Influence of Infection Percent on Improvement of Fusiform Rust Resistance in Slash Pine

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Abstract

Open-pollinated progenies of 143 slash pines selected for growth and form traits but unselected for fusiform rust resistance (population-A) and 92 slash pines selected for rust hazard locations in Georgia and Florida over a two-year period. Fusiform rust incidence data three and five years after establishment were subjected to binomial analysis and to analysis of plot means transformed to the square root of the arcsin.

There was substantial variation among families of both populations. Estimates of heritability increased linearly as infection percentage increased in binomial data, while heritability estimates from the transformed plot mean analysis were maximum at 60% infection. Predicted gains up to 20% were estimated from intensive mass selection as applied in population-B, and these closely paralleled realized gains in this population. Gain estimates were also calculated for mass selection plus progeny testing and for selection in the second generation. A high percentage of infection was the most important factor for maximization of gain with highest gain expressed in tests with approximately 70% infection. Alternative resistance improvement procedures are discussed.

Key words: Cronartium fusiforme, Pinus elliottii, resistance breeding, selection, heritability estimation, genetic gain estimation.

Zusammenfassung

Frei abgeblühte Nachkommenschaften von 143 Pinus *elliottii*, die nach Wachstums- und Formeigenschaften ausgewählt worden waren, ohne die Resistenz gegen Cronartium fusiforme zu berücksichtigen (Bestand A) und 92 P. elliottii, die auf Rostresistenz selektiert worden waren (Bestand B), wurden für die Dauer von 2 Jahren in drei Gebieten mit hoher Wahrscheinlichkeit des Auftretens von Rost in Georgia und Florida ausgepflanzt. Die Daten über den Auftritt von Chronartium fusiforme, 3 und 5 Jahre nach dem Pflanzen, wurden einer binomialen Analyse sowie einer Analyse von Diagramm-Mittelwerten, die in die Quadratwurzel des arc. sin umgeformt wurden, unterzogen.

Es traten grundlegende Abweichungen innerhalb der Familien beider Populationen zutage. Die Schätzung des Heritabilitätsfaktors nahm linear zu, in dem Maße, wie der Infektionsprozentsatz der binomialen Daten anstieg. Andererseits gingen die betreffenden Schätzwerte für die Heritabilität aus der Analyse der umgeformten Diagramm-Mittelwerte nicht über ein Infektionsmaximum von 60% hinaus. Die vorhergesagten Steigerungen bis zu 20% wurden aufgrund intensiver Massenauslese, wie auf Bestand B angewendet, geschätzt und kamen tatsächlich sehr nahe an die festgestellten Steigerungen in diesem Bereich heran. Steigerungsschätzungen wurden ebenfalls für Massenauslese und Nachkommenschaftsprüfung sowie die Auslese der zweiten Generation durchgeführt. Ein hoher Infektionsprozentsatz war der wichtigste Faktor für das Erzielen der Höchststeigerung, wobei sich die höchste Steigerung in Tests mit ungefähr 70% Infektion zeigte. Alternativverfahren zur Verbesserung der Resistenz werden diskutiert.

Introduction

The widely adopted intensive management system of short rotation plantation forestry in southern United States has resulted in the establishment of many million acres of loblolly (Pinus taeda L.) and slash (P. elliottii Engelm.) pine plantations. Both species are highly susceptible to fusiform rust caused by the fungus Cronartium fusiforme (Hedge and Hunt ex Cumm.) (Czabator, 1971). During the same period the plantation system was being established, there was a drastic reduction in wildfires, and controlled burning and other treatments failed to prevent widespread regeneration and growth of oak species, alternate hosts to the rust fungus. In the presence of abundant, young susceptible pine hosts and prevalance of alternate hosts (Dinus, 1974; Hollis and Schmidt, 1977; Squillace et al., in press) fusiform rust has increased to epidemic proportions in many locations (Squillace, 1976). There is evidence that the incidence of rust is increasing with each year of planting (Schmidt et al., 1974; Griggs and Schmidt, 1977). Dollar impact was estimated at 28 millioa annually in the Southeast United States (Powers et al., 1974). This situation has made increased genetic resistance to fusiform rust a major objective of loblolly and slash pine improvement programs.

