THE OAK DECLINE COMPLEX

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Live oak decline has captured much attention in Texas in recent years. Even more recently the syndrome has become more captivating due to efforts to relate it to oak wilt. During this same period it has been observed in other parts of the United States, primarily in California, Florida, and Illinois. There has been concern expressed by oak enthusiasts that the disease may compete with Dutch elm disease for top honors among tree killers.

The first occurrence of oak decline in the United States was reported from Pennsylvania by Ingram (12). This was followed over a decade later in New York, Connecticut, and New Jersey (1, 7, 37, 38). The first expression of concern came from Texas (26, 27). Later concern came from Virginia (2) and more observations from Texas (5, 8, 9, 21). By 1960 oak decline was becoming a national problem, not limited to regional environments or species classes.

Nutrient deficiencies have been considered to be predisposing factors (17, 18). Oates has presented evidence that fertilization aids recovery of oaks from decline. van Arsdel (33) tried to create some order out of the symptomology chaos by using diagnosis of nutrient deficiencies through leaf symptoms as a diagnostic method for determining oak decline. Some of the best descriptions of oak decline symptoms have been given by Dunlap and Harrison (5), Feder et al (6), Lewis and Oliveria (17) and van Arsdel (33).

This disease seems to be most prevalent and severe under drought conditions or shortly thereafter. Many investigators through the years (10, 12, 17, 19, 20, 25, 27, 35, 38) have observed a close relation between oak decline and low moisture availability. Lewis and Oliveria (17) have indicated that symptoms of drought stress can be confused with those of oak decline. Taubenhaus (26, 27), and van Arsdel and Halliwell (35) considered drought stress to be a precursor to oak decline. This was supported by Rhodes and Tainter (19) reporting damaged cells

in growth rings the year following drought in declining white oaks. That drought predisposes oaks to decline appears well established.

Temperature relations remain unclear. Several investigators (15, 17, 19, 25) have observed that temperature has a direct impact on the development of oak decline. This may be due, in part, to the temperature requirement of the pathogen. Lewis and Oliveria (17) observed that Botryodiplodia theobromae causes decline symptoms at 32°C, but not at 26°C. Growth of Hypoxvlon atropunctatum is also enhanced by high temperatures (25). Lewis (15), and Lewis and Oliveria (17), reported that Ceratocystis fagacearum can be isolated from declining trees in the spring and fall, but only imperfect forms of this fungus associated with decline can be isolated in the summer in Texas. This raises an interesting aspect concerning the relationship of C. fagacearum to oak decline.

Of greatest concern, however, have been the pathogens related to the fate of the live oaks in Texas. Numerous investigators (4, 5, 8, 11, 17, 27, 29, 36) have isolated several vascular invaders from trees showing symptoms of oak decline. At least 13 genera have geen associated with the oak decline complex from different parts the United States: Botrydiplodia, Cephalosporium, Diplodia, Dothiorella, Endothia, Fusarium, Hyalodendron, Hypoxylon, Nectria, Pestolotia, Phialophora, Verticillium, and Verticicladiella, occurring separately or in combination. Some of these organisms may be imperfect forms of C. fagacearum, also included in this myriad of organisms associated with oak decline (15).

Botryodiplodia theobromea was recognized by Schmidt and Fergus (21) as a branch canker and dieback pathogen of *Quercus prinus*. Lewis (16) later indicated that this organism was associated with dieback of *Q. virginiana* in Texas, possibly as a part of the decline complex. It also has been observed associated with decline in California oaks

(31).

Since 1964, when Horne and Halliwell (11) first attributed the cause of oak decline in Texas to *Cephalosporium diospyri*, Texas investigations have been directed toward this organism (8, 13, 15, 32, 36). After isolating *C. diospyri* from oaks in Illinois, Florida, and California, Thomas (29) has attributed this organism to be one of several in the general oak decline complex throughout the continental United States.

