Measurement of Expanding Oak Wilt Centers in Live Oak

D. N. Appel, R. C. Maggio, E. L. Nelson, and M. J. Jeger

Associate professor, Department of Plant Pathology and Microbiology; associate professor, Department of Forest Science; and graduate research assistant, Computer Science Department, Texas A&M University, College Station 77843; and U.K. Overseas Development Natural Resources Institute, London, U.K.

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ABSTRACT

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Local, tree-to-tree spread of *Ceratocystis fagacearum* was monitored in live oak (*Quercus fusiformis*) with sequential, color infrared aerial photography during 1982–1987. A computer-based system for analyzing expanding foci was developed to measure rates of crown defoliation and mortality, as well as expansion distances. Four foci expanded radially an average of 11–16 m/yr, with longer maximum distances of expansion (up to 40 m/yr) commonly occurring. One focus that expanded most rapidly increased from 0.3 to 3.6 ha over 5 yr, affecting 10,774 m^2 of crown cover. This was initially the smallest focus, and it had the greatest live oak density. The largest initial focus had a lesser oak density and increased from 1.5 to 6.6 ha, affecting 11,396 m^2 of crown cover. Crown survival in 1987 ranged from 4 to 26% for trees that originally showed symptoms in 1982. A strong linear correlation between the area of affected crown cover and total area occupied by each focus was noted. The rapid rates of focus expansion were attributed to a high potential for root grafting and the occurrence of common root systems among clonally propagated live oaks.

Additional keywords: *Ceratocystis fagacearum*, epidemiology, *Quercus fusiformis*.

Oak wilt "centers" are classical examples of disease foci, or local concentrations of diseased plants about primary sites of infection (10). As defined, the size and shape of foci tend to influence patterns of further spread. Contaminated insects (Coleoptera: Nitidulidae) initiate foci in new areas by acquiring *Ceratocystis fagacearum* (Bretz) Hunt from inoculum sources in previously established centers (11). After initial infection, these foci then expand from perimeter trees to adjacent trees by fungal transmission through functional root connections (16,25,26). The connections are usually root grafts, but common root systems arising from vegetative root suckering may also contribute to fungal spread in live oaks (*Quercus fusiformis* Small) (2).

Transmission of *C. fagacearum* through root connections is termed "local" spread and is usually specified as any occurrence of a diseased tree within 15.2 m (50 ft) of the originally infected source tree (6). Local spread causes the majority of tree losses in the midwestern United States (1,19). The same is believed to be true of live oak mortality in Texas, but attempts to quantify the rates of local spread and resultant losses have not been reported. In a previous survey, patches of oak wilt in Texas live oak averaged 3.6 ha, sometimes reached 80 ha, and contained hundreds of trees (2,4). These large patches are often conglomerates of numerous foci with long, rapidly expanding, and irregular perimeters. The large sizes make local spread difficult to predict and control, particularly in urban locations (3).

Remote sensing with color infrared (CIR) aerial photography has proven to be a valuable tool in studying spatial patterns of forest disease extending over large areas (23). In an aerial survey of Texas oak mortality, discrete oak wilt foci were easily discerned on CIR photography (4). The expansion of individual foci was also clearly observed when sequential photographs were taken in subsequent surveys (5). A computerized system was developed for detecting patterns of mortality and measuring rates of expansion in actively growing foci, as monitored by CIR aerial photography. The objectives of this project were: to investigate the potential for utilizing aerial photography to predict patterns of fungal spread; to gain a better understanding of fungal behavior in live oak; and to facilitate the placement of barriers for disease control. This report describes the system and illustrates some important aspects of oak wilt epidemiology in Texas.

MATERIALS AND METHODS

The sequential, 1:12,000 scale, CIR aerial photography was described in the report of a previous survey (4). The first photo mission was flown in August 1982 and followed by similar missions in August 1983, October 1985, and September 1987. Photointerpretation was done on a Krones LZK Photomultiplier (Krones, Inc., Franklin, WI) at approximately a 7.5 times enlargement of the original photographs. The crowns of individual trees and clumps of trees were delineated as polygons and rated as healthy (0), diseased (1 or 2), dead (3), or trees of other species (4). When removal of diseased trees was detected, they were recorded as dead. A geographic information system (Micro-GIS) was used to digitize both spatial and attribute data into a microcomputer (18). These data were then transferred to a mainframe computer for further analysis.

