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COMPUTER SIMULATED FUSIFORM RUST LOSSES FROM EARLY INFECTIONS IN LOBLOLLY PLANTATIONS[1/]

F. Thomas Lloyd[2/]

Abstract.--A computer simulation model that predicts the fates of individual trees within stands appears promising for projecting fusiform rust losses in infected loblolly pine plantations. In one plantation, observed yield loss between age 10 and 20 was 265 cubic feet and loss predicted from a modified growth simulator for the same period, planting density, and site index was 317 cubic feet. The degree of accuracy could be improved if a measure of stem-gall severity rather than just presence of a stem gall could be devised. Extrapolation of the simulation over a range of 0-90 percent incidence of stem galls at age 3 indicated a linear relationship of loss to early infection amounting to 82 cubic feet between age 10 and 20 for every 10 percent increase in stem gall incidence. Long-term studies would be required to check that projection.

Additional keywords:--Transition probabilities, individual-tree-based model, mortality, stocking, periodic increment, *Pinus taeda*.

In areas of high hazard of fusiform rust caused by *Cronartium quercuum* (Berk.) Miyabe ex Shirae f. sp. *fusiforme*, forest managers need a system for measuring rust infection early in the life of a loblolly pine (*Pinus taeda* L.) plantation and deciding whether to liquidate the stand. The decision requires prediction of effects of early infection percentages on subsequent mortality and growth. The goal of my research is to modify the mortality component of a plantation loblolly yield model to predict the impact of rust-associated mortality. Some preliminary results are reported here.

The model selected for modification was a computer simulator developed by Daniels and Burkhart (1975) and generally referred to as PTAEDA. This model predicts mortality and growth of individual trees within stands. A model of this type is particularly appealing for predicting fusiform rust impact because it might be implemented with less data than a stand-based model. The latter would require both rust incidence and tree size measurements from a considerable number of infected stands. The data used to

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[2/] Biometrician, Southeastern Forest Experiment Station, USDA Forest Service, Charleston, South Carolina 29407.

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modify PTAEDA comes from a genetics study designed to field test family resistance to fusiform.[3] The scope of this genetics study is restricted to a single planting density (436 trees per acre) on one 8.26-acre tract having a uniform site index of 70 feet (age 25). These limitations confine the inferences from the investigation to the same extent that rust-associated mortality rates are related to planting density, site productivity, and incidence level.

Since PTAEDA predicts the fate of individual trees, mortality information derived from the genetics study had to be put in a form compatible with simulating growth of individual trees. My approach was to estimate average death rates for rust-infected trees given both their present age and their age when first infected with a stem gall. These probabilities will hereafter be referred to as transition probabilities (TP's) because they represent the average annual rate that living, rust-infected trees die--that is, the rate of change or transfer from a living to a dead state.

Estimates of these TP's are simply the ratios of the number of trees with stem galls that die during a single year over the number with galls that were alive at the beginning of the year. Since rust incidence and mortality in the genetics study were observed only at ages 3, 5, 10, 15, and 20, it was necessary to fit the cumulative survival and mortality rates at these observed ages and then use these regression equations to interpolate annual values of numbers living and numbers dying. Times of infection, therefore, were in four periods: 0-3, 4-5, 6-10, and 11-15 years. The time of infection factor was analyzed by grouping trees according to the period they received their first stem gall. The resulting TP estimates are presented in Table 1. We should, at this point, remember that the TP's might change with stocking, site, and incidence level--possibilities we are unable to check by analyzing the present data.

MODIFYING THE SIMULATOR

Rust-associated mortality was incorporated into PTAEDA by using the above-described TP's. Each tree with a stem gall at the beginning of each growing season was subjected to the risk of death by generating a random number (u) between 0 and 1 and ceasing growth (that is, killing the tree) if $u < q_{ij}$, where q_{ij} is the TP for the i^{th} growing season of trees infected during the j^{th} infection period.

The mortality mechanism needed one additional change because field observation of rust-associated mortality cannot distinguish mortality due to natural competition from that caused by rust. Both of these causes are present in the TP's yet PTAEDA has its own built-in natural mortality mechanism based on tree competition status, as measured by a competition index described in Daniels and Burkhart's (1975) publication. PTAEDA's natural mortality component was retained because it was designed to cover a range of stocking, so the TP's had to be adjusted for this mortality.

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[3] The genetics study was installed and maintained by the Tree Improvement Project SE-1403, USDA Forest Service, Macon, GA.

