PATTERNS OF FUSIFORM RUST INCREASE AND THEIR IMPLICATIONS FOR SELECTION AND BREEDING

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Abstract.--When increase of fusiform rust was observed for 12 years in two progeny tests of six open-pollinated slash pine families, family rankings did not change appreciably from the 3rd year after planting. Noncumulative estimates of rust incidence reflected family rankings as accurately as cumulative estimates. Noncumulative and, especially, cumulative estimates were highly correlated with results from artificial inoculations. Cumulative records, however, are necessary for accurate second generation selection. Without individual tree histories, substantial numbers of selections would be escapes or previously galled trees that became rust free through natural pruning. Such errors are quite frequent in progeny tests with light to moderate infection levels. The course of rust-associated mortality paralleled that for total infection and stem infection. Infected trees in one family were better able to withstand and survive infection than those in a similarly infected family.

Additional keywords: Cronartium fusifoLme, Pinus elliottii var. elliottii, P. taeda, tolerance, epidemiology.

INTRODUCTION

Fusiform rust (Cronartium fusiforme Hedge. & Hunt ex Cumm.) limits efficient management of slash (Pinus elliottii var. elliottii Engelm.) and loblolly (P. taeda L.) pines over much of their commercial range. Genetic variation in resistance is substantial in both pines (Rockwood and Goddard 1973, Stonecypher et al. 1973) and resistance breeding is widely used for reducing losses.

Despite much progress, commercial breeding programs still rely on few resistant parent trees, and much remains unknown about different types of resistance and their relative frequencies. Pathogenic variability in C. fusiforme is considerable, and forms are capable of negating resistance in some slash pines (Dinus et al. 1975). Increased emphasis must therefore be placed on acquiring more resistant parents and determining the nature of their resistance so that future planting stock contains a variety of resistant genes.

This report describes the course of rust infection in two open-pollinated slash pine progeny tests. Patterns of rust increase over the 12 years after

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planting illustrate how much infection occurred in each of six families, when and how rapidly infections accumulated, and how each family responded. Results suggest several types of resistance are available even in this limited sample of slash pine. Implications concerning selection for second generation breeding and relationships between artificial inoculation results and field performance are also discussed.

MATERIALS AND METHODS

Open-pollinated progeny tests of six south Mississippi slash pines were established in 1963 near Gulfport, Mississippi (Location 1), and in 1964 near Bogalusa, Louisiana (Location 2). All six families were planted at Location 1 in a randomized block design. Each family is represented by a 30-tree plot in each of seven blocks. At Location 2, five families, excluding Family 4, are represented by 30-tree plots in each of five blocks. Early field performance and its relationship to artificial inoculation results were described earlier (Dinus 1969).

Galled trees, trees with stem galls, and trees killed by rust at Location 1 were counted annually the first 6 years after planting, and at the end of the 9th, 10th, and 12th growing seasons. The same counts were taken annually the first 4 years at Location 2, and at the end of the 6th, 10th, and 12th growing seasons. Data were summarized to give three cumulative estimates and one noncumulative estimate of rust incidence and severity.

Cumulative estimates included: (1) cumulative percent galled (CPG)--total trees having had at least one gall as a percentage of trees living plus those killed by rust; (2) cumulative percent stem galled (CPSG)--total trees having had at least one stem gall as a percentage of trees living plus those killed by rust; and (3) cumulative percent rust-associated mortality (CPRAM)--trees killed by rust as a percentage of trees living plus those killed by rust. In developing cumulative estimates, trees dying of causes other than rust were removed from the data during and after the year in which they died. Such mortality was greatest in the first 2 years after planting, was not correlated to relative family resistance (Dinus 1969), and is not expected to influence outcome of the analyses.

The noncumulative estimate was percent galled (NCPG)--the number of trees having at least one gall on a given observation date as a percentage of trees living at that time. NCPG is a point-estimate variable and has been used frequently to evaluate rust incidence in progeny tests, provenance trials, and commercial plantations (Wells and Wakeley 1966, Rockwood and Goddard 1973, Stonecypher et al. 1973).

