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Host Relationships of Fusiform Rust Disease

II. Genetic Variation and Heritability in Typical and South Florida Varieties of Slash Pine

By C. D. NELSON[1]), R. C. SCHMIDTLING and R. L. DOUDRICK

USDA Forest Service, Southern Institute of Forest Genetics, 23332 Hwy 67, Saucier, MS 39574, USA

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Summary

Seedlings of wind-pollinated families from 6 sources of slash pine (*Pinus elliottii* var. *elliottii* and var. *densa*) were tested for development of fusiform rust disease using 2 sources of *Cronartium quercuum* f. sp. *fusiforme* inocula. The seed source origins ranged from 24.5 °N latitude at the southern tip of Florida (*P. e. densa*), to 30.25 °N latitude, in north central Florida (*P. e. elliottii*). All seedlings received basidiospores of both inocula; inoculum of each source was applied to a single, separate shoot. Differences among varieties of slash pine were significant, with less pycnial sporulation present on seedlings from southern seed sources. Differences between inocula were significant within *P. e. elliottii* only, suggesting increased specificity for *P. e. elliottii* hosts and these inocula, although inocula x family-within-seed-source interactions were not significant. Heritability estimates for infection or sporulation on an individual seedling basis ranged from 0 to 0.45 within *P. e. densa* and from 0.20 to 0.59 within *P. e. elliottii.* On a family mean basis, heritability estimates were higher, ranging up to 0.58 within *P. e. densa* and 0.71 within *P. e. elliottii.* Diverse sources of reaction to *C. q. fusiforme* appear to be present in *P. e. densa,* suggesting a backcross breeding approach in which genes for reaction are introgressed into fast-growing populations of *P. e. elliottii.* The relatively large individual tree-based heritability estimates in *P. e. elliottii* should help to expedite the introgression process.

Keywords: Pinus elliottii var. *elliottii, Pinus elliottii* var. *densa, Cronartium quercuum* f. sp. *fusiforme.*

FDC: 181.41; 165.53; 443; 165.3; 172. *Cronartium quercuum;* 174.7 *Pinus elliottii.*

Introduction

Fusiform rust, caused by *Cronartium quercuum* (BERK.) MIYABE ex SHIRAI f. sp. *fusiforme*, is the most serious disease affecting the commercially important southern pines. Research efforts have identified sources of genetic resistance to the disease (JEWELL and MALLETT, 1964; WELLS and WAKELEY, 1966; KINLOCH and STONECYPHER, 1969; DINUS, 1971; SCHMIDT et al., 1981; POWERS and KRAUS, 1986; SLUDER, 1986; POWERS and KUHLMAN, 1987; KUHLMAN and POWERS, 1988; HODGE et al., 1989). Breeding programs have attempted to incorporate this resistance into elite families (ZOBEL et al., 1971; POWERS et al., 1979; WEIR and GODDARD, 1986). Recent research efforts have focused on defining the nature of the interaction of host and pathogen with hopes of developing better strategies for controlling the disease in high-production environments (NANCE et al., 1992; NELSON et al., 1993).

In artificial inoculation experiments, disease development is usually scored as the percentage of galled seedlings. Some efforts have been made to quantify the size and shape of galls as a further indication of resistance. SNOW et al. (1990) suggested that round galls on loblolly pine (*Pinus taeda* L.) seedlings indicated restricted pathogen development and, therefore, more host resistance than long, fusoid-shaped galls. WALKINSHAW et al. (1980) identified several symptoms that, when combined in regression equations, served to better predict field resistance of loblolly and slash pine (*P. elliottii* ENGELM. var. *elliottii*) families. In a unique effort to quantify *C. q. fusiforme* aecial sporulation on slash pine, GRIGGS (1984) scored wind-pollinated progenies of several elite parents in 2 field environments and found some families with high percentages of infection and low percentages of sporulation and vice versa. These results were verified in a second test of

[1]) Present address: Southlands Experiment Forest, International Paper Co., Bainbridge, GA 31717, USA

control-pollinated progenies of the same parents (C. D. NELSON, unpublished).