Table 1.--Probabilities used in PTAEDA to reflect higher death rates of stem-gall infected trees

Growing season	Infection periods			
	0-3 years	4-5 years	6-10 years	11-15 years
	- - - - - - - Transition probabilities[a] - - - - - - -			
4	0.213	--	--	--
5	0.144	--	--	--
6	0.110	0.025	--	--
7	0.090	0.026	--	--
8	0.078	0.027	--	--
9	0.071	0.028	--	--
10	0.068	0.029	--	--
11	0.066	0.030	0.002	--
12	0.066	0.031	0.004	--
13	0.069	0.032	0.005	--
14	0.072	0.032	0.007	--
15	0.077	0.033	0.008	--
16	0.082	0.034	0.012	0.004
17	0.086	0.035	0.014	0.004
18	0.086	0.036	0.017	0.004
19	0.080	0.036	0.019	0.005
20	0.070	0.037	0.022	0.005

[a] These rates include both natural and rust-caused mortality.

The approach was to first subject all trees to the built-in natural mortality risk factor and then use the numbers of trees killed in that particular annual growth period to adjust the TP's. The adjusted TP is

$$q^*_{ij} = (q_{ij}n_{ij} - m_{ij})/n_{ij} \quad , \text{ for } m_{ij} < q_{ij}n_{ij}$$

$$= 0 \quad , \text{ for } m_{ij} \geqslant q_{ij}n_{ij}$$

where n_{ij} is the number of surviving, rust-infected trees at the beginning of the i^{th} growing season that were first infected during the j^{th} infection period. The symbol m_{ij} represented the number of the n_{ij} trees killed by PTAEDA's built-in mortality function during that growth period.

All that remained was to infect the trees with rust. The probabilities associated with the four infection periods represent the rate of first-time formation of stem galls on the stem-gall-free trees available at the beginning of each infection period. The observed rates for this genetics study were $p_1 = 0.03$, $p_2 = 0.25$, $p_3 = 0.74$, and $p_4 = 0.19$, where the p_i's correspond to the four infection periods, respectively.

Infection was randomly distributed by a process similar to the above-described mortality mechanism. That is, random numbers (u) between 0 and 1 were generated for each planting position and the tree subsequently assigned to that position was assumed to be infected if $u < p_i$. Infection was carried out at the beginning of the simulation, before trees are assigned to planting positions.

USING THE MODIFIED SIMULATOR

The first application was a check of the simulated rust impact against the observed losses calculated from the genetics study. This was done by averaging the results of 27 simulation runs using a planting density of 436 trees per acre (10-foot by 10-foot spacing), site index of 70 feet (age 25), and infection rates by period of 0.03, 0.25, 0.74, and 0.19. The age 10-15 and 10-20 growth periods were used because tree size data in the genetics study were only measured at ages 10, 15, and 20 and the method of estimating rust-associated loss required initial size measurements. The results of this check are presented in Table 2. The simulator worked well when applied to the narrow range of conditions used to modify it.

Table 2.--Total cubic foot volume losses (outside bark) attributed to rust-associated mortality

Source	Periodic increment	
	Age 10-15	Age 10-20
	- - - - - cubic feet - - - - -	
Observed (genetics study)	83	265
Simulated (modified PTAEDA)	82	317

A second application uses the modified simulator to extrapolate rust impact inside a range of early rust incidence of 0 to 90 percent for both the 0-3 and 4-5 year infection periods. This example used 600 planted trees per acre (instead of 436) because PTAEDA performed best at this value relative to three other yield models (Burkhart et al. 1972, Feduccia et al. 1979, and Lenhart and Clutter 1971). Site index was retained at the 70 foot (age 25) value found in the genetics study, and the length of the projection was limited to 20 years. The response variable was periodic growth from age 10 to 20. Figure 1 shows average values of periodic growth from nine runs at each infection level of 0, 10, 20,..., 80, and 90 percent. The averaging of the nine runs was an attempt to smooth out the variability resulting from the stochastic elements built into the simulator.

The trends for both the 0-3 and 4-5 year infection periods appear linear. Future validation of such a trend would mean that there is no thinning advantage resulting from rust-associated mortality in "lightly" infected stands planted at 600 trees per acre. A plausible explantion for this is that rust-associated mortality occurs across the diameter range. Another obvious visual attribute of Figure 1 is the difference in the slopes of the growth-over-infection-rate relationship between the 0-3 and 4-5 infection periods. It raises the question of why infections occurring before age 3 would have a higher mortality rate. It might be related to the size of the tree, or possibly due to many of these early infections having occurred in the nursery rather than in the field after the seedling had become established in the plantation.

By using average 10-year volume increment of a rust-free stand as the maximum potential growth, the manager could choose what is deemed an acceptable percent of this potential and then solve the fitted linear regressions of the data in Figure 1 for the corresponding percent incidence. A refinement to this process would be to use the variability found in the simulations to set a tolerance interval around the linear regression. This tolerance interval could then be used to establish the region of uncertainty depicted by the question mark in Figure 2. This region is found by solving where the line of acceptable growth intersects this tolerance interval.

