It might be thought that the very low level of attack recorded here was due to the much larger number of trees included. The other survey areas were admittedly selected because they initially showed disease. It could be argued that, if they had been extended to include 2,000 to 3,000 instead of 50 to 300 elms, they also would have given much lower figures of diseased and dead. In a few places this might have been true. An extension of the kind envisaged, however, could have brought Ugley, Stansted and possibly Newport all into the same group; the same could have happened with the two Wimpole areas, and Longstowe. Most certainly it would have combined the two Casewick counts. These combinations would obviously have made little difference in the general recorded level of disease. General observation around the areas actually surveyed suggested that they were quite typical of the much larger numbers of elms in their immediate vicinity, and that likewise the low intensity of disease at Blenheim was actually a true reflection of conditions in that particular locality.

In some places the disease made very slow progress during the survey, although it had reached quite high intensity before the survey started. This was the case in both the Wimpole counts (Figs. 12 and 15) and in the larger survey at Casewick (Fig. 14). In all these areas, however, deaths slowly rose to quite a high level.

Several of the individual tree records showed a sudden onset of severe disease and sometimes death, after long periods of exposure to disease, during which they were, at the worst, only mildly affected. The same phenomenon is displayed on a larger scale by the small population at Longstowe (Fig. 10). In 1943 35 per cent of the elms showed active disease, but none had died. Thereafter the disease fell to a very low level of activity, only to rise again in 1948, when 43 per cent were actively diseased. This had resulted by 1955 in the death of 16 per cent of the trees, although by then the disease level had again fallen. Some of the later local epidemics, such as that experienced in the Isle of Sheppey in Kent, about 1949–50, must have been of the same nature. The disease had certainly been present in that area for many years previously.

It is clear, therefore, that local populations show variations in behaviour almost as great as those found between individual trees, and that there is only limited evidence of anything more than a local pattern of behaviour. It will be seen later, when the position over the country as a whole is considered, that there is some significance in the "peak" years which were always experienced simultaneously in more than one of the survey areas, and in the general tendency of the disease to make much slower progress or even to decrease in virulence since the nineteen-thirties.

The Effect of Environment

It is an obvious possibility that the differences in disease behaviour between local populations and between individual trees might be due to variations in environment. Several suggestions have been made, linking the disease with climatic catastrophes such as drought, but this aspect has been less stressed than with most other tree diseases. In general, workers have taken the view that *Ceratostomella ulmi* is primarily a pathogen not dependent on favourable external circumstances. However, Burger (1938) considered variations in the severity of the disease in different regions of Holland to be due to differences in soil.

The author noted at an early stage the extremely wide range of conditions under which the disease occurred. Active cases were observed on heavy clays and on dry sands, and even in the middle of a stream where all the roots appeared to be under water. Later it was observed that recovery also took place regardless of soil type. Foulden, where some of the most spectacular recoveries occurred, was on dry sand; Bridge End, another area with a high recovery rate, was on clay. In fact, virtually no evidence has been found to connect individual or local variations in disease severity and behaviour with the soil conditions under which the trees were growing.

There is no doubt, of course, as will be discussed later, that climate, and in particular the summer temperature, does markedly affect the course of the disease. But the climate is sufficiently uniform over the main elm area in England to affect all localities in the same way and roughly to the same degree. Summer temperatures therefore produce variations in the disease level as between one year and another over the country as a whole, rather than local variations in any one year.

There is at least one other instance where environment seems to play a part. No serious outbreaks have ever developed in really large towns. For instance the disease has been found at various times in Regent's Park, in Kew Gardens, in Kensington Gardens, on Tooting Common and in other parts of London, but there has been no further spread from the one or two trees initially attacked in each case. It seems possible, though no investigation has been made on this point, that the beetles will not tolerate polluted conditions, so that the means of further spread is lacking. At any rate elm planting is certainly safer in large cities than elsewhere. It is also possible that extreme exposure may interfere with beetle feeding, so that elms very near the coast may be less liable to infection. No evidence on this point has been collected in Britain.

Progress over the Country as a Whole

The Earlier Surveys

After each of the surveys prior to 1947 an effort was made to evaluate the disease over the country as a whole, with particular reference to those areas in the southern half of England where elms predominate. These estimates of disease progress were based to some extent on the local surveys discussed above. It seemed probable, however, that these would not give an overall picture with any degree of accuracy, and general observations made on journeys from one survey area to another, and in the course of other work, were also used. On this basis the position was summarised each year as being much worse, slightly better, etc., than the previous year. These admittedly rather vague estimations have been used to produce a rough graph showing the average progress of the disease in the years 1928–55 (Fig. 18). In this graph, therefore, a rise in the dotted line indicates that the disease was getting worse and a fall that it was getting better, the rate of deterioration or improvement being indicated by the steepness. Figure 18 has also been used to show a possible relationship between the disease level and summer temperature. This will be discussed later in the bulletin. The information for 1948 onwards comes from the later more accurate surveys. From the graph it will be noted that the disease reached a peak in 1931 and again in 1936 and 1937, since when it has irregularly and slowly declined. The peaks in 1931, 1936, 1937, 1940 and 1943, which appeared in the records of some of the local elm populations discussed above, appear clearly as general peak years. This graph may be taken to apply to the southern half of England. Information from other parts of Britain was too meagre and irregular to be taken into account.