Given the potential for shifting pathotype frequencies in populations of *C. q. fusiforme* (SNOW et al., 1975, 1976), it is important to investigate the relationship between pine infection and subsequent sporulation of the pathogen. Pathotypes of *C. q. fusiforme* that are not only capable of infecting pines but also of subsequently sporulating (pycnia and aecia) may cause a relatively rapid increase in frequency of virulent pathotypes in the fungus population. In the present study, we tested seedlings of wind-pollinated families from six sources of slash pine distributed throughout its native range in Florida, with the objective of determining the genetic variance and heritability of host reaction to *C. q. fusiforme* in the native slash pine population, which may be useful in planning breeding strategies. In the accompanying article by DOUDRICK et al., an analysis of geographic variation of these seed sources and sources from related species native to the Caribbean basin is provided.

Materials and Methods

Seedlings

Seeds of 6 sources of slash pine, 3 *P. e. elliottii* and 3 *P. e. densa* were obtained for study (*Table 1*). The origins of sources FL1 to FL6 ranged from 24.5 °N latitude on Big Pine Key, Monroe County at the southern tip of Florida for *P. e. densa* to 30.25 °N in Baker County in north-central Florida for *P. e. elliottii.* From each source, an attempt was made to collect 25 to 30 wind-pollinated cones from 16 trees. All collections, separated by parent tree, were made in the late summer of 1991 (late August in the south to early September in the north). Trees were not selected for any characteristic except the availability of cones and a spacing of at least 50 m from another parent tree. Each source (area ≤ 1 mi^2) represented a natural stand or parts of a few adjacent natural stands.

The reduced numbers of families and seedlings per family, especially in source FL1, were due to low numbers of filled seeds per cone and low germination rates. Most cones on the FL1 trees had opened prior to cone collection, resulting in collections from fewer trees and in fewer extractable seeds per tree. This problem was also evident, but to a lesser degree, in sources FL2 and FL3 (*Table 1*).

Seeds were germinated and grown in a glasshouse (described in detail in the companion paper by DOUDRICK et al.). Seedlings were hedged when the epicotyls had elongated 6 cm to 8 cm to produce 2 or more shoots for inoculation, and inoculated when new shoots emerged 1 cm to 2 cm.

Twelve weeks after inoculation, seedlings were planted in the nursery at Harrison Experimental Forest (HEF). Seedlings were lifted and scored for disease development in November 1993. A total of 1,149 seedlings representing 77 families were inoculated with the 2 inocula of *C. q. fusiforme* (*Table 1*).

Inoculation

Two inocula of *C. q. fusiforme* were used. Both inocula originated as collections of aeciospores from single galls on 10-year-old trees planted in Madison County in north central Florida; 2 galls on loblolly pine (CCA), origin Livingston Parish, Louisiana; and 3 galls on slash pine (SC) of local origin. The 2 inocula were applied on each of 10 hedged seedlings per family per replication, with 2 replications. From 9 to 16 families per source of slash pine were inoculated (*Table 1*), with all seedlings receiving basidiospores of both inocula, one shoot for each inoculum. The methods for producing inocula and the inoculation techniques are described in detail in the accompanying article by DOUDRICK et al.

Data

Presence or absence of galls and spores (pycniospores) for each inoculum was scored as phenotypes of disease development. These data were used to obtain nine binomial variables for analysis--CCA-Gall, SC-Gall, GALL, CCA-Spore, SC-Spore, SPORE, CCA-Sp, SC-Sp, and SP-- where the prefix *CCA* or *SC* indicate the inoculum. *Gall* is based on the total number of seedlings and is 1 for infected and 0 for not infected. *Spore* is 1 for spores present and 0 for no spores. *Sp* is the same as *Spore* except it is based only on infected seedlings. Reaction variables in uppercase and without the inoculum prefix have the same

Table 1. – Locations and varieties of slash pine seed sources and number of wind-pollinated families per source tested for reaction to two inocula of *Cronartium quercuum* f. sp. *fusiforme.*