Diplodia quercina has long been considered a weak branch canker pathogen in oaks. Rolan (20) suggested it as a possible cause for oak decline in France. Hecht-Poinar et al (10) implicated this pathogen as the principal cause of oak decline in California. In their investigations repeated isolations of D. quercina were made from branches and petioles of affected trees, no isolations were reported from roots or root crowns. Inoculations with D. quercina into branches confirmed pathogenicity in such structures only.

Dothiorella quercina was the first reported as a cause of decline, expressed as a twig blight (12). It has long been considered by forest pathologists as a weak twig canker pathogen. Thomas (28, 29) has found this organism commonly associated with oak decline in California following root infection as well as branch infection. Lewis and Oliveria (17) reported *Endothia* spp. from advanced dieback in Texas live oaks and attributed this as a possible pathogen in the decline complex.

Fusarium oxysporum has been associated with wilt-decline of oaks in California (29), and apparently enters into the decline complex. It has been isolated from oaks in Illinois, Missouri, Florida, Texas, Oregon, and California (31).

Hyalodendron sp. was reported by Halliwell (8) as a possible entity in the oak decline complex in Texas. Hypoxylon sp. also was implicated by Lewis and Oliveria (17), despite the fact that Hypoxylon symptomology differs widely from that normally recognized as oak decline (30).

Pestalotia macrospora has been isolated from declining oaks in Florida, Texas, and California, and is included among the pathogens associated with oak decline (29). This fungus has not been reported by other investigators. Thomas (29), and Lewis and Oliveria (17) have reported *Phialophora* sp. as a possible pathogen in the complex.

Thomas (29) has obtained it from live oaks in Florida, Texas, and California, and considers it one of the more difficult pathogens in the complex to suppress.

Verticillium albo-atrum is not considered a pathogen of oaks by many investigators, but it has been associated with oak decline in Illinois (22) and California (29). Thomas (29) includes this, together with Verticicladiella sp., as part of the oak decline complex.

There is some discrepancy concerning the amount of time required for oak decline to kill a tree. Halliwell (9) indicated that death was a matter of a few weeks after infection under field conditions. On the other hand, van Arsdel et al (34 believed that more than 10 years are required for the demise of a tree infected with oak decline. This is in line with Crowley's (3) report that 10-20 years are required for decline-infected trees to die. The rate of decline seems to be regulated as much by environmental conditions as by the pathogens. Clearly there is need for additional investigation concerning the multitude of factors affecting oak decline.

The purpose of this investigation is to demonstrate the pathogenicity of some of the fungi associated with oak decline, to delineate some of the symptoms resulting therefrom, and to report some environmental conditions related to the development of the disease.

Materials and Methods

The fungi used in this investigation included isolates obtained from field samples taken from coast live oaks (*Quercus agrifolia*), valley oaks (*Q. lobata*), and California black oaks (*Q. kelloggii*) exhibiting the various symptoms ascribed to oak decline in California: marginal chlorosis, foliar yellowing on individual branches, marginal necrosis of leaves, rapid wilt, slow wilt, shortening of internodes, and complete dieback of individual branches. All source trees had characteristic brown, reddish brown, or metallic gray vascular discoloration in branches, stems, and roots. All had demonstrated vascular resistance on at least one side of the tree by a Shigometer vascular probe.

Isolates included Verticillium albo-atrum, Pestolotia macrotricha, Dothiorella guercina,

Cephalosporium diospyri, Phialophora sp., and Fusarium oxysporum. Trees challenged by inoculations with these fungi were 18 Q. lobata and 18 Q. agrifolia 3-0 nursery stock grown in one-gallon cans. From potato dextrose agar slant cultures of each test organism 1 cm sq. blocks were transferred to bark wedges made approximately 2 cm above the root crown of three saplings of each tree species in early October, 1981. The wedges were sealed to the stem with sterile medical tape and wrapped with Saran to keep them moist. Triplicate randomized blocks of each combination of tree and fungus were used. There were three uninoculated controls for each tree species.

