Intravascular Injection with Propiconazole in Live Oak for Oak Wilt Control

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ABSTRACT

Appel, D. N., and Kurdyla, T. 1992. Intravascular injection with propiconazole in live oak for oak wilt control. Plant Dis. 76:1120-1124.

The fungicide propiconazole was evaluated for oak wilt control in live oak by in vitro growth inhibition of *Ceratocystis fagacearum*, greenhouse inoculation trials on treated trees, and intravascular injection of field-grown trees. The EC_{50} values for four pathogen isolates were consistently less than 20 μg a.i./ml and as low as 2 μg a.i./ml. Container-grown live oaks infused with two fungicide formulations and artificially inoculated with *C. fagacearum* exhibited significantly less disease severity and fewer numbers of diseased trees than untreated controls. In field testing with natural infection, injected live oak plots and subplots consistently had lower levels of crown loss than untreated plots for 9–36 mo following treatment. Average levels of crown loss in seven treated plots containing 57 trees ranged from none to 41%, whereas crown loss in five untreated plots containing 43 trees ranged from 61 to 100%. Differences in disease progress between treated and untreated plots were apparent within 12 mo after injection. Tree injections at the presymptomatic, preventive stage resulted in better disease control than injections of trees with incipient symptoms.

There are several measures available to manage oak wilt, caused by the vascular parasite *Ceratocystis fagacearum* (T.W. Bretz) J. Hunt. These include reducing availability of inoculum to nitidulid vectors, eliminating fresh wounds as infection courts, and creating barriers to root graft transmission among diseased and healthy trees (2,8,12,19). However, these recommendations have limitations when applied to live oaks (*Quercus virginiana* Mill. and *Q. fusiformis* Small) in central Texas (3). Although useful for reducing losses in large populations, they are impractical for protection of individual trees or small groups at high risk of infection. Access and maneuvering heavy equipment needed to sever root connections are difficult in the rugged terrain and thin, rocky soils of central Texas. Also, these measures are undesirable in urban environments, because they require sacrificing valuable specimen trees and are often disruptive to the landscape.

Localized epidemics of oak wilt cause enormous losses of live oaks in central Texas (4). The high value of live oak and impact on property values (20) continue to stimulate research on control alternatives such as intravascular injection with fungicides. Although injection systems using substituted 2-aminobenzimidazoles are recommended for control of Dutch elm disease, caused by *Ophiostoma ulmi* (Buisman) Nannf., there are no similar methods for oak wilt (16, 25,26). Early attempts to protect deciduous red oaks (subgenus *Erythrobalanus*) from artificial inoculation with *C. fagacearum* by chemical injections resulted in only slight success (22). Antibiotic injections resulted in prolonged latent periods and delayed symptoms, but treated trees eventually died at the same frequency as untreated trees. In later studies, benomyl injections reduced or prevented symptom development in treated, inoculated red oaks compared with untreated trees after one growing season (14). Also, some therapeutic value was observed in treated trees with 5–10% symptom development.

Injections of benzimidazoles were unsuccesfully tested for oak wilt prevention and therapy on live oaks in Texas (18). Symptoms were delayed in injected trees, and therapy on live oaks in Texas (18). Symptoms were delayed in injected trees, but mortality rates in treated and untreated trees were similar 15 mo following injection. Results were attributed to uneven fungicide distribution by the vascular systems (18), although no assays were conducted on trees to determine fungicide movement. Subsequent studies on uptake and distribution of thiabendazole demonstrated that a large degree of variability occurred in the crowns of injected live oaks (23). The season of injection and climatic conditions at time of treatment influenced the detection of fungicide in branches, and thiabendazole failed to satisfactorily control the disease in most treatments.

A relatively new group of fungicides, the triazole derivatives, has become important in controlling a wide range of economically important plant diseases (5,10,27). These compounds inhibit ergosterol biosynthesis in fungi, are extremely fungitoxic at low concentrations, and are systemic in treated plants (15). These properties were considered important for a potential fungicide to successfully treat live oaks at high risk of infection by *C. fagacearum*. The following report describes in vitro and in vivo experiments to test the use of the triazole-derivative propiconazole for use in intravascular injection for oak wilt control. Preliminary studies on the use of propiconazole as a soil drench for oak wilt control were reported previously (9).

