Dr. Richard J. Campana, left foreground, participates in tree planting ceremony at 1967 International Shade Tree Conference at Fairmount Park, Philadelphia. Dr. Campana, chairman of the department of botany and plant pathology at the University of Maine and one of the foremost researchers actively searching for new methods of control for Dutch elm disease, is the immediate past president of ISTC. He agreed to relate his experiences and observations regarding the national DED program at the request of WEEDS TREES AND TURF magazine. The editors are happy to publish this extensive insight into the problem by Dr. Campana.

Dutch Elm Disease:

A Matter of Priorities

By DR. RICHARD J. CAMPANA
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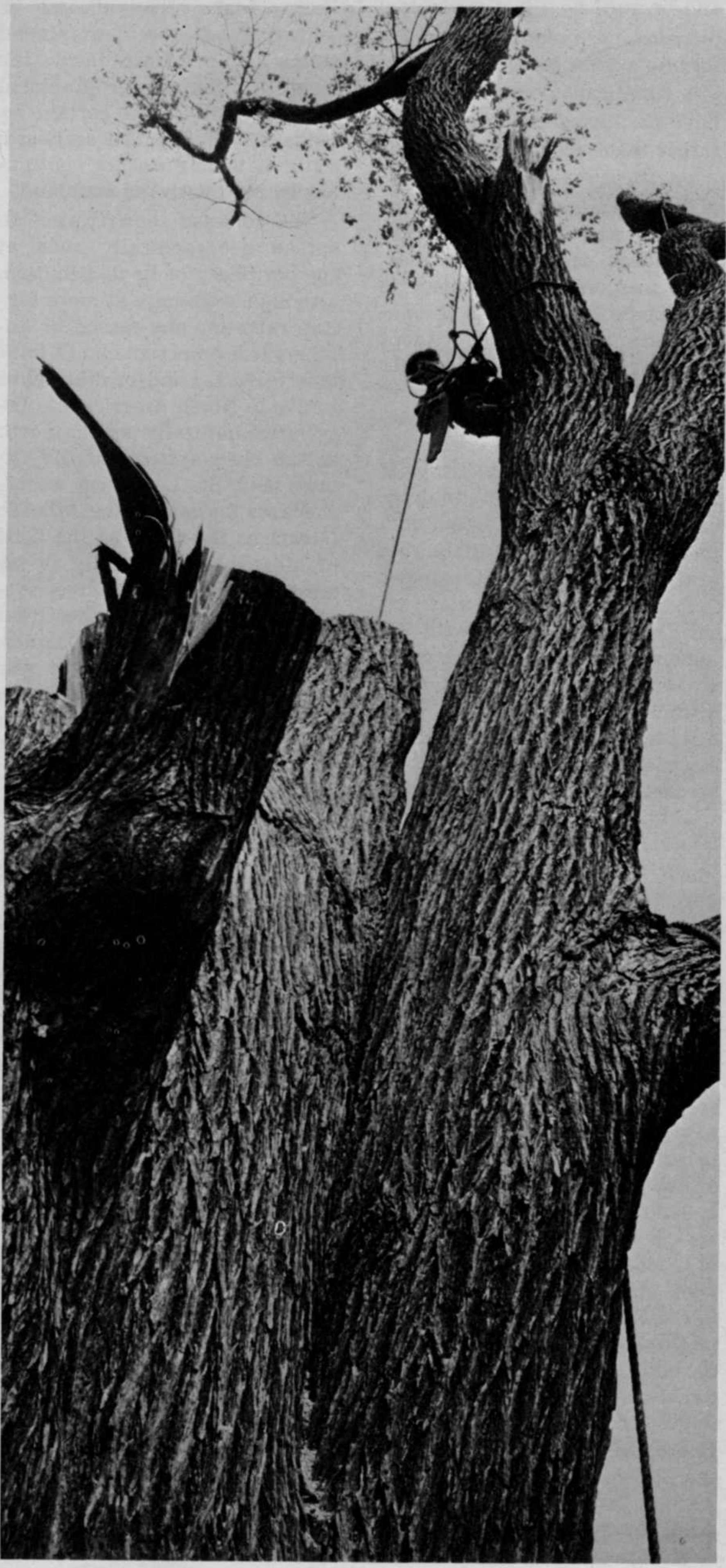
Orono, Maine

THE DUTCH elm disease is an American tragedy. Introduced inadvertedly to the United States from Europe by 1930, it rages unabated through native elms of field and forest, and seems slowed only temporarily and sporadically in relatively few urban areas at great cost in time, labor and money.