A Fortran program (Interactive Oak Wilt Analysis Program, IOWAP) was prepared to reproduce a map of the disease center and to compute simple statistics (5). The IOWAP software package includes tools for data verification, high quality graphics output, and interactive analysis. Because of the expected large sizes of the input data sets, most of the computation is done in an interactive environment on the mainframe. The package does provide additional analyses for interaction on a microcomputer. The underlying goal for the analyses is to determine an origin and the distances from it to the perimeter of the focus. The perimeter distances, areas, and yearly health categories for individual polygons are listed by the program, and total areas are computed for each disease category in every year. The polygon selected as a reference point (origin) from which linear rates of expansion were measured is chosen on the bases of least distance from all other polygons and poorest health rating; it was considered the hypothetical original infection point.

In this study, four foci confirmed as oak wilt in previous surveys were selected to illustrate the capabilities of the system and various aspects of the disease in live oak. The sizes and characteristics of the crown cover comprising each disease center are given in Table 1. Foci sizes were measured with a planimeter on a simulated map of diseased and healthy crowns. Two population parameters were used to describe live oak stand characteristics. The first is the area of live oak crown cover, expressed as a proportion of total crown cover, including other species. The second was relative density of live oak, defined as the area of live oak crown cover per total area of the foci in 1987. Species of trees other than live oak were identified by their defoliated condition using winter aerial photography.

To measure linear expansion, each focus was divided into octants as described by Cobb et al (9). Two different measurements were made to define expansion of the foci. The average expansion rate was determined by analyzing the changes in annual distances from diseased crowns to the original infection in all octants where susceptible hosts existed on foci perimeters. The maximum expansion rate represented similar measurements for only those octants where spread actually occurred. Octants where no opportunity existed for spread, such as those adjacent to open fields or major geographic boundaries, were not included in determining rates of expansion.

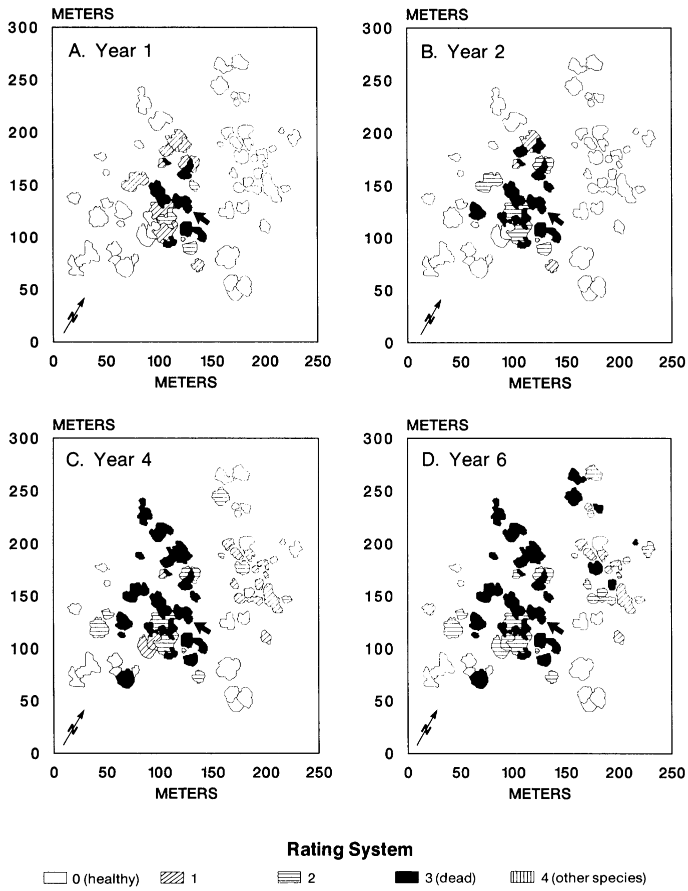

Fig. 1. Computer maps of expansion of oak wilt foci KBBP282 during 1982–1987. **A,** Year 1, 1982. The arrow indicates the polygon selected as the hypothetical origin, or first infection. **B,** Year 2, 1983. **C,** Year 4, 1985. **D,** Year 6, 1987.

