



# Evaluation of Laurel Wilt Disease in Georgia: Progression in Redbay and Sassafras – 2008-2010



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Cover photo: A unique high density stand of large redbay devastated by laurel wilt disease near Claxton, GA, photographed on 2/11/2009.

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## Background

Laurel wilt disease (LWD) caused by the fungus *Raffaelea lauricola* and vectored by the redbay ambrosia beetle (RAB), *Xyleborus glabratus*, has spread rapidly throughout the coastal maritime forests in Georgia, killing nearly all large, previously abundant redbay (*Persea borbonia*)<sup>1</sup> trees in its path. As this disease spreads inland, it is moving into more diverse habitats, often with scattered and smaller redbay trees, as well as other host plants in the laurel family. Although surveys and research have revealed much about this disease, many questions remain about the disease process and its impacts on host plants.

## Objectives

The goals of this laurel wilt monitoring project are to: 1) determine the fate of plants in the Lauraceae family, primarily redbay and sassafras, in areas where laurel wilt disease has already moved through, 2) document the continuing spread of the disease in Georgia, 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 abundance of redbay ambrosia beetles in areas with varying stages of disease progression.

Initial objectives included evaluating the effects of LWD on a number of plants in the Lauraceae family in GA, including redbay, sassafras (*Sassafras albidum*), pond-spice (*Litsea aestivalis*), pondberry (*Lindera melissifolia*), and camphor (*Cinnamomum camphora*). However, due to difficulties encountered with locating and accessing pond-spice and pondberry sites, attention has been focused almost exclusively on documenting the progression of the LWD in redbay and sassafras.

## Methods

**LWD Distribution and Spread in Georgia.** The advance of LWD in 2009 and 2010 in Georgia was documented through observation of dead and dying redbay and sassafras trees by GFC foresters, contacts with landowners, and directed road surveys and visits to monitoring plots by GFC Forest Health personnel. The presence of LWD in previously uninfected counties and instances of uncertain diagnosis were confirmed through laboratory isolation and identification of the pathogen by Steve Fraedrich, USDA Forest Service, Athens, GA.

**Long-term Monitoring Plots.** Permanent plots were established in early 2009 and revisited on about a 6-month schedule to investigate the laurel wilt disease process as it spreads into new areas and through individual groups of redbay and sassafras trees, and to determine the fate of these two species after the disease has moved through an area. Plots were located in areas initially fitting each of three **disease process stages**: 1) **absent** (ahead of the advancing front where no disease is known to be present), 2) **active** (where the redbay ambrosia beetle and the laurel wilt pathogen are present and killing trees), and 3) **old** (areas where large host trees have died and begun to decay and redbay ambrosia beetles have presumably emerged from old host material).

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<sup>1</sup> Some taxonomists distinguish redbay (*P. borbonia*) and swamp bay (*P. palustris*) as separate species. For this study, these taxa are recognized as one species and referred to as redbay.

***Plot distribution and classification.*** Sixteen monitoring plots were installed in redbay habitat from late-winter through early summer 2009: A) Five ahead of the advancing front in areas in which LWD was apparently “absent,” B) six plots with LW diseased trees (“active”) just inside the advancing disease front, and C) five in “old” infection areas, well within the current active disease front. One very unique site located near Claxton, GA with nearly pure, large redbay trees that had been killed by LWD prior to 2009 (“shock and awe” site) was visited and photographed periodically, but a plot was not established due to extremely hazardous conditions and the large amount of fallen tops and stems, which made the area almost impenetrable.

Five separate plots were established in sassafras stands (thickets) after leaf-out in May 2009, three in the absence of disease and two with active disease episodes in progress. One additional active sassafras site was periodically monitored and photographed, but a plot was not established due to a profusion of poison ivy in and around the sassafras trees.

Among the initial 16 redbay plots, two had sassafras trees mixed with redbay. Thus, three additional sub-plots (modules, described below) with mostly sassafras (two at one site and one at the other) were established to expand the area in which to monitor the disease progression through both species on the same site. Active disease was present at initiation in redbay, but not in sassafras, on the site with one additional sub-plot. The other two sassafras modules and adjoining redbay plot are still disease free.

One additional plot was established in a unique area with four lauraceous species (redbay, sassafras, pondspice and pondberry) growing adjacent to each other on the edge of an upland pond located about 40 km beyond the advancing disease front.

