

EXOTIC PATHOGENS OR SHIFTS IN CLIMATE? LAUREL WILT AND BUR OAK BLIGHT

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ABSTRACT

Two new diseases in eastern USA, laurel wilt (caused by *Raffaelea lauricola* T.C. Harr., Fraedrich & Aghayeva) and bur oak blight (BOB, caused by *Tubakia iowensis* T.C. Harr. & D. McNew), recently have appeared in eastern USA. A new disease on a native host suggests that the pathogen is exotic or that there has been a substantial change in the environment that favors pathogen over host. A few principles help distinguish between the two. Introduced pathogens generally go through severe genetic bottlenecks and show limited diversity compared to native pathogens. Also, in native pathosystems, especially with foliage diseases, susceptible host populations typically show substantial variation in resistance, with resistant and susceptible individuals in proportion to the disease pressure, often determined in part by rainfall and humidity.

Mortality of redbay (*Persea borbonia* (L.) Spreng.) on Hilton Head Island, South Carolina, was first noticed in 2003. Although initially attributed to drought, Steve Fraedrich of the US Forest Service found that the affected trees had vascular streaking typical of a wilt disease. True vascular wilt diseases (systemic colonization of the host through non-living vessels in the early stages) are generally rare on native trees.

Cultures of an unusual fungus with a slimy yeast phase were isolated from the xylem discoloration and sent to Iowa for identification. Analysis of rDNA sequences showed that the fungus was related to *Ophiostoma* spp., a genus that includes numerous saprophytic bark beetle associates but also the causal agents of two wilt diseases, Dutch elm disease and black stain root disease on conifers, both of which involve bark beetle vectors. The redbay pathogen was most closely related to *Raffaelea* spp., which are fungal symbionts of ambrosia beetles, close relatives of bark beetles.

The pathogen was named *R. lauricola* and the disease was named laurel wilt because all tested American members of the family Lauraceae appeared to be susceptible to systemic colonization. These hosts include sassafras (*Sassafras albidum* (Nutt.) Nees), pondspice (*Litsea aestivalis* (L.) Fern.), pondberry (*Lindera mellisifolia* (Walter) Blume), California bay (*Umbellularia californica* (Hook. & Arn.) Nutt.), and avocado (*Persea americana* Mill.). Redbay and swampbay (*Persea palustris* (Raf.) Sarg.) appear to be uniformly susceptible, all cultures look identical, and there is no genetic variation in the pathogen.

Several ambrosia beetle species were associated with wilted redbay, and one of them, the redbay ambrosia beetle, *Xyleborus glabratus* Eichhoff, was a recent arrival. A specimen had been indentified from near Savannah, Georgia in 2002, and the beetle was likely introduced in solid wood packing material. This Asian ambrosia beetle proved to be the vector of the pathoge. Adult ambrosia beetles generally produce brood galleries in the sapwood of dead and dying trees and inoculate the walls of their tunnels with symbiotic fungi carried in special sacs called mycangia. Larvae and adults of ambrosia beetles feed primarily or exclusively on fungal growth in the galleries. Most ambrosia beetles have *Raffaelea* species as their fungal symbionts. Isolations from *X. glabratus* collected in the Southeast and in its native range in Taiwan and Japan found *R. lauricola* to be the primary inhabitant of the mandibular mycangia, but five other *Raffaelea* species have also been isolated from samples in the Southeast. The mycangial flora of the Southeast populations of *X. glabratus* was very similar to that of the Japanese and Taiwanese populations, but another part of Asia was a likely source for the Savannah introduction.

