

INTRODUCTION

Laurel wilt disease (LWD) is caused by the fungus *Raffaelea lauricola* and vectored by the redbay ambrosia beetle (RAB), *Xyleborus glabratus* (Fraedrich and others 2008). The pathogen and vector were apparently introduced from Asia through the Port of Savannah, and the disease has spread rapidly throughout the lower coastal plains forests in Georgia, killing nearly all large, previously abundant redbay (*Persea borbonia*)¹ trees in its path. Although research and prior surveys have revealed much about this disease, little is known about how it will progress in more diverse habitats with more scattered and smaller redbay trees and other hosts in the laurel family, particularly sassafras (*Sassafras albidum*).

The goals of this monitoring project are to (1) follow the progression of the disease in Georgia; (2) monitor mortality of redbay and sassafras sprouts and trees in areas through which LWD has already moved; (3) establish a methodology and document changes in vegetation composition resulting from the elimination of redbay and associated hosts by this disease; (4) monitor the rate of mortality in redbay, sassafras, and other host plants as the disease spreads inland; and (5) monitor the relative numbers of RAB in areas with varying stages of disease progression.

¹ Some taxonomists distinguish redbay (*P. borbonia*) and swamp bay (*P. palustris*) as separate species. For this study, these taxa are both recognized as redbay.

METHODS

LWD distribution and spread in Georgia

The advance of LWD in Georgia from early 2009 through the end of 2011 was documented by noting dead and dying redbay and sassafras trees through contacts with landowners, directed road surveys, and assessments of monitoring plots by Georgia Forestry Commission Forest Health personnel. The presence of LWD in previously uninfected counties was confirmed by submitting samples from symptomatic trees to Steve Fraedrich, U.S. Department of Agriculture Forest Service, Athens, GA, for laboratory culture of the pathogen, *R. lauricola*.

LWD long-term monitoring plots

Standardized permanent plots were established using modified Carolina Vegetation Survey protocol (Wentworth and others 2008) procedures in redbay and sassafras habitats to document the LWD process, vegetation changes, and host regeneration survival within sites and across the landscape in southeast Georgia. Sixteen redbay plots were established in the spring of 2009 and revisited six times through early spring of 2012. Each plot consisted of four contiguous 10-by-10 m modules (total 400 m²) in which all host trees > 2.5 cm diameter at breast height (d.b.h.) were marked, measured,

CHAPTER 11.

Progression of Laurel Wilt Disease in Georgia: 2009–11

(Project SC-EM-08-02)

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and assessed for tree health status and numbers of live and dead sprouts. Redbay and sassafras regeneration (< 2.5 cm d.b.h.) was tallied in 10-m^2 subplots in each module. Vegetation composition in each plot was characterized by estimating the percentage of overstory and understory cover by plant species. Eight sassafras plots were installed using similar procedures, with one or two modules instead of four, and, on three plots with dense thickets of sassafras, trees were tallied by diameter class and not followed individually.

Redbay plots were established in three disease status categories: (1) **absent**—ahead of the advancing front where no disease was known to be present (five plots), (2) **active**—where RAB and the laurel wilt pathogen were present and killing trees (six plots), and (3) **inactive**—areas where large host trees had died and begun to decay and where RAB had presumably emerged from dead host material (five plots). Five sassafras plots were established on absent sites and three plots were established on active sites. Three of the sassafras plots were located adjacent to redbay plots where redbay and sassafras were growing together. All of the absent redbay monitoring plots were located within 15 km of areas known to have LWD and RAB in redbay, sassafras, or both. The location and host species present for each long-term monitoring plot are illustrated in figure 11.1 (two sassafras plots established less than 100 m apart at two locations are represented by single symbols on the map).