Monitoring plots were established on both private and public properties and landowners/managers were consulted to obtain approval for installation and monitoring of plots on their land. Finding appropriate combinations of adequate host plants, designated stage of disease progression, and landowner approval was difficult. The location, primary species, and disease stage (as of September 2010) for each of the long-term monitoring plots are illustrated in Figure 1. Details, including date initiated, dates visited, disease status, host species present, county location, and eco-region are listed for each plot in Table 1.

***Plot installation and data collection methods.*** Long-term plots were established and vegetation described using simplified methods adapted from the Carolina Vegetation Survey protocol. Plots generally consisted of four 100 square meter (10 m x 10 m) **modules** in a row (10 m x 40 m), or side-by-side in a 4-module square (20 m x 20 m), depending on the distribution of host plants. The corners of each monitoring plot module were marked with PVC pipe and the four corners of the aggregate plot were additionally marked with brass rods. GPS coordinates for the lower left corner of module one and the bearing of the plot center line (side of module one) were recorded at plot establishment along with general site characteristics. Number, location, DBH (diameter at breast height – 1.37 m (4.5 ft.)) and tree condition: 1) live, healthy, 2) LW symptomatic, 3) LW dead, 4) LW dead, fallen apart, 5) dieback by other causes, or 6) dead by other causes, along with numbers and condition of sprouts, were noted for each redbay and sassafras tree greater than 2.5 cm DBH in each plot module.



Figure 1. Location of laurel wilt disease long-term monitoring plots, primary host species, disease stage in September 2010, and RAB trap sites.

Each redbay tree was numbered and plotted on a sketch map and also marked in the field plot with an aluminum tag placed on a wire pin at the base of each tree or group of trees. Since five plots were established in areas where redbay trees had already decayed and broken apart with only the trunk or stump and sprouts remaining, the original tree DBH was estimated to characterize the original redbay component in these plots. The fate of host regeneration was monitored by recording the numbers and condition of host plants <2.5 cm DBH in one-meter strips along one side of each module at each assessment. Redbay and sassafras less than 1.4 m tall were counted and recorded as live or dead “seedlings”, and host plants taller than 1.4 m, but <2.5 cm DBH, were recorded as live or dead saplings. No attempt was made to distinguish between sprouts and seedling plants. Percent understory (<2.5 m) and overstory (>2.5 m) canopy cover was noted by plant species.

Table 1. List of LW monitoring plots and RAB trapping sites, inspection dates, disease status, hosts present, counties and eco-region classification.