It is perhaps a defect of the earlier surveys that apart from the special study of individual trees, no records were kept of the severity of the disease on the trees observed, so that trees of all severity classes were lumped together. Had the graph been based on severity of symptoms as well as on the number of trees showing them, there is no doubt that the fall in level after 1937 would appear more marked. There has been a slow but definite decrease since then in the number of trees displaying active symptoms, but there has also been a much more marked decrease in the number of trees with severe or moderate symptoms, as compared with slight or very slight. The fact that the disease is far less conspicuous now than in the nineteen-thirties is due more to the reduction in severity of symptoms than to the reduction in numbers actively diseased.

The Later Surveys

In 1947 it was decided to introduce a new method of survey involving a nearer approach to random sampling. Road routes were selected to include some of the major elm areas. The routes described below are those used in 1948 and thereafter. They cover all the areas visited in 1947 with the addition of 18 new plots.

Route 1. Starting at Basingstoke, it passed through part of north Hampshire, south Berkshire, and across central Wiltshire and Somerset to Bridgwater; thence it turned south and ended at Taunton in Somerset.

Route 2. Starting at Bristol, it went northwards through Gloucestershire, Worcestershire and Shropshire to Ellesmere; thence it turned north-east to Nantwich in Cheshire, and then south-east through Staffordshire and Warwickshire to Banbury in north Oxfordshire.

Route 3. Starting at Reading in Berkshire, it went up the Thames valley to Oxford, and thence through part of Oxfordshire and south Northamptonshire to Northampton.

Route 4. Starting at Market Harborough in south-west Leicestershire, it went across Northamptonshire to Bedford, and thence to Cambridge; it then turned north-west to Huntingdon and ended at Stamford in the extreme south of Lincolnshire.

Route 5. Starting at Downham Market in west Norfolk, it swung across the north of that county to Norwich and then dropped south across Suffolk to Colchester in Essex; it then turned west across Essex and Hertfordshire to end at Hatfield.

The plots were selected by driving ten miles from the start and then going on more slowly till an area with more than ten elms, all within a reasonably small compass, was located. The same process was repeated after twenty miles had been reached and so on. If no suitable area was found within the first five miles, no plot was made. Thus the first plot had to lie between 10 and 15 miles from the starting point, the second between 20 and 25 miles and so on. In this way, apart from an occasional omitted plot, none could be more than 15 miles from the next or nearer to it than five miles. The elms in each plot were then mapped so that they could be recognised as individuals in future years, and assessed according to the scheme given below.

Current Year's Symptoms	*Dead Wood*
None	None
Very slight	A small amount
Slight	¼ dead
Moderate	½ dead
Severe	¾ dead
Dead	Dead

The evaluation of current year's symptoms was a matter of personal observation, but all the surveys were carried out by the author.

This new type of survey, initiated in 1947, and extended in 1948 to include a larger number of plots, was repeated in 1949, and then stopped owing to