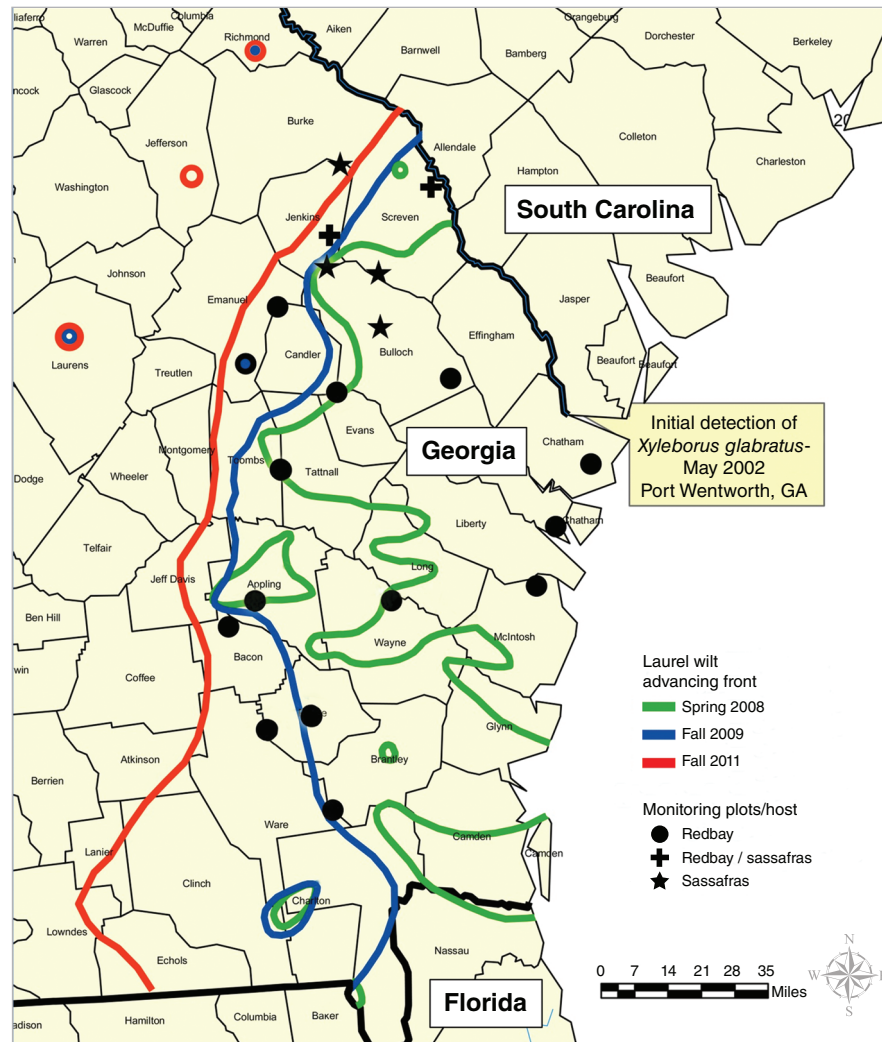


Figure 11.1—Georgia laurel wilt monitoring plot locations and host species, and delineation of the westward-advancing disease fronts in the spring of 2008 (determined by a grid survey), fall of 2009, and fall of 2011 (estimated from progression in monitoring plots and directed road surveys).

RAB monitoring

The relative abundance of RAB in redbay and sassafras habitats with varying stages of disease development (absent, active, or inactive) was assessed by deploying Lindgren 8-funnel traps baited with commercial manuka oil lures (Synergy Semiochemicals Co. P385-Lure M) throughout the month of August (30 days) at 22 sites in 2009 and 23 sites in 2010. Disease development stage designations were determined for each site depending on the status during the trapping period. Dr. Jim Hanula (Forest Service, Athens, GA) sorted trap catches and reported numbers of RAB caught in each trap for both trapping intervals.

RESULTS AND CONCLUSIONS

Distribution and spread

The expanding distribution of LWD in Georgia from the spring of 2008, based on a systematic grid survey (Cameron and others 2008), through the end of 2011, based on assessments of LWD monitoring plots and directed road surveys (Cameron and others 2010), is illustrated in figure 11.1. The rate of spread varied greatly along the disease front in Georgia from less than 10 km/year in the upper coastal plain in the north to more than 35 km/year in the southern coastal plain, where large numbers of more uniformly distributed redbay trees have been killed across a broad area behind the advancing front. At least five isolated disease incidents have been documented well beyond the previously

known distribution of LWD and likely originated from human-assisted dispersal of the RAB vector via movement of infested wood.

During the past 3 years, most new disease infections along the northern advancing front in Georgia have occurred in isolated pockets of redbay and in scattered groups (thickets) of sassafras trees, where redbay is relatively scarce or absent. Although LWD has not spread rapidly through this area, it has steadily infected additional dispersed groups of sassafras, indicating that LWD can infect sassafras in the absence of redbay and may spread beyond previously predicted limits (Koch and Smith 2008).

