Proceedings of the Iowa Academy of Science

Volume 61 | Annual Issue Article 16

1964

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This paper is intended to review critically and briefly the research being conducted on the oak wilt disease caused by the fungus, *Endoconidiophora fagacearum* Bretz. Although conditions and work in Iowa will be referred to constantly throughout the course of this review, much of the material will be applicable to the other oak wilt areas of this country.

Many old time woodsmen maintain the presence in oak stands at the turn of the century of what we now call oak wilt; but the first definite occurrence of this disease is indicated by photographs taken in the McGregor area in 1932 (Dietz and Young 1948). By 1943, oak wilt became a serious threat to the oak areas in northeastern Iowa and in the state parks of Dolliver and Pilot Knob. Since that time, oak wilt has been found to be present throughout the oak range in Iowa. At this writing, nationally, this disease is considered the most serious forest tree disease in the eastern half of the United States.

Research was begun on this disease in the early 1940's by workers in Iowa and Wisconsin with the support of their respective conservation commissions. As the disease became known in other areas of the country, more research programs developed. In 1950 the National Oak Wilt Research Committee, a group of men in the hardwood industry, was formed and has financially supported oak wilt research at six research institutions since that date. The federal government as well as other state and local agencies have also actively supported oak wilt research in the oak regions of this country. At this time, eleven states are active in research on this disease.

Research on oak wilt has been grouped within four major topics. These include the determination of the host and geographical distribution of the disease, its causal agent and the behavior of this organism in nature and artificial culture, the transmission of this disease, and its control under given conditions.

[1]Journal Paper No. J-2515 of the Iowa Agricultural Experiment Station, Ames Iowa. Project No. 1047. Research sponsored jointly by the National Oak Wilt Research Committee, the Iowa State Conservation Commission, and the United States Department of Agriculture.

Of all the species of oak that have been tested for their susceptibility to oak wilt, none have shown any resistance to it (Barrett 1947, Bretz 1952a, Hoffman 1953b). Both the American and Chinese chestnut (Bretz 1951, Bretz and Long 1950, Ernst and Bretz 1953) have also shown susceptibility to the disease. Workers in Ohio have reported the ability to re-isolate the oak wilt fungus near the original inoculation site from seven other tree species three months after they had inoculated the trees. Although these trees did not contract the disease, the fungus was able to maintain itself in the respective wood species.

Within the oak genus, the reaction to the disease is different. Oak wilt will kill trees of the red oak group within one growing season. Those of the white oak group will have the disease over a period of years with a few branches dying each year. The explanation for this difference within the oaks has dealt with their lateral translocation rates. By means of dyes, (Beckman and Kuntz 1951) the trees of the white oak group have been shown normally to translocate material very slowly in the lateral direction, while in those of the red oak group, the opposite is true. Therefore, translocation of the fungus in diseased trees seems to be one factor involved in these different diseased reactions. More research is needed on this basic problem for complete clarification.

The geographical distribution of oak wilt has been determined by aerial and ground surveys. Under the leadership of the Division of Forest Pathology of the United States Department of Agriculture survey techniques, particularly aerial, have been greatly improved the past few years. The need for surveys for oak wilt and pole blight of western white pine was the stimulus for research in this field (Fowler 1952). At the present time, oak wilt has been found in eighteen states east of the Rocky Mountains (Fowler 1953).

The fungus causing oak wilt was originally described by Henry (1944). It was given the imperfect name, *Charala quercina.* In 1951, Bretz found the perithecial stage of the fungus in artificial culture. The perfect stage is also found commonly in nature (Stessel and Zuckerman 1953). It was then given the name, *Endoconidiophora fagacearum* (Bretz 1952b). The fungus was shown to be a heterothallic hermaphrodite with the two compatibility groups being designated as A and B (Hepting *et al.* 1952). A white mutant was found in Minnesota and was shown to be an unisexual male (Barnett 1953). Other white mutants have also been found but they have behaved normally (Barnett *et al.* 1954).