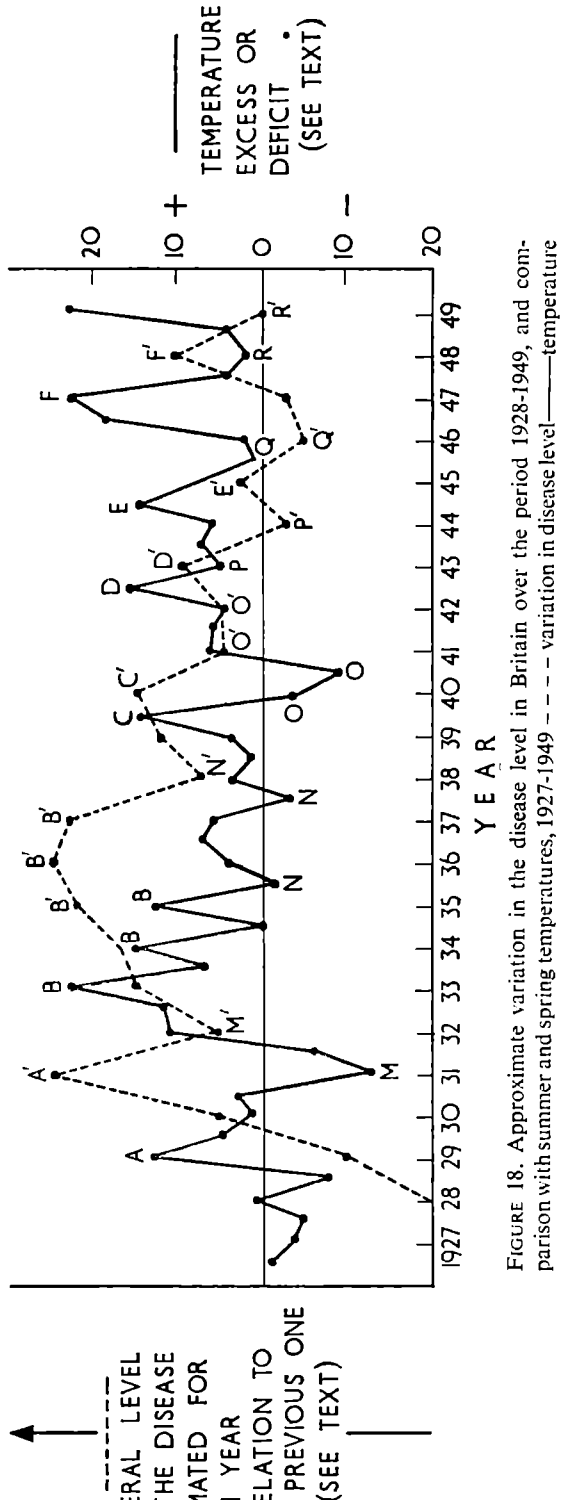

FIGURE 18. Approximate variation in the disease level in Britain over the period 1928-1949, and comparison with summer and spring temperatures, 1927-1949 – – – variation in disease level——temperature excess or deficit.

pressure of other work. It was again repeated in 1955, though by then there was some doubt in a few places as to the fate of trees that had disappeared since 1949. In 1947, 58 plots were surveyed, giving a total of 1,416 elms; in 1948 a further 18 plots were added, bringing the total to 76 and the total of elms to 1,837. The same 76 plots were reviewed in 1949 and 1955. Inevitably there was some loss each year of elms felled for various reasons, often unconnected with the disease. To counterbalance this, some new trees were brought into the survey. These were always small elms falling within the area originally planned, but earlier considered too small to have separate identities. No additions were made in 1955. Thus there was some alteration in the number of trees included in the surveys each year, and in the identity of the trees recorded. These changes are set out in Table 7.

The results of these surveys are set out in Tables 8 to 11. In the first three tables, when individual trees are under consideration, the numbers falling into different categories are expressed as percentages of the total. In Table 11, where *whole* plots are considered, the actual number in each category is given. In general it will be noted that the disease was much worse in 1948 than in 1947, that the position was somewhat better in 1949, and that by 1955 the disease was right back to the low level of 1947. These results have been used to complete the graph showing the variation in disease level from year to year (Fig. 18).

In 1947 the percentage of trees showing active symptoms was low, but within that low figure the proportion showing moderate and severe symptoms was unusually high as compared with subsequent years. This was probably due to the very hot summer of 1947, which tended to accentuate the symptoms on those trees that had been infected. Possibly the same hot summer, by encouraging the breeding of *Scolytus* beetles, was also responsible for the increase in the number of trees actively diseased in 1948 as compared with 1947.

Considering dead wood (Table 9), it is clear that the rather severe injuries often suffered by those trees that were infected in 1947, left an aftermath in the number of cases showing dead wood in 1948. Slight cases often recover the following year without any development of dead wood. In 1949 there was a further increase in the cases showing dead wood, presumably as a result of the more active attack in 1948, but it is noticeable that the proportion of trees with a lot of dead wood had fallen, which reflects the lower level of individual severity in 1948, as compared with the hot summer of 1947.

The same tendency for 1948 to develop a lot of new cases and 1949 to show a lot of dead wood is illustrated by Table 10, where the two classes of symptoms are combined. This table also shows that, while the general position was restored by 1955, there were then fewer active cases and more trees showing dead wood than in 1947, further evidence of the gradual decline of the disease. More surprising is the comparatively large number of disease-free plots and of plots showing only dead wood in 1955 as compared with the earlier years. Admittedly 75 trees known to have had the disease had been removed since 1949, though it is quite certain that many of these were felled for reasons other than disease damage. Even allowing for these, however, the increase in disease-free and disease-inactive plots is distinctly encouraging.

These later surveys inevitably give a distorted picture as regards the very low percentages of dead elms. Although these are true figures for the elms still standing in 1947, they take no account of elms felled because of the disease in earlier years. It is almost certain that the disease has on the average killed more than 10 per cent of the elm tree population over the southern half of England. To some extent this loss has been made good by new elms arising mainly from hedgerow suckers, and the final effect of the disease on our elm population, except in a few of the worst afflicted areas, has really been surprisingly small. These replacement elms, being young, are of course small and do not yet really take the place of the much larger elms which succumbed. The general effect of the disease on our elm popula-

CHANGES IN NUMBERS OF ELMS SURVEYED

TABLE 7

	1947	1948	1949	1955
Trees felled since previous survey:				
Possibly because of Elm Disease	—	3	17	75
Probably for other reasons	—	40	51	274
Young trees freshly included	—	37	31	—
Total number of elms in survey	1,416	1,837	1,800	1,451
Total number of plots surveyed	58	76	76	76

1947–55 SURVEY—CURRENT YEAR'S SYMPTOMS

TABLE 8

Active Symptoms	1947	1948		1949		1955	
		1947 *plots*	*All plots*	1947 *plots*	*All plots*	1947 *plots*	*All plots*
	% %	% %	% %	% %	% %	% %	% %
None	88	77	76	83	83	89	90
Very slight	3	9	9	9	8	5	5
Slight	4	7	8	4	4	3	2
Moderate	3	3	3	2	2	1	1
Severe	1	2	2	—	1	1	1
Total with symptoms, as above	11	21	22	15	15	10	9
Dead	1	2	2	2	2	1	1
Grand Total	100		100		100		100