Disease process in redbay

The LWD process in redbay habitat initially develops slowly, starting in one to a few infected (symptomatic) trees. Once a tree becomes symptomatic, it generally wilts rapidly and is colonized by ambrosia beetles within a few months. The rate at which the remaining trees become infected depends in part on host size and density in the area, but the process usually takes a year or more. LWD progression is most rapid in areas with high volumes of redbay, and it is slower in the presence of fewer, smaller, and more scattered redbay trees. The time for the disease to run its course through an area with abundant host (from the first symptomatic trees to inactivity, when all large redbay are dead and have fallen to the ground) ranges from about 3.0 to 4.5 years.

LWD kills nearly all redbay trees greater than a few centimeters d.b.h.; however, abundant sprouts and seedling regeneration are present on most sites after the disease runs its course (fig. 11.2). Sprouts attached to the base of dead redbay trees generally die, but those further out on the root flare often remain alive, although many die back from attacks by *Xylosandrus compactus*, another Asian ambrosia beetle. A few areas have been observed on the coastal plain in Georgia where numerous redbay trees greater than 2.5 cm ranging up to 12 cm d.b.h. are still healthy; however, the continued presence of scattered trees and clumps of sprouts with LWD indicate that the disease and RAB are still active at low levels many years after the initial epidemic.

Disease process in sassafras

LWD infects sassafras in an apparently haphazard fashion, killing some individual trees and entire thickets while others remain apparently healthy. The largest sassafras trees tend to be affected first, and spread is sometimes rapid in dense thickets, apparently through interconnected lateral roots (fig. 11.3). Leaves on sassafras trees with LWD droop, shrivel, or turn shades of yellow, orange, or red and fall off shortly after symptoms are first manifested, leaving crowns bare within months (fig. 11.4). Infected sassafras will often leaf out in the spring with small leaves that soon shrivel and die. Characteristic black staining in the wood can be present on the surface of the outer sapwood and can be embedded beneath a nonsymptomatic current year's growth ring (fig. 11.3). Epicormic

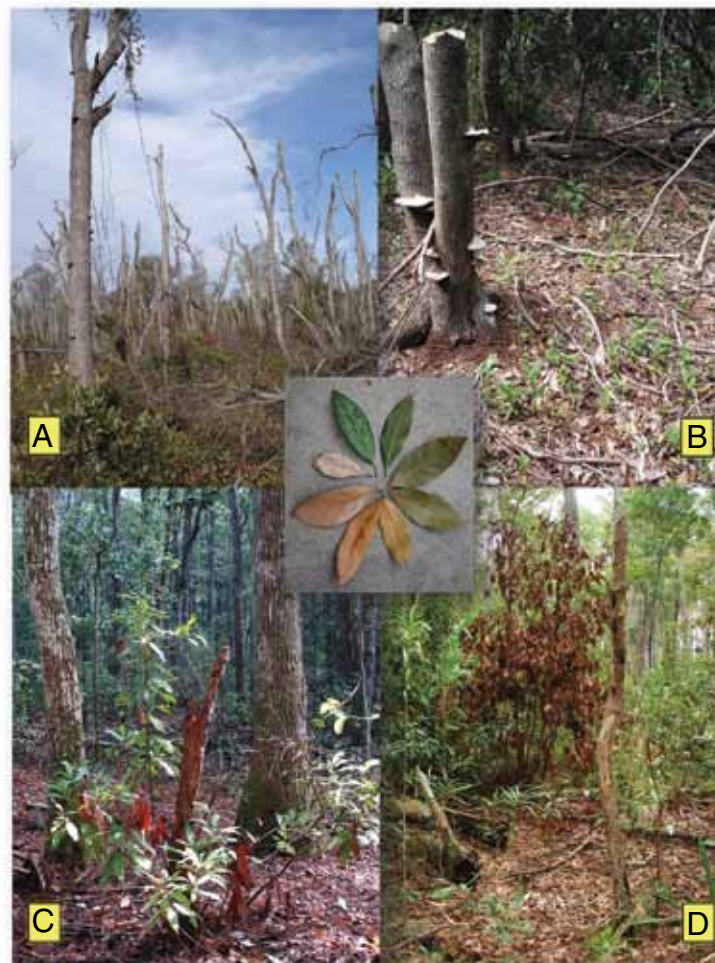


Figure 11.2—Laurel wilt disease, or LWD, in redbay illustrating progression of leaf symptoms (center) and (A) total mortality and collapse in a dense stand of large redbay, (B) seedling regeneration after the overstory was killed, (C) sprouts around long-dead redbay, and (D) stump sprouts killed by laurel wilt years after the first wave of LWD. (Photos A, B, C, and D courtesy of Scott Cameron, Georgia Forestry Commission; center inset photo courtesy of Chip Bates, Georgia Forestry Commission.)

