

PHYTOPHTORA CINNAMOMI THE “STEALTH” KILLER

Joseph James

Clemson University, Clemson, SC

- PHYTOPHTORA is not a fungus, but an oomycete related to red and brown algae.
- There are probably 250 to 450 species worldwide.
- Cinnamomi is clearly the most virulent of the known species.
- *C. Crenata*, *Mollisima*, *Sequinii*, and *Henryi* evolved with *Cinnamomi* in Southeast Asia and are largely resistant.
- *P. cinnamomi* and perhaps several other species were most likely introduced to North America with experimental potted plants shipped to plantation owners around Charleston, SC circa 1780 to 1800.
- Wherever P.C. was spread it completely wiped out the pure American chestnut; this event went completely undetected by the scientific world until the 1940's.
- Through the hybridization process trace amounts of resistance derived from the original Chinese and Japanese trees have been found to persist in the various backcross generations. (James and Jeffers)
- By the use of random screening of large numbers of hybrid seedlings from different families, a few trees have been shown to survive *P. cinnamomi*.
- Early data shows that by breeding these survivors with each other the progeny's survival rate increases by a factor of 4 to 8 fold over their parent's generation. Herein lies the hope for the future.

Though difficult to prove, *P. cinnamomi* probably evolved in Southeast Asia. The area around Charleston, SC seems a quite logical point of entry; especially when one considers the extensive plantation culture with its accompanying interest in gardens, and with the introduction of exotic tropical plants to be developed for agricultural profit. These plants would have been potted if they were to survive the long ocean voyage. Any number of other exotic microorganisms including other *Phytophthoras* would have gained entrance also.

This large scale screening is being done on Chestnut Return Farm in Oconee Co. South Carolina. The work is entirely based on the premise that genes for resistance to *P. cinnamomi* would have been derived from the original Chinese trees. All the Chinese genes, including those for *phytophthora* resistance, would have been carried in progressively diluted form. Each successive backcross generation would statistically have an average reduction of Chinese genes of 50%. The F1 cross= 50% Chinese genes; B1= 25%; B2= 12.5%; B3= 6.25% Chinese genes. All survivors are not created equal. Some thrive while others just “get by”. The difference is probably a reflection of the different resistance alleles a given hybrid seedling might carry. How many different alleles contribute to “complete” resistance is currently unknown. It is TACF's goal to eliminate most of the undesirable Chinese alleles while maintaining the genes for

resistance. When dealing with *P. cinnamomi* we have one strong factor in our favor, DEATH. Only those few seedlings that have, by chance, still retained sufficient genes for resistance from their Chinese ancestor will live **. These surviving trees will be grown in an open-pollinated orchard and will breed only with other B3F3 survivors. The seedlings in each generation will be inoculated directly with *P. cinnamomi* and trees with too many non-resistant genes will be eliminated. This natural culling process will concentrate the resistance alleles eventually toward 100% frequency and reduce the non-resistant alleles toward 0%.

We have now entered our third year of B3F3 screening. The results obtained by direct seeding in the field appear to closely mimic the results obtained from our standard tub tests. But there was glaring difference that I observed between Rows 3 and 4 from the 2011 planting. That first year of direct seeding of B3F3's in the field was supposed to result in the usual 70% or so die-off of seedlings in the first summer. That did not happen and I became worried that the existing level of field inoculum was too low. So, I decided to inoculate Row 4 but leave Row 3 uninoculated to gauge any differences in health and growth rates. The seedling count in Row 3 dropped from 103 to 80 living trees by April of 2012. The remaining trees in Row 3 continued to grow exceedingly well. 26 of 80 reached between 7.5 and 9 ft. in height by 15 mos. from the time of sprouting. Their individual biomass, I would estimate, was at least 4X greater than the best trees in Row 4. Could it be that we are missing a portion of the complete genetic complement for resistance?