Percentages for each variable were calculated on a plot basis and subjected to analyses of variance for the randomized block design. Analyses combining locations were not attempted because location effects would have been confounded with different planting dates and numbers of families. Family differences at various times were compared by Duncan's multiple range test. Relationships between variables were quantified by simple regression analyses of plot

percentages. Similar analyses were used at various times to relate mean family performance after artificial inoculation to CPG and NCPG. All tests of statistical significance were at the 0.05 level.

RESULTS AND DISCUSSION

Cumulative disease increase

Plots of CPG against time indicate that few trees were galled during the first 2 years after planting, but that the number of galled trees increased rapidly between the second and sixth seasons, and then more slowly until it approached a maximum within 10 years (fig. 1). Differences between locations are apparent in that the largest increases at Location 2 occurred a year or two after those at Location 1. Nevertheless, maximum CPG was greater at Location 2. Our findings confirm earlier descriptions (Griggs and Schmidt In press) and conform to patterns of disease increase observed in other host/pathogen systems (Kranz 1974).

At both locations, the largest increases occurred when height and crown size were increasing rapidly. Rates of increase lessened thereafter because many new infections occurred on trees already galled. Some susceptible but previously rust-free trees were infected each year, but fewer were as the trial progressed.

Patterns of increase were similar for all families (fig. 1), but degree of infection among families has been significantly different since the 2nd year, and rankings have remained the same or become more definite (table 1). Throughout the trial, Family I had significantly fewer galled trees than any other family. Families 5 and 6 were always most frequently galled, while Families 2, 3, and 4 were intermediate. In terms of galls per galled tree, however, Family 2 had nearly three times more potentially sporulating galls 6 years after planting than other intermediate families. The implications are twofold. First, families like Family 2 can be expected to be in greater danger of damage or death than families with similar amounts of infection but fewer galls per tree. Second, such materials may serve to intensify disease by producing more inoculum than others with equivalent infection. This observation also suggests, albeit indirectly, that families with fewer than average galls may occur and that they should be sought for future breeding.

Patterns of increase in CPSG mirror those for CPG with only minor exceptions (fig. 1). The curves are essentially identical until the second or third growing season--an observation not unexpected because planted slash pines have relatively few branches available for infection until then. Also, CPSG levels off later and at lower levels. The slower increases are expected because several years are 'required for branch galls to grow into the stem.

Family rankings have been stable; differences have been significant and have continued to increase since the 2nd year after planting. At last measurement, Family I had the fewest trees with stem galls (table 1), Families 5 and 6 were the most frequently galled, while the others were intermediate.