The trees, kept under natural conditions during the winter, were exposed to a historical surplus of rain until the following April. At no time after inoculation were the trees under moisture stress until early in April. At that time the containers were moved under cover and watered thoroughly every 14 days. Soil moisture was measured with an Aquaprobe daily from April 1 to May 11. Average daily temperatures were recorded. The trial was terminated on May 11, 183 days after inoculation, when final symptoms were recorded.

Isolations after 183 days were made from (a) root crown, (b) 1 cm above the inoculation site, and (c) the branch crown from each sample, including uninoculated control trees. Isolates were cultured on standard potato dextrose agar in 30 cm Petri plates. The plates were incubated 14 days at 25°C.

Experimental Results

Prior to 148 days after inoculation the soil moisture had remained at 98%-100% due to recurring rainfall, and the average maximum air temperature was less than 10°C. After the test trees had been put under cover, there was some variation in soil moisture. The weekly average soil moisture dropped to 60% field capacity, and the maximum air temperature rose above 10°C.

Three days before the first symptoms were evident (159 days) soil moisture averages were between 47%-55%, and maximum air temperatures were above 18°C (Fig. 1). The decrease of soil moisture below 60% favored symptom development, reflecting the moisture

stress level. Beyond the date of first symptom development average weekly soil moisture dropped to 35%, and never rose above 49% during the duration of the test. During this period average maximum air temperatures remained consistently above 18°C. Maximinum decline followed a maximum 28°C temperature 176 days after inoculation.

The test trees remained asymptomatic to 159 days. The first symptom determinations were made at 162 days, the trees inoculated with *D. quercina* having the most prominent symptoms, especially in *Q. lobata* (Fig. 2).

After 169 days trees inoculated with D. quer-

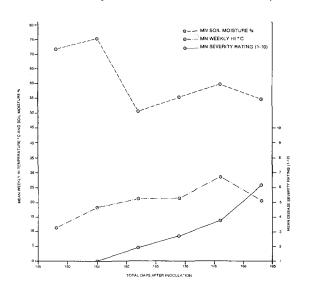


Figure 1. Association of soil moisture and air temperature to oak decline symptom development.

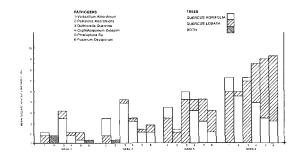


Figure 2. Oak decline symptom development caused by six pathogens in Quercus agrifolia and Quercus lobata.

cina had the most prominent symptoms, being most severe in *Q. lobata*. Symptoms in trees inoculated with *V. albo-atrum* or *C. diospyri* had less severe symptoms. They were more severe in *Q. agrifolia* inoculated with *V. albo-atrum* than in *Q. lobata*, while the reverse was true in trees inoculated with *C. diospyri*.

Within 176 days the most severe symptoms were evident in trees inoculated with *V. alboatrum*, *D. quercina*, or *C. diospyri*. This was most evident in *Q. agrifolia* inoculated with *V. alboatrum* or *D. quercina*, while *Q. lobata* had the most severe symptoms following inoculation with *C. diospyri*.

At the termination of the trial after 183 days the most severe symptoms were evident in *Q. lobata* inoculated with *C. diospyri* (Fig. 3), *Phialophora* sp. (Fig. 4) or *F. oxysporum* (Fig. 5); symptoms were less severe in *Q. agrifolia*. Less severe symptoms were evident in *Q. agrifolia* inoculated with *D. quercina* (Fig. 6) or *V. albo-atrum* (Fig. 7); *Q. lobata* were even less affected. Symptoms were only moderate in *Q. lobata* inoculated with *Pestalotia* (Fig. 8), and even less severe in *Q. agrifolia*.