1947–55 SURVEY—DEAD WOOD

TABLE 9

	% %	% %	% %	% %	% %	% %	% %
None ..	87	84	82	81	80	84	85
A small amount	7	9	8	11	10	6	6
¼ dead	3	3	3	3	4	6	5
½ dead	2	2	3	2	2	2	2
¾ dead	—	—	2	1	2	1	1
Total with dead wood, as above	12	14	16	17	18	15	14
Wholly dead	1	2	2	2	2	1	1
Grand Total	100	100	100	100	100	100	100

1947–55 SURVEY—CURRENT YEAR'S SYMPTOMS AND DEAD WOOD

TABLE 10

Symptoms	1947	1948		1949		1955	
		1947 *plots*	*All plots*	1947 *plots*	*All plots*	1947 *plots*	*All plots*
	%	%	%	%	%	%	%
None	82	71	70	74	74	80	82
Current year's only ..	5	13	12	6	7	3	2
Dead wood only	6	5	6	10	9	8	8
Current year's and dead wood ..	6	9	10	8	8	8	7
Dead	1	2	2	2	2	1	1
Total	100	100	100	100	100	100	100

1947–55 SURVEY—CONDITION OF PLOTS

TABLE 11

Symptoms	1947	1948	1949	1955
None	6	5	6	15
Current year's only	5	7	4	4
Dead wood only	5	4	9	11
Current year's and dead wood	42	60	57	46
Total	58	76	76	76

tion and on the continued use of elms is discussed at a later stage.

The new method of survey was adopted in the hope that it might give a more correct evaluation of the general position. It is therefore of some interest to compare it with the results of the earlier surveys. There appeared to be two possibilities. The earlier survey areas were selected originally because the disease was active within them. At the time of their selection they almost certainly had a worse incidence of disease than the average throughout the country. It might be that this position still persisted as late as the mid-nineteen forties, so that the old plots would still show higher disease percentages than the new ones. On the other hand, in some of them the disease appeared to have run its course and it was therefore possible that they might give a lower percentage of actively diseased trees than the general average, though of course still a higher percentage of dead ones. For comparative purposes percentage figures for free, actively diseased and dead trees were prepared by totalling all the figures for the old areas re-surveyed in 1945 and 1946. One abnormal area was deliberately omitted. This was Blenheim, where the percentage of infected trees was very low and the total number so large that it would have completely outweighed all the other areas together. Percentages for the remainder are set out in Table 12 in comparison with the 1947 figures from the new survey.

It appears from this table that by 1945 the old plots had reached a disease level at which they were roughly representative of the main elm area as a whole. The higher percentage of dead trees in the old plots arises from the length of time they had been under observation. During this period all trees which died were noted and remained on the records. The fact that the death percentage of 8 per cent given for the old surveys in the table is lower than the estimate, given above, of over 10 per cent for the country as a whole, can be attributed to the fact that many of the old surveys were started after 1931, and therefore failed to record the numerous deaths that occurred up to, and including, that year. However, allowing for the differences in percentages dead, the figures for the old-type surveys in 1945 and 1946 are on the same general level as those for the new-type survey in 1947.

It is clear from Table 7 that the number of elms felled for reasons other than Elm Disease in recent years far exceeds the disease losses. This is so, even if we assume that all those trees previously having active disease or a substantial quantity of dead wood were felled for those reasons. It is almost certain that many of the trees placed in the first line of the table, "Trees felled possibly because of Elm Disease", were in fact cut down for other reasons. The design of the new survey does, however, bring in a very definite bias. All the plots were along roads, most of them main roads, and in such places felling for road widening, house-building and so on is likely to be at a higher level than in places well away from road influence. But a good deal of the felling was certainly for timber purposes, and even if we attribute half of it to the roadside siting of the plots, it would still be true to say that man is removing far more elms than the disease. This is now also the case in Holland, though in the nineteen-thirties the diseased trees removed as part of the sanitation programme definitely outnumbered trees felled for other purposes (Went 1954).

COMPARATIVE PERCENTAGE OF TREES SHOWING ACTIVE SYMPTOMS IN 1945, 1946 AND 1947

TABLE 12

	Old Survey		*New Survey*
	1945	1946	1947
	%	%	%
Free	77	84	88
Actively diseased	15	8	11
Dead	8	8	1
Total number of trees involved	1,756	1,291	1,416

Past and Future Trends

The observed variations in the level of the disease in Britain, based partially on the survey figures, and partially on the comparisons with previous seasons, which were recorded each year, are shown in Fig. 18. Apart from this it was clearly apparent that the severity of the symptoms developing in those trees which had become infected depended to some extent on the summer climate. In hot summers, when the transpiration rate was high, the symptoms tended to be severe, and it was noted during the course of the surveys that in some cases there was a tendency for peaks in the number of trees infected by Elm Disease to occur in years following hot summers. This was certainly the case when the high peak of the disease in 1948 followed the exceptionally hot summer of 1947. This probably reflects the favourable influence of the hot weather on the beetle population. In some years, notably 1940 and 1943, disease peaks followed warm weather in the spring and early summer of the same year. This again is likely to be an indirect effect, depending on a climatically controlled increase in the beetle population. In order to show both these effects, the mean monthly temperatures at two meteorological stations, one at Oxford and the other at Cambridge, were used. These two stations can be taken as representative of the area where the Elm Disease surveys were carried out. The figures actually used were the deviations from average of the mean monthly temperatures. The figures for the two stations were first added together and then totalled for the months of April, May and June and again for the months of July, August and September. The former were taken to represent spring temperatures, which might affect beetle breeding and therefore Elm Disease in the same year, the latter to represent the summer temperatures, the effect of which on the disease would not be apparent till the following year.