Numerous studies have indicated genetic variation in resistance to fusiform rust in both loblolly and slash pines (Dinus, 1969; Goddard and Schmidt, 1971; Jewell, 1959; Kinloch, 1968; Powers et al., 1976). Loblolly pine exhibits variation in resistance due to geographic source of seeds (Wells and Wakeley, 1966) and to genetic differences among individual trees (Kinloch; 1968). Geographic variation in resistance appears to be lacking in slash pine (Goddard and Wells, 1977) but there are strong individual tree differences (Dinus and Griggs, 1975). This paper is concerned with partitioning of genetic variance in resistance in slash pine and exploitation of resistance found for development of planting stock with reduced susceptibility to this disease.

Materials and Methods

Populations

Selection of superior phenotypes (mass selection) was initiated in natural slash pine stands throughout the species range by participants in the Florida Cooperative Forest Genetic Research Program. Scions from selected trees were used for orchard establishment. Althought tree infected with fusiform rust were avoided, there was little selection pressure for resistance due to low disease incidence in these stands. Indeed, early progeny test results indicated that progenies of selected trees on the average were no more resistant than those of unselected slash pine controls (Schmidt and Goddard, 1971). Open pollinated seeds from orchards were collected for establishment of rust resistance trials. In the following discussion, these parent trees as a group are called population A.

A second population (Population B) was obtained from a plantation of local origin in Wayne County, Georgia, where over 90% of the trees were infected. In this populatioa mass selection was applied with improvement of rust resistance as the primary objective. All infected trees were removed and seeds were collected from the remaining rust free trees

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two or more years later. We have previously reported highly significant differences in rust resistance between progenies of populations A and B (Goddard *et al.*, 1975).

Planting sites

Three planting locations in areas with a history of high rust incidence were selected. The planting sites were in Bulloch County (east Georgia), Webster County (west Georgia), and Taylor County (north Florida). Starting in January, 1971, and annually thereafter, these sites were used to establish screening trials for field resistance to fusiform rust. Open pollinated progenies of selections from populations A and B were planted in 1971 and 1972 as indicated in *Table 1*. Of the 143 population A families, 13 were planted both years in all three locations. The 92 population B families were all planted once at each location.

Table 1. — Range of family mean fusiform rust infection percent and average infection percent of two slash pine populations established in two planting years at three locations.

Population	Year of Planting	No. of Families	Location	Age	Infection Percent Range	Infection Percent Mean
				(yrs)	(%)	(%)
A	1971	92	Webster	3	41-100	85.6
				5	62-100	91.2
			Taylor	3	0-91	40.4
				5	9-100	51.5
			Bulloch	3	0-89	51.1
				5	11-93	57.2
	1972	64*	Webster	3	25-96	61.7
				5	25-96	69.2
			Taylor	3	12-83	46.2
				5	12-83	51.4
			Bulloch	3	0-44	13.1
				5	4-70	24.9
B	1971	92	Webster	3	27-96	62.0
				5	31-100	70.3
	1972	92	Taylor	3	0-57	26.8
				5	3-61	29.7
			Bulloch	3	0-19	5.8
				5	0-28	12.4

*13 of these families were planted the previous year also.

Seedlings from both populations were planted randomly in ten-tree row plots, replicated in three blocks in each location. Spacing was 0.6 m within rows 2.4 m apart. The 1972 plantings were adjacent to but not randomized with the 1971 plantings.

Data collection and analyses

Rust infection in all plantings was assessed three and five years after estabilshment. The number of stem and limb galls was recorded for each infected tree. A gall on the main stem can cause reduction in product value and directly or indirectly lead to tree mortality. A limb gall has negligible impact on growth or product value unless the limb gall extends to the main stem. However, limb galls are equally as indicative of susceptibility as stem galls. Reporting on fusiform rust resistance in loblolly pine, Blair[2]) found close coincidence of family ratings as indicated by three indexes: a severity score, number of galls per tree, and percentage of trees infected. Because of the superior accuracy of classifying a tree as infected or not infected and the simplicity of analysis and interpretation of this procedure, analyses were based on whether or not a tree was infected, a threshold or "all-or-none" approach.

Even so, alternative analytical procedures are available and data were subjected to two separate procedures:

1. Binomial analysis — each tree classified as "1", infected, or "0", not infected, and these data subjected to analysis of variance. This procedure permits estimation of both individual and family heritabilities.

2. Arcsin square root transformation of the percentage of trees infected per plot. Family heritability can be directly estimated from analysis of variance by this method. Individual heritability was estimated by use of constant binomial variance.