RESULTS

The four foci in Table 1 were chosen for illustration because they were distinct, relatively small, and surrounded by susceptible hosts. The opportunity for spread was not the same in each location. For example, all eight octants in foci KBBP282 contained susceptible hosts immediately adjacent to diseased trees during 1982–1983 and 1983–1985 (Table 1), but during the first year, spread occurred in only one octant (Fig. 1). During the second and third years (1983–1985), KBBP282 spread in seven directions. As this focus expanded, the opportunity for new infections was reduced to five octants in the fourth and fifth years; spread occurred in only three. A similar trend was observed at the other foci, where expansion systematically depleted susceptible hosts in various directions. Although delays of a year or more sometimes occurred, healthy trees on perimeters eventually became diseased (Fig. 1). The least opportunity for spread was found in KBB26b because the center was initiated along a fencerow on the edge of a mixed oak stand (*Q. fusiformis* and *Q. stellata*) bounded by an open field. Other barriers to expansion included deep streams and gullies, major highways, or abrupt changes in species composition.

Because of the limited opportunity for spread, the size increase for KBB26b (from 0.8 to 2.8 ha) was the smallest of the four

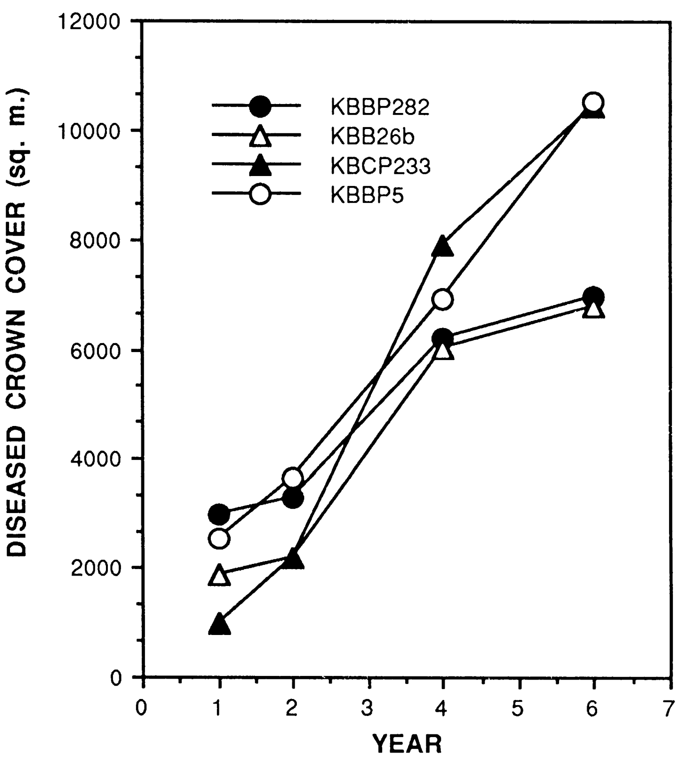

Fig. 2. Yearly increases in affected (diseased) crown cover at four oak wilt foci in Texas, 1982–1987.

TABLE 1. Live oak stand characteristics photointerpreted from 1:12,000 color infrared aerial photographs of four central Texas oak wilt foci

Center	Size (ha)		Total crown cover (m)	Proportion live oak[a]	Relative density[a]	No. of functional octants[b]		
	1982	1987				82–83	83–85	85–87
KBBP5	1.5	6.6	11,396	0.98	0.17	5(7)	7(7)	2(4)
KBBP282	0.8	3.7	7,398	1.00	0.20	1(8)	7(8)	3(5)
KBB26b	0.8	2.8	9,142	0.82	0.27	1(4)	4(4)	1(4)
KBCP233	0.3	3.6	10,774	0.97	0.30	4(7)	4(7)	2(4)

[a] The proportion of live oak is based on the ratio of live oak crown cover to total crown cover, while relative density refers to the area of live oak crown cover divided by the total area occupied by the foci in 1987.

[b] The first number represents the number of octants in which spread was observed and the number in parentheses refers to the number of octants containing high-risk, healthy live oaks on the perimeter of the foci.

foci (Table 1). KBB26b also had the greatest proportion of species other than live oak (0.28), but the density of live oak was high, relative to that of the other foci. The other three disease centers were nearly pure live oak stands (Table 1), a common feature of the oak forests of central Texas.