Seed source	County	Cooperator	Latitude °N	Variety	No. of families	Seedlings No./Family
FL1	Monroe	USFWS	24.5	*P. e. densa*	9	5.4
FL2	Collier	Collier Ent.	26.3	*P. e. densa*	12	13.2
FL3	Polk	USAF	27.5	*P. e. densa*	11	14.0
total				*P. e. densa*	32	
FL4	Orange	Disney	28.3	*P. e. elliottii*	14	16.3
FL5	Marion	USDA FS	29.3	*P. e. elliottii*	15	17.5
FL6	Baker	USDA FS	30.3	*P. e. elliottii*	16	18.6
Total				*P. e. elliottii*	45	
Grand total					77	
Inoculum source						
SC	Madison	USDA FS	30.4	Collected on *P. e. elliottii*		3 Trees
CCA	Madison	USDA FS	30.4	Collected on *P. taeda*		2 Trees

Table 2. – Means and F-test results[a]) comparing varieties of slash pine and seed sources-within-varieties for reaction to 2 inocula of *Cronartium quercuum* f. sp. *fusiforme.*

		Variety differences[b]		Source differences[b]					
		P. elliottii		*P. e. densa*					*P. e. elliottii*
Inoculum	Trait	*densa*	*elliottii*	FL1	FL2	FL3	FL4	FL5	FL6
		---------- Percent ----------							
CCA	Gall	73	60 n.s.	75	72	71	61	59	58 n.s.
SC	Gall	61	65 *	72	44	66	58	69	69 ***
Either	GALL	86	80 n.s.	92	78	89	76	81	83 **
CCA	Spore[c]	29	39 ***	36	21	30	33	43	40 n.s.
SC	Spore	30	45 ***	41	13	34	34	49	53 ***
Either	SPORE	35	58 ***	49	29	45	46	65	64 ***
CCA	Sp[d]	41	63 ***	53	31	40	51	73	65 **
SC	Sp	47	67 ***	56	31	53	56	71	74 ***
Either	SP	59	70 ***	64	37	52	56	80	76 ***

[a]) Statistical model, Y = block + var + block • var + source(var) + block • source(var) + error, all factors were considered fixed.
[b]) Significance levels: *) = P < 0.05, **) = P < 0.01, ***) = P < 0.001, and n.s. = not significant.
[c]) Sporulation expressed as a percentage of *total* number of seedlings with pycnial sporulation.
[d]) Sporulation expressed as a percentage of *infected* seedlings with pycnial sporulation.

meaning but are for seedlings infected by either inocula. All variables were analyzed by a general linear models procedure (SAS, 1990).

Heritability estimates were calculated for each variety on an individual (h_i^2) and half-sib family (h_f^2) mean basis (BECKER, 1984) with the following formulas:

$h_i^2 = 4 \bullet v_{f(s)} / (v_{f(s)} + v_{b \bullet f(s)} + v_e)$ (1) Individual tree basis

$h_f^2 = v_{f(s)} / (v_{f(s)} + v_{b \bullet f(s)} / r + v_e / rn)$ (2) Half-sib family mean basis

where $v_{f(s)}$ = family-in-seed source variance component,

$v_{b \bullet f(s)}$ = block X family-in-seed source variance component,

v_e = error variance,

r = number of blocks, and

n = number of trees per plot.

Phenotypic, genetic, and environmental correlation coefficients were calculated for the following pairs of variables: CCA-Gall by SC-Gall, CCA-Spore by SC-Spore, CCA-Gall by CCA-Spore, and SC-Gall by SC-Spore (FALCONER, 1982).

Results and Discussion

Differences among varieties of slash pine were significant ($P < 0.05$) for SC-Gall and all sporulation variables (*Table 2*). Seedlings of *P. e. elliottii* incurred more infection with SC inoculum and also more pycnial sporulation than those of *P. e. densa.* Differences among seed sources-within-varieties were significant for all variables except CCA-Gall and CCA-Spore (*Table 2*). In general, a north-south trend was observed among seed sources for reaction to both inocula, with more infection and sporulation occurring in the northern sources with the exception of the southernmost source, FL1. The deviation of FL1 from the north-south trend may be due to introgression of genes from *P. caribaea* MORELET (see accompanying article by DOUDRICK et al.).

