Advances in Understanding the Host-Pathogen Interaction in Fusiform Rust Disease of Loblolly Pine

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Abstract

Fusiform rust disease continues to be the most destructive disease in southern US pine plantations. Our cooperative research program is designed to identify, map, and clone the interacting genes in both the host and pathogen. Eight resistance genes (*Fr1* - *Fr8*) have been genetically mapped in loblolly pine (*Pinus taeda*) (Wilcox et al. 1996; H. V. Amerson unpub.). In addition, DNA marker analysis has proven extremely useful for understanding and manipulating the pathogen, *Cronartium quercuum* fsp. *fusiforme* (Cqf). Recently, we showed that mixtures of spores avirulent and virulent towards *Fr1* are capable of causing multiple infections on seedlings artificially inoculated under standard conditions (Kubisiak et al. 2005). However, only virulent spores are capable of causing disease on seedlings with the *Fr1*allele, while both types of spores cause disease on seedlings without *Fr1* (i.e., *fr1/fr1)*. These results encouraged us to hypothesize the presence of an avirulence gene (*Avr1*) corresponding to *Fr1* and suggested a means for proving its existence. In an ongoing study we inoculated a family segregating for *Fr1 (Fr1/fr1)* with an isolate segregating for avirulence and virulence to *Fr1*. Bulk segregant analysis (Michelmore et al. 1991) was performed on pycniospore drops collected from galls on trees with and without the *Fr1* allele within a single full-sib family. In total, 1,200 RAPD primers were screened against the bulk samples. Preliminary analyses suggests that four RAPD markers are significantly linked to *Avr1*, with one marker showing two recombinant progeny out of 96 and the other three markers showing eight recombinants. In addition, the eight recombinant progeny observed for the three cosegregating markers differ from the two observed for the most closely linked marker, indicating that *Avr1* is most likely within the genomic region flanked by these markers. We plan to use this information along with a recently developed fosmid (40 kb average insert size) library for Cqf to identify, clone and sequence the *Avr1* gene. Once achieved, *Avr1* allele-specific markers will be developed for use in assessing the occurrence and frequency of *Avr1* alleles across the pathogen population. Such information, for all known Avr genes, should provide a reliable means by which informed management decisions can be made regarding the deployment of host resistance genes.

References

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