Accepted for publication 2 June 1992.

MATERIALS AND METHODS

In vitro tests for efficacy. The effect of propiconazole on vegetative growth of *C. fagacearum* was tested on fungicide-amended potato-dextrose agar. Four isolates of the pathogen (TAMU 502, 526, 932, and 1777) were grown on potato-dextrose agar in petri plates amended with 0.0001, 0.00025, 0.0005, 0.001, 0.0025, and 0.005 μg/ml of propiconazole or an unamended control. Propiconazole rates are expressed here and in the remainder of this report as active ingredient (a.i.). The agar was amended by adding appropriate amounts of a propiconazole stock solution either before autoclaving (trial I) or just before pouring the agar (trial II). Growth was estimated after 4, 10, and 14 days at 23 C by measuring mycelial extension on two perpendicular axes passing through the center of the colony. There were five inoculated plates for each isolate at each fungicide rate and two growth measurements for each plate. Growth inhibition on amended plates was compared with normal growth on unamended controls. The EC_{50} for the isolates was determined from linear regression analysis of percent inhibition (probit scale) versus the concentration of the chemical in the medium (log_{10} scale) (11). In a separate experiment, *C. fagacearum* isolate 589 and an *O. ulmi* isolate were grown under similar conditions at four propiconazole concentrations between 0.00 and 0.001 μg/ml to compare the sensitivities of the oak wilt and Dutch elm disease pathogens. Growth on the amended plates was measured at 2, 6, 8, and 10 days and analyzed by analysis of variance (ANOVA) and Duncan's multiple range test at each measurement (24).

Inoculation of treated, immature live oaks. A greenhouse test was performed on 30 3-yr-old live oaks grown in containers in the greenhouse. An infusion technique was used to deliver 1 ml of fungicide into the vascular systems of 20 trees, and the remaining 10 were infused with distilled water. A hole was drilled with a 0.16-mm bit approximately 3 mm into the sapwood and 2 cm above the soil line on a downward angle so that

a micropipette tip filled with the fungicide solution could be inserted. Two formulations of the fungicide were used to treat 10 trees for each formulation. One consisted of propiconazole in an organic solvent formulated as a 1.1 EC (13% a.i., formulation A). The other (formulation B) was water-based as a 1.24 MEC (micro-EC, 14% a.i.). The concentrations of the solutions were 131 and 121 μg/ml for formulations A and B, respectively. One milliliter of solution was placed in the pipette tip and allowed to drain into the tree. Two weeks after infusion, trees were inoculated with a spore suspension of *C. fagacearum* (approximately 5×10^6 conidia per milliliter) by drilling another hole at the soil line and placing a drop of the conidial suspension in the wound. Disease development was subsequently recorded as numbers of symptomatic trees and extent (percent) of symptoms in the crowns. Average crown loss values for each group were analyzed for differences using ANOVA and Duncan's multiple range test (24).

Injection of high-risk, mature live oaks. Field tests with propiconazole injections were performed in natural stands of mature live oaks growing in urban and rural environments. In total, there were 57 treated and 43 untreated trees arranged in two experimental designs to test the efficacy of propiconazole injections. In complete plots, all trees were injected and compared with complete plots of untreated trees. In split-plots, treated and untreated trees were intermingled and analyzed as subplots (designated T for treated or U for untreated). All of the plots were located on the perimeters of actively expanding oak wilt foci so that the risk of natural infection was comparable among groups. None of the treated trees was more than 78 m from an infected tree. There were three split-plot locations: Round Rock (subplots 1T and 1U), Comfort (subplots 7T and 7U), and the North West Hills (subplots 2T and 2U) neighborhood of Austin, Texas (Table 1). Of the six complete plots, four were treated and two were untreated. They were in the North West Hills (plots 3, 5, and 8) and Camp Mabry (plots 4, 6, and 9) neighborhoods of Austin. Average tree diameters are in Table 1.