All trees but the uninoculated controls expressed some symptoms. Severity of symptom expression was significant in all inoculated *Q. lobata* (Table 2). Significantly severe symptoms in *Q. agrifolia* were caused only by *V. albo-atrum*, *P. macrotricha*, or *D. quercina* under conditions of the trial.

Wilt symptoms were evident in all *Q. lobata* inoculated 183 days prior with any of the test organisms (Table 1). Wilt also was evident in all *Q. agrifolia* except those inoculated with *F. oxysporum*. Rapid wilt, expressed as a greenishgray discoloration of the leaf blade, was prevalent in those *Q. agrifolia* inoculated with *Phialophora* sp.

Stunting, as determined by obvious shortening of internodes, generally was apparent in *Q. lobata* inoculated with *D. quercina*, *C. diospyri*, *Phialophora* sp., or *F. oxysporum*. It was not evident in *Q. lobata* inoculated with *V. albo-atrum* or *P. macrotricha*.

Minor chlorosis was general in all *Q. agrifolia* inoculated with any of the test organisms. *C. diospyri.* however, failed to cause chlorosis in *Q.*

lobata. In the Q. lobata inoculated with P. macrotricha or D. quercina, however, chlorosis was evident in only the basal leaves as a senescence syndrome.

Dieback was evident only in those *Q. agrifolia* inoculated with *D. quercina* or *C. diospyri*. Terminal dieback was general, however, in all *Q. lobata* inoculated with any of the test organisms.

No symptoms were evident in any of the uninoculated control trees.

Isolations made on May 11, 183 days after inoculations, were positive in all cases except from the control plants. The pathogens were obtained from the upper crowns, inoculation sites, and root crowns. These results demonstrated the capacity of each of the test organisms to cause decline in *Q. agrifolia* or *Q. lobata*, and that infection by each organism could be systemic. Symptoms varied according to tree species and pathogen.

Discussion

These investigations demonstrated clearly that oak decline may be expressed by *Q. agrifolia* and *Q. lobata* following infection by any of several different fungi. In this case positive inoculations and isolations were obtained with *Verticillium alboatrum*, *Pestolotia macrotricha*, *Dothiorella quercina*, *Cephalosporium diospyri*, *Phialophora* sp., and *Fusarium oxysporum*. Each was able to infect systemically all portions of the inoculated plants. Symptoms varied according to tree species and pathogens.

Since each organism used in this investigation was isolated from all parts of the inoculated trees beyond the inoculation site, it would follow that each was systemic in pathogenicity, although some of these organisms have been considered localized in the past (i.e., Pestalotia, Dothiorella). It may be that some of the other organisms reported in the past as associated with oak decline, such as Botyrodiplodia, Diplodia. Hyalodendron, Hypoxylon, Nectria, and Verticicladiella may be in complex with some of the pathogens included in this investigation, and that they may be systemic under certain conditions. This spectrum of oak decline pathogens requires further investigation. The relation of some of these organisms as imperfect forms of C. fagacearum emphasizes an even greater need for additional in-

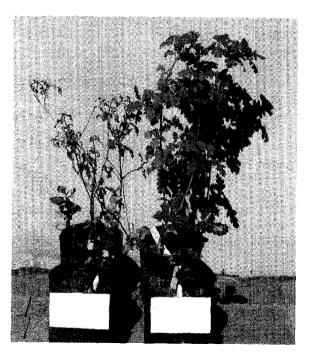


Figure 3. Quercus lobata and Q. agrifolia on left inoculated with Cephalosporium diospyri (182 days).

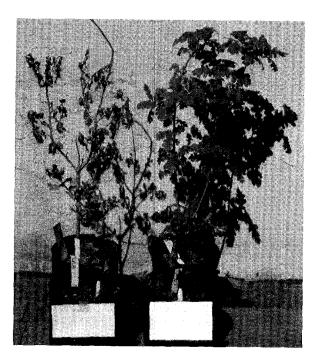


Figure 4. Quercus lobata and Q. agrifolia on left inoculated with Phialophora sp. (182 days).