Differences among families-within-seed sources (by varieties) were significant for all variables in *P. e. elliottii* and for CCA-Gall and GALL only in *P. e. densa* (*Table 3*). Heritability estimates, being primarily a function of among-family

Table 3. – F-test results[a]) comparing families-within-seed sources of 2 varieties of slash pine and heritability estimates for reaction to 2 inocula of *Cronartium quercuum* f. sp. *fusiforme.*

		P. e. densa			*P. e. elliottii*		
Inoculum	Trait	Difference[b]	Individual	Family	Difference[b]	Individual	Family
			----------h^2--------			---------h^2---------	
CCA	Gall	***	0.45	0.58	**	0.34	0.54
SC	Gall	n.s.	0.20	0.32	*	0.20	0.41
Either	GALL	**	0.39	0.53	**	0.38	0.59
CCA	Spore[c]	n.s.	0.05	0.10	*	0.33	0.51
SC	Spore	n.s.	0.00	0.00	**	0.41	0.60
Either	SPORE	n.s.	0.17	0.25	***	0.59	0.71
CCA	Sp[d]	n.s.	0.19	0.26	*	0.35	0.39
SC	Sp	n.s.	0.00	0.00	**	0.43	0.50
Either	SP	n.s.	0.20	0.27	**	0.53	0.58

[a]) Statistical model, Y = block + source + block • source + fam(source) + block • fam(source) + error, all factors were fixed, except fam(source) and block • fam(source).
[b]) Significance levels: *) = P < 0.05, **) = P < 0.01, ***) = P < 0.001, and n.s. = not significant.
[c]) Sporulation expressed as a percentage of *total* number of seedlings with pycnial sporulation.
[d]) Sporulation expressed as a percentage of *infected* seedlings with pycnial sporulation.

variance, generally reflected these differences--the more significant the family differences, the higher the heritability estimates (*Table 3*). Heritability estimates associated with significant ($P < 0.05$) F-tests for families were considered to be significantly greater than zero. Within *P. e. elliottii,* heritabilities were moderate to high, as expected, given the hypothesized complementary genetic interaction of host and pathogen (KINLOCH and WALKINSHAW, 1990; DOUDRICK and NELSON, 1993; NELSON et al., 1993). Heritability estimates within *P. e. densa* were quite variable and generally smaller than for *P. e. elliottii,* although seed source differences within *P. e. densa* were substantial (*Table 2*). No heritability trends were apparent with respect to Gall vs. Spore or CCA vs. SC.

Phenotypic, genetic, and environmental correlation coefficients are presented in *table 4* for CCA-Gall by SC-Gall, CCA-Spore by SC-Spore, CCA-Gall by CCA-Spore, and SC-Gall by SC-Spore. A lack of family variance for SC-Spore within *P. e. densa* precluded the opportunity to calculate genetic and environmental correlations involving SC-Spore. Most of the genetic estimates were ≥ 1.0, indicating that the same genes are largely responsible for host reaction to the 2 inocula and expression of the 2 reaction variables with the same inoculum. An exception was within *P. e. elliottii* for CCA-Gall by CCA-Spore, although the estimate was large (+0.82).

The environmental correlations were essentially zero for all pairs involving the same reaction variables, but were positive and moderately large for different reaction variables. The large environmental correlations in these cases are indicative of strong auto-correlation between the variables for the same inoculum. In each case, the phenotypic correlations were positive and similar, ranging from 0.21 to 0.38 for Gall by Gall and Spore by Spore class of correlations and from 0.41 to 0.68 for Gall by Spore correlations. Phenotypic correlations are not very good indicators of the genetic relationships in terms of host reaction to the 2 inocula. The predictive value, R^2, of one inoculum to the other on different shoots of the same seedling (i.e., clonal basis) is <15%.

Differences between inocula were not significant on seedlings of *P. e. densa,* but were significant for Gall and Spore on those of *P. e. elliottii* (*Table 5*). Within *P. e. elliottii,* inoculum SC produced more infection and sporulation than did CCA (*Table 2*). Inoculum x seed source interaction effects were significant for Gall on seedlings of both varieties and for Spore on those of *P. e. densa* only. In general, SC produced higher infection and sporulation of pycnia percentages on seedlings from northern sources than southern sources, whereas the opposite was true for CCA (*Table 2*). It may be significant that the SC inoculum was collected on slash pine very close to the origin of FL6, whereas CCA was collected at the same location, but on loblolly pine (see accompanying by DOUDRICK et. al.). Inoculum x family-within-seed-source interactions were not significant for any variable in either variety (*Table 5*), which was expected because the genetic correlations between inocula were very large for both Gall and Spore (*Table 4*).