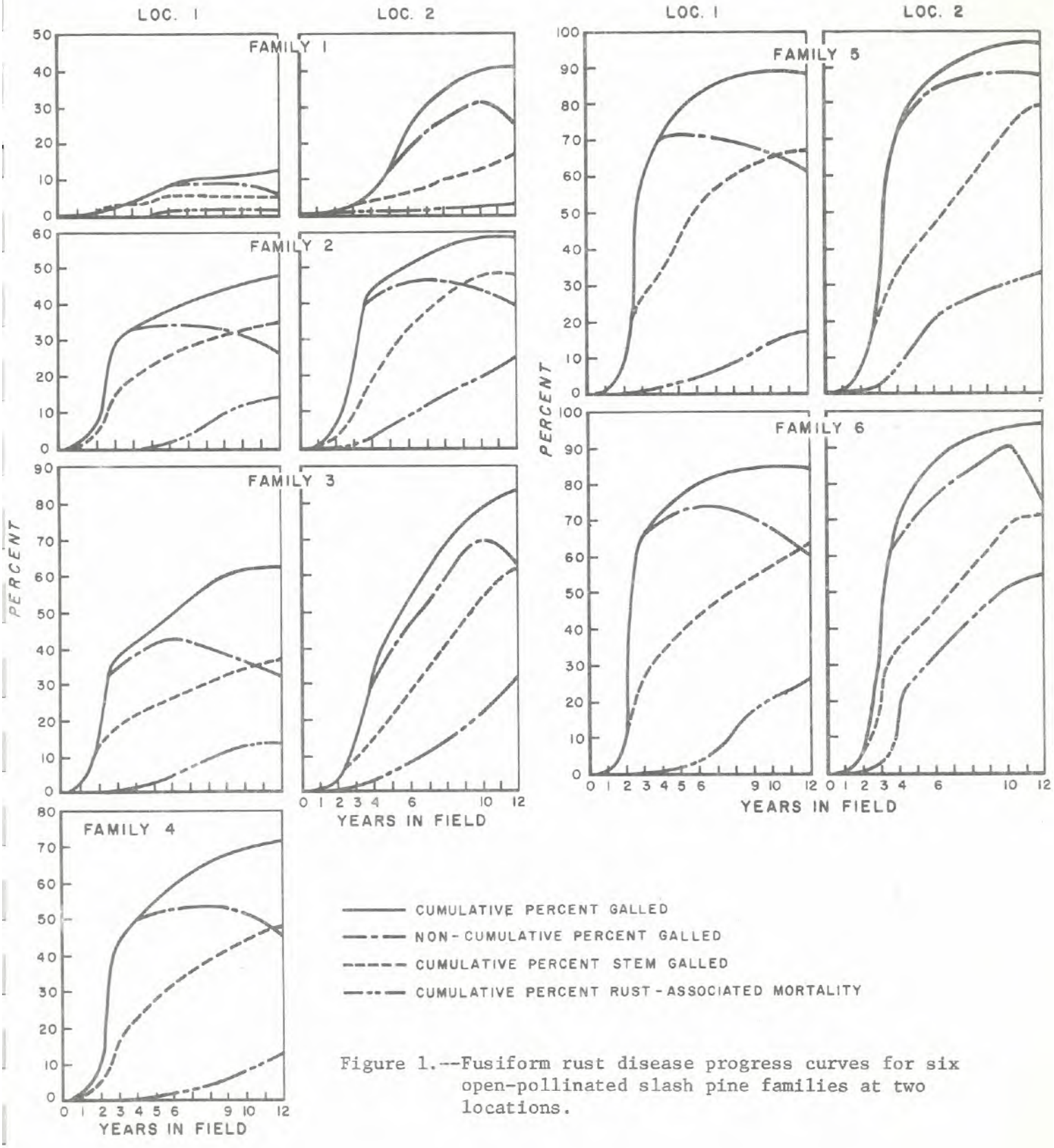

Figure 1.--Fusiform rust disease progress curves for six open-pollinated slash pine families at two locations.

the least and Family 6 the most deaths (table 1). Family 5 proved intermediate, though it was one of the most susceptible families in terms of CPG and CPSG. In fact, Family 5 had significantly less CPRAM than Family 6 despite having a higher frequency of trees with stem galls. Hence, infected trees within Family 5 withstood and survived infection better than trees in an otherwise equally susceptible family, and as well as families with 20 percentage points less infection in terms of CPG and NCPG (fig. 2). This greater survival resulted in an average plot volume approaching that of families with far less overall infection. Moreover, this tolerance was observed at both locations and was particularly evident in the most heavily infected plantation. These results strongly suggest that tolerance is heritable.