It is the most serious and devastating shade tree disease in North America. Not only has elm been one of the most populous species in most cities and towns in midwestern and northeastern United States; but elms are natively distributed throughout the eastern half of the U.S. as a forest tree principally along streams. The disease is unusually serious because it kills most trees once the main stem is affected, it continues to spread and intensify each year, all species of elm are susceptible to some degree, it creates ideal conditions for proliferation of elm bark beetles which make its greater distribution possible, and at present there is no known cure for trees affected. The disease is characterized by wilting of foliage caused by clogging of water conducting vessels, and is caused by a microscopic fungus, *Ceratocystis ulmi* (Buism.) C. Moreau, which multiplies and moves in such vessels. The fungus may be spread from diseased to healthy trees by root grafts between such trees, or by either one of two elm bark beetles (the native American, *Hylurgopinus rufipes* Eichh.; or the introduced European, *Scolytus multistriatus* Marsh.) The origin of the disease is obscure. It appeared unexpectedly and unknown in war-torn areas of Western Europe following World War 1. In Europe it spread throughout the continent and destroyed extensive elm stands wherever it occurred. In Canada and the U.S. it spreads unchecked through native elms of field and forest, and continues to decimate urban elm populations in the absence of effective measures to prevent its spread. It is truly a tragedy of monstrous proportions which continues to unfold before our eyes with each succeeding disastrous period of new infections. The disease may be controlled only in the sense that its toll may be limited, and only within limited areas requiring substantial effort on the part of local residents.

As the Dutch elm disease continues to spread, it probably now destroys millions of trees annually. Moreover, it continues to stimulate public interest and emphasizes clearly the inability to prevent its spread from one area to another. Unfortunately there is much misunderstanding and widespread ignorance about many aspects of the disease. Even among research entomologists and plant pathologists there is disagreement on its many aspects. It is obvious, that among scientists many points of conflict arise, either where there is no evidence at all, or where the evidence is merely indicative but not conclusive. In any event, after 16 years of continuous work with the disease, it has become increasingly apparent to the writer that Dutch elm disease is far more complex as a biological phenomenon than most realize. During this period, many of us have accepted too easily without critical evaluation, not only ideas for miraculous prevention



and/or cure by chemical magic, but also more plausible ones affording a false sense of security. The significant point is, that we have often accepted ideas as facts, rather than recognizing them for what they really were, as hypotheses, assumptions or conclusions derived from assumptions. In practice, perhaps the most serious mistake we have made is to educate the public to the advantages of spraying, without stressing strongly enough either the meticulous thoroughness and timing required for effective application of suitable chemicals, or the relative inadequacy of spraying without prior and proper attention to sanitation, and treatment for potential root grafts. The purpose of this paper is to present some new views on the Dutch elm disease with the hope that they may help to clarify certain aspects of this most difficult biological puzzle. Various aspects of the disease will be discussed under the subtopics of: spread and distribution, susceptibility, symptoms, diagnosis, transmission and control. There is no attempt here to discuss any of these topics at length; emphasis is placed only where the writer believes there has been misunderstanding and possible misinformation.

Spread and Distribution:

The disease continues to spread at three levels: from state to state; from area to area within states; and from one section of urban control zones to others. It is now known to be within an area bounded by: the provinces of Quebec, Ontario, New Brunswick and Nova Scotia, Canada, in the North; the Atlantic Ocean in the East; the states of North Carolina, Tennessee, Oklahoma, Arkansas and Texas in the South; and the states of Kansas, Nebraska and South Dakota in the Midwest. Isolated infections were reported from Denver, Colorado over ten years ago, and only within the past year from Boise, Idaho. The disease is known to be present within all states and provinces inside the larger, contiguous, geographical area, as well as on certain islands off the eastern seaboard, some of which are not contiguous by road with the mainland.

The disease continues to spread geographically even at the periphery of its distribution, although seemingly at more limited rates for one reason or another. The American elm *(Ulmus americana* L.*)* and/or other elms native to North America are distributed naturally as far north as the river systems of the Ottawa and St. Lawrence watersheds, as far east as the Atlantic Ocean, as far south as the Gulf of Mexico and generally as far west as the numerous river systems will alow in the the Great Plains (i.e. in The Dakotas, Nebraska, and Kansas). It was surprising to the writer to learn that the American elm is even known to be present in the foothills of the Black Hills of Wyoming.

