Evaluation of Slash Pine for Resistance to Pitch Canker

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Abstract.--Two- to three-year-old orchard open-pollinated seedlings from 46 slash pine (*Pinus elliottii* Engelm. var. *elliottii*) clones were evaluated for resistance to *Fusarium moniliforme* var. *subglutinans* (FMS). These families, representing fast growing and/or fusiform rust resistant genotypes, were planted near Gainesville, Florida. Terminal and lateral shoots of 21 to 24 seedlings per family were wounded and inoculated with a polymix of four isolates of FMS. Family mean symptom expression ranged from 16.6 to 91.7%; shoot mortality ranged from 4.2 to 91.7%. Strong individual and family heritabilities suggested that genetic resistance may be useful in management of pitch canker. Estimated gains from four improvement options are presented. There were no significant correlations between pitch canker resistance and either fast-growth or fusiform rust resistance.

Additional Keywords: *Pinus elliottii* var. *elliottii*, *Fusarium moniliforme* var. *subglutinans*, heritability, genetic variation, genetic gain, fusiform rust, *Cronartium quercuum* f. sp. *fusiforme*.

INTRODUCTION

FMS infects many southern pines. This fungus has been especially damaging in slash pine, inciting resin-soaked cankers on the branches and main stem resulting in shoot dieback, stem deformity, reduced growth, and mortality (Blakeslee et al. 1980, Blakeslee and Oak 1979, Dwinell et al. 1985, Phelps and Chellman 1976). Prospects for long-term control of pitch canker are strengthened by studies showing genetic variation within pine species for resistance to pitch canker (Barnett and Thor 1978, Blakeslee and Rockwood 1978, Dwinell and Barrows-Broaddus 1979, 1981 and 1983, Dwinell et al. 1977). This paper reports further evidence of genetic variation in slash pine and estimates genetic gains that could be obtained from using resistant genotypes.

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MATERIALS AND METHODS

Open-pollinated progeny of 46 fast-growing and/or rust-resistant clones were planted in a randomized complete block design on a fertile, well-drained site in north central Florida in 1981 and 1982. The trees were planted at a 10' x 2' spacing in 10-tree row plots that were replicated three times.

From September 24 through October 18, 1983, the terminal and one lateral shoot of each tree were inoculated with an aqueous suspension of conidia of FMS. At the time of inoculation, the trees averaged 7.25 feet in height and were vigorous and healthy. The eight most vigorous of the ten trees planted per row plot were selected for inoculation. Precipitation and ambient temperature were monitored on the site during the inoculation period.

The inoculum consisted of four proven-pathogenic isolates of FMS obtained from slash pines in Volusia, Franklin and Gilchrist counties in Florida. The isolates were single spored and grown on carnation leaf-water agar for about 10 days prio to use. An aqueous suspension of conidia from each source (1-1.5 x 10 conidia/ml) was prepared daily, and equal aliquots of each source were combined just prior to use. Post-inoculation germination checks were made daily, and viability consistently exceeded 96%.

Prior to inoculation, shoots were surface sterilized by spraying with 95% ethanol and allowed to dry. Each shoot received two wounds at the same level, located on opposite sides of the third flush of 1983 growth. The wounds were made with an 18-gauge needle, and two drops of inoculum were placed in each wound. Control branches, selected at random within the family, were treated in the same manner except that sterile water was used in place of the inoculum.

At about 18-day intervals for the next eight months, each shoot was examined for symptom development. At each observation, the condition of the shoot was rated as 1 (no symptoms), 2 (foliage discolored) or 3 (foliage brown and shoot dead). So that maximum disease expression could be obtained for all trees, the shoots were harvested according tc disease severity, with dead shoots being harvested and processed in the laboratory before living shoots were harvested. In July 1984 when neu symptom expression had essentially ceased, all remaining shoots were collected and brought to the laboratory for detailed examination and isolation.

Two responses on terminal shoots were analyzed; percent of trees with pitch canker symptoms (conditions 2 and 3) and percent of trees with pitch canker induced mortality (condition 3 only). To determine significance of blocks and families, analysis of variance was conducted suing plot means. For genetic analyses, these two binomial responses were handled in the same manner as percent rust-infected data (Rockwood and Goddard 1973). Individual tree and family mean heritabilities were calculated on the assumption of half-sibs. Genetic gains employed techniques presented by Shelbourne (1969). Selection intensities assumed were: 10% in seed production areas and for tested clonal orchards, 2% for untested clonal orchards and 40% in existing clonal orchards.

Correlations between traits were based on family means. Clonal evaluations for growth and rust resistance included in the correlations were weighted, standardized comparisons from numerous progeny tests involving open-pollinated progenies and an unimproved check lot.

RESULTS AND DISCUSSION

Symptom development followed a typical disease progress curve (Figure 1). Observed symptoms included those regularly associated with pitch canker on slash pine, via, discoloration and death of needles around the inoculation sites, exudation of pitch from infected tissues surrounding the inoculations, discoloration and death of needle and shoot tissues distal the the cankers, and, for those trees where advanced symptom development did not occur until the following spring, a second flush of symptoms coinciding with the spring flush of growth.