Figure 5. Quercus lobata and Q. agrifolia on left inoculated with Fusarium oxysporum (182 days).

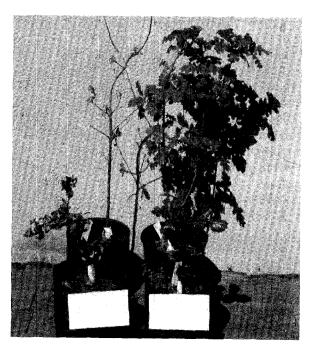


Figure 6. Quercus lobata and Q. agrifolia on left inoculated with Dothiorella quercina (182 days).



Figure 7. Quercus lobata and Q. agrifolia on left inoculated with Verticillium albo-atrum (182 days).

vestigation. If mycologists could clarify further the relation of these organisms to *C. fagacearum*, this complex may be more simple than it is at present.

Symptoms varied according to tree and pathogen species. The differences often were discreet, while at the other times significant. It would be unwise for the field diagnostician to attempt distinguishing organisms involved in the oak decline complex on the basis of symptoms alone. More than one pathogen may be involved in the expression of decline syndromes. It thus is imperative that isolation of the pathogen or pathogens involved be performed before the final diagnosis.

Nutrient and moisture stress symptoms can be confused with those expressed by infections by decline-related pathogens. Symptoms were expressed following successive periods of drought when weekly soil moisture levels dropped below 60% field capacity, development being inversely proportional to soil moisture. This confirms earlier reports that drought stress predisposes oaks to decline expression (10, 15, 19, 25, 27, 35, 38).

The timing of this investigation during California winter conditions failed to clarify the role of temperature in affecting symptom development.



Figure 8. Quercus lobata and Q. agrifolia on left inoculated with Pestalotia macrotricha (182 days).

There was an obvious increase in maximum air temperature over 13°C prior to symptom expression, the maximum symptom development occurring 5-8 days after a 28°C maximum. There is a direct relation between decline expression and air temperature, which is a logical expression of transpirational stress. Under the conditions of these investigations it was not possible to determine what temperature stimulated symptom expression or initiated decline. Nor was it clear how long after the occurrence of that initiating temperature the first symptoms were expressed. Maximum air temperatures over 13°C were followed, however, by initial symptoms within 10 days.

"Oak decline" is a name for a disease based on a complex of symptoms, not actually on the pathogens involved. This investigation has demonstrated that any one pathogen can cause "decline" syndromes of various and interposed types. It is clear that ascribing a particular organism to oak decline in any form is hazardous. Any of several pathogens can cause similar syndromes. "Oak decline" then becomes a condition caused by any one or a combination of pathogens, which can become lethal to oaks.

Table 1. Development of symptoms in two Quercus species 7 months after inoculation with one of six fungi associated with oak decline.

Pathogen	Wilt		Stunt		Chlorosis		Dieback	
	agrifolia	lobata	agrifolia	lobata	agrifolia	lobata	agrifolia	lobata
Verticillium	+	+	+		+	+		+
Pestalotia	+	+	+		+	+1	_	+
Dothiorella	+	+	+	+	+	_	+	+
Cephalosporium	+	+	+	+	+	+ 1	+	+
Phialophora	+2	+	_	+	+	+	_	+
Fusarium	_	+	+	+	+	+	_	+

¹basal leaves only ²rapid wilt

Table 2. Severity of symptoms of decline in 2 species of oaks incurred by 6 species of pathogens.