CONCLUSIONS

Research needs include large, long-term mortality studies in rust-infected loblolly plantations and some measure of severity of infection that predicts mortality more accurately than simple presence of a stem gall. The presence or absence of galls produces very small TP's, which translates to poor prediction accuracy. It would also be helpful in the study of mortality to measure one tree dimension (preferably diameter) from the very first observation, and to remeasure every 2 years.

An individual-tree simulator offers some efficiency in this kind of research in that it simplifies and reduces the amount of tree size data needed. A simulator approach also allows the pulling together of many separate studies. This research suggests that the two-pronged approach of observing mortality over time in stands covering ranges of planting density, site index, and early incidence levels coupled with a proven simulator is an efficient strategy for studying fusiform rust impact.

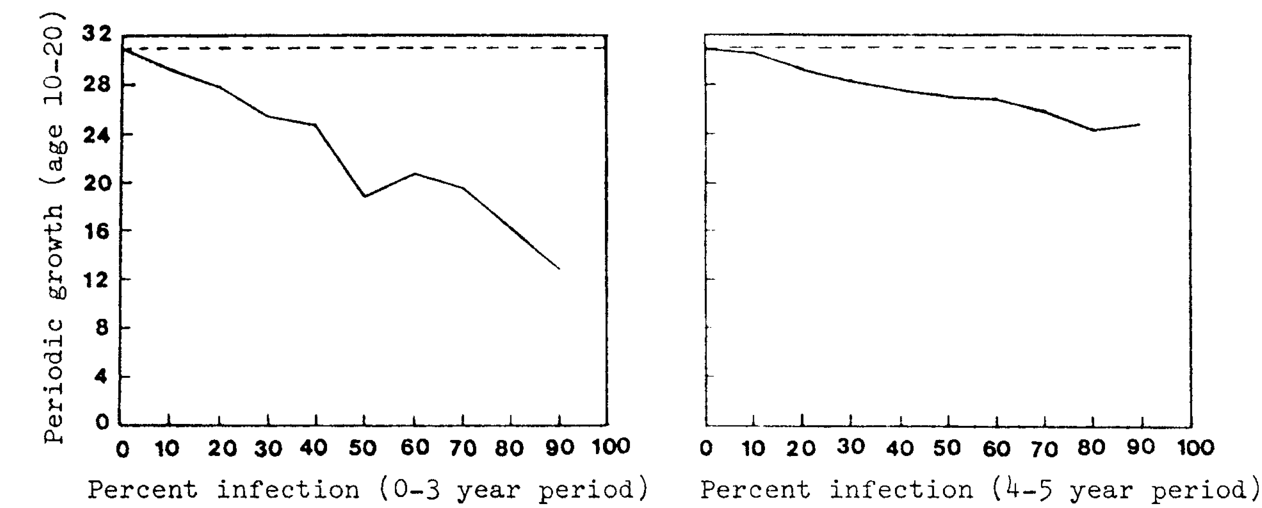

Figure 1.--Simulated 10-year increment in 100's of total cubic feet (outside bark) obtained by averaging the predictions from nine runs of the modified version of PTAEDA (the yield predictions vary because of stochastic elements in the simulator).

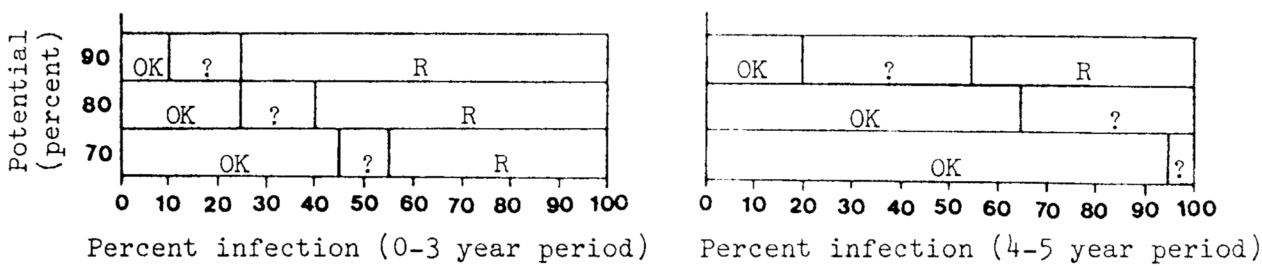

Figure 2.--Hypothetical management decision guide based on early percent stem infections. R = replant. Percent of maximum growth (called potential) determined by the manager.

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