The pitch canker pathogen was reisolated from about 90% of the inoculated shoots and 2% of the control shoots, thus indicating that the observed symptoms were due to the pathogenic action of the introduced

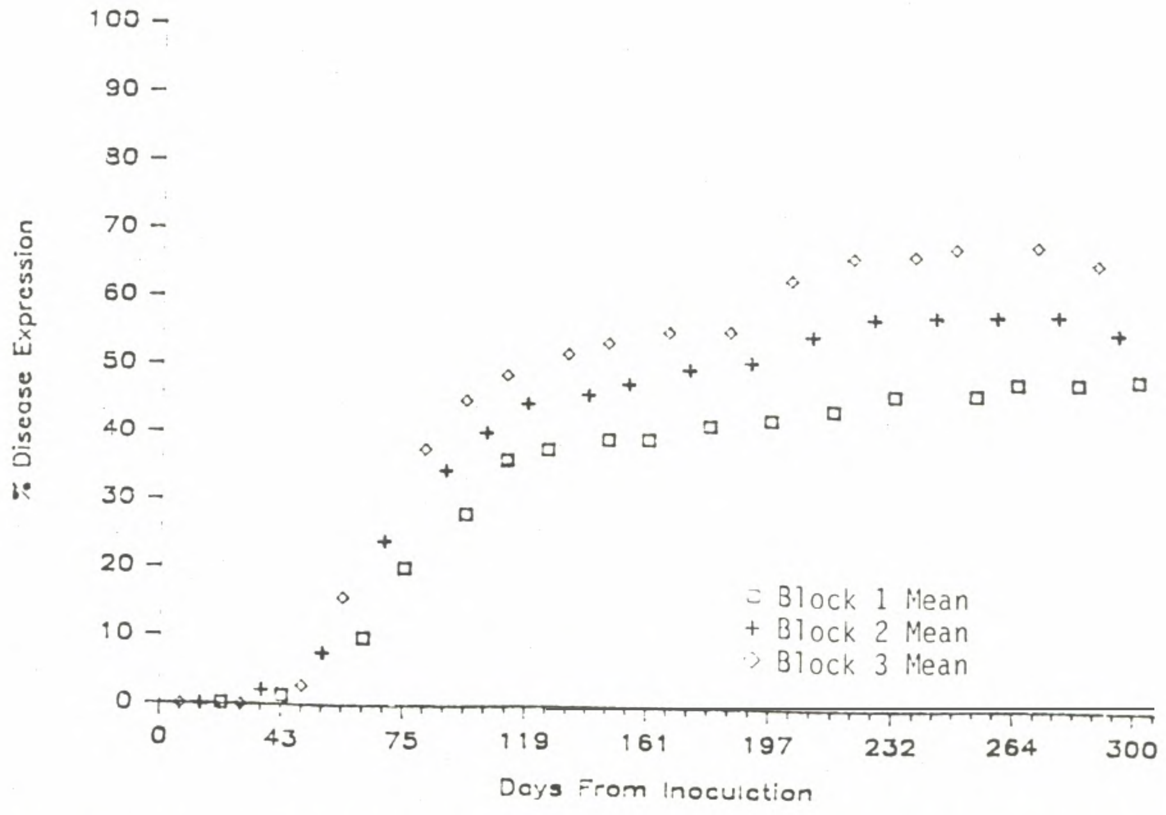

Figure 1. Pitch canker symptom expression disease progress curves based on block means.

fungus. Failure to obtain higher yields from the inoculated shoot reisolation may be due to incomplete isolation from the canker, or loss of viability of the fungus due to active defense of host, thus limiting tissue available for colonization. Reisolation of the pathogen from a small percentage of the control shoots can be readily attributed to the presence of idigenous FMS inoculum that can infect susceptible tissue when wounded.

Family mean responses to the pathogen were normally distributed, ranging from highly susceptible to highly resistant (Table 1). A similarily wide range of incidence has been observed in loblolly pine clones (Dwinell et al. 1977). However, there were certain trees that became infected but progressive tissue colonization did not occur as the canker was stabilized and the infection delimited by the production of hypertrophic callus around the canker. Those trees, based on mean symptom expression, would appear to be poor performers, but when evaluated with respect to shoot mortality, appear to have functional resistance. Overall, there was a strong correlation between symptom expression and mortality response.

Significant differences existed among families and among blocks (Table 2). The differences between blocks may be related to differences in edaphic conditions, topography, variations in rainfall during the inoculation period or the slightly higher percentage of younger (two-year-old) trees included in the third block.

Table 1. Slash pine family means for percent pitch canker symptom expression (Symp) and mortality (Mort) of terminal shoots.