In the North it is likely that spread of the disease is limited by extremes of cold which preclude or limit occurrence of one of the insect vectors *(Scolytus multistriatus* Marsh.*)*, considered by many to be the more effective of the two known carriers of the causal fungus. In the East the only deterrent to spread of the disease seems to be the Atlantic Ocean, which limits to a great degree transportation of contaminated elm wood, as well as aerial transmission of bark beetles and viable inoculum. However, road access to large islands, such as Long Island in New York and Mt. Desert Island in Maine, makes such islands easy targets. On the other hand, to the extent that they have native or introduced elms, islands isolated by substantial stretches of open water are less easily invaded by the disease.

In the south the disease ap-

"Why did he go out? He had all the business he could handle!"

pears to be limited both by temperature and less dense populations of both native and planted elms. In transmission from elm to elm the fungus on the body of the insect vector is expected to remain viable longer under conditions favoring slow desiccation. Also, the fungus is limited in growth and development by high temperatures. To the extent that the degree and duration of the heat of the South favor rapid desiccation of viable spores, and limit growth and reproduction of the fungus on or in the host trees, they should be expected to act as deterrents. On the other hand, possibly the heat and high humidity of the South could favor the European elm bark beetle, so that it may produce more than two broods per year. But even if this were possible, it would not be expected to increase the probability for spread to a significant degree, because of the primary importance of the first brood. However, I have seen no data on this.

In the West spread of the disease seems only to be limited by native occurrence of the elm itself. However, its occurrence beyond native distribution, into urban areas first in Colorado and more recently in Idaho should be instructive. It reemphasizes the role that man plays in spread, and thus should serve as another living warning that elm populations, however remote beyond the range of native elms, are still open to invasion with the help of man in all his multitudinous activities and travels. However careful we should try to be, it is not unlikely that the disease could be introduced inadvertedly into the relatively cool, wet and most favorable climate of the Northwestern states of Oregon and Washington, and the province of British Columbia. The apparent elimination of infections in Denver, and the possibility of elimination in Idaho, should not lull us into a false sense of security where more favorable conditions may occur for survival of the fungus. If the disease gets into coastal Washington, Oregon and/or British Columbia, only the occurrence and distribution, or lack thereof, of elm populations would seem to be significant as natural factors favoring control.

Susceptibility:

The American elm has long been recognized by many authorities as the most susceptible to Dutch elm disease of all elms known to man. Also, it is conceded generally that native European and especially native Asiatic elms have greater resistance to the disease than do most North American species. Various hybrid selections of European and Asiatic elms have been reported to have a degree of resistance close to immunity. However, it should be clear, that it is almost impossible to be certain of high resistance under all conditions, since variation in strains, and thus virulence, of the fungus may change. We have every reason to believe that sexuality and hybridization by the causal fungus are common, thus new strains and capacities of virulence are possible continuously. However, this should not discourage us from seeking resistant varieties, even in the most susceptible American elm.

With the American elm in particular, a considerable degree of resistance to one or more strains of the fungus is becoming increasingly apparent. This is evident, when, of hundreds of trees inoculated with a single fungus strain, only a fraction become diseased, and many of the diseased trees recover. On the other hand, with certain other strains more often than not, most of several hundred trees inoculated do become diseased and die. Unfortunately, it is probable that most naturally diseased trees are infected by multiple fungus strains with varying degrees of virulence. This is apparent where sexual fruiting structures of the fungus are found naturally produced in many diseased trees. This phase of the life cycle of the fungus is possible when 2 different, but compatible mating strains are present in the same tree, indicating the distinct possibility of different genetic capacities for disease causation. Another example, illustrating variability in virulence, may well be the single large tree surviving, when all others surrounding it are killed. Such a tree is often easily infected and killed if inoculated deliberately with a pure culture of the fungus. In the past we have often regarded such trees as "fortuitous escapes" lucky enough to have been inoculated naturally. While this may often be so, it is more probable that such trees are actually exposed to no more than a single fungal strain. It is also possible that they either failed to become infected, or did become infected, but recovered. They could then easily become diseased later from a different fungal strain. Fortunately, in many cases, they may never

Recent Symposiums on Dutch Elm Disease

Two major symposiums on Dutch elm disease have been held during the past year, one a regional meeting during June, 1967, at the US Forest Service Laboratory, Delaware, O., and an international symposium, February, 1968, at Iowa State University, Ames, Ia. Copies of proceedings are available respectively from John W. Peakcock, recording secretary, Northeastern Forest Experiment Station, Box 365, Delaware, O., and Iowa State University Press, Iowa State University, Ames, Ia.

again become exposed to any strain.