Disease progress during the 5 yr for all four foci is depicted in Figure 2. The most rapid rate occurred in KBCP233, which increased from 983 to 10,451 m^2 of affected crown cover. Although focus KBBP5 increased to a similar level of disease (10,520 m^2), it was initially more than two times larger (2,537 m^2) than KBCP233. The least expansion occurred in KBBP282, from 2,994 to 6,978 m^2 of affected crown cover. KBB26b exhibited a pattern

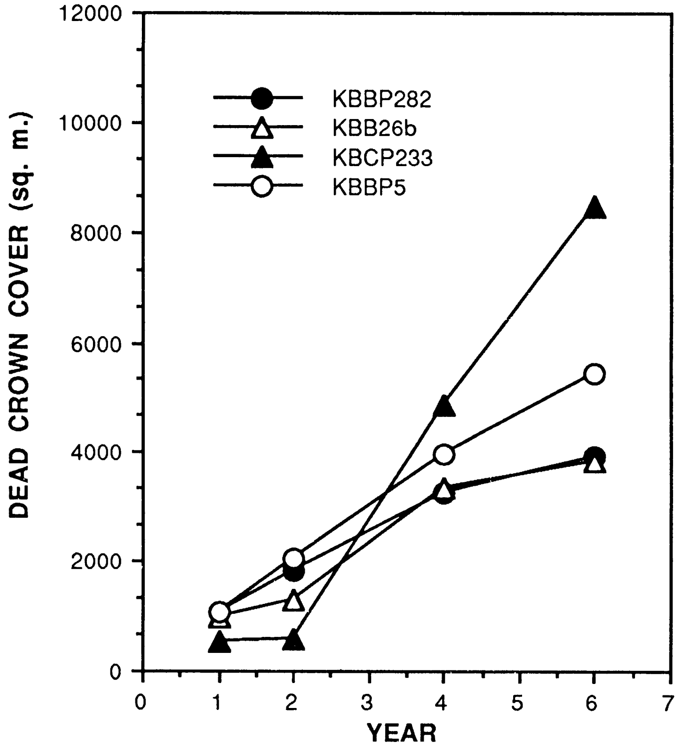

Fig. 3. The yearly rates in increase of dead crown cover for four oak wilt foci.

of disease increase very similar to that of KBBP282. The patterns of mortality in the foci (Fig. 3) followed patterns of disease increase fairly consistently, with the exception of trees in KBCP233. In 1987, 81% of the affected trees were dead in KBCP233, whereas mortality in the other three foci ranged between 52 and 57% of the total affected crown cover. This difference is attributed to the large number of trees harvested from KBCP233 as the disease progressed.

Linear regression was used to analyze the relationship between affected crown area and focus size at each site. A strong positive correlation was found between affected crown cover and the total area of land occupied by the diseased trees (Fig. 4). The steepest slope was exhibited by KBCP233, whereas the least slope of diseased crown cover in relation to area was in KBBP5. However, because of the small sample size, a test for homogeneity of slopes did not indicate that they were sufficiently different to be statistically significant (24).

The photography clearly showed that individual live oaks within foci die at different rates. Therefore, trees in various states of crown loss were discerned throughout all of the foci during 1987. Some trees detected as diseased on the 1982 photography were still surviving after 5 yr (Table 2). Of the diseased crown cover found surviving in foci KBBP5 and KBBP282 in the 1987 photographs, 4 and 26%, respectively, were initially infected before 1982. Tree removal in the other two foci prevented them from being used for this analysis. Some trees survived infection during every year for each focus with one exception; all of the trees showing symptoms during 1982–1983 in KBBP282 died. A few unaffected "escape" trees were also observed in some of the foci.

The average yearly distances of expansion varied from 11 m/yr (KBBP282) to 16 m/yr (KBCP233) (Table 3). The greatest distance of annual expansion measured for any period was over 40 m during 1982–1983 for focus KBCP233. Generally, foci expanded in all directions in which susceptible oaks were available, as illustrated by the depiction of KBBP282 in Figure 1.