These deviations are also shown graphically in Figure 18, which shows a certain measure of correspondence between the peaks and dips in the graph thus created, and that for the annual level of the disease. In some cases, however, a peak in temperature shows a year or even more before a peak in disease level. This may mean that a build-up in the beetle population, taking more than one year to develop, is triggered off by a hot season. This could arise because the increased severity of the injury on those trees that were infected in the hot summer would provide greater breeding facilities for beetles, which would then cause increased numbers of infections in the following years.

If it is assumed that the general level of the disease rose to a peak around 1936 and then fell, the deviations of the disease level in each year above and below this general trend are significantly correlated with the summer temperatures of the *previous* year, but not with the spring temperatures of the *same* year. Nevertheless there are several cases where high spring temperatures have been immediately followed by a rise in the disease level. Peaks in the temperature curve are lettered A, B, C, etc., and the peaks in the disease level, which may be connected with them, A', B', C', etc. Similarly depressions in the temperature curve are lettered M, N, O, etc., and the possibly resultant depressions in disease level M', N', O', etc.

It is obvious, however, that even the immediate future course of the disease cannot be foretold merely by a study of temperatures. On the other hand there is a strong probability that a peak year will follow a period of exceptionally hot spring or summer weather. There must certainly be other factors affecting the year to year level of the disease, besides the indirect effect of climate on the beetles that carry the causal fungus.

Apart from these annual variations in the disease, there has certainly been a general decline in the disease level since 1937. Since this is more a matter of a reduction in severity of attack than of a fall in numbers affected, it does not appear as clearly as it should from the survey figures. To the author, who over the survey period developed an "eye" for diseased elms, the decline is abundantly apparent. In the early years up to 1931 the disease was so virulent that it frequently reached the headlines of the national press, and led to many somewhat ill-founded prophecies of the eventual extinction of the elm in Britain. During the rest of the decade and even later, the disease had a habit of suddenly flaring-up in a new locality. Each time this happened it produced requests for advice and often an alarmist story for the local press. Since the war, on the other hand, the disease has attracted practically no attention, the occasional occurrence of symptoms being regarded almost as part of normal elm behaviour. Enquiries reaching the author about the disease now number only one or two each season.

Little information is available on the long-term behaviour of the disease in other countries. Went (1954) took the view that in Holland the disease was not decreasing and would eventually wipe out all their elms. In the United States it was progressing steadily in many areas up to 1945, despite active efforts to control it by felling, which were not abandoned till 1940. Zentmyer, Horsfall and Wallace (1946) found that its progress was steady and arithmetical, a quite different state of affairs from our experience in England. In America the disease is still increasingly active on the margins of its range as it moves into new areas (Anon. 1956; Campana & Carter 1957). On the other hand Mańka (1954) states that the disease has decreased in the Polish province of Poznán since the period 1947–49. In

most parts of the Continent much less is heard of the disease now than before the 1939–45 war, and it is almost certain that there has been a general reduction in severity. In most countries this cannot be attributed to control measures, and it certainly appears that in Europe the disease is generally on the decline. It is a great pity that more accurate information is not available.

The outcome in America may be different, since they have an elm population of higher and more uniform susceptibility and one growing under ideal climatic conditions for the development of this particular disease.

Unfortunately there is no certainty that the decline in Britain will continue or even that the disease will remain at its present tolerable level. This means that the planting of new elms is still fraught with a degree of risk which we cannot estimate. Even if we argue that the disease has declined because it has attacked and killed all the most susceptible trees, it is clear that by new planting we may provide a fresh batch of susceptible individuals.

Actually the above assumption is not supported by the facts. Trials, described above, of supposedly resistant, surviving elms were very disappointing. Plantings, such as the avenues at Trowbridge and Casewick, which are almost certainly clonal, showed enormous variations in severity of attack between individuals. This must have depended on some circumstance other than the inherent resistance of the tree. Trees have survived unharmed in heavily infected areas and then suddenly succumbed when the peak of the local attack had passed. Other trees have been nearly killed by the fungus and have then recovered, sometimes despite fresh infections. In fact, even with our considerable knowledge of the disease, we still cannot say how it will behave in the future in the individual tree, let alone in the elm population as a whole.

It would be more reasonable to suggest that earlier attacks have wiped out those individuals in which there was a tendency for the disease to recur without necessarily involving fresh beetle infection, and in which therefore repeated annual damage eventually led to death. Lacking exact information on the method of infection in the trees under observation, none of the survey data can really throw light on this idea. It is hardly possible that it can be the major factor in the observed reduction of the disease.