Plot ID#	Estab. Wint/Spr 2009	Summer 2009	Spring 2010	Summer Fall 2010	Traps Aug 2009	Traps Aug 2010	Initial Stage	Rb **	Sas	Ps	Pb	County	Eco-region **
111	1/30/09	8/11/09	2/6/10	9/21/10	X	X	1	1				Emanuel	65l
112	2/10/09	8/11/09	4/14/10	9/21/10	X	X	1	1	1			Jenkins	65l
113	2/12/09	8/9/09	3/14/10	9/19/10	X	X	1	1				Bacon	75h
114	4/6/09	8/12/09	2/6/10	9/21/10	X	X	1	1				Emanuel	65l
115	6/23/09	8/8/10	2/4/10	9/18/10	X	X	1	1				Ware	75h
121	2/3/09	8/11/09	4/14/10	9/23/10	X	X	2	1	1			Screven	65l
122	2/4/09	8/13/09	3/16/10	9/19/10	X	X	2	1				Bulloch	65l
123	2/4/09	8/9/09	3/16/10	9/21/10	X	X	2	1				Tattnall	65l
124	2/12/09	8/12/09	3/14/10	9/19/10	X	X	2	1				Appling	75h
125	3/24/09	8/8/09	2/4/10	9/18/10	NA	X	2	1				Brantley	75f
126	3/25/09	8/8/09	2/4/10	9/18/10	X	X	2	1				Pierce	75h
131	2/7/09	8/14/09	2/3/10	9/16/10	X	X	3	1				Bryan	75j
132	2/8/09	8/9/09	2/3/10	9/16/10	X	X	3	1				Chatham	75j
133	2/22/09	8/14/09	2/3/10	9/16/10	X	X	3	1				McIntosh	75j
134	2/13/09	8/9/09	2/6/10	9/16/10	X	X	3	1				Bulloch	75f
136	4/30/09	8/8/09	3/14/10	9/18/10	X	X	3	1				Wayne	75f
211a	5/14/09	8/11/09	4/14/10	9/21/10	NA	NA	1	1	1			Jenkins	65l
211b	5/14/09	8/11/09	4/14/10	9/21/10	NA	NA	1	1	1			Jenkins	65l
212a	5/1/09	8/10/09	4/15/10	9/23/10	X	X	1		1			Jenkins	65l
212b	5/1/09	8/10/09	4/15/10	9/23/10	NA	NA	1		1			Jenkins	65l
213	5/14/09	8/11/09	4/19/10	9/23/10	X	X	1		1			Jenkins	65l
221	5/13/09	8/10/09	4/15/10	9/23/10	X	X	2		1			Bulloch	65l
222	5/13/09	8/10/09	4/17/10	9/23/10	NA	NA	2		1			Screven	65l
223	5/13/09	8/11/09	4/14/10	9/23/10	NA	NA	2	1	1			Screven	65l
224	5/2/09	8/6/09		9/24/10	X	X	3		1			Bulloch	65l
311a	5/27/09	8/12/09	5/10/10	NA	NA	NA	1	1	1		1	Wheeler	65l
311b	5/27/09	8/12/09	5/10/10	NA	NA	NA	1			1		Wheeler	65l
135	NA	NA	NA	NA	X	X	3					Evans	65l
116	NA	NA	NA	NA	X	X	1					Emanuel	65l
117	NA	NA	NA	NA	X	X	1					Wheeler	65l
				TOTALS	22	23		20	10	1	1		
Key*	Absent	Adjacent	1st sympt	Active									
	Old	Multi-spp.	Sass adj	Sass adj									
* Absent=no known LW near, Adjacent=disease near, but not in plot, 1st sympt=first LW symptomatic trees in plot, Active=multiple trees with LW symptoms, ambrosia beetles active, Old=disease moved through area, RAB emerged, host trees fallen apart, Multi-spp.=more than 1 host species in plot, Sass adj=added module to include more sass - plot number color indicates redbay/sass plot association. Initial stage: 1=live, healthy, 2=LW symptomatic, 3=LW dead.													
** Rb=redbay, Sas=sassafra, Ps=pondspice, Pb=pondberry, Eco-region (Griffith et al. 2001).													

Procedures were modified for sassafras stands (thickets) with large numbers of tightly spaced small trees. One or two modules with dimensions adjusted to the configuration of the stand were established and tallies of live and dead plants by diameter class were recorded instead of tagging and measuring each individual tree. In the one plot with four host species present, numbers of live and dead stems were recorded for two individual clumps of pond spice, numbers of live and dead pondberry stems were recorded in a one meter sample strip in each module. Redbay and sassafras trees were marked and recorded as in all the other redbay plots.

**Redbay Ambrosia Beetle (RAB) Monitoring.** The presence and relative abundance of RAB in each of the disease process stages for redbay and sassafras habitat were assessed by deploying Lindgren 8-funnel traps baited with commercial Manuka oil lures (Synergy Semiochemicals Co. - P385-Lure M) adjacent to 18 of the LW monitoring plots throughout the month of August (30 days) in 2009 and again in August 2010 (Fig. 6-D). Traps were deployed at four additional locations, two “absent” redbay sites beyond the advancing front, one declining active redbay site (“shock and awe” near Claxton, GA), and one active sassafras for a total of 22 sites in 2009 (Figure 1, Table 1). The twenty-two traps deployed in 2009 were allocated to disease stages and host species as follows: Redbay-Old (4), Redbay-Active (8), Redbay-Absent (6), Sassafras-Active (3), and Sassafras-Absent (1). One additional active permanent monitoring plot site was added in 2010. Disease stage associated with each trapping site was assigned according to the status at the time of trap deployment each year in data summaries. Thus, the disease stage for several sites differed from the original 2009 designation and changed from 2009 to 2010 due to changes in activity after over time (Table 2). Manuka oil baits were provided by Dr. Jim Hanula, USDA Forest Service, Athens, GA. Dr. Hanula also sorted through trap catches and provided RAB counts each year.

## Results

**Distribution of LWD in Georgia.** The laurel wilt disease front and outlying disease centers in GA as of September 2009 and October 2010 are delineated by the broad red lines on the maps in Figures 2 and 3, respectively. For comparison, LWD distribution maps based on surveys conducted during the winters of 2006-2007 and 2007-2008 in GA are illustrated in Figures A-2 and A-3, respectively. The 2009 and 2010 LWD surveys reported here concentrated on delimiting the advancing disease front into new areas, while the 2006-2007 and 2007-2008 systematic surveys involved visiting many evenly spaced sample points over a large area in GA, which facilitated a finer resolution of disease distribution on maps. A south-wide perspective of the historical distribution of LWD in GA, FL, and SC is illustrated in Figure A-1 (Appendix A).