DISCUSSION

Oak wilt is traditionally managed by detection and eradication of established foci (1). A thorough understanding of focus expansion is therefore necessary if local spread is to be controlled effectively by creating barriers to root transmission. The sizes and expansion rates of oak wilt foci vary at different locations, partially because of differences in frequencies of root grafting

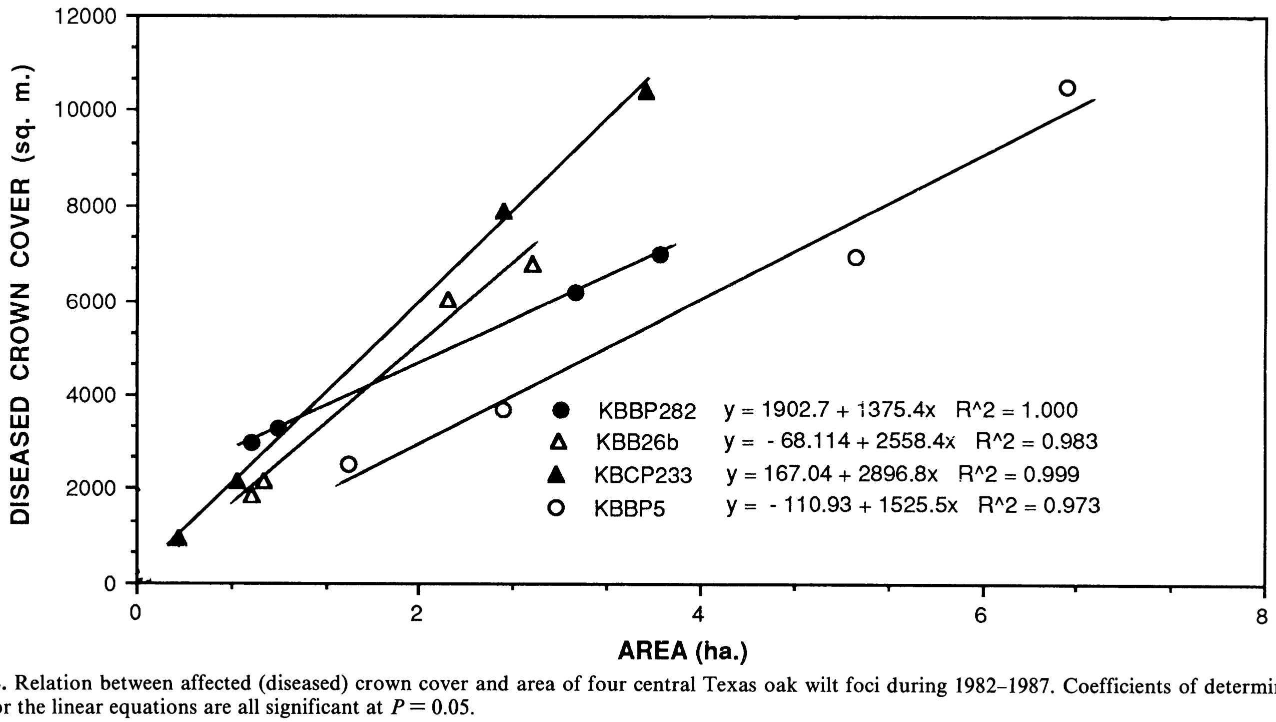

Fig. 4. Relation between affected (diseased) crown cover and area of four central Texas oak wilt foci during 1982–1987. Coefficients of determination (r^2) for the linear equations are all significant at $P = 0.05$.

influenced by stand density (11,23). In the Appalachian Mountains of the eastern United States, disease foci usually involve one to six trees and cover less than 0.2 ha (6,8,21). In the midwestern and north central United States, foci are often larger (17) and commonly expand at rates of 7.5 m/yr (11). These sizes and rates involved oak wilt in deciduous *Quercus* spp. By contrast, the average rates of expansion in the four foci in Texas containing semideciduous live oaks ranged from 11 to 16 m/yr, with much higher maximum yearly distances of spread. Apparently, *C. fagacearum* is transmitted more efficiently in live oak. The patterns, as opposed to rates, of local spread are qualitatively similar to those previously described for other oaks, where the fungus spreads by root grafts. Tree-to-tree expansion at the focus perimeter is observed consistently in homogenous live oak stands; "satellite" infections originating from nearby existing foci do not occur. Fungal mats for inoculum production are not found on live oaks, so the lack of overland spread would be expected in the absence of any vectors other than the sap-feeding nitidulids.

The foci in live oak were located on dry, shallow, rocky soils typical of the Hill Country in central Texas (13). These conditions may encourage lateral extension of root systems and root crowding and may potentially increase the numbers of root grafts among trees (12). More importantly, live oaks have a clonal habit of reproducing by vegetative root suckers (22); if maintained to maturity, the common root systems resulting from this habit add significantly to the potential for local spread. These factors are probably sufficient to account for the extremely high rates of expansion recorded in live oak.