Symptoms:

Although much has been written on early foliage symptoms of Dutch elm disease, most descriptions either are merely repetitious of earlier writings, or are so superficial that they do not reflect different symptom patterns that may signify differences in degree and timing of infection. The so-called typical "first evidence of infection," as the single wilting branch "flagging" in sharp contrast to the remainder of the foliage, has often been used as a model to educate the public. In reality, however, the first evidence of the disease in a newly infected tree is probably not often visible to anyone but an investigator, who knows precisely when an inoculation was made. According to this writer, the first actual evidence of a new infection, when terminal crotches are inoculated, is a slight distortion or curling of leaves or leaf margins nearest and terminal from the point of introduction and development of the fungus (in research, the inoculation point). As such leaves bend inward on themselves, they show only a faint loss of green and their dull green undersides are exposed, giving them a dull, grey-green tint. This distortion and faint discoloration without other symptoms are rarely noticed, as they occur only in terminals, and are not readily discernible at this point except to a trained observer. Shortly thereafter, both yellowing and/or browning of foliage is often common, as well as premature abscission of leaves at all stages of infection, although many leaves do not abscise at all. But even such later symptoms may escape notice, since they occur only on branch ends distal to small twig crotches, often only high in the crown and not visible from the ground. In fact, it is not uncommon for many such infections to go unnoticed in the year of inoculation, because further symptoms may not occur that year at all. More often than not, in studies by the writer with various single strains of the causal fungus, this has happened with hundreds of inoculations. But this is an account only of terminal inoculations in twig crotches in wood of 2 and 3 years of age, designed specifically to simulate introduction of the fungus where the European elm bark beetle does it naturally.

When the fungus is introduced into larger stems supporting larger areas of foliage, similar symptoms as described above are more sudden, conspicuous and easily detected. This type of inoculation is often described as producing such severe wilting that the succulent terminal collapses, bends and becomes the so-called "shepherds crook," so often described as typical. In the view of the writer this symptom is neither common nor typical in the year of inoculation. When it does occur, it signifies that the fungus had time to build up a "massive head of wilting steam," and such a symptom can occur only while terminals are succulent and when active growth is in progress (i.e., early in the growing season). Thus,

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Dead trees such as these killed by Dutch elm disease demonstrate the tremendous problem and potential high cost of removal.

Dutch elm disease

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such a symptom is indicative to the writer that the fungus had been present in stems larger than twigs for some time, and is more suggestive of an infection that had occurred initially in the previous year, rather than one occurring in the present one. While the writer has been able to produce such "shepherds crooks" with massive inoculations of large stems, in eight years of successive inoculations he has never seen such a symptom from terminal inoculations.

Diagnosis:

The Dutch elm disease is so common over the landscape in the northeast, the midwest and adjacent parts of Canadian Provinces, that almost everyone who observes trees can and does accurately pinpoint much genuine Dutch elm disease. Accordingly, the point is often raised, that positive diagnosis is only of academic significance, because diseased elms from any origin are potentially dangerous as sources of bark beetles carrying the causal fungus. There is much truth to this, and positive diagnoses are only required under certain conditions. They are often required by state law for partial compensation, and must be continued by those concerned with reimbursement for tree removals, as well as by those seeking to avoid legal action, to placate a client, to satisfy ones curiosity, or to ascertain the cause in any case.

However, there is often unnecessary misunderstanding about the nature of diagnosis and the significance of results. In a few words, samples of an ailing tree suspected of the disease are treated in the laboratory to favor growth and development of the causal fungus. If present, the fungus most often grows out from the sample and may be identified either in pure culture, or by characteristic asexual structures. Since other fungi also cause similar field symptoms, and separation of the causal fungus from them is necessary, only trained persons can make diagnoses with confidence. Unfortunately, it is not often appreciated by amateur pathologists, who can learn easily to recognize characteristic features of the causal fungus, that there may be other fungi present that resemble *Ceratocystis ulmi*, and that there are atypical strains of the causal fungus as well. Every pathologist or mycologist who works with the causal fungus comes to recognize these variations, and at one time or another has had serious reservations and disagreements with colleagues as to what *is* it, and what *is not* it.