Risk of infection was estimated by measuring the distance of each tree in a plot to the nearest symptomatic tree (Table 1). The plots and split-plots were located to test preventive treatments before development of symptoms on foliage. However, six trees were symptomatic at injection and were treated therapeutically; none were in any stage of defoliation or crown loss. Four symptomatic trees each were located in subplots 2T and 2U and one each in plots 5 and 6.

Trees were injected at low pressure (7–10 kg/cm^2) into holes (0.8 cm) drilled approximately 2 cm deep into the sapwood of root flares exposed by excavation below the soil surface. The fungicide was contained in 11-L garden sprayers modified with a standard harness kit of plastic injection ports and polyvinyl tubing. Treatment dates and fungicide rates are given in Table 1. Propiconazole used in the test was formulated as a 1.1 EC in an organic solvent carrier and diluted to 1–3 ml/L of water per 2.5 cm/dbh (diameter at breast height). In two complete plots and two subplots, trees were treated a second time approximately 1 yr after the first injection (Table 1). Fungicide concentrations were determined on the basis of propiconazole solubility and phytotoxicity tests conducted in immature trees in the greenhouse (*unpublished*). Disease progress was estimated by identifying diagnostic oak wilt symptoms (1) and measuring the amount of crown loss (defoliation and dieback) or mortality following infection.

RESULTS

In vitro growth inhibition. In preliminary experiments, mycelial growth of *C. fagacearum* was suppressed by propiconazole at concentrations of 0.1 μg/ml. The EC_{50} values for the four *C. fagacearum* isolates from Texas in trial 1 ranged from 0.002 to 0.004 μg/ml. When repeated, the values were slightly higher, ranging from 0.0038 to 0.0157 μg/ml. Growth on unamended agar was lower for each isolate in the second trial. No isolate demonstrated any significant reduced sensitivity to the fungicide (Table 2).

When compared with growth on unamended agar, growth suppression was observed within 2 days following inoculation of plates containing 0.0005 μg/ml of propiconazole (Table 3). After 10 days' growth on plates containing 0.0005 and 0.001 μg/ml, *C. fagacearum* exhibited 5% and 9% reduction in growth, respectively. *O. ulmi* exhibited an even greater sensitivity to the fungicide (Table 3). At 6 days on agar amended with 0.00025 μg/ml of propiconazole, growth was 28% below that on unamended agar. The level of suppression was 40% after 10 days' growth on agar amended with 0.001 μg/ml of propiconazole.

Fungicide efficacy in immature trees. In container-grown trees inoculated and then treated with either fungicide formulation, there were fewer symptomatic trees than in the untreated controls (Table 4). Disease severity was also re-

Table 1. Treatment dates, rates, and tree attributes for plots and subplots with native live oaks used for testing the efficacy of propiconazole for control of oak wilt

Plot no.	Treatment date[u]	Disease rating (mo)[v]	dbh[w] (cm)	Distance[x] (m)	Rate[y]
1T	23 July 1987	36	28.7	13.0	0.02–0.09
1U	Control	36	15.5	13.2	...[z]
2T	9 Aug. 1987	27	21.0	6.5	0.10–0.30
2U	Control	27	30.0	6.6	...
3	27 May 1988	26	43.9	16.1	0.15–0.20
4	24 Sept. 1988	22	41.6	15.7	0.16
5	25 Mar. 1989	21	50.5	8.4	0.09–0.30
6	23 May 1989	14	64.5	13.6	0.13–0.36
7T	17 Oct. 1989	9	32.8	17.4	0.10
7U	Control	9	35.5	14.9	...
8	Control	26	...[z]	9.5	...
9	Control	22	17.8	...	...

[u] All trees in plots 1T and 2T were reinjected approximately 1 yr following the initial treatment; four trees in plot 3 and one tree in plot 4 were also reinjected.
[v] Time after treatment initiation at which disease ratings were taken.
[w] Diameter at breast height, as the average for all trees in the plot.
[x] The average distance of all the trees in the plot from a symptomatic tree.
[y] Range in fungicide concentrations used on trees in the plot in grams a.i./cm dbh.
[z] No measurements taken.