	Pitch Canker			Pitch Canker	
Family	Symp	Mort	Family	Symp	Mort
1-60	16.6	8.3	56-56	62.5	33.3
23-59	20.8	16.7	327-56	64.9	26.2
M-835	29.2	8.3	102-57	66.7	29.2
57-56	33.3	12.5	261-56	66.7	29.2
239-56	37.5	25.0	285-55	66.7	41.2
M-204	40.3	13.9	69-56	66.7	29.2
205-55	45.8	12.5	163-57	68.3	38.3
24-60	45.8	16.7	173-57	70.8	25.0
13-59	47.0	30.6	33-58	70.8	41.2
89-57	48.2	17.6	48-59	70.8	37.5
M-308	50.0	8.3	60-56	70.8	29.2
16-59	50.0	25.0	B-106	75.0	20.8
163-58	50.0	12.5	65-56	75.0	45.8
31-60	50.0	29.2	91-58	75.0	50.0
66-73	50.0	20.8	M-109	76.4	45.8
27-58	50.8	14.3	13-56	79.2	66.7
M-817	54.2	4.2	293-55	79.2	54.2
64-56	54.2	37.5	106-56	83.3	58.5
357-56	55.4	26.8	265-55	83.3	66.7
57-61	58.3	25.0	76-58	83.3	45.8
100-56	62.5	29.2	130-60	87.5	30.9
330-56	62.5	41.7	347-56	87.5	75.0
342-56	62.5	50.0	70-56	91.7	91.7

Table 2. Analyses of variance, heritabilities and genetic gains for percent pitch canker symptom expression (Symp) and mortality (Mort) in slash pine.

		Pitch Canker			
		Symp		Mort	
		Analysis of Variance			
Source	DF	MS	F	MS	F
Block	2	3857.6	10.40*	1574.8	5.58*
Families	45	927.8	2.50*	1070.2	3.79*
Error	90	371.0		282.5	
		Heritabilities			
Individual		.253		.383	
Family Mean		.600		.736	
		Genetic Gains			
		----------(% of Mean)----------			
Short-term Options -					
Seed Production Area		39.4		107.7	
Tested Clones in Existing Seed Orchard		16.8		41.4	
Long-term Options -					
Orchard of Untested Select Trees		54.5		148.9	
Orchard of Tested Clones		60.7		149.3	

*Significant at the 1% level.

Family differences were somewhat greater for percent mortality than for percent symptoms, and this relationship was further evidenced in heritabilities and genetic gains (Table 2). Individual tree heritabilities were strong and family heritabilities also suggested potential for genetic gain.

A variety of short- and long-term options are available for realizing the genetic potential for reducing pitch canker incidence. Short-term alternatives include seed production areas in heavily-infected plantations or natural stands in epidemic areas, a very successful strategy for developing fusiform rust resistance (Goddard et al. 1975), and collection of seed from tested clones in established seed orchards. The more productive alternative appears to be seed production areas (Table 2). Assuming that 20 clones of a typical 50-clone orchard will be resistant and contribute seed, existing orchards provide a relatively small, but still meaningful, gain.

The long-term options involving new clonal orchards offer greater improvement. An orchard of proven pitch canker resistant clones would provide slightly more gain than an orchard of untested clones derived from pitch canker epidemic areas.

Simultaneous implementation of short- and long-term options, if possible, is desirable for developing progressively resistant planting stock. Conversion of stands to seed production areas is a relatively low cost alternative applicable to some forestry organizations. Initiation of new orchards is immediately possible due to ongoing screening efforts which have identified more than 25 resistant trees.

Correlations between pitch canker resistance, rust resistance, fast growth and tree height were insignificant (Table 3). Lateral shoot response showed a strong relationship to the response of terminal shoots (Table 3) notwithstanding the differences in shoot size and phenology . These results suggest that fast growth and rust resistance need not be compromised in selecting for pitch canker resistance. Several of these 46 clones were superior in all three characteristics.

Additional testing involving different environments and additional clones will be conducted in 1985-86. Data obtained will permit expanded examination of the results reported in this paper.

CONCLUSIONS

These results from inoculations of vigorous, healthy, field-grown trees suggest that genetic resistance to pitch canker is present in slash pine and that significant genetic gains may be realized.

Pitch canker resistance appears to be unrelated to either rust resistance or fast growth, indicating that fast-growing, rust-resistant and pitch canker resistant trees may be selected.

Table 3. Correlation coefficients among slash pine family means for terminal (Term) and lateral (Lat) shoots pitch canker symptom expression (Symp) and mortality (Mort), tree height and clonal rust and growth evaluations.

	Shoots				Clonal Evaluation	
	Term.	Lat.				
Trait	Mort	Symp	Mort	Tree Height	Rust	Growth
Term - Symp	.78*	.79*	.72*	.10	-.01	.05
Mort		.73*	.77*	.09	-.01	-.05
Lat - Symp			.87*	-.14	.03	-.04
Mort				-.11	-.01	-.01
Tree Height					.02	-.08
Clonal Rust						-.25

*Significant at the 1% level.

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