

Section 2 Moderator's Report

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This was a very interesting and useful conference. I was especially impressed by the fact that the latest technology is being used to study chestnut blight, and by the dedication and high talent of the researchers. The molecular genetics approach is the obvious one for progress in understanding the disease, and it offers hope for practical controls.

There is one area of research that needs more attention, in my opinion. I refer to studies on biochemical basis of interactions between *Cryphonectria parasitica* (Murr.) Barr and the chestnut cell. A better understanding of the factors responsible for pathogenicity by the fungus and resistance

in the host could be very important as background for molecular genetics manipulations, and for general understanding. In general, such work does not seem to be popular. The conference had only a few presentations concerned with the biochemistry of host-pathogen interactions. One was the paper by Vannini, et al., who studied polyphenoloxidase and oxalate production. However, the roles of these factors still have not been clarified. There were presentations on cutinase and ethylene as potential factors in disease. Hopefully, these works and others will be followed further.

Why emphasize such background work? There are good examples of how information on host-pathogen interactions can lead to significant understanding. One example is the leafspot disease of maize caused by *Cochliobolus carbon* = R.R. Nelson. Some years ago *C. carbonum* was found to produce a selective toxin that is required for pathogenicity to the host (certain genotypes of maize). Ability of the fungus to produce this toxin is controlled by a single gene, and resistance in maize to the toxin (and to the fungus) is controlled by one dominant gene. Recently, other researchers have found that the fungal gene produces an enzyme required for toxin production, and that non-pathogenic isolates lack this enzyme (Panaccione et al., Proc. Natl. Acad. Sci. 89:6590-6594). The gene has been isolated. Also, workers from the same laboratory found

that resistant maize has a gene (which was isolated) for production of an enzyme that destroys toxin; the enzyme is lacking in the susceptible host (Meeley and Walton, Plant Physiol. 97:1080-1086; and Meeley et al., The Plant Cell, 4:71-77). Thus, the leafspot on maize, caused by *C. carbonum*, is now perhaps the best understood of all plant diseases.

I do not suggest that similar mechanisms are involved in the chestnut/fungus interaction, but this and other examples illustrate the potential of biochemical background studies. I suggest that more work is needed on factors responsible for pathogenicity of *Cryphonectria parasitica*. This clearly is a difficult and frustrating approach, as work so far has demonstrated. There must be a mechanistic basis and such work has potential rewards.