But aside from the identification itself, what is the significance of a positive test? We can say with confidence that the Dutch elm disease fungus has been isolated from a certain diseased tree. Usually this means the tree will die if the fungus has entered the main stem. With most such cases in the past, I never doubted that such trees should be destroyed. However, within the past few years I have seen more than one elm recover where the fungus had invaded the main stem, and for this reason, I have become more cautious about such a recommendation. It is possible for the causal fungus to be limited to the growth ring of one year, and if for any reason it is unable to cross into the growth ring of the following year, the tree may and often does recover on its own.

But what of a negative test result? Does it mean that Dutch elm disease is not present? Not necessarily, often another sample will be positive. In one situation where I had examined the tree in the field, I refused to be satisfied with three successive negative tests and obtained a positive only on the fourth one. However, if one isolates another known pathogen than the Dutch elm disease fungus, there is good reason to consider the test truly negative; but if no microorganism is obtained, and all field symptoms are indicative, another sample should be taken. It is my opinion that at least one kind of microorganism must be isolated from discolored elm wood, or the test is void.

Transmission:

Fungus transmission is the vital point in spread and distribution of the disease, and is presently the focal point of control. Until recently spread of the fungus was believed to occur almost exclusively through the activity of either one of the 2 elm bark beetles (the native, *Hylurgopinus rupifes* Eicch., and the European, *Scolytus multistriatus* Marsh.). Although transmission of the fungus in the vascular system by root graft between noninfected trees had been demonstrated over twenty years ago, and some transmission in this manner was known to occur, it was given little attention until quite recently. However, studies have shown that probability of root grafts among closely spaced urban elms may be substantially high, and spread of the fungus by this means must now be given careful consideration. Unfortunately it has not been given the attention that it deserves. Although the writer is unaware of any studies on the speed of graft formation and subsequent transmission, he has seen evidence to suggest that grafting may be possible within a 2-year period from the first contact between adjacent root systems. Also, field observations by others suggest that speed of fungus transmission by spore movement may be rapid. Unfortunately, there is as yet relatively little evidence on actual frequency of transmission of the causal agent and subsequent development of disease. A question is raised as to whether or not actual transmission occurs naturally by forces within the grafted system, by active growth or migration of the fungus, or by mechanical disruption occasioned by cutting one of the 2 grafted trees. Even before the frequency of root grafts had been explored by others, the writer had seen positive evidence in the field of root transmission of the fungus only following cutting of a known diseased tree. Of course grafted healthy trees may become diseased eventually without the cutting of attached diseased ones, but transmission is most probably accelerated by such cutting. This is the basis for the recommendation made over ten years ago by the writer and others, that immediately on confirmation of Dutch elm disease, action be taken to sever potential root connections between nearby healthy trees *before* (and not after) the diseased tree was removed. This point emphasizes the need for further studies on the nature and speed of spore movement across such grafts.

Returning to beetle transmission of the fungus, we know that some beetles may be carrying enormous loads of viable fungus spores, but we know also that some beetles may carry none at all. We know further that all spores are not viable, and that viability is to some degree a function of desiccation. It is also apparent that spores deposited in wounds made by feeding beetles do not always gain access to vessels, and it is suspected by the writer on the basis of studies, that many viable spores either do not travel far in the vascular system or are inactivated at the infection point by antagonism from other microorganisms.

Another aspect of beetle transmission that requires comment is the timing of beetle emergence and activity. Depending on climatic conditions at various latitudes, beetle emergence usually begins from late April to mid-May. At least two broods of either beetle are possible, but the European species may have two and one half or even three. Much emphasis has been placed on peak emergence of broods, so that some people actually believe that these are the only significant times that beetles are available for transmission. However, beetles are reported to be emerging and active over the entire growing season from early May at least to early frost. Thus the emphasis on peak emergence may be misleading, unless interpreted accurately. The first peak emergence around mid-June is unusally significant in that this is the period of maximum available fungus-carrying beetles precisely in the middle of the most susceptible period for the elms with respect to new vessel development. Although a second brood of beetles may occur in August, this one has much less significance because the elms are beyond maximum susceptibility. However, this does not mean that infection cannot occur at this time; it only means that it is less probable. The writer has had no difficulty inoculating elm in August, with positive disease development. However, such late season infections are often likely to be walled off by the tree and remain isolated. When this occurs, they are in effect inactivated.