Barriers to local spread can be established by disrupting root connections or poisoning trees on the perimeters of oak wilt foci (14,15). The prospects for successful control through the placement of barriers have been improved in some states through the development of models and decision aids to determine the likelihood of spread between adjacent trees (7,14,19,20). These aids are based on intertree distances and the diameters of the potentially connected trees. The use of CIR aerial photography to measure affected crown cover does not provide sufficient data to develop similar aids for controlling the unique spread observed in diseased live oak stands. The system does give an indication of what can be expected in terms of outward expansion of foci, but not the functional intertree distances involved during that expansion. Based on these indications, it is recommended that the traditional concept of local spread be increased from 15.2 m (50 ft) to at least 22.9 m (75 ft) when oak wilt is encountered in live oak.

A variety of different sizes, shapes, and stand densities were represented by the four foci analyzed in this study, but no distinct pattern accounted for the observed differences in disease progress. Focus size, the proportions and densities of live oak, and the opportunity for spread are probably the most important factors in determining rates of disease progress. The focus with the highest stand density, KBCP233, had a high proportion of live oak and was the smallest focus in 1982. KBCP233 exhibited the greatest increase in size (Table 1) and also attained a relatively high level of affected crown cover (Fig. 2). By contrast, KBBP5 had a relatively low density and a proportion of live oak similar to that of KBCP233. The increase in affected crown cover in KBBP5 was similar to that of KBCP233 (Fig. 1), but the larger initial size and lower tree density caused the total size of KBBP5 to become very large. The differences in the two foci are reflected further in the slopes for linear equations describing the relationship between affected crown cover and focus size. Analyses of larger numbers of rural and urban foci over longer periods of time would be useful to determine whether patterns in disease progress can be predicted on the basis of stand and site characteristics.

Foci were found to expand in any direction where susceptible hosts occurred. The extremely high risk for live oaks growing on the perimeters of actively expanding foci and the large focus sizes have made oak wilt a particularly notorious tree disease for urban and rural landowners in central Texas. The aerial photography and data generated by the system were extremely useful for quantifying rates of spread and, if the size of the disease center and crown cover characteristics are known, may be used to estimate potential losses (Fig. 4). Additional sequential photography is needed to verify this relationship for larger and older foci. In addition to studying the influences of various stand and site characters on foci expansion, the system also would be useful for quantifying the success of disease control efforts in central Texas.

TABLE 2. Proportions (Prop.) of diseased crown cover and areas (m^2) surviving in 1987 following infection before 1982 and during 1982–1983, 1983–1985, or 1985–1987

	Period of initial infection							
	Before 1982		1982–1983		1983–1985		1985–1987	
Foci	Prop.	m^2	Prop.	m^2	Prop.	m^2	Prop.	m^2
KBBP5	0.04	177	0.07	371	0.32	1,621	0.57	2,895
KBBP282	0.26	801	0.00	0	0.56	1,094	0.18	555

TABLE 3. Average[a] and maximum[b] distances of expansion for four Texas oak wilt foci

		Distances (m)			
Foci	Spread	1982–1983 (± SD)	1983–1985 (± SD)	1985–1987 (± SD)	Av. yearly rate (± SD)
KBBP5	Av.	22 ± 22	37 ± 37	28 ± 33	
	Max.	30 ± 20	35 ± 35	57 ± 6	15 ± 9
KBBP282	Av.	0[c]	46 ± 32	16 ± 16	
	Max.	0	53 ± 29	26 ± 5	11 ± 12
KBB26b	Av.	2 ± 5	52 ± 37	15 ± 30	
	Max.	10 ...	52 ± 37	59 ...	14 ± 12
KBCP233	Av.	23 ± 29	44 ± 53	26 ± 44	
	Max.	40 ± 27	78 ± 46	53 ± 53	16 ± 11

[a] Average (Av.) spread is calculated from distances of expansion in all octants where the opportunity for spread existed due to the occurrence of live oaks adjacent to diseased trees on the focus perimeter.
[b] Maximum (Max.) spread refers to the average distance (m) calculated for only those octants where spread actually occurred.
[c] Spread occurred in one octant but was negligible.

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