Only *X. glabratus* has been shown to regularly carry a lethal vascular wilt pathogen in its mycangium. Some exotic ambrosia beetles have proven to be pests when they have built up large populations and have

attacked and killed horticulturally important trees or stressed forest trees. Generally, ambrosia beetles are attracted to ethanol and other “stress compounds” emanating from dead and dying trees. But there appears to be a unique relationship among *X. glabratus*, *R. lauricola* and their aromatic hosts in that the beetle is attracted to host volatiles from living trees. The beetle will bore into xylem of healthy branches and stems of Lauraceae but abort the galleries and not lay eggs. It is during this unique tunneling phase that the pathogen is introduced into the host, and after systemic colonization, wilting occurs and another generation of *X. glabratus* attacks the weakened or killed tree for egg-laying. In this way, laurel wilt is similar to Dutch elm disease, in which the young bark beetle vectors go through a period of maturation feeding and inoculate the pathogen into the twigs of healthy elms during maturation feeding, and a later generation of beetles breed in the wilted trees.

Redbay and the closely related swampbay are highly susceptible to laurel wilt and are good hosts for *X. glabratus*. The epidemic has spread rapidly on these hosts, which are native to the coastal plains of the Atlantic and Gulf Coasts, but spread has been slower on sassafras. The epidemic has moved along fronts at more than 20 miles per year, and there have been numerous satellite centers that likely were initiated by movement of infested wood, such as firewood or craftwood. The disease is known from North Carolina south to the commercial avocado production area of Miami-Dade County, Florida and west to Mississippi. Only the smallest redbay and swampbay survive the advancing fronts. To the west, the Mississippi Delta area has little native Lauraceae, so there should be some effort to prevent movement of the vector beyond this line. California bay is susceptible, and it is believed that many important tropical and subtropical Lauraceae native to Mexico, Central and South America could be severely affected by the disease.

The second, less dramatic disease emerging in the eastern USA is a late-season leaf blight on bur oak, *Quercus macrocarpa* Michx. It was first noted in southwest Wisconsin and southern Minnesota in the late 1990s, with later reports in Iowa and eastern Nebraska. It was thought that the leaf pathogen *Tubakia dryina* (Sacc.) Sutton was responsible, but we looked closer and found that there are a number of undescribed species of *Tubakia* on oaks and chestnut in the eastern USA. We described the bur oak pathogen as a new species, *T. iowensis*, and the disease was named bur oak blight, or BOB for short.

Variation in culture morphology and in rDNA sequences suggested that the pathogen was native to Iowa. The close relatives of the BOB pathogen also seemed to be native to eastern USA. The disease was found to be most severe on mature bur oak on former savanna sites and more severe on upland sites than on bottomland sites. But even on the most severely affected sites, most of the trees appeared to be relatively resistant. Severely diseased and apparently disease-free trees were side-by-side, with the same susceptible trees showing symptoms each year. Variation in the pathogen and some resistance in natural host populations suggested that this was a native disease.

With the help of many cooperators and funding from the Northeastern Area of the US Forest Service, Doug McNew and I established that *T. iowensis* was relatively common on former upland savannah sites across most of Iowa, northern Illinois, southern Wisconsin, much of Minnesota, eastern South Dakota and eastern Nebraska (<http://www.public.iastate.edu/~tcharrin/BOB.html>). Scattered sites in northeastern Kansas, northern Missouri and western Illinois also had BOB. The distribution of the fungus appears to coincide with the distribution of a poorly studied variety of bur oak, *Q. macrocarpa* var. *oliviformis* (Michx. f.) A. Gray, which is an upland variety with relatively small acorns. The olive-shaped acorn of var. *oliviformis* is a fraction of the size of the acorns of var. *macrocarpa*, which is primarily a bottomland species that is eastern and southern to the distribution of var. *oliviformis*. A third variety, var. *depressa* (Nutt.) Engelm., also has small acorns and is native to the Great Plains, but it may not be susceptible to BOB. Although other species of *Tubakia* may cause veinal necrosis on var. *oliviformis* and var. *macrocarpa*, *T. iowensis* appears to be specific to var. *oliviformis*. However, oaks, including bur oak, hybridize readily, many populations may be mixtures of two species or varieties, and oak taxonomy is controversial.