Results from this study suggest that *P. e. densa* may contain useful genes for host reaction to fusiform rust disease. This finding may be especially so when considering the percentage of seedlings infected and producing pycnia spores. It appears that the highest frequency of useful genes for the inocula tested occur south of the FL3 source, within the transition zone between the 2 varieties (SQUILLACE, 1966). The phenotype for resistance to *C. q. fusiforme* in *P. e. densa* does not appear to be strictly related to slower growth rates as seedlings of FL2 grew faster than those of FL1, but FL2 seedlings showed a lower percentage infection (see accompanying article by DOUDRICK et al.). Large genetic differences for infection percentages within

Table 4. – Phenotypic (r_P), genetic (r_G), and environmental (r_E) correlation coefficients between 2 inocula of Cronartium quercuum f. sp. fusiforme and 2 reaction variables on families within 2 varieties of slash pine.

	P. e. densa			*P. e. elliottii*		
	r_P	r_G	r_E	r_P	r_G	r_E
CCA-Gall by SC-Gall[a]	0.21	1.0	-0.14	0.25	1.00	-0.01
CCA-Spore by SC-Spore	0.38	--[b]	----	0.35	1.00	-0.03
CCA-Gall by CCA-Spore	0.41	1.0	0.36	0.67	0.82	0.59
SC-Gall by SC-Spore	0.52	---	----	0.68	1.00	0.57

[a]) CCA and SC identify inocula. The Gall trait is the percentage seedlings infected; the Spore trait is the percentage of total seedlings with pycnial sporulation.
[b]) The lack of family variance precluded of genetic and environmental correlations involving the trait SC-Spore.

Table 5. – F-test results[a]) comparing 2 inocula of *Cronartium quercuum* f. sp. *fusiforme* for disease development on seedlings of families-within-seed sources of 2 variaties of slash pine.

	P. e. densa			*P. e. elliottii*		
	Gall[b]	Spore[b]	Sp[b]	Gall[b]	Spore[b]	Sp[b]
Inoculum	n.s.	n.s.	n.s.	*	**	n.s.
Seed source	**	***	**	n.s.	***	***
Inoc X Source	*	*	n.s.	*	n.s.	n.s.
Family(Source)	***	n.s.	n.s.	**	***	**
Inoc X Fam(S)	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.

[a]) Statistical models, Y = block + inoc + source + all interactions + residual, all effects were fixed, and Y = fam(source) + block • fam(source) + inoc • fam(source) + block • inoc • fam(source) + error, all effects were random.
[b]) Significance levels: *) = $P < 0.05$, **) = $P < 0.01$, ***) = $P < 0.001$, and n.s. = not significant.

families with rapidly growing seedlings were also found, and these differences appear not to be related to growth rate (C.D. NELSON, unpublished). Successful use of the *P. e. densa* germplasm in a breeding program would require a backcross breeding strategy, where genes for host reaction from *P. e. densa* are introgressed into fast-growing *P. e. elliottii* populations. The moderately high individual heritability estimates within *P. e. elliottii* should help to expedite this breeding process.

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Loss of Genetic Diversity Following Selection from Populations with a Family Structure

By R.-P. WEI[1])

Department of Forest Genetics and Plant Physiology, Swedish University of Agricultural Sciences, S-901 83 Umeå, Sweden

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Summary

Loss of genetic diversity for selection from populations of unrelated families was analytically formulated for a series of selection strategies. Two components are identified, one caused by selection and another by random drift. The total loss of genetic diversity increases as selection becomes more based on family information. Selection dominates the loss except in cases where significantly more within-family deviations are used, where random drift becomes more important. When heritability is higher and the members of families are more closely related, selection effect becomes stronger but drift effect becomes weaker. The total loss of diversity and selection effect become worse with large family number and size. Drift effect decays with small family number and large family size. The significance of controlling the loss of genetic diversity in breeding populations is discussed in both breeding and conservation biology aspects.

[1]) Present address: Department of Renewable Resources, University of Alberta, Edmonton, Alberta, Canada T6G 2H1