The natural fruiting structure of the fungus was not found until 1951 (Curl *et al.* 1953). It consists of a mycelial mat and a center gelatinous pad. Both endoconidia and perithecia are found on the mat. The pad consists mainly of large, ovoid to spheroidal, brown-walled cells which are arranged in a pseudoparenchymatous manner. These fruiting structures are found between the bark and wood of oak wilt killed trees. Mats and pads are formed both on the outer wood and the inner bark in a mirror image arrangement. The pressure exerted by the cushion-like pads is enough to crack the bark, thus exposing the fruiting bodies to the external environment. These structures exude a cidery like odor which attracts birds, rodents and insects.

At this point, it would be best to outline the life cycle of the oak wilt fungus in nature. A diseased tree of the red oak group will be used as an example. In this way some of the basic problems we have yet to solve can be demonstrated in an orderly fashion.

After the fungus has been inoculated into the tree, on the average, about three weeks elapse before visible external symptoms appear in the crown of the tree. What happens during this three-week period is a basic problem which is found in all vascular diseases. In oak wilt, tyloses and gums develop in the vessels a few days before visible wilting. (Struckmeyer *et al.* 1954). It must be pointed out also that the fungus can be isolated from all the wilting parts of the crown. Therefore, the invading pathogen has been able to reproduce itself and either grow or be carried throughout the vascular system of the tree.

Another theory for the direct cause of wilting has been the one involving a toxin which is produced by the pathogen. Experiments involving artificial fungous culture extracts and oak cuttings or seedlings (Hoffman 1951b) suggest this theory but it also implies, basically, too simple an answer. There is no reason not to believe that both water deficiency and a toxic principle produced by the invading fungus are present. Could not the fungus incite the formation of tyloses and gums by such a metabolic product? Such is a basic problem whose solution needs to be approached before other phases of oak wilt knowledge become clear.

During the period approximately two to ten months after the tree has exhibited symptoms, the mats and pads may appear. Although the factors needed for their production are not known yet, recent observations point to wood moisture and air temperature as two possible factors. A recently killed tree may produce these structures the same fall of the year it became diseased and/or the

following spring. During the development of the mats, the endoconidia are first to appear. They are usually followed by perithecial development. Research which has been recently reported indicates that only one compatibility type of the fungus is present in the tree at one time (Boyce and Garren 1953, Fergus 1953). Therefore, the mat would represent only one type and would have to be spermatized by spores from the other type being produced on another killed tree. These spores could be carried by insects (Leach *et al.* 1952), other animals or the wind since perithecia have not been found on unexposed mats. Although experimental evidence using insects suggests this premise, the apparent "blooming" of perithecia that has appeared in Iowa suggests the possibility of other methods. Perhaps the sexual phenomenon in this organism has been simplified too much. Further study is certainly indicated along these basic lines.

Another interesting problem involving the mats and pads is the formation of the gelatinous pad. It is an unique structure among the fungi. Although it is produced very commonly in nature very few structures resembling it are found in artificial culture. Academically, at least, a morphologic study of its formation would prove quite stimulating.

After the mats and pads are produced and later deteriorate to black carbonaceous masses, little is known about the presence of the fungus in the wood of the tree. The ignorance of these facts has produced many problems in the oak industry. At least one foreign country, Portugal, has imposed quarantine regulations on shipments of oak lumber from oak wilt areas of the United States. Recent experiments indicate that properly dried and seasoned oak lumber is free of the oak wilt fungus (Spilker 1953). Other experiments with the fungus alone also suggest this. The point, between a killed tree with mats and kiln-dried lumber, where the oak wilt fungus is still alive has not been determined. The solution to this problem is of the highest priority this year.

Oak wilt is transmitted in two ways; by root grafts between healthy and diseased trees and by some type of overland vector. The pattern of disease spread within areas of infection and the translocation of dyes and poisons between root grafted oaks clearly suggests root grafts as a means of oak wilt transmission (Kuntz and Riker 1950 a, b).