The statistical model assumed is indicated in *Table 2*. Note that the analyses, which provided the basis for heritability estimation, involved only Population A, considered essentially random in so far as resistance to fusiform rust is concerned. Similar analyses were completed separately for the 1972 plantings. Analyses were also conducted of each planting individually. Least squares analyses were used to correct for missing data. A combined analysis of both planting years was excluded because only 13 families were planted both years, creating an extremely unbalanced design. Data from the two planting years provided separate estimates of variance components. Estimates of heritability. Variance components were partitioned for population-A and used for estimations of heritability. Since population-B was selected for rust resistance, it was inappropriate for estimation of genetic parameters.

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[2]) Blair, Roger L., 1970: Quantitative inheritance of resistance to fusiform rust in loblolly pine. Ph. D. thesis, North Carolina State University.

Table 2. — Analysis of variance of fusiform rust infections at age 3 years as a threshold character (binomial data) in slash pine population-A and variance components estimated.

Source of Variation	Degrees of Freedom[a]	Mean Squares[b]	Variance Components Estimated[c]
Locations	2	124.95**	$\sigma^2_t + 8.99\sigma^2_{b(\ell)f} + 27.61\sigma^2_{\ell f} + 761.37\sigma^2_{b(\ell)} + 2277\theta^2_L$
Blocks (Locations)	6	1.16**	$\sigma^2_t + 8.96\sigma^2_{b(\ell)f} + 25.17\sigma^2_{\ell f} + 759.48\sigma^2_{b(\ell)}$
Families	91	1.26**	$\sigma^2_t + 8.95\sigma^2_{b(\ell)f} + 74.23\sigma^2_f$
Locations x Families	182	.28*	$\sigma^2_t + 8.76\sigma^2_{b(\ell)f} + 24.75\sigma^2_{\ell f}$
Blocks (Locations x Families)	518	.22**	$\sigma^2_t + 8.44\sigma^2_{b(\ell)f}$
Within Plots	6037	.18	σ^2_t

[a] Degrees of freedom of some sources reduced due to missing data.

[b] * indicates significant at the 0.05 level of probability.
** indicates significant at the 0.01 level of probability.

[c] Coefficients for the variance components were determined using SAS Varcomp procedure.

σ^2_t = within plot variance.
$\sigma^2_{b(\ell)f}$ = variance due to interaction of families and blocks within locations.
$\sigma^2_{\ell f}$ = variance due to interaction of families and locations.
σ^2_f = variance among families.
$\sigma^2_{b(\ell)}$ = variance among blocks within locations.
θ^2_L = variance among locations.

Using the binomial analyses, individual tree heritability of rust resistance was estimated as:

$$h^2_i = \frac{4\sigma^2_f}{\sigma^2_t + \sigma^2_{b(l)f} + \sigma^2_{lf} + \sigma^2_f}$$

Family heritability was estimated as:

$$h^2_f = \frac{\sigma^2_f}{\frac{\sigma^2_t}{l \cdot b \cdot k} + \frac{\sigma^2_{b(l)f}}{l \cdot b} + \frac{\sigma^2_{lf}}{l} + \sigma^2_f}$$

The various components of both formulas were calculated as indicated in *Table 2*.

For transformed plot mean data, basically the same formulas were used except binomial sampling variance was used for calculation of within plot variance. With the proportions infected transformed to arcsins, the binomial sampling variance is constant and $\sigma^2_{bin} = 821$ (Fisher and Yates, 1948). With the transformed data, the mean square for blocks within locations × families interaction estimates $1/k\,\sigma^2_{bin} + {}^2\sigma_t$.

Standard errors of variance components were approximated as follows (Anderson and Bancroft, 1952):

$$\mathrm{Var}\,(\sigma^2_g) \cong \frac{2}{K^2_1} \sum_g \frac{MS^2_g}{df_g + 2}$$

$$\mathrm{S.E.}\,(\sigma^2_g) = \sqrt{\mathrm{var}\,(\sigma^2_g)}$$

where, K_1 = coefficient of the variance component,
MS_g = g^{th} mean square used to estimate the variance component,
and df_g = the degrees of freedom of the g^{th} mean square.

Genetic gain predictions

Heritability estimates obtained were used to calculate genetic gain or response to selection. Gain is influenced by heritability and the intensity of selection as well as by the extent of genetic variation present in a population. Gains for three different selection procedures were estimated.