Another possibility is that there has been a general lowering of the average virulence of the fungus. This is more probable than the idea mooted above, that there has been a rise in the average resistance of the elm population, but its probability rests only on the general trend of events. Detailed investigation immediately throws doubt on its validity. Since 1945, a number of inoculation experiments have been carried out, using various strains of the fungus, isolated mostly from slightly attacked elms in the vicinity of Farnham, Surrey. These have invariably produced strongly developed disease symptoms, when inoculated into susceptible young elms. Even now one can still find violent manifestations of the disease on individual trees in nature, though the last recorded extensive local outbreak was in 1949–50. Under these circumstances it is difficult to believe that the fungus really is losing its virulence.

A very feasible explanation of the reduction in the disease has been suggested to the author by Mr. James Macdonald. In the early days of the disease in Britain dead and dying elms were often left standing for long periods, because no sanitation measures were in force, and the market for elm timber was saturated. War-time timber shortages entirely altered this position. Nearly all severely diseased or dead elms which were still fresh enough for beetle breeding were felled either for timber or firewood. After the war the market for elm timber remained good, and there has been much less tendency to leave dead and dying trees standing. In any case once a reduction in the beetle population had been initiated, it would tend to become continuous, since each reduction in the number of diseased trees would decrease the number of beetles, and each reduction in beetles would decrease the number of new infections. It is very possible therefore that the beetle population may be falling in concert with the disease.

Unfortunately no comparative numerical studies of variations in the beetle population from year to year are available. We have therefore no means of checking whether there has in fact been a fall in the number of *Scolytus* beetles. In the author's opinion it is almost certain, on circumstantial evidence, that such a reduction has taken place. Even if we assume such a reduction, it would nevertheless be rash to assert that it provides the sole explanation of the fall in the number of trees diseased, and of the severity of the symptoms on them.

Elm still ranks as one of our commonest and best hedgerow trees, and there is a steady demand for its timber. If, as seems desirable to many, there is a revival in hedgerow planting, elm should play a considerable part. This is not only because of its inherent suitability and timber value, but also because in many places it can be raised cheaply, simply by letting existing hedgerow suckers grow up into trees. This course would almost certainly lead under present conditions to some of the resultant trees being attacked by the disease, a very few of them seriously. This probability need not deter anyone from hedgerow planting, though it does still throw doubt on the advisability of using elms for formal avenues, where the loss of a single tree can disfigure

the whole layout. Whether a widespread recrudescence of hedgerow elms might lead to a redevelopment of the disease in a virulent form, is a question that could be answered only if we knew more exactly why the disease has decreased since 1937. Since the number of hedgerow elms could be increased comparatively cheaply, the risk of an upsurge of the disease is probably worth taking.

Chapter 6

THE CONTROL OF THE DISEASE

Economic Justification

Control measures must of course always be related to their cost, their effectiveness, and the money which they save. The first two considerations vary according to the method used, and are therefore dealt with under methods of control below. But it is pertinent here to examine what exactly we should achieve if we could bring Elm Disease under full control. We might consider two slightly unreal extremes, a country where the disease wipes out virtually every elm and another where some method or methods of control save them.

In the first country there is a substantial loss of amenity, which may have quite a high monetary value; there is a smaller loss in that a useful timber genus can no longer be planted because of the disease risk, but provided the elms die over a period there should be little loss of timber. Diseased or freshly dead elms can be utilized just as well as healthy ones.

The country where control has been entirely successful has, of course, preserved its amenity; it can continue to plant elms, and it can market its existing elm timber over a longer period. But it must weigh virtually the entire cost of control against the preservation of amenity or the ability to continue to grow elms.

In the United States, where elm is a most valuable shade tree, and where trees for shade are really required, or in Holland where elm is the tree most resistant to exposure in some of the seaside districts, high expenditure on control might be justified. In Britain where elm is a valuable, but surely not essential, part of our landscape, heavy expenditure would be less easy to justify.

In any case, the argument set out above assumes that the control proves successful. If it is not fully successful, it may merely serve to spread the effect of the disease over a longer period than it would have taken if unhindered. The final result measured in loss of elms may well be the same.

Methods of Control

Both the early ravages of the disease and the importance of elm in northern temperate regions stimulated a great deal of work on control. A considerable number of methods have been investigated, but they are discussed briefly below only in so far as they are applicable to British conditions.

Felling

Quite naturally when the disease first appeared as a serious cause of damage to elms in Europe, it was assumed that the proper action was to fell all diseased elms. This was attempted on a rather haphazard basis in many countries, but was accepted as official policy in Holland to the extent of felling all moderately and badly diseased elms, and also for a time in Germany. After the disease had reached the United States, on unbarked timber imported from Europe, eradication was attempted there.

In Britain, it was decided during the summer of 1928 that the disease was too widespread for eradication to be possible, but felling of diseased trees as a voluntary measure was still recommended. By the autumn of 1928 even that practice was under criticism, on the very reasonable grounds that we did not then know what the fate of infected trees might be. The phenomenon of recovery became apparent in 1929, and led to the abandonment of all recommendations to fell, except for such trees as were obviously beyond hope of recovery. The desirability of felling badly diseased trees became more obvious, when the means of transmission by *Scolytus* beetles was discovered and it was apparent that such trees were potent sources of infected beetles. The present recommendations given in Forestry Commission Leaflet 19 (Anon. 1958) are that elms should not be removed till they are dead or unsightly, but that in parks and gardens and with street trees efforts should be made to remove dead wood and thus lessen beetle breeding.