Control:

Of all the different aspects of Dutch elm disease on which we have definite information for guidance, control seems to me to be the least understood. As a general concept the word implies more than it really means for Dutch elm disease. In this sense it is an unfortunate choice of a term, that may not only enlist public support for a worthy cause, but may lead also to bitter disillusion and total lack of support when a misunderstanding becomes clear. Among those who try to understand the disease in all of its immense biological complexity, control means only a limitation of disease among a limited population of elms within a limited area, given careful and systematic application of tested procedures. But few public officials understand the complexities involved and in practice are allowed to believe that they have done or are doing the proper thing to insure disease control.

As currently understood by the writer, there are only 4 control measures accepted generally as effective both in theory and actual test. Two of these are indirect and are designed generally to reduce the probability of fungus-carrying elm bark beetles in or nearby healthy trees to be protected. The most significant of these is sanitation, which involves the elimination not only of all diseased and/or beetle breeding elm wood, but of all such wood potentially hazardous for such breeding as well. In effect this means elm wood diseased or weakened from any cause. The significance of this measure is the need for wholesale cleaning of weakened elm wood by constant cutting, trimming and pruning generally. This in itself when properly done is a most formidable chore, and for this reason can only be done effectively for limited numbers of highly valued elms. The other indirect method is general maintenance involving watering and fertilizing when and where essential to prevent or minimize weakening of branches sufficient to allow invasion by beetles. In a well managed shade tree population, these practices should be routine and systematic, but here, too, numbers of trees so treated will be limited by time and resources.

The remaining 2 methods of control are direct. Spraying is designed to coat every square millimeter of bark annually with a chemical that will kill every visiting beetle, and by so doing prevent infection completely. The theory is sound, experimental results have been proven, but in practice results have often been disappointing. Economics, insecticidal controversy, cluttered streets, parked cars, logistics, weather, time available labor, ignorance, irresponsibility of labor, *et al. ad infinitum* combined, appear to preclude the precision and meticulous care necessary for effective spraying of large numbers of trees with due attention to all required conditions. Adequate manpower and resources within time limits alone, are rarely available for the assignment. The result seems to be a diffusion of effort and spray over a widely scattered elm population, too large for the resources used. If spraying for Dutch elm disease is to retain or regain confidence in the eyes of the public, it is my view that it must be more limited and more carefully applied.

Unfortunately, spraying has been used too often as a control measure at the expense of both sanitation and root graft treatment. It has often been shown to be ineffective for this reason alone. Since good sanitation complements spraying, in its near or total absence, even moderately good spraying may be relatively ineffective. Under extremely intensive beetle pressure it is difficult to see how even excellent spraying can be perfect, and many contaminated beetles, with viable spores should be expected to survive for successful inoculation.

Concerning root grafts, it should be obvious that overhead spraying will be wasted, if the fungus is moving underground. And yet it is my understanding that treatment for root graft is widely neglected, and in some areas is not used. As with all control measures economics plays a major role here. But again this emphasizes a point that keeps recurring in control, i.e. the inadequacy of money, manpower, machines and time to exercise all proper control measures.

The question of pruning a single infected limb from an otherwise healthy tree as a control measure, has been raised by many with hopeful anticipation. As with many pathologists I have seen severely wilted limbs pruned and complete recovery of the tree, especially where the fungus apparently had been removed completely. On the other hand, I have seen scores of trees die following pruning. Saving a tree by pruning still seems to be a relatively rare phenomenon in spite of many successful cases. In one situation, a large severely wilted branch was pruned but discoloration of the vascular system was evident in the main stem. The owner was informed that the tree would die, but the tree was not removed, so that its demise could be studied. Two years later the tree still lives in a perfectly healthy condition. However, such cases are rare. In most cases, by the time a tree wilts conspicuously enough to be sampled, the chances are good that the fungus is by then deep in the tree, has been able to cross from one growth ring to another, and cannot be removed by pruning. Thus, there is little likelihood that such a tree will survive. However, there is much here that we do not know. I have altered my own views here radically, and no longer condemn a desirable elm to the axe until I am certain the disease will not cease, either with man's aid or none at all. A few words of warning, however, known diseased trees cannot be left unattended. At the very least they must be pruned carefully. Also, every tree owner who elects to try to save a diseased tree must be informed fully, so that the practicing arborist will not assume an unwarranted responsibility that might result in embarassment or even legal action.