The first procedure is mass selection. For rust resistance, this involves selection of all rust-free trees or a random sample of rust free trees in a population. Population-B is a typical example of mass selection as only rust-free trees were selected and used for production of seed for the next generation. Data from progenies of population-B provide a comparison of realized gain with predicted gains. Predicted genetic gain from mass selection was calculated as:

$$\Delta G_1 = \frac{i_1 K\ \sigma^2_A}{\sigma_1}$$

where ΔG_1 = expected genetic gain
i_1 = selection intensity or proportion of trees selected
K = fraction of the total additive variance in the covariance of additive value
σ^2_A = additive variance = $4\sigma^2_f$
$\sigma_1 = \sqrt{\sigma^2_t + \sigma^2_{b(l)f} + \sigma^2_{lf} + \sigma^2_f}$ (σ^2_t, $\sigma^2_{b(l)f}$, σ^2_{lf}, σ^2_{ft} as defined in *Table 2*)

The value of K for population-B is 1, since both parents were selected for rust resistance.

The second procedure involves mass selection combined with progeny testing. In population A, this provides an estimate of gain from a clonal seed orchard rogued on the basis of progeny performance. The gain equation used is:

$$\Delta G_2 = \Delta G_1 + 2i_2 \frac{1/4\sigma^2_A{}'}{\sigma_2}$$

where,
i_2 = selection intensity determined by the proportion of trees selected among those originally selected.

$\sigma^2_{A}{}'$ = additive genetic variance of the selected parent population

$= \sigma^2_{A}(1\text{-}Bv')$

where $B = h^2_i$ and v' is a variable dependent on i_1 and is tabulated by FINNEY (1956). $\sigma^2_{A}{}'$ can be assumed to be the same as σ^2_{A} in population A, but not in population B.

$$\sigma_2 = \sqrt{\frac{\sigma^2_{t}}{k \cdot b \cdot l} + \frac{\sigma^2_{b(l)f}}{b \cdot l} + \frac{\sigma^2_{lf}}{l} + \frac{1}{4}\sigma^2_{A}{}'}$$

The third procedure is selection in the second generation population for establishment of a new clonal orchard. The procedure involves selection of the best individuals in the best families. Selection of the best families is based on general combining ability and is applicable to population-A and population-B. The pertinent gain formula is:

$$\Delta G_3 = \Delta G_1 + i_2 \frac{1/4\,\sigma^2_{A}{}'}{\sigma_2} + i_3 \frac{3/4\,\sigma^2_{A}{}''}{\sigma_3}$$

where i_2, $\sigma^2_{A}{}'$ and σ_2 are as defined above and

i_3 = selection intensity determined by the proportion of uninfected individuals within families

σ^2_{3} = within half-sib family variance

$\sigma^2_{A}{}''$ = additive variance of the population after selection. It is assumed $\sigma^2_{A}{}'' = \sigma^2_{A}$, since genetic recombination has occurred.

Results and Discussion

For both populations A and B and both planting years, the level of infection varied greatly among planting locations *(Table 1)*. The most severe infection percentage was observed in Webster County where 91% of population A trees planted in 1971 were infected by age 5. In all locations, a much higher percentage of population A trees were infected than population B. However, the variation among family means was comparable for both populations except for the 1972 Bulloch County planting where infection percentages were very low.

Analyses of variance for 1971 plantings of population A *(Table 2)* indicated highly significant effects of locations and of families. Similar results were obtained from 1972 plantings. Comparable conclusions can be drawn from both analytical procedures except for the location × family interaction. In the binomial analysis of the 1971 planting, this interaction was significant at $p < 0.05$. For the other analyses, the F value for this genotype-environment interaction was slightly below this level of significance. In no case was the interaction strong although there were some shifts in family ranking at different locations.

Effects of locations and years are confounded in this study. In both Webster and Bulloch Counties, a higher percentage of trees were infected in 1971 plantings than 1972 plantings, but an almost entirely different group of families was involved. Thus, possibly different effects of years and locations are combined as environmental effects. Observations of infection at two ages indicate a fairly consistent average 7 percent increase in infection from the third to the fifth year *(Table 1)*.

Variance Components

Analyses were made of population-A data from individual planting locations and years, and for combined data from each planting year. Variance components were estimated for individual plantings and for the combined ana-

Table 3. — Family and family × location components of variance and standard error of components of fusiform rust infection in planted slash pine as estimated by two analytical procedures.

