Simultaneous Laurel Wilt Disease Biology and Resistance Research

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Abstract

Laurel wilt (LW) is a devastating, emerging disease of native and non-native members of the Lauraceae family in the southeastern United States. Currently, the fungal pathogen (*Raffaelea lauricola*) and its vector (*Xyleborus glabratus*) are found in Alabama, Florida, Georgia, Mississippi, and North and South Carolina. The wilt is decimating native stands of redbay (*Persea borbonia* (L.) Spreng.), and causing significant damage to planted and native sassafras (*Sassafras albidum* (Nutt.) Nees) and avocado (*Persea americana* Mill.). Research has addressed significant knowledge gaps that exist for the biology and management of this disease. To date, effective fungicidal management of LW has been limited to the expensive, preventive treatment of high-value landscape trees with systemic fungicides. Long-term management in landscape and avocado plantings may rely on a combination of sanitation practices and the use of disease-resistant germplasm. The susceptibility/tolerance of different taxa and avocado germplasm has been assessed in artificial inoculation work in the field. Host range experiments have assessed the response of 35 taxa in the Annonaceae, Fagaceae, Lauraceae, Magnoliaceae, Moraceae, and Sapindaceae, which include known hosts of the vector in southeast Asia and their relatives. In general, members of the Lauraceae that are native to the southeastern United States have been most susceptible, whereas those in the family from Asia and in other families have been resistant. Forty-one cultivars of avocado, representing the three races of the species (Guatemalan, West Indian, and Mexican) and hybrids thereof, have been screened for disease response. Unfortunately, West Indian cultivars that predominate in Florida have been most susceptible. Host-pathogen interactions have been examined in greenhouse and field studies. Results from this work indicate that: (i) systemic colonization by the pathogen, without apparent internal or external symptom development, occurs in some hosts; (ii) wilting is associated with reduced hydraulic conductivity in the xylem; (iii) vascular dysfunction results from host responses, not occlusion of vessels by the pathogen; and (iv) the pathogen does not produce wilt-inducing toxins. In ongoing research, greater understandings are sought for how susceptible and tolerant/resistant host plants respond to this disease.

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