Conclusion:

Over the past sixteen years the writer has observed with interest the operation of many Dutch elm disease programs. In spite of some noteworthy successes in control over a long period of time, even the best programs sustain substantial losses in certain years. Unfortunately, more programs than not have failed disastrously. This has led the writer to ask himself if it is really possible to control Dutch elm disease satisfactorily on a community-wide basis. In evaluating each of the control methods necessary for a successful program, it is now my view that the community will indeed be rare that will actually support in practice the kind of complete and thorough control program necessary according to theory. If there is any community in the U.S. that, over a period of years, has practiced complete, thorough and timely spraying, complete thorough and timely root graft treatment where needed, and complete and timely maintenance under conditions of adverse environmental stress, such as drouth, I have yet to hear of a detailed report. In practice most control programs appear to involve at least some of these measures, some use even one or 2 thoroughly, most use but one or 2 on a piece meal basis, but how many, if any, use all as prescribed?

In my experience, much sanitation is inadequate or too late, because of the sheer enormity of the wood to eradicate. Most spraying is inadequate in deposition or untimely in application. Root graft treatment seems to be generally ignored, except for certain areas, and complete maintenance with water and fertilzer when and where needed for an entire population of elms seems prohibitive economically and physically.

In effect, where community-wide control of the disease is in

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Aquatic Weed Control

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program which would utilize both federal and state facilities and financing, state supervision of control on a statewide basis, and the authority to perform field operations in any area not covered by a specific local program and where control is necessary to safeguard the state as a whole.

Frank Wilson, director of the Polk County Mosquito Control District, Bartow, Fla., was elected as new president. Blackburn, the retiring president was named vice-president, and Paul R. Cohee, Hercules, Inc., Orlando, Fla., continued as secretary-treasurer. New directors elected for the Society were: Stan Abramson, Southern Mill Creek, Tampa, Fla., John W. Woods, Florida Fish and Game Commission, Tallahassee, Fla.; and R. P. Blakeley, director of Old Plantation Farms, Plantation, Fla. James D. Gorman, Tampa, Fla., is the retiring vice-president, and retiring directors are Fred W. John, Belle Glade, Fla., and Dr. Fred W. Zurburg, Lafayette, La. Dr. Lyle Weldon, ARS, USDA, Fort Lauderdale, Fla., continues as editor and will publish proceedings of the entire annual meeting. These are automatically mailed to Society members and are available on a purchase basis for non-members.

Members voted to stage their 1969 annual meeting during June at the Holiday Inn, Palm Beach, Fla.

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Dutch Elm Disease

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trouble, it is not for failure or proven control methods, but instead of failure to apply all measures needed as prescribed. Failure to do so seems to result from a combination of factors involving both human and physical relationships. I believe that current effort, time and money now diffused over the community with but limited success, can best be used on limited numbers of highly valued trees. Many trees now pruned and sprayed are not worthy of this attention, because they are not only potentially hazardous for disease spread, but are also without aesthetic attraction. Such trees should be destroyed rather than protected. A reassessment of priorities is clearly necessary for control programs to be more successful. Priorities should be shifted from selectivity of control methods, to value and location of selected, desirable elms to be protected. *All* control measures should be used only on *limited* numbers of elms, and current selectivity of only one or two control measures for all elms should be abandoned as undesirable and hopeless. Ultimately, the disease can be expected to reduce all urban elm populations to fewer numbers of elms that may be protected with complete care, but if current practice continues some of the most beautiful and desirable trees will have gone and many grotesque ones may live on. The message here is to insure complete protection to those trees for which shade tree care has a purpose, anything less will ultimately jeopardize the integrity of arboriculture.

New Jersey Now Growing 3000 Acres of Sod Yearly

The New Jersey Department of Agriculture reports that its state sod growers now have more than 3000 acres of "instant lawn" under production.

Of the Garden State's 31 sod farms, 15 are located in central counties, 9 in northern, and 7 in southern.

Sod production got underway in the state about 30 years ago; since that time, local and out-of-state markets have expanded to include industry, businesses, athletic fields, golf courses, landscapers, garden centers and, of course, private homeowners.