In the one year period between fall of 2009 and fall of 2010, laurel wilt advanced about 16 km or less along most of the disease front in GA, apparently less than in prior years. Three notable exceptions include isolated disease incidents (outlying infections) located in Richmond County to the north, Laurens County to the west, and the corners of Lowndes, Lanier, Clinch counties in the south, all of which are about 65 km ahead of any known LWD and likely involved man-assisted dispersal of the RAB vector. The southern and western outlying infection centers are in redbay habitat, but the northern outlier involves only a few sassafras trees located on a sandy upland site adjacent to a busy highway, far from any known redbay or LWD.

Between spring 2008 and fall 2009, LWD caused particularly heavy losses in areas of high density redbay behind the advancing front in Brantley, Wayne and Appling counties in southeastern GA. In



2010, areas with especially heavy redbay mortality were observed in a swath through portions of western Brantley, Pierce, and Bacon counties. LWD incidence generally was more scattered and less severe throughout most of the remaining area of disease distribution, especially along the northern extent of the disease in GA.

During the past 2-3 years, most new disease infections along the advancing front in northern Bulloch, southern Jenkins, and northern Screven counties have occurred in scattered groups or thickets of sassafras trees, where redbay is relatively scarce. While LWD has not spread rapidly through this area, it has steadily infected additional groups of sassafras. The disease is approaching other areas along the western front where redbay is scarce or absent, but there is a considerable population of sassafras. These areas are particularly important to follow, since sassafras could serve as a host for LWD over large areas in the eastern U. S.

Griffith et al. (2001) developed a map for Georgia delineating eco-regions and sub-regions with similar geology, physiography, vegetation, climate, soils, land use, fauna, and hydrology, which are useful in describing the advance of LWD in GA (Fig. A-4). LWD steadily spread westward through the northern half of the Southern Coastal Plain (Sea Islands and Sea Island Flatwoods) in GA and now is well situated to rapidly expand in the remaining uninfected portions of the inland Southern Coastal Plain (southwestern portions of the Bacon Terraces and the Okefenokee Plains) in Bacon, Ware, Clinch, Atkinson, Lanier, Lowndes, and Echols counties. The spread of LWD slowed but has continued to spread into Southeastern Plains, Atlantic Southern Loam Plains (Upper Coastal Plain) north of the Altamaha River, where it is running out of high density, large redbay. As LWD spreads farther westward in central GA it will encounter less redbay and greater amounts of sassafras in scattered pockets and spread through these ecotypes is yet to be determined.

**Laurel Wilt Disease Spread Predictions.** The factors that influence the spread rate and distribution of LWD were considered in a previous GFC report on LWD in GA (Cameron et al. 2008). The potential for rapid natural spread of the disease is far greater in areas on the coastal plain where densities and size of redbay are substantially greater than inland where host trees are generally smaller and more widely scattered.

Coder (2006) developed a laurel wilt risk prediction map for GA based primarily on availability of susceptible host and climate (Fig. A-5). The spread of LWD disease in GA to date has largely been restricted to the highest and second highest risk areas (zones 1 and 2), with the exception the northern expansion of the disease where it already has moved through zones 1, 2, 3 and into the edge of the second lowest risk area, zone 4, largely in sassafras. Hanula et al. (2008) did not find the RAB to be attracted to cut stems of sassafras and speculated that this might slow the spread of laurel wilt in areas outside the southeastern Atlantic Coastal Plain. Indeed, the advance of laurel wilt in the Upper Coastal Plain in GA has slowed, but most of the spread along the northern advancing front has largely been in sassafras. LWD also has been observed in sassafras in geographic isolation from diseased redbay in SC and FL (Smith et al. 2009).

Koch and Smith (2008) developed a model and map which predicts the rate of spread and eventual geographical distribution of laurel wilt (Fig A-9). These estimates are based on a number of factors including: 1) climate, 2) density of redbay and sassafras (Figs. A-6 and A-7), 3) an assumption that RAB will not spread in the absence of redbay, and 4) a spread rate derived from the historical county spread maps for FL, GA, and SC. LWD and RAB have spread somewhat slower than predicted by

the model. Spread rate to the north in SC also has been slower than predicted. Long distance spread aided by human activities, such as the recently discovered LWD in FL and MS, is certainly beyond the predictive abilities of the model.