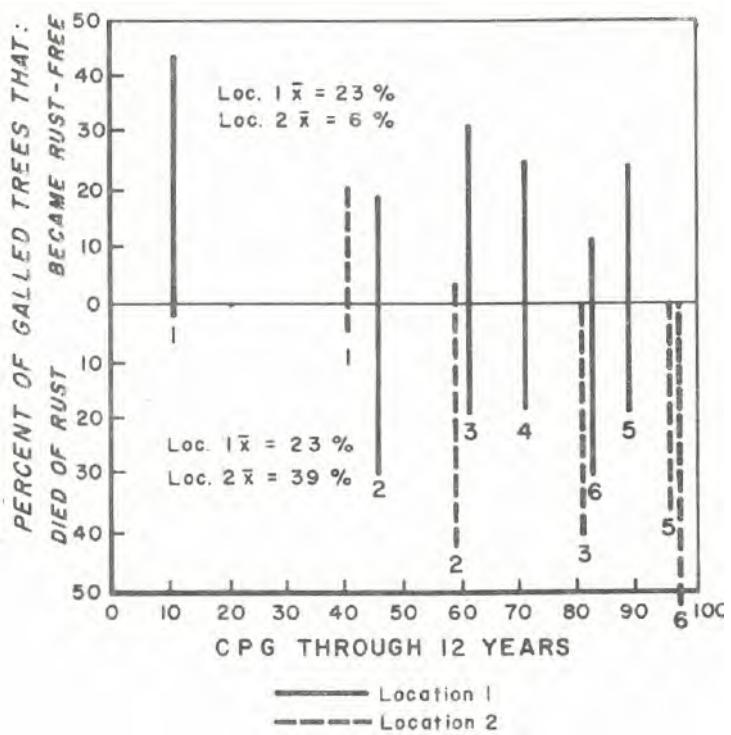

Figure 2.--Effect of rust-associated mortality and natural pruning of galled branches on divergence of noncumulative from cumulative measures (percent) of rust incidence.

The implications are clear--tolerant families should be identified in present and future progeny tests. Once inheritance of tolerance has been confirmed, steps should be taken to incorporate it with other types of resistance now available or becoming available. Such actions seem particularly important because similar phenomena in agronomic crops have proved stable despite shifts in pathogenic virulence (Browning 1974). A word of caution--tolerant trees used alone could intensify disease in certain situations. The greater survival of galled trees could increase inoculum abundance and, consequently, the potential for infection in surrounding plantations.

The overall parallelism of CPG, CPSG, and CPRAM curves implies that early infection may provide a basis for predicting rust-associated mortality.

Simple regressions computed separately for each location support this inference and confirm previous findings (Sluder 1977, and Wells and Dinus In press), though our correlations were not as strong. Variation in CPSG 4 years after planting at Location 1 explained 56 percent of the variation in CPRAM at 12 years. The correlation for Location 2 was 49 percent, also significant. Correlations involving CPG and CPRAM were weaker than for CPSG and CPRAM because CPG includes some trees having only branch galls. Low degrees of freedom precluded analyses within families, but scatter diagrams suggested linear associations for most families. Nature of the relationships, however, appeared to vary among families. For example, the apparent slope for Family 5 was lower than that for Family 6--another indication of the tolerance of Family 5.

Noncumulative disease increase and its relationship to CPG

Plots of NCPG against time indicate that cumulative and noncumulative measures of percent infection generally are identical through the third or fourth growing season (fig. 1). Thereafter, the curves begin to diverge because of rust-associated mortality, natural pruning of galled branches, or some other form of recovery. Both CPG and NCPG continue to increase for several years, but NCPG increases more slowly and eventually declines. This is similar to patterns observed by Schmidt et al. (1974) for slash pine and by Wells and Dinus (In press) for loblolly pine.

Degree of divergence between NCPG and CPG differed between locations and among families (fig. 1 and table 1). Differences in CPRAM account for much of the variation. Infection was heavier at Location 2 and more galled trees died than at Location 1--39 to 23 percent (fig. 2). Also, galled trees in susceptible and intermediate families had a higher probability of death than those in the resistant family.

Divergence also depended upon the frequency with which previously galled trees became and remained rust free, a phenomenon more prevalent at Location 1 than at Location 2 (fig. 2). In four of the six families at Location 1, this form of recovery contributed as much or more to divergence than rust-associated deaths. In contrast, few such trees remained rust free for very long at Location 2 because new infections occurred much more frequently and consistently.

Numbers of galled trees that became and remained rust free also varied among families (fig. 2). This may be because of heritable resistance but, because of difference between sites in environment and inoculum load, it is difficult to assess. Family 1, the most resistant entry, was the only one for which most of the divergency consistently could be attributed to galled trees having become and remained rust free.

Family rankings on the basis of NCPG accurately reflect those for CPG. Few rank changes have occurred in terms of either variable since the second growing season. Hence, relative family resistance can be gauged quickly and inexpensively by point estimates of percent galled.