It is quite certain that had the felling of diseased

elms been practised on a large scale in Britain our losses, resulting in this case indirectly from the disease, would have been much higher than the direct loss that has occurred. For we can hardly assume that the policy would have been completely successful at an early stage, so that the fungus was eradicated. American and Dutch experience suggests that eradication is impossible. Difficulties of detection and the widespread occurrence of elms in out-of-the-way places make it clear that to attempt such a task would have been outrageously expensive, and that it would have been certain to fail. Partial eradication of diseased elms would certainly have lessened the initial fury of the disease. It would, however, have been a constant task, the abandonment of which at any stage might well have resulted in an immediate upsurge. In the course of such measures it is certain that a great many elms, which have now recovered, would have been felled.

The Dutch, following a policy of felling, designed not to eradicate the disease, but merely to slow up its progress, have lost two-thirds of their elms (Went 1954). It is not entirely fair to compare their experience with ours, because the conditions in Holland are obviously different. Their street plantings were nearly always clonal stocks, which were thought to show uniform and often low resistance to the fungus. This hypothesis remains unproven, as does the suggestion that Britain has escaped lightly because its elms are genetically very mixed and therefore very variable in their resistance. Evidence has already been presented that these ideas do not fit in with the known behaviour of the disease in Britain. The author is prepared to hazard that the Dutch would have more elms left alive and standing today, if they had restricted their felling to dead and really severely damaged trees. But this is an individual view and there are others who hold firmly that the practice of sanitation measures will lead to a greater survival in the end.

In America there is disagreement as to the effect of the felling policy that was followed until 1940. Zentmyer, Horsfall and Wallace (1946) consider that it had little effect. Marsden (1953), while agreeing that its abandonment as a national policy was inevitable, feels that it is still a worthwhile measure for any township really interested in its elms. He supports this suggestion by comparing the progress of the disease in two towns with slightly differing control programmes, a doubtful basis of argument in view of the well-known erratic behaviour of the disease.

In some parts of the United States very active sanitation programmes are still in progress (Anon. 1956). There is so little information available on the possibility of recovery of American elms, that it would obviously be rash to suggest that it would have been better to fell only severely infected and dead elms. It is quite certain that such a policy would have saved a great deal of money.

There is definite evidence, however, that severe infections do take place immediately around accumulations of material suitable for beetle breeding. This has been noted on numerous occasions in Britain (Plate 33), the sources varying from standing dead trees, or logs in timber yards, to piles of elm firewood. As recently as 1957 a small but violent outbreak was observed around a large dead elm that had been left standing. This phenomenon of intense local spread from a source of infected beetles has been described on a more exact basis in America (Parker *et al.* 1947; Liming, Rex and Layton 1951). There is certainly strong evidence here for the avoidance of such foci of infection by the early removal of dead trees, and if possible of large dead limbs, and by the storage of elm firewood in closed sheds or well away from elms. But that is a very different matter from the wholesale felling of all diseased elms.

Pruning

Elm disease can be arrested by pruning provided the infection is not far advanced (Zentmyer, Horsfall and Wallace 1946). Late infections, which do not progress very far before the end of the summer, are the easiest to deal with in this way. There is considerable doubt, however, whether such infections would prove seriously damaging to the trees in any case, since they are almost certain to recover naturally the following year. It is the early, severe infections, which cannot be controlled by pruning out diseased wood, that are most likely to cross over and continue as active cases the following season.

Where pruning is tried, and it could be attempted on small trees of special value, it should be done so that all the wood showing markings in the current year is removed. In some cases this may involve cutting out parts of the tree which are not showing active external symptoms, so that the final damage to the tree may be substantially greater than would have been caused by the disease, if it had been left alone.

Quarantine

It is obviously desirable that any elm-growing country, which is still free of Elm Disease, should endeavour to prevent its importation. This is probably best done by a complete prohibition on the entry of elms of any kind, for even supposedly resistant varieties may carry the disease in a suppressed form. In Britain such action was taken in 1926, very soon after the alarm had been raised on the Continent. It is now known, of course, that the fungus was already present in Britain at that time. This prohibition remained in force until 1949, since

there was always the possibility that some strain of the fungus might arise abroad which was more virulent than those we already possessed. Lacking evidence of any such occurrence, the ban was lifted, so that elms could be imported under licence, but only from Europe, mainly in order to allow the easy entry of any disease-resistant clones that might be produced in Holland. The continued prohibition on import from countries *outside* Europe was designed mainly to prevent, if possible, the entry of Phloem necrosis, the serious virus disease now widespread in the central United States.

Selection and Breeding

The possibilities of selection and breeding for resistance, and the work that has already been done, mainly in Holland, has already been discussed. Accounts of the Dutch work have been given by Went (1954) and Heybroek (1957).