Table 2. In vitro inhibition (EC_{50}) of four *Ceratocystis fagacearum* isolates on propiconazole-amended agar after 2 wk growth

	Trial I			Trial II		
Isolate no.	Growth[y] (mm)	EC_{50}[z] (μg a.i./ml)	R^2	Growth[y] (mm)	EC_{50} (μg a.i./ml)	R^2
TAMU 526	66.0	0.0041	0.99	32.7	0.0157	0.99
TAMU 932	79.7	0.0020	0.83	71.7	0.0038	0.79
TAMU 1777	61.1	0.0033	0.87	58.2	0.0060	0.78
TAMU 502	78.1	0.0030	0.91	61.5	0.0075	0.91

[y] Average radial growth for each isolate (five plates/isolate) on unamended agar.
[z] Determined from a plot of percent inhibition (probit scale) versus the concentration of the chemical in the medium ($\log_{10}$ scale).

duced in both treated groups. At 67 days following inoculation, trees treated with formulation B had only 4% average crown loss, which was significantly less than the 35% exhibited by untreated controls. The levels of 17% and 10% crown loss exhibited at 160 days by trees treated with formulations A and B, respectively, were both significantly less than the 45% crown loss in the control group.

Mature live oak treatments. The subplots (1T, 1U, 2T, 2U, 7T, and 7U) were designed to compare disease progress in individual, randomly selected, treated trees with similarly chosen, untreated trees intermingled in the same plot. Complete plots were established to compare disease progress where trees were treated as a group (plots 3, 4, 5, and 6) with other groups of untreated trees (plots 8 and 9). All treated trees were compared with untreated trees in the same oak wilt focus and were selected to minimize variation in risk of infection by the pathogen. Average distances from symptomatic trees were similar for treated and untreated trees, so that exposure to challenge by the pathogen was presumed to be the same. The presence of extremely large trees in some plots disproportionately influenced average sizes (Table 1).

Crown loss in the untreated control plots and in untreated trees in the split-plots ranged from 61% (subplot 2U) at 27 mo following initial observation to 100% (plot 9) after 22 mo (Tables 5 and 6). In treated plots and treated trees in split-plots, average crown loss ranged from none in two complete plots (3 and 6) to 41% in plot 5 and subplot 7T. In every case, crown loss in the treated trees was significantly less than in the corresponding untreated group. The highest level of disease control in the split-plots was observed at Round Rock, where crown loss was 88% in the untreated trees (subplot 1U) and 2% in the treated ones (subplot 1T). The lowest level of control was at Comfort, where the untreated subplot (7U) had 88% crown loss and the treated group (7T) had 41% crown loss. Significant reductions in crown loss can also be observed by comparing the results from the complete plots in the same neighborhoods (Table 6). For example, crown loss in the two treated North West Hills plots was none and 37% in plots 3 and 5. Untreated plot 8 in North West Hills had 79% crown loss.

Mortality was also lower in treated than untreated plots (Tables 5 and 6). Dead trees were observed in only two of the seven treated plots and subplots; two trees out of nine and three of 11 died on plots 4 and 5, respectively. In contrast, all of the untreated plots and subplots contained dead trees. Mortality ranged from 37% (subplot 2U) to 100% (plot 9) in the untreated trees.

Differences in disease progress for treated and untreated plots could be distinguished within 12 mo following initial observations and injections. These differences are depicted in disease progress curves for the five plots and split-plots in North West Hills (Fig. 1). Disease progress for the untreated trees in subplot 2U and plot 8 increased for 20 mo and then declined. Complete protection was observed in one treated plot (plot 3), whereas trees in the remaining two treated plots stabilized after 6 and 20 mo at average crown loss levels below those observed in the untreated controls.