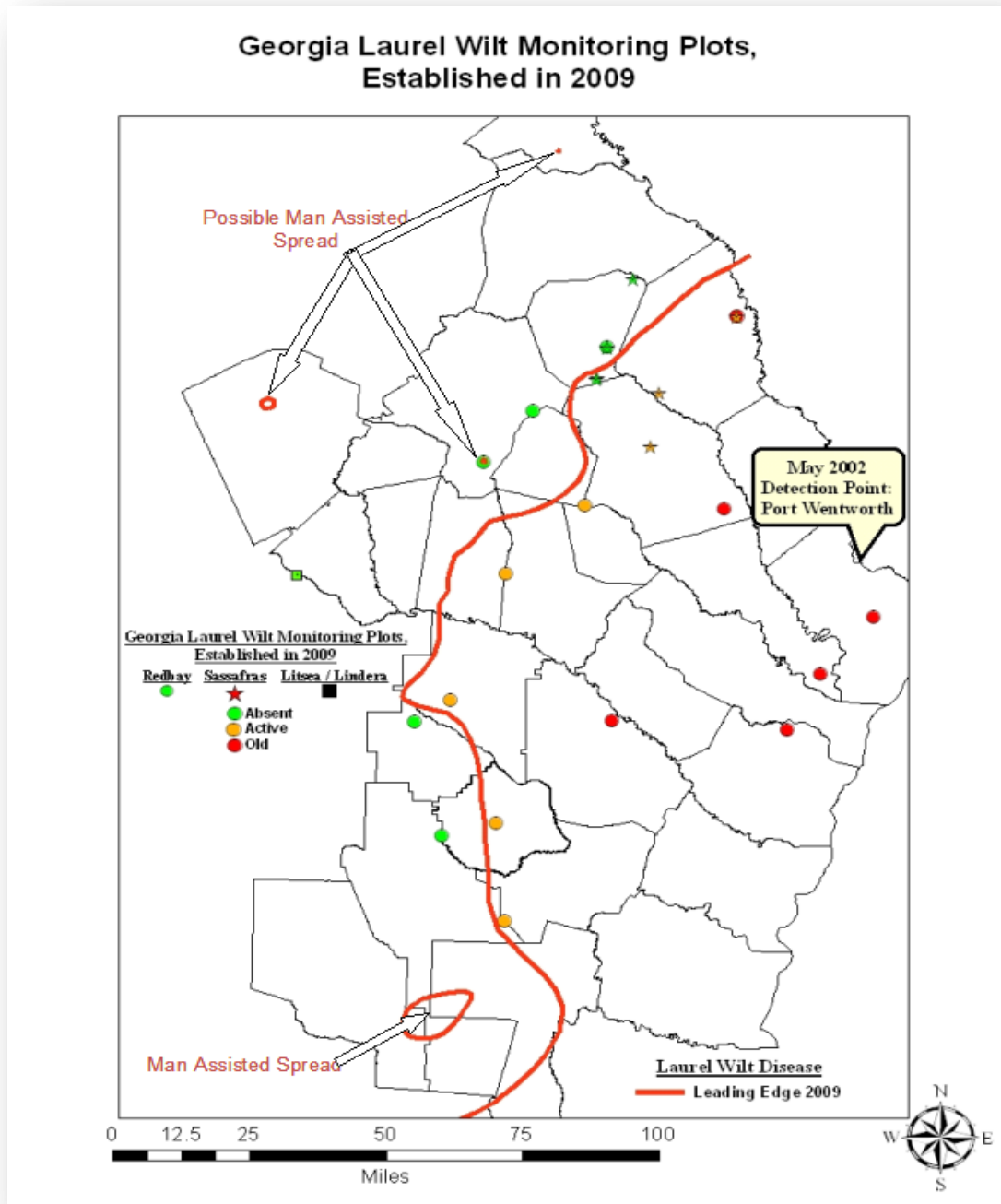


Figure 2. 2009 Georgia laurel wilt survey permanent plot locations depicted by circles for redbay plots, stars for sassafras plots, and colors indicate stage of infection (green =absent, orange=active and red =old). The red line delineates the approximate advancing front toward the west as of September 2009 and the irregular red outlines indicate isolated infection centers possibly spread by human activities.

## Georgia Laurel Wilt Monitoring Plots and Disease Front October 2010