	Quercus sp.	Ave. severity						
		Days after inoculation						
Pathogen		148	155	162	165	176	183	
Verticillium	agrifolia	1.0	1.0	2.0	3.3	4.3	7.0	
	lobata	1.0	1.0	1.7	1.7	3.3	5.7	
Pestalotia	agrifolia	1.0	1.0	1.0	1.3	2.3	5.3	
	lobata	1.0	1.0	1.7	1.3	2.0	5.7	
Dothiorella	agrifolia	1.0	1.0	3.3	4.7	5.7	7.0	
	lobata	1.0	1.0	4.0	5.0	5.0	6.7	
Cephalosporium	agrifolia	1.0	1.0	1.7	3.0	4.0	4.7	
	Iobata	1.0	1.0	2.0	3.3	5.0	8.3	
Phialophora	agrifolia	1.0	1.0	1.3	2.0	3.0	3.3	
	Iobata	1.0	1.0	2.0	2.3	5.0	8.7	
Fusarium	agrifolia	1.0	1.0	1.3	2.0	2.0	3.0	
	Iobata	1.0	1.0	1.3	2.7	4.0	9.0	
Controls	agrifolia	1.0	1.0	1.0	1.0	1.0	1.0	
	lobata	1.0	1.0	1.0	1.0	1.0	1.0	
Mean air temperature (°C)		9.5	10.2	15.5	16.3	17.0	15.3	
Mean soil moisture (%)		59.5	62.5	38.0	45.5	49.0	44.0	

L.S.D. 5% level by Student's t test = 3.96

Drought may be a precursor to this problem. The relationship of other environmental conditions as predisposing factors clearly requires more investigation. Obviously, however, "oak decline" is not limited geographically, or specifically, as the literature testifies.

Literature Cited

- Beattie, R.K. and G.F. Gravatt. 1933. Diseases of trees: epidemic tree diseases. Plant Dis. Rptr. (Suppl.) 85: 61-67.
- Boyce, J.S., Jr. and D.F. Speers. 1960. Oak dieback in Virginia. Plant Dis. Rptr. 44: 351.
- Crowley, Jim. 1977. Southern oak wilt linked to fungus. Weeds, Trees & Turf. Nov. pp. 18-22.
- 4. Dooling, O.J. 1961. Oak wilt identified in Texas. Plant Dis. Rptr. 45: 749.
- Dunlap, A.A. and A.L. Harrison. 1949. Dying of live oaks in Texas. Phytopathology 39: 715-717.
- Feder, W.A., Gary W. Moorman and T.A. Tatter. 1980. English oak decline on Cape Cod, MA. (Abstr.) Phytopathology 70: 462.
- Graves, A.H. 1929. Twig blight of oak. 18th Ann. Rpt. Brookyn Bot. Gard., 1928. Brooklyn Bot. Gard. Rec. 18: 59.
- 8. Halliwell, R.J. 1965. *Oak decline in Texas.* (Abstr.) Phytopathology 55: 1060.
- Halliwell, R.J. 1966. Association of Cephalosporium with oak decline in Texas. Plant Dis. Rptr. 50: 75-78.
- Hecht-Poinar, Eva I., J.D Britton and J.R. Parmeter, Jr. 1981. Dieback of oaks in California. Plant Dis. 65: 281.
- 11. Horne, C.W. and R.J. Halliwell. 1964. Oak wilt in Texas. Plant Dis. Rptr. 48: 419.
- 12. Ingram, D.E. 1914. A twig blight of Quercus prinus and related species. J. Agric. Res. 1: 339-347.
- Kaufman, J.W. 1978. A comparison of reaction to Cephalosporium diospyri in four tree species. Ph.D. Thesis, Tex. A&M Univ., College Station, TX. 73 pp.
- van Arsdel, E.P. 1977. Reaction of Siberian elm and three American trees to Cephalosporium diospyri. Proc. Am. Phytopathology Soc. 4: 224-225.
- 15. Lewis, Robert, Jr. 1977. Oak wilt in Central Texas. Proc. Am. Phytopathology Soc. 4: 225.
- Lewis, Robert, Jr. 1978. Botryodiplodia theobromae associated dieback in Texas live oaks. (Abstr.) Phytopathology News 12: 206.
- Lewis, Robert, Jr. and F.L. Oliveria. 1979. Live oak decline in Texas. J. Arboric. 5: 241-224.
- Oates, James T. 1978. The essentials of municipal arboriculture. J. Arboric. 1: 148-150.
- Rhodes, D. and F. Tainter. 1979. Interaction of precipitation, temperature, and annual radial growth of white oak in Northwest Arkansas. (Abstr.) Phytopathology 69: 531.
- Roland, G. 1945. Etude faite sur une tracheomycose de Chene occasionee par un Diplodia. Parasitica 1: 11-36.
- Schmidt, R.A. and D.L. Fergus. 1965. Branch canker and dieback of Quercus prinus L. caused by a species of Botryodiplodia. Am. J. Botany 43: 731-737.