In those plots where symptomatic trees were treated, injections of asymptomatic trees were consistently more successful in reducing crown loss than treatments of symptomatic trees. Figure 2 depicts average crown loss for trees in split-plot 2. The trees in each subplot (2T and 2U) were grouped according to their state of health at injection or initial observation. Four trees in treated subplot 2T were symptomatic when injected and four were asymptomatic. In the untreated trees in subplot 2U, there were also four symptomatic and four asymptomatic trees. Average crown loss levels after 27 months for the symptomatic and asymptomatic untreated trees (subplot 2U) were 67% and 55%, respectively. In subplot 2T, the trees treated prophylactically (asymptomatic) had an average crown loss of 19% after 27 mo, whereas those receiving therapeutic treatments (symptomatic) had 36% crown loss (Fig. 2).

DISCUSSION

Risk of infection and survival of untreated trees are particularly important considerations when testing a fungicide for oak wilt control in live oak. Asymptomatic, unaffected trees in actively expanding disease centers are rare,

Table 3. Mycelial growth of *Ceratocystis fagacearum* and *Ophiostoma ulmi* on propiconazole-amended potato-dextrose agar

Propiconazole (μg a.i./ml)	Days of Growth (mm)			
	2	6	8	10
C. fagacearum				
Control	13.0 a[y]	46.3 a	61.2 a	75.4 a
0.00025	12.7 ab	45.7 a	61.2 a	75.1 a
0.0005	12.5 bc	44.2 b	58.5 b	71.8 b
0.001	12.1 c	41.9 c	55.4 c	68.6 c
O. ulmi				
Control	...[z]	32.5 a	...	64.8 a
0.00025	...	23.5 b	...	50.9 b
0.0005	...	21.0 c	...	45.4 c
0.001	...	17.7 d	...	38.9 d

[y] Values represent the mean of 20 observations on 10 plates for each fungicide rate; those followed by different letters are significantly different ($\alpha = 0.001$) according to Duncan's multiple range test.
[z] No measurement taken.

Table 4. Numbers of diseased trees and levels of symptom development in container-grown live oaks treated with propiconazole and inoculated with *Ceratocystis fagacearum*

Formulation[x]	Time following inoculation[w]			
	67 Days		160 Days	
	No. of diseased trees[y]	Percent crown loss (av.)[z]	No. of diseased trees	Percent crown loss (av.)
Control	10	35.5 a	9	45.0 a
A	4	18.5 ab	6	17.0 b
B	3	4.0 b	5	10.5 b

[w] Trees were inoculated on August 8–9, 1990.
[x] Formulation A is a 1.1 EC with an organic solvent base; formulation B is a 1.24 MEC in water; the control trees consisted of infusion with sterile, distilled water. Trees were treated with 1 ml of either the fungicide solution or water.
[y] Ten trees were initially inoculated in each treatment.
[z] Values represent the mean of 10 observations; those followed by different letters are significantly different ($\alpha = 0.05$) according to Duncan's multiple range test.

Table 5. Disease development in live oaks located in treated (T) and untreated (U) subplots

Plot	No. of trees		Percent crown loss (av.)
	Total	Dead	
1T	8	0	2 a
1U	8	5	88 b
2T	8	0	27 a
2U	8	3	61 b
7T	6	0	41 a
7U	6	3	88 b

[z] For each subplot, numbers within each column followed by a different value are significantly different ($P = 0.05$) according to a t test procedure.

but some trees do survive infection. In a previous survey, 4–26% of untreated trees in the interiors of four large oak wilt foci were surviving 5 yr after infection (4), but all of these survivors had some degree of crown loss before remission. Similarly, in the present study, there were various levels of crown survival in diseased, untreated live oaks even though the risk of infection by the pathogen was the same (Table 1). Complete mortality was observed in only one out of five untreated plots and subplots. To evaluate any treatment, survival rates in treated trees must be compared with survival in untreated trees. Also, all trees must be located on the perimeters of actively expanding disease centers to minimize variability in exposure to the pathogen. When those conditions were met, tree survival in propiconazole-treated plots was better than in any untreated plots (Tables 5 and 6). Complete or nearly complete protection was observed in three of seven treated plots and subplots from 14 to 36 mo following injection. These results are supported by data from in vitro growth inhibition studies of the pathogen (Table 2) and protection of container-grown, inoculated trees by propiconazole (Table 4). The evidence was considered sufficient to recommend propiconazole injection for oak wilt control in live oak. Although propiconazole soil drenches have produced promising results for oak wilt control in live oak (9), environmental concerns make injection a preferred technique. The manufacturer (Ciba-Geigy Corp., Greensboro, North Carolina) is currently marketing propiconazole under the tradename Alamo for oak wilt in Texas.