All *Tubakia* species form pycnothyria on necrotic areas on leaves. These asexual fruiting bodies produce rainsplashed conidia on the underside of a shield-like structure. A unique feature of *T. iowensis* is a second asexual fruiting body, a crustose layer enclosing conidia that develop slowly over the winter. Leaves

killed by *T. iowensis* in late summer first show small areas of necrosis at the petiole base, then the necrosis girdles the petiole and the leaf dies. Many of these killed leaves hang on the branches through the winter, and the crustose fruiting bodies form on the petioles and release their spores for rain dispersal in the spring. If there is abundant rainfall as the new shoots expand in spring, then conidia from the crustose fruiting bodies may infect the undifferentiated shoots. Infection of expanding shoots apparently leads to a prolonged endophytic or latent phase, lasting for two months or more. Eventually the petioles become necrotic at their attachment point to the twigs, and the leaves are cast off or remain attached on the branches through the next spring. Fully expanded leaves infected by the primary inoculum may develop necrotic spots or veinal necrosis, in which case conidia soon form along the veins, thus producing a secondary inoculum. However, the primary inoculum and infection of expanding shoots in the spring appears to be critical to the dramatic blight symptoms seen on some trees, where every leaf on the tree is symptomatic by August.

If BOB is a native disease (variation in the pathogen and host resistance), it is surprising that it went unrecognized until recently. One theory is that a shift in climate is responsible for the current epidemic. One of the most significant changes in Iowa's climate has been increased rainfall, especially in the spring, the period when bur oak breaks bud and new shoots are expanding, when the host appears to be most susceptible to endophytic infections. Rainfall during this critical period of shoot expansion leads to spore release, dispersal of the spores, and leaf wetness for germination and infection.

A fundamental principle of native forest tree diseases, especially for foliar diseases, is that there is variation in resistance, i.e., there are resistant and susceptible individuals in host populations. If the environment is conducive and disease pressure high, a high amount of resistance is expected in the host population. If the environment is not conducive, then little resistance is expected because there is a "cost to resistance." Bur oak blight is primarily a disease in relic savanna forests that are often dominated by bur oak of 125 years or more. In the previous century, there may have been little disease pressure because spring rainfall was low, or at least there occasionally were consecutive years of low spring rainfall and probably little disease, so even susceptible individuals would likely produce good acorn crops and pass on susceptibility. However, those relic trees are now in a wetter climate. Iowa has not had consecutive dry springs since the 1988/89 drought, and the last 12 years (including 2012) have had average or above spring rainfall. Predictions are for increased rainfall for much of the Midwest and Northeast.

American forests are seeing increased disease pressure from exotic pathogens and shifts in climate. Not all of these threats are against commercially important tree species, but their ecological importance should warrant further investigation and mitigation.

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FOREWORD

This meeting was the 24th in a series of annual USDA Interagency Research Forums that are sponsored by the Forest Service, Animal and Plant Health Inspection Service, National Institute of Food and Agriculture, and Agriculture Research Service. The Group's original goal of fostering communication and providing a forum for the overview of ongoing research among the Agencies and their cooperators is being realized and facilitated through this meeting.

This proceeding documents the efforts of many individuals: those who organized and sponsored the meeting, those who provided oral and poster presentations, and those who compiled and edited the contributions. The proceedings illustrates the depth and breadth of studies being supported by the agencies and their many cooperators and demonstrates the benefits and accomplishments that can result through the spirit of collaboration

International in scope, this meeting attracts a diverse audience of scientists and managers from North America and abroad because we are sharing invasive species issues that are common to all of us. In addition to presentations by scientists from agencies within USDA, presentations were made by scientists from France, Italy, Great Britain, Austria, Russia, and Canada and included representatives from the following:

- Food and Agriculture Organization (FAO), Rome
- European & Mediterranean Plant Protection Organization (EPPO), Paris
- Foreign Agricultural Service
- National Research Council, Board on Agriculture and Natural Resources
- Russian Forestry, Siberian Branch
- FS-International Forestry
- Embassy of Peru, Agricultural Attaché
- Costa Rica
- Tribal Enterprises, WI
- Canadian Food & Inspection Service, Natural Resources Canada, Canadian Forest Service

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