- 22. Shurtleff, Malcolm. 1976. Detecting and controlling tree diseases and pest: oaks. Grounds Maint. Oct. p. 32.
- Solomon, W.D., F.I. McCracken, R.L. Anderson, R. Lewis, Jr., F.L. Oliveria, T.H. Filer, and R.J. Barry. 1980.
 Oak pests a guide to major insects, diseases, air pollution and chemical injury. USDA, Forest Service, General Report SA-GR11.
- Tainter, F.H. and W.D. Gubler. 1973. Natural biological control of oak wilt in Arkansas. Phytopathology 63: 1027-1037.
- Tainter, F.H. and R. Lewis, Jr. 1982. Nonstructural carbohydrate contents of trees affected with Texas live oak decline. Plant Dis. 66: 120-122.
- Taubenhaus, J.J. 1934. Live oak disease at Austin, Texas. Tex. Agric. Exp. Sta. 47th Ann. Rpt. pp. 97-98.
- Taubenhaus, J.J. 1935. Live oak disease at Austin, Texas. Tex. Agric. Exp. Sta. 48th Ann. Rpt. pp. 99-100.
- 28. Thomas, W.D., Jr. 1949. Field manual of forest and shade tree diseases. Colo. A&M College 154 pp.
- Thomas, W.D., Jr. 1978. Oak decline. TIPS Pest Leaflet #4. Forest. Ag, Lafayette, CA.
- Thomas, W.D., Jr. 1979. Characteristics of root systems in California oaks. Proc. Symposium on the ecology, management, and utilization of California oaks. Claremont, CA. pp. 178-179.
- 31. Thomas, W.D., Jr. 1983. Ten years of observations in plant clinic isolations (in prep.).
- van Arsdel, E.P. 1970. Live oak decline, its identification and some possibilities of control. Proc. 3rd Ann. Tex. Conf. on Insects, Plant Diseases, Weed and Brush Control. pp. 56-61.
- van Arsdel, E.P. 1978. Oak pathology using leaf symptoms to diagnose recondite maladies. J. Arboric. 4: 228-233.
- 34. van Arsdel, E.P., D.L. Bush and T.W. Jares. 1974. Previsual detection of oak decline and rust diseases in Texas with infra-red photography. (Abstr.) Proc. Am. Phytopathology Soc. 1: 110.
- van Arsdel, E.P. and R.J. Halliwell. 1970. Progress in research on live oak decline. Plant Dis. Rptr. 54: 669-672.
- van Arsdel, E.P., S.D. Lyda and T.W. Jares. 1972. Treatment of Cephalosporium wilt disease with benomyl fungicide. (Abstr.) Phytopathology 62: 807.
- Waterman, A.M. 1937. Diseases of shade and ornamental trees: summary of specimens received in 1935 and 1936 at the New Haven Office, Division of Forest Pathology. Plant Dis. Rptr. 21: 33-37.
- White, R.P. 1936. Notes on new or unusual outbreaks of diseases of ornamentals in New Jersey in 1935. Plant Dis. Rptr. 20: 79-81.

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