Some of the most recent Dutch selections are being tested under conditions of natural infection in Britain. These are clones which have shown a fairly high experimental resistance, but have occasionally succumbed to inoculation. It is expected that they will prove sufficiently resistant in the field to be used with safety. Some of them appear to be trees of very good form, and they may eventually prove very useful for park and street planting, where the relatively high cost of specially propagated clonal stocks would be justified. They can hardly be expected to replace our hedgerow elms, so many of which arise spontaneously from suckers, aided by man only to the extent that he does not cut them back in the course of hedging. But it is surely reasonable to take a risk of disease with trees that have cost us nothing to plant, provided we have resistant stocks available for sites where we must use and pay for new plants. It is to be hoped that the Dutch selections will eventually allow us to plant along streets and in avenues without the present possibility of disease-caused gaps.

Spraying against Scolytus Beetles

Since the spread of the fungus is almost entirely due to *Scolytus* beetles, it should be possible to control the disease indirectly by measures against these insects. There is even less hope of eradicating the beetles than there is of stamping out the fungus, and so control work has been largely aimed at preventing the beetles feeding on elms and infecting them. Practicable methods have been elaborated in the United States (Swingle, Whitten & Young 1949; McCallum & Stewart 1951; Welch & Matthysse 1955) and are being used there on a limited scale. One method involving two sprays, one in the early spring and the other in the summer, was tried on quite a large scale in Britain (Peace 1954). The results were generally, but not consistently, successful, but the cost was £1 per tree per year for trees only 20–30 ft. high. Since spraying of this nature is purely protective, it would have to be repeated every year. An expenditure of £1 per year is too much to charge against any normal tree, so that the method appears to be of little practical use, though it could be adopted to protect small individual trees of high ornamental or sentimental value.

Chemotherapy

A good deal of work has been done in America on the possibility of controlling the fungus by injecting systemic chemicals into elms or watering them onto the ground over the roots (Zentmyer, Horsfall & Wallace 1946; Dimond *et al.* 1949; Potter 1956). A number of substances have given quite good results in small trees, but in larger trees they have not been so successful. McCallum and Stewart (1951) considered that these methods could not yet be recommended for general use. Recent advances in the study of systemic fungicides, especially in the use of antibiotics, do suggest some future possibilities; but in any case the question of cost again arises. This is also a protective measure which to be effective would have to be applied every year. The cost is likely to be of the same order as that for spraying, somewhere in the region of £1 per tree per year. Such a cost is so high that it inevitably restricts the method to individual small trees of very special value.

Chapter 7

SUMMARY AND CONCLUSIONS

This Bulletin is mainly devoted to describing the surveys in Britain of Elm Disease caused by the fungus *Ceratostomella ulmi*. These were started in 1928, and continued with some interruptions until 1955. To provide the necessary background a brief description of the disease is given, followed by a consideration of its history and distribution with particular reference to Britain. Very short descriptions are given of other diseases of elm, which might be confused with that caused by *Ceratostomella ulmi*. Variability in the fungus and differences in resistance in the host are described, with particular

reference to their influence on the general behaviour of the disease. The progress of the disease as disclosed by the surveys is considered in relation to (*a*) the individual tree, where the phenomenon of recovery is particularly discussed, (*b*) local populations, and (*c*) the country as a whole. A final section is devoted to methods of control.

It is concluded that the disease has declined erratically, but continuously, since 1936–37, not only in the number of elms affected, but also more particularly in the severity of the damage caused. It is believed that this reduction has also occurred on the Continent. The reasons for the decline are not properly understood, and there is evidence against its being due either to loss of virulence by the fungus or to the higher resistance of the surviving elms. It may be connected with the wartime clearance of diseased elms for timber and firewood and the favourable post-war market, which still makes the felling of dying elms an economic possibility. This may have led to a reduction in suitable breeding sites for *Scolytus* beetles, and consequently in the number of infections which they cause. At the moment man is removing elms, by felling them for timber and to clear ground for road widening or building, much faster than the disease is killing them. Despite its early violence the total effect of the disease on the elm population has been surprisingly small. The total loss probably lies somewhere between 10 and 20 per cent of tree elms, and this is being made good by the growth of young sucker elms in hedges. Only in a few localities has the disease materially altered the landscape.

In view of the very frequent recovery of attacked elms, the author is convinced that the loss would have been much higher if a policy of felling diseased elms had been put into action in Britain, instead of one of virtual *laissez faire* that was adopted. It is possible that widespread felling was a mistake in Holland, and even in America.

There is no certainty that the disease will continue to decline, but equally there is no reason to expect a resurgence. If the present level of attack remains constant, establishment of elms in hedgerows, especially by the cheap method of encouraging suckers to grow up, is perfectly reasonable. Plantings in avenues or along streets may be disfigured by loss of individual trees. Such plantings might perhaps await the field testing of Dutch resistant selection now in progress. Some of these will probably provide elms of good form, with the higher level of resistance necessary for sites where each tree has an individual value.

Control should be limited to the removal of elms which are dead, nearly dead or unsightly, since these may act as centres of spread for infected beetles. Where possible dead limbs, which serve the same role, should also be removed. Unbarked logs and elm firewood should never be left in the vicinity of living elms. These precautions should lessen the possibility of severe local outbreaks.

The disease may long continue as a minor nuisance, but unless it completely changes its present trend of behaviour it will never bring about the disaster once considered imminent. Indeed in order to wipe out most of the elms in Britain it would have to achieve an even higher level of virulence than it did in its worst years in the nineteen-thirties.

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