	Variance Components and Their Respective Standard Errors[a]			
	$\hat{\sigma}^2_{f}$	$S(\hat{\sigma}^2_{f})$	$\hat{\sigma}^2_{\ell f}$	$S(\hat{\sigma}^2_{\ell f})$
Binomial Analysis				
1971 planting				
Webster	0.0120	0.0024		
Taylor	0.0186	0.0047		
Bulloch	0.0157	0.0043		
Combined	0.0140	0.0025	0.0024	0.0012
1972 planting				
Webster	0.0142	0.0042		
Taylor	0.0161	0.0050		
Bulloch	0.0034	0.0017		
Combined	0.0110	0.0025	0.0006	0.0012
Transformed Plot Mean Analysis				
1971 planting				
Webster	58.3	12.8		
Taylor	77.1	19.4		
Bulloch	59.4	16.0		
Combined	62.8	10.9	8.1	5.6
1972 planting				
Webster	57.2	17.4		
Taylor	71.3	20.6		
Bulloch	13.8	8.4		
Combined	45.8	10.4	N.C.[b]	5.4

[a] $S(\sigma^2)$ = standard error of estimate of indicated component.

$\hat{\sigma}^2_{f}$ = estimate of family variance component.

$\hat{\sigma}^2_{\ell f}$ = estimate of family x location interaction component.

[b] N.C. = negative estimate of component.

lyses (*Table 3*). Standard errors of the family component (σ^2_f) were relatively small in all plantings as estimated by both statistical procedures. All estimates of family × location interaction components were relatively small and had relatively large errors of estimate.

Heritability Estimates

Estimates of individual tree heritability ($\hat{h}^2_i$) varied, depending on analytical procedure, planting year and location, ranging from 0.07 with plot mean data from the 1972 Bulloch County planting, which had a low infection percent, to 0.39 with the binomial analysis of data from the 1971 Webster County planting having the highest percent infection. Estimates from combined analyses by both analytical procedures were similar with the 1971 plantings giving higher estimates than 1972 plantings. In each case, the $\hat{h}^2_i$ estimate from combined analyses was intermediate to estimates from individual locations, approximating a mean value.

Estimates of family heritability ($\hat{h}^2_f$) were consistently high except from the 1972 Bulloch planting. Analyses combined over locations all gave estimates of 0.78 to 0.80.

Relationship Between Genetic Parameters and Mean Infection Percentage

One of the objectives of this study was to compare genetic parameters at different percentages of infection. Genetic parameters were regressed on infection percent as the independent variable. Third year data from all plantings were used, and, to cover a wider range of infection percents, fifth year data of the 1971 Webster County and the 1972 Bulloch County plantings were also included. Each genetic parameter was expressed as a function of the infection percent based on the regression model which provided the best fit. Results of these analyses are summarized in *Table 4*.

Binomial data. The regression line with best fit for total phenotypic variance was expressed as a parabolic function with maximum values at about 50% infection, tailing off symetrically with higher and lower infection percents (*Figure 1*). Estimates of additive genetic variance increased up to 40 percent infection, remained rather constant up to 80 percent, and decreased slightly at higher infection. As $\hat{h}^2_i$ is the ratio of these two parameters, the sharp reduction of total phenotypic variance at higher infection, accompanied by very gradual decrease in additive genetic variance, resulted in a straight line increase in heritability with increase in infection percent (*Figure 2*). Estimates of $\hat{h}^2_f$ responded similarly.

Transformed plot mean data

Estimates of additive genetic variance computed from the plot mean analyses had a relationship to mean infection percent similar to estimates of this component from bino-

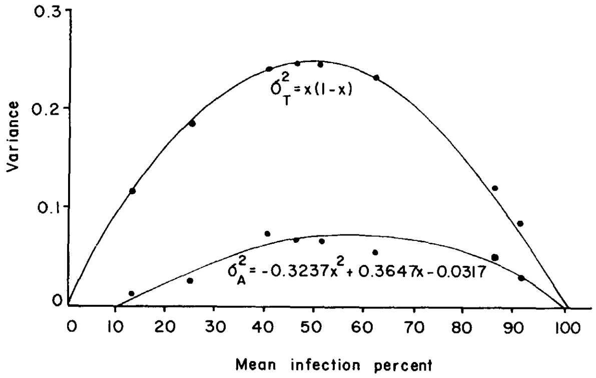

Figure 1. — Regressions of total phenotypic variance (σ^2_T) and additive variance (σ^2_A) of fusiform rust resistance computed from binomial scale data on mean infection percent.