This consistency also suggests that NCPG would be as accurate a predictor of CPRAM as CPG. Indeed, Wells and Dinus (In press) and Sluder (1977) have found strong correlations between percentage of stem infection observed 5 years after planting and rust-associated mortality at the 10th year in both loblolly and slash pines.

The few rank changes that have occurred were limited to susceptible and intermediate family groups. Such shifts were most prevalent at Location 1 where infection was lighter and more galled trees became and remained rust free. Similar shifts have been observed, particularly in progeny tests involving more families, families with less striking differences in resistance, or low infection rates (Schmidt and Goddard 1971). Our results indicate that CPG may prove more reliable than NCPG in such circumstances and underscore the desirability of establishing progeny tests on hazardous sites (Sohn et al. 1975).

Implications for advanced generation selection

Though relative performance of families can be estimated reliably by cumulative or noncumulative assessments within 3 to 5 years of planting, selecting rust-free individuals within the best families for advanced generation breeding requires much greater care to avoid selecting escapes or previously infected trees that are currently rust free. The potential for each error varies according to amounts of early infection, duration of exposure, and the manner in which rust data are collected.

New infections continue to occur until at least 10 years after planting regardless of family or location (fig. 1). For example, CPG for Family 1 at Location 2 increased from 24 percent in the 6th year to 40 percent in the 10th, showing that 16 of every 100 trees supposedly resistant at year 6 proved susceptible upon further exposure. Early selection based on resistance in field trials therefore does not seem particularly efficient. The danger of selecting escapes was greater at Location 1.

The probability of selecting escapes can be reduced by delaying final selection until 10 or more years after planting. CPG records are then required to avoid the second error of selecting previously galled trees which have become and remained rust free. Divergence of CPG and NCPG curves was not restricted to susceptible families (fig. 1). Some divergence, resulting mainly from loss of galled branches through natural pruning,occurred for even the most resistant Family 1 (fig. 2). These results underscore both the need to maintain histories of disease for individual trees for at least 10 years and the need to establish resistance trials for second generation selection on hazardous sites.

Artificial inoculation and its relationship to CPG and NCPG

Family rankings in terms of both CPG and NCPG also agreed with those from artificial inoculations (table 1). Correlations between family performance at Location 1 and artificial inoculation results have been significant since the 3rd year after planting. The correlation between Location 2 and artificial inoculation results was strong but not significantly so until the 10th year because rapid increase in infection occurred later at

Location 2. Regardless of location, such correlations were strongest for CPG and strengthened with years of exposure.

Variation in results from artificial inoculations explained 81 percent of variation in CPG 12 years after planting at Location 1 as compared with 69 percent of variation in NCPG. At Location 2, artificial inoculation results explained 92 percent of the variation in CPG and 85 percent of that in NCPG. The higher correlations at Location 2 probably result from the tendency of infection levels there to approach those caused by artificial inoculation (table 1).

Our results indicate that artificial inoculations more accurately reflect family rankings than previously supposed (Dinus 1971, Wells and Dinus 1974), especially when field infection is measured and expressed in cumulative terms. That field infection levels eventually match those following artificial inoculations further suggests that in either type of test a similar number of trees would remain rust free and those trees might have similar genotypes. Hence, selection of rust-free survivors from artificial inoculations (Dinus and Griggs 1975) may provide an effective shortcut to field evaluation and selection.

CONCLUSIONS

The foregoing results indicate substantial variation among families in each observed measure of disease incidence. In general, resistant materials can be identified on the basis of percent infection after exposure under artificial or field conditions. Though productive as a first step, such simplified approaches have not taken and cannot take full advantage of the considerable resistance in slash and loblolly pine. An exception is the so-called C--score rating used in some tree improvement programs (Stonecypher et al. 1973). Because numbers of galls per tree and extent of damage are noted, such an index provides more information than the usual procedures. Even these ratings, however, are averages and do not allow breeders to find, isolate, and intercross materials with different forms of resistance.

Detailed evaluation of disease progress curves can identify parents with different types of resistance, including tolerance, and clarify the significance of their resistance in the epidemiology of fusiform rust. Once the nature and inheritance of different resistance types are understood, planned combinations can be made to insure that future seed orchards and plantations contain a variety of resistance genes.

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