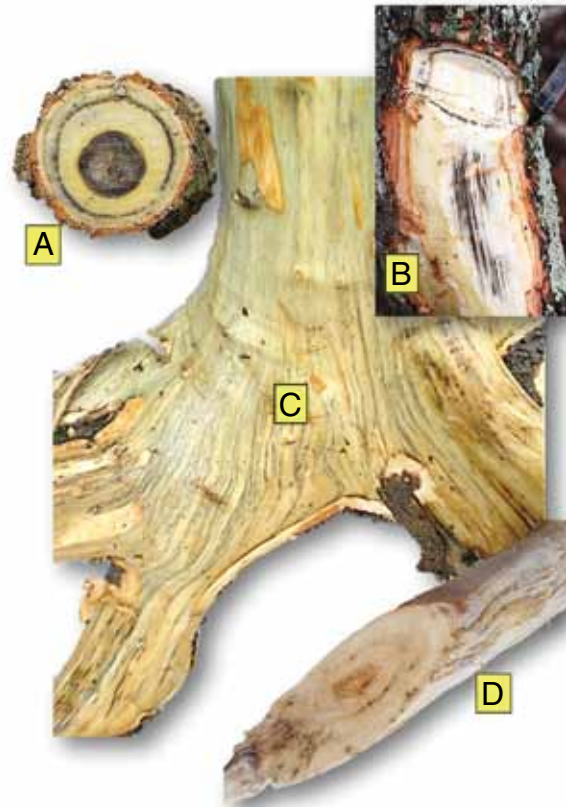


Figure 11.3—Portions of four separate sassafras trees infected with *Raffaelea lauricola*, illustrating black staining resulting from a reaction of the host to the presence of the fungus: (A and B) embedded within the growth ring of the previous growing season; (C) visible on the outside of the wood exposed by removing the bark at the base of a tree, root flare, and roots; and (D) in a long lateral root through which the fungus apparently infected a small symptomatic tree at the edge of an expanding disease center. (Photos A and C courtesy of Chip Bates, Georgia Forestry Commission; photos B and D courtesy of Scott Cameron, Georgia Forestry Commission.)