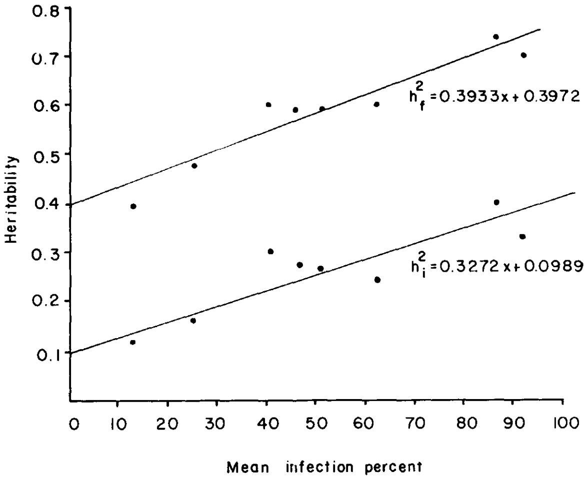

Figure 2. — Linear relationships between mean infection percents and the estimates of heritability of individual trees (h^2_i) and family means (h^2_f) computed from binomial data.

Table 4. — Analyses of variance for regression of variance components and heritabilities on infection percent.

Source of Variation	Binomial data								Transformed plot mean data			
	σ^2_T		σ^2_A		h^2_i		h^2_f		h^2_i		h^2_f	
	df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.
Regression	2	0.0202**	2	0.0010**	1	0.0308**	1	0.0615**	2	0.0207**	2	0.0402**
Deviation from Regression	5	0.0000	5	0.0001	6	0.0027	6	0.0062	5	0.0020	5	0.0016
R^2		0.999		0.855		0.743		0.909		0.805		0.909

**Significant at the 0.01 level.

Table 5. — Realized and expected percentage point gains from mass selection in progeny tests according to their mean infection percent computed from two analyses methods, transformed plot mean and binomial data.

Location	Age	Infection level (%)	Realized gain (% point)	Expected gain (% point)	
				Transformed plot mean data	Binomial data
Webster (1971)	3	85.6	23.6	18.1	20.5
	5	91.2	21.1	15.3	17.3
Taylor (1972)	3	46.2	19.4	21.4	19.2
	5	51.4	21.7	21.6	20.5
Bulloch (1972)	3	13.1	7.3	4.1	7.4
	5	24.9	12.5	11.4	12.0

mial analyses. However, total phenotypic variance estimates did not decline sharply with high infection. Consequently, the continuous increase in heritability estimates with infection percents higher than 70% was not evident *(Figure 3)*.

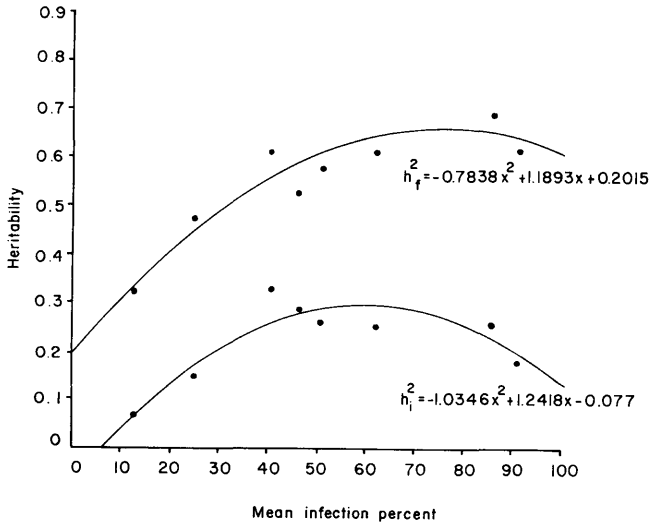

Figure 3. — Regressions of mean infection percents with the estimates of heritabilities of individual trees (h^2_i) and family means (h^2_f) computed from transformed plot mean data.