Overland transmission studies have been of first priority the past two years. Last year much was accomplished when experimental evidence was placed against some members of the animal kingdom

as possible mechanical vectors of the fungus. A few insects (Dorsey *et al.* 1953, Norris 1953, Griswold and Bart 1953) principally those of the family Nitidulidae were shown able to transmit the disease experimentally. Later in the year, the Illinois group was able to transmit the disease experimentally by the use of squirrels (Norris 1954).

Certain conditions appear necessary for successful overland transmission in the majority of cases (Norris 1954). Firstly, fungus inoculum has to be present in nature. Thus, mats and pads have to be present and sporulating. Secondly, the mode of entrance of the pathogen into the healthy tree is through a fresh wound. The wound may either be made by the vector or by some other agent such as wind. The same group of insects are found on such wounds as on the mats and pads. Thirdly, the tree must be in its annual, maximum growth period. Lastly, the sexual spores, ascospores, must be present on the mats. These last two conditions are open to much debate but successful insect transmission work in Iowa has occurred only in May and June, the only time these two conditions coincide. Much further study needs to be done on transmission. A few problems which are being undertaken in Iowa this year are the relation of the age of the wound to transmission, do these Nitidulid bettles travel from fungus fruiting structures to wounds commonly and are ascospores and early wood formation important or coincidental in transmission.

Birds have also been suspected as carriers of the oak wilt fungus. To date, though, this fungus has not been found on 306 birds examined (Tiffany *et al.* 1954).

Control studies have consisted mainly of sanitation, eradication and chemotherapy. Both means of spread of the disease suggest sanitation and eradication as control methods. Certainly, the removal and the destruction of the diseased tree would destroy the fungus inoculum which is needed for future spread. But, in the case of root graft transmission, the fungus may have already been transmitted to neighboring, healthy trees. Therefore, means of breaking these root connections have been studied. This can be done by cutting the roots with a blade, cutting the trees down which are within fifty feet of the diseased tree or poisoning this ring of healthy trees with a silvicide.

The latter two show great promise under many conditions (Bragonier 1953). Much silvicide work has been done in Iowa which shows, at the present, a 10% solution of 2,4,5-T in kerosene applied to basal frills at dormant and budbreak conditions as the

best. Two big questions remain in silvicide studies. Are the roots being killed along with the tops? If they are not, can the fungus be transmitted through them? Indications are that the roots are alive after the tops have been killed, but it is not known for how long.

Besides eradication of the diseased tree, spraying and better care of healthy trees have been suggested for control of overland spread. The protection of healthy trees against wounds and the spraying of such trees against insects which might be attracted to such wounds seems reasonable from what knowledge is available at the present. No studies on this type of control have been undertaken.

Another type of control practice has been indicated where root grafts appear not to be very common. This is to keep the diseased-killed trees mat and pad free and to discourage insects from populating these trees. Although, little is known on the conditions needed for mat and pad formation, studies (Boyce 1954) have begun on the use of a combination insecticide-wood preservative spray on such trees.

The other control studies in oak wilt research have been on chemotherapy (Hoffman 1951a, 1952). This constitutes the application to a plant of a chemical which is absorbed by the plant and acts against the pathogen or its products within the plant. The chemical may either act as an eradicant or a protectant. It may be applied as a foliage spray or to the soil for root absorption. Although much empirical testing has been done to date, no chemical shows any promise on large trees. Optimism has been expressed over some results using seedling oaks (Hoffman 1953a), but the reaction of small trees to this disease is too variable for clear-cut results. This writer believes that until the fundamental questions concerning the reaction between the host and the pathogen are answered the chances for success in the chemotherapy of this disease are very small. This is a good illustration of the need a practical problem has for a firm foundation of basic research knowledge.

The previous paragraphs have attempted to present a clear, concise picture of oak wilt research as it stands today. This review has shown the need for the answering of basic questions in order to fill the gaps in our present knowledge of the oak wilt disease. I hope it has challenged you as well as the writer toward these ends.

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