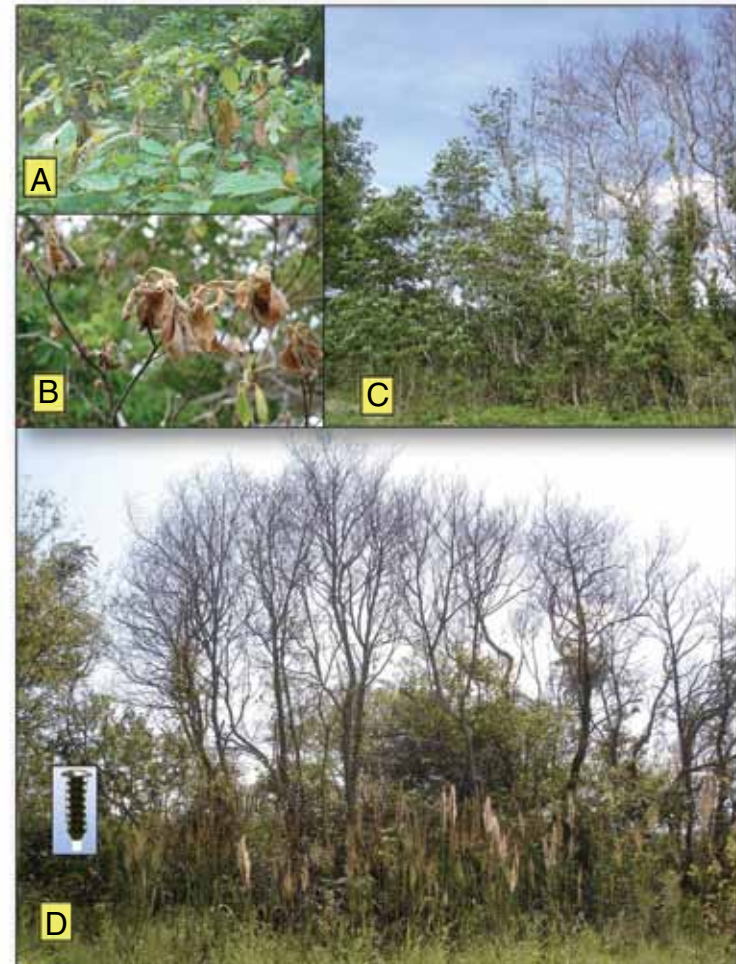


Figure 11.4—Progression of laurel wilt disease in groups (thickets) of sassafras illustrating (A) initial leaf symptoms, (B) nonpersistent dead leaves, (C) rapid spread through thicket, and (D) 1 year following collective mortality, and position of a Lindgren funnel trap used to monitor redbay ambrosia beetles associated with disease centers in different stages of development. (Photo A courtesy of Chip Bates, Georgia Forestry Commission; photos B, C, D, and inset courtesy of Scott Cameron, Georgia Forestry Commission.)

shoots are common on the lower stems of diseased sassafras trees, and ambrosia beetle attacks are most abundant at the base of sassafras trees killed by LWD.

Vegetation changes associated with LWD

Redbay trees killed by LWD decay quickly and begin falling apart within 1 to 2 years. Major, rapid canopy cover changes occur on sites that have a large redbay overstory component before being impacted by LWD (fig. 11.2). Basal sprouts were abundant and persisted around the majority of redbay trees killed by LWD on all monitoring sites. However, redbay seedlings only proliferated on two sites where redbay was a significant component in the overstory and where there was little understory vegetation prior to the LWD epidemic. The impact of LWD on the original vegetation composition is far less on sites with small or sparse redbay in the overstory. When sites have a dense overstory and/or understory of other species, redbay regeneration remains sparse after the passage of LWD.

Redbay ambrosia beetle trapping

Traps baited with manuka oil and deployed throughout August 2009 and August 2010 were effective for monitoring and comparing relative numbers of RAB among the three disease status categories. RAB were rarely captured in absent (apparently disease-free) locations in advance of the disease front (table 11.1). Numbers of RAB caught near active LWD sites varied greatly but were roughly correlated with the number and size (volume) of dead host trees in the vicinity

of the trap, similar to the findings of Hanula and others (2011). Few RAB were trapped on sites where host trees were recently symptomatic and ambrosia beetles were beginning to colonize the trees, while large numbers of RAB were captured on a few sites where new brood beetles were apparently emerging from large volumes of dead host trees. Small numbers of RAB were frequently caught in traps deployed in inactive areas where the disease had moved through many years before, indicating that low populations are being maintained in small redbay trees, or possibly in other, undetermined host material. Few RAB were caught in most traps located near groups of small sassafras trees infected with LWD in the absence of nearby redbay trees. Numerous RAB, however, were caught in one trap adjacent to a large group of sassafras recently killed by LWD (fig. 11.4), supporting the conclusion that RAB attack,

Table 11.1—Numbers of redbay ambrosia beetles, *Xyleborus glabratus*, caught in manuka oil-baited traps located adjacent to redbay and sassafras stands experiencing varying stages of laurel wilt disease development during August 2009 and August 2010

Host type	Disease stage ^a	Number of traps	Total number RAB	Mean number RAB/trap	Standard deviation
Redbay					
	Absent	12	3	0.3	0.6
	Active	15	650	43.3	104.0
	Inactive	10	13	1.3	1.6
Sassafras					
	Absent	2	0	0.0	0.0
	Active	6	121	20.2	43.6

RAB = redbay ambrosia beetles.

^a Disease stage assigned at the time of trap deployment.

transmit LWD, and produce brood in sassafras, although it may not be a favored host (Hanula and others 2008; Mayfield and Hanula 2012).

This summary represents the data collection and analyses completed under the funding of the evaluation monitoring project. Since the preparation of this summary, additional research and analyses have been conducted and an updated, more detailed paper is in preparation.

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