Individual tree heritability estimates from the various plantings in this study span nearly the entire array of published estimates for fusiform rust resistance of slash and loblolly pine. Barker[3]) and Blair[4]), in particular, have partitioned genetic variances and estimated heritability of rust resistance in loblolly pine. Their estimates range from 0.02 to 0.39, depending on the resistance index used, location, and year of planting. Rockwood and Goddard (1973) reported heritabilities of rust resistance in slash pine ranging from 0.05 to 0.24, depending on location and year. We suggest that many of the differences in heritability estimates may be due to differences in infection level in the various studies. The environment of the study site has a pronounced effect on phenotypic variation, and regarding rust resistance, the relative abundance of rust inoculum is a major component of the environment.

Logically, if the infection percent is low, the chance for trees to escape infection is high, whether or not they are resistant, and low heritability values would be indicated. This is evident in heritability estimates calculated from both analytical procedures. If infection percents are high due to high virulence or quantities of inoculum in the area, most trees are infected and degrees of resistance may not be expressed. Thus, differences attributed to locations or to years may be largely due to differences in inoculum load.

Estimation of Genetic Gain

A major objective of partitioning variance to estimate heritability is to provide an estimate of genetic gain and to compare alternative breeding procedures. Gain implies some restriction of the breeding population, i.e., application of selection. Gain is dependent upon heritability of a trait and the selection pressure applied. In this study, considering fusiform rust resistance as an "all-or-none" trait, only disease-free trees were selected to establish population-B. As over 90% of the original population was infected, the selection intensity was 1.75 (i, in the gain formula for mass selection) (from Nanson, 1967). Thus, selection intensity was directly related to infection percent in the population where selection pressure was applied. As a result of low infection and low selection pressure, genetic gain for rust resistance in parent population-A was practically zero.

Using genetic parameters estimated from population-A (considered random for rust resistance) and the selection intensity applied in population-B, expected gains from mass selected population-B were calculated from both binomial and transformed plot mean data *(Table 5)*. Performance of progenies of population-B provided an indication of realized gain. Expected gains calculated from the binomial analyses were in closer agreement with realized gain at all locations than estimates from the transformed plot mean analyses. Expected gains, however, were silghtly but consistently lower than realized gains.

It is noteworthy that expression of gain is associated with infection percent in the progeny population although

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[3]) Barker, J. A., 1973: Location effects on heritability estimates and gain predictions for ten-year-old loblolly pine. Ph. D. thesis, North Carolina State University.

[4]) Blair, R. L., 1970: Quantitative inheritance of resistance to fusiform rust in loblolly pine. Ph. D. thesis, North Carolina State University.

not as closely as selection intensity is associated with infection percent in the parent population. If there were few infections in progeny populations, differences between resistant and susceptible progenies were low and genetic gain was not fully expressed. Mass selection with various selection intensities and gains in progeny populations exposed to a range of infection percents are graphically depicted in *Figure 4.*

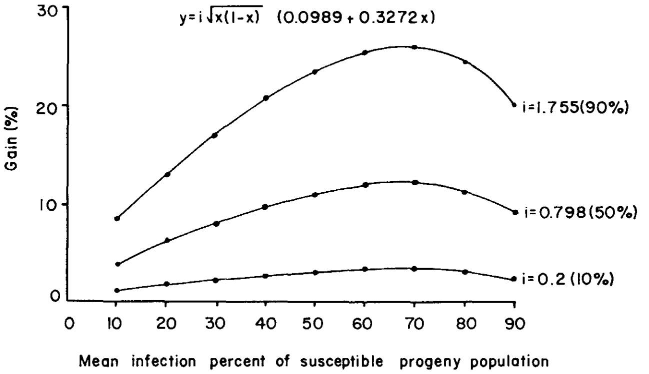

Figure 4. — Variation of expecetd gain from mass selection with different mass infection percents in progeny populations. Different i values represent the selection differentials in rust infection in the parent population when mass selection was performed.

Results reported here reinforce Barker's conclusing that heritability and gain estimates based on a single planting have limited reliability. This is particularly true if the estimates are used to predict genetic improvement over a range of planting locations. For practical application, gain estimates based on the combined analysis of data from several locations or years are more likely to be realistic. However, the full potential improvement from use of resistant planting stock will not be expressed in planting locations with low potential infection levels.

Mass selection plus progeny testing

Although there was no mass selection gain in population-A, selection on the basis of progeny performance could provide substantial improvement in rust resistance. Additional selection in population-B would also be beneficial. Selection intensity in this case is determined by the proportion retained of trees originally selected. Calculations were made assuming 25% of the parents would be retained. The procedure provides estimates of genetic gains if the best parent trees on basis of progeny performance were used to establish a new orchard (*Table 6*). Twenty-five percent of the trees in population-A, established in a new orchard, would produce progenies with 21 to 25% genetic gain if progenies were planted in areas with substantial exposure to rust.