Propiconazole exhibits many properties that are considered to be essential for a fungicide to control vascular pathogens in trees (26). For example, no phytotoxic vascular discoloration, typical of tree injections with benzimidazole compounds (16,21), was observed beyond that expected of normal wounding. Vegetative growth of four geographically diverse *C. fagacearum* isolates and an *O. ulmi* isolate were extremely sensitive to very low concentrations of the fungicide in vitro (Tables 2 and 3). Similar sensitivities have made propiconazole and related triazoles effective fungicides for control of numerous other plant pathogens (6,11,17,27). In addition to the influence on growth of the pathogen, triazole compounds have plant growth regulator properties reportedly due to inhibition of gibberellin biosynthesis in treated plants (7). Plant growth inhibition, increased chlorophyl content, and increased tolerance to dryness, frost, and salt stress have been attributed to the activity of propiconazole (5,10,15). Plant growth regulation by propiconazole could play a significant role in the successful control of oak wilt and should be further investigated.

Although results of this study were considered sufficient to recommend propiconazole for oak wilt control in live oak, there are several limitations to this use of injection for disease management. Although not specifically addressed in the experimental design, propiconazole injections clearly did not act as a barrier to inhibit transmission of the pathogen through root connections between treated and untreated trees; many treated trees clearly became infected. The treat-

Table 6. Disease development in complete plots of treated and untreated live oaks located in two Austin, Texas, neighborhoods

Plot	Treatment	No. of trees		Percent crown loss (av.)[z]
		Total	Dead	
North West Hills				
3	Injected	6	0	0 a
5	Injected	14	3	37 b
8	Not treated	6	4	79 c
Camp Mabry				
4	Injected	9	2	22 a
6	Injected	6	0	11 a
9	Not treated	15	15	100 b

[z] For each neighborhood, numbers within each column followed by a different value are significantly different ($P = 0.05$) according to Duncan's multiple range test.

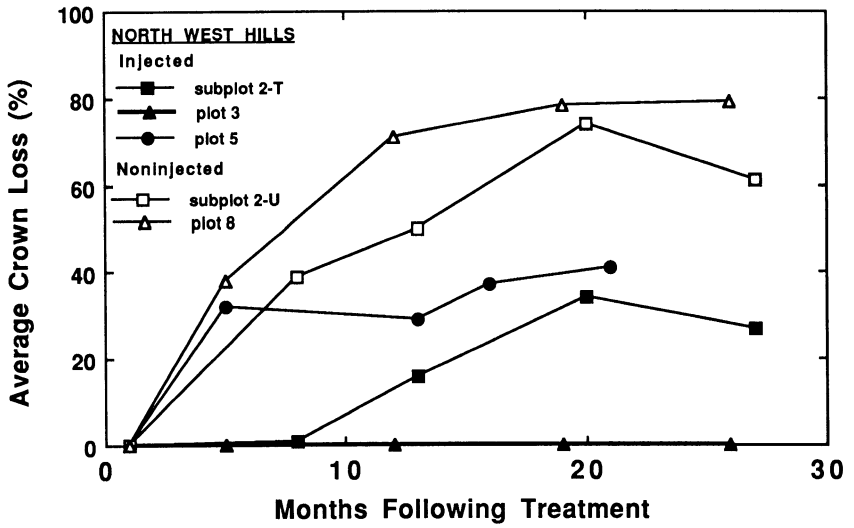

Fig. 1. Average disease progress (percent crown loss) in treated trees (subplot 2T, plots 3 and 5) and untreated trees (subplot 2U and plot 8).

