



**SCREENING FOR RESISTANCE TO *PHYTOPHTHORA CINNAMOMI*
IN HYBRID SEEDLINGS OF AMERICAN CHESTNUT**

S. N. Jeffers¹, J. B. James², and P. H. Sisco³

¹Department of Entomology, Soils, and Plant Sciences; Clemson University
120 Long Hall, Clemson, SC 29634-0315, USA

²Chestnut Return
260 Steve Nix Road, Seneca, SC 29678, USA

³The American Chestnut Foundation, Southern Appalachian Regional Office
One Oak Plaza, Suite 308, Asheville, NC 28801, USA

American chestnut (*Castanea dentata*) once was one of the primary hardwood tree species in forest ecosystems in the eastern USA. However, in the 1800s, Phytophthora root rot (PRR; also known as ink disease), caused by *Phytophthora cinnamomi*, resulted in widespread death of chestnut in the Piedmont region of southeastern states where clay soils are dominant. This was followed in the early 1900s by chestnut blight, caused by *Cryphonectria parasitica*, which almost eliminated chestnut from its primary mountain habitat. Since 1989, the American Chestnut Foundation (TACF) has been producing hybrid chestnut seedlings by crossing Chinese chestnut (*C. mollissima*) with American chestnut and then backcrossing progeny to *C. dentata* in an attempt to produce American-type chestnut trees resistant to *C. parasitica*. In recent years, hybrid seedlings planted in the field in southeastern states have died from PRR before they could be challenged by *C. parasitica*. Therefore, in 2004, we began screening hybrid seedlings for resistance to *P. cinnamomi*. In 2004 to 2006, hybrid seeds from known crosses were obtained from TACF cooperators, and seeds from *C. dentata* and *C. mollissima* were collected in the field. Seeds were stratified and then planted outside in April in replicate 568-liter plastic tubs filled with soilless container mix at a field site in Oconee Co., SC. Inoculum was produced by growing two isolates of *P. cinnamomi*, originally recovered from chestnut seedlings, on autoclaved rice grains. Seedlings were inoculated 12 to 14 weeks after planting. Inocula were combined, mixed thoroughly, and then evenly distributed in 1- to 3-cm-deep furrows between rows of seedlings. Seedlings were watered as needed throughout the study period, and the container mix in each tub was brought to saturation at least once while plants were actively growing. Plants were evaluated for PRR symptoms in December when fully dormant.

Each year, seedlings started dying approximately 3 weeks after inoculation and continued to die throughout the summer months; symptoms were typical of PRR. *C. dentata* seedlings consistently were susceptible, *C. mollissima* seedlings consistently were resistant, and hybrid seedlings varied from susceptible to resistant. Resistant seedlings were planted in the field for further evaluation. Preliminary results suggest that resistance is incompletely dominant and regulated by one gene. Moreover, the genes for resistance to *P. cinnamomi* and *C. parasitica* do not appear to be linked. Screening efforts have been expanded in 2007 and will continue in coming years.