Selection in a secound generation

Selection among progenies for establishment of a second generation orchard is a widely proposed procedure for obtaining continued genetic improvement of forest trees. For calculation of potential gains through this procedure, selection of the best 25% of the families for rust resistance was assumend. Within family selection pressure was dependent on the extent of infection at each study location. Selection of disease-free trees among the best families is assumed. Selection of the best families will provide a rather consistent gain in all but areas of very low infection. Within family selection pressure, however, is closely related to disease level and only in Webster County with the highest disease incidence is the expected gain from second generation selection higher than that expected from selection of

Table 6. — Prediction of gain in fusiform rust resistance from three methods of selection in two slash pine populations.

Procedure	Location and Planting Year	Expected gain (% point)	
		Population-A	Population-B
Mass selection[a]	Webster (1971)	0	20.5
	Taylor (1972)	0	19.2
	Bulloch (1972)[c]	0	12.0
Mass selection plus progeny testing[b]	Webster (1971)	22.6	20.5 + 16.2 = 36.7
	Taylor (1971)	24.8	
	Bulloch (1971)	23.2	
	Webster (1972)	21.5	
	Taylor (1972)	23.6	19.2 + 16.8 = 36.0
	Bulloch (1972)[c]	14.4	12.0 + 10.2 = 22.2
Family and within family selection in second generation[b]	Webster (1971)	11.3 + 13.2 = 24.5	20.5 + 8.1 + 9.3 = 37.9
	Taylor (1971)	12.4 + 6.7 = 19.1	
	Bulloch (1971)	11.6 + 6.2 = 17.8	
	Webster (1972)	10.8 + 6.7 = 17.5	
	Taylor (1972)	11.8 + 5.8 = 17.6	19.2 + 8.4 + 3.3 = 30.9
	Bulloch (1972)[c]	7.2 + 1.6 = 8.8	12.0 + 5.1 + 0.7 = 17.8

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[a] More than 9/10 selected in population-A on basis of rust resistance, 1/10 in population-B.

[b] For both population, 1/4 trees or families selected on basis of progeny tests. Within family selection dependent upon infection percent at test site.

[c] Calculated on basis of infection percent at age 5, all others at age 3.

the best parents for a new orchard. Second generation selection gain estimates were substantial in all locations with infection higher than 50% and opportunity is provided for selection for other traits within the resistant families. It should be emphasized, however, that for highest gain in resistance, selection must be performed in areas where fusiform rust is most severe.

Conclusions

Comparisons of analyses of data taken at the third and fifth years suggest that genetic parameters of rust resistance are well expressed by the third year. Slash pines are most susceptible to fusiform rust at early ages, particularly to infections likely to cause severe loss or mortality. Only when early infection levels were low, resulting in low variances, did accumulation of additional infections over the additional two years have appreciable effect on heritability estimates.

Both statistical procedures used in data analysis allow similar conclusions to be drawn. Transformation of plot means to the square root of arcsins weights family and other means somewhat differently than with the binomial analysis, depending upon the number of trees surviving per plot. However, in all plantings reported here, survival was uniformly near 90% (trees killed by rust were counted as infected) and weighting had only slight effect on the analyses. It was suspected that the continuous increase in heritability at very high infection percents indicated by the binomial analysis would overestimate gain at extremely high infection percentages. However, realized gain agreed closely with predicted gain from the binomial analyses. For this reason we conclude that the binomial analysis is the superior analytical procedure.

Lacking means to identify resistant trees other than freedom from fusiform rust galls, intensive mass selection can be applied only where the disease is abundantly present. Selection intensity is thus directly related to the infection percent in the population from which selections are made. The gain to be achieved from such selections can be fully expressed only when the progeny population is also heavily exposed to the disease.

Therefore:

1. Mass selection for rust resistance should be done in heavily infected stands.

2. Progeny tests should be conducted in areas with moderate to heavy exposure to rust if progeny test results are to be used for reselection among parent trees. Several such tests are preferable for reliable assessments of trees in the parent population.

3. For good gains in resistance from selections made in the progeny population, the second generation population must be severely exposed to rust so that the within-family selection different will be high. Otherwise, with comparable intensities of family selection, higher resistance gains can be achieved through reselection in the parent population.

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