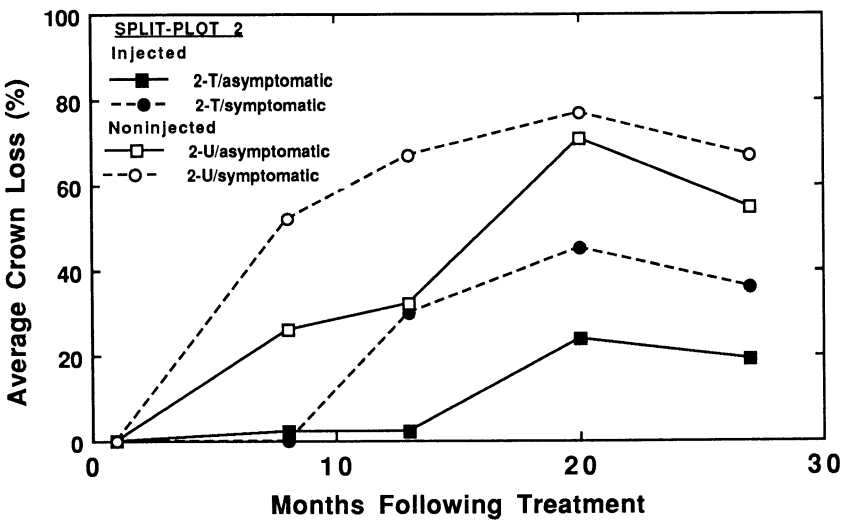

Fig. 2. Average disease progress in split-plot 2. There were four symptomatic trees injected (therapeutic treatments) and four symptomless trees treated (preventative treatments) in subplot 2T and an equal number of symptomatic and symptomless trees in subplot 2U.

ments were most efficacious when administered to asymptomatic trees prior to infection on a preventive basis, but even a small proportion of those trees still exhibited dieback. There is currently no method to predict in which trees injections will fail. In some cases, treated trees will not retain sufficient levels of crown to remain as viable landscape specimens. Therefore, the recommendation to use propiconazole for oak wilt control in live oak must be accompanied by proper expectations for success. Our understanding of the treatment is still insufficient to rule out the necessity of a second treatment. Propiconazole injected into symptomatic trees appears to have the potential to reduce or stop crown loss (Fig. 2), but therapeutic treatments could probably be improved with further research on rates, volumes, and alternative injection methods. Based on the results of these and previous studies (4), it should be presumed that an asymptomatic live oak located immediately adjacent to a symptomatic tree is potentially infected and to be protected must receive prophylactic treatment. The fungicide is currently labeled as a 1.1 EC (formulation A, Table 4) at rates of 2 ml/L of water per 2.5 cm dbh (preventive treatment) and 3 ml/L of water per 2.5 cm dbh (therapeutic treatment), but there is evidence that the water-based formulation B may be a viable alternative. Formulation B is desirable because it lacks organic solvents used to solubilize the active ingredient.

The results of in vitro testing, greenhouse inoculation trials, and field testing indicate that propiconazole may have potential to control oak wilt in other *Quercus* species as well as Dutch elm disease caused by *O. ulmi*. However, live oaks are unique among *Quercus* species in their responses to the pathogen; fungicide injections may be supplementing those mechanisms that are responsible for the natural survival rates observed in untreated live oak populations. It is imperative that these results not be used to assume propiconazole will apply to deciduous oaks, especially the highly susceptible red oaks, without appropriate research. Furthermore, propiconazole injections in live oak should be used in conjunction with other control techniques (3,13) to ensure that all suitable means are used to manage the disease and reduce losses.

ACKNOWLEDGMENTS

This work was supported in part by the Parks and Recreation Department, City of Austin, Texas; the USDA Forest Service Southern Hardwoods Laboratory, Stoneville, Mississippi; and the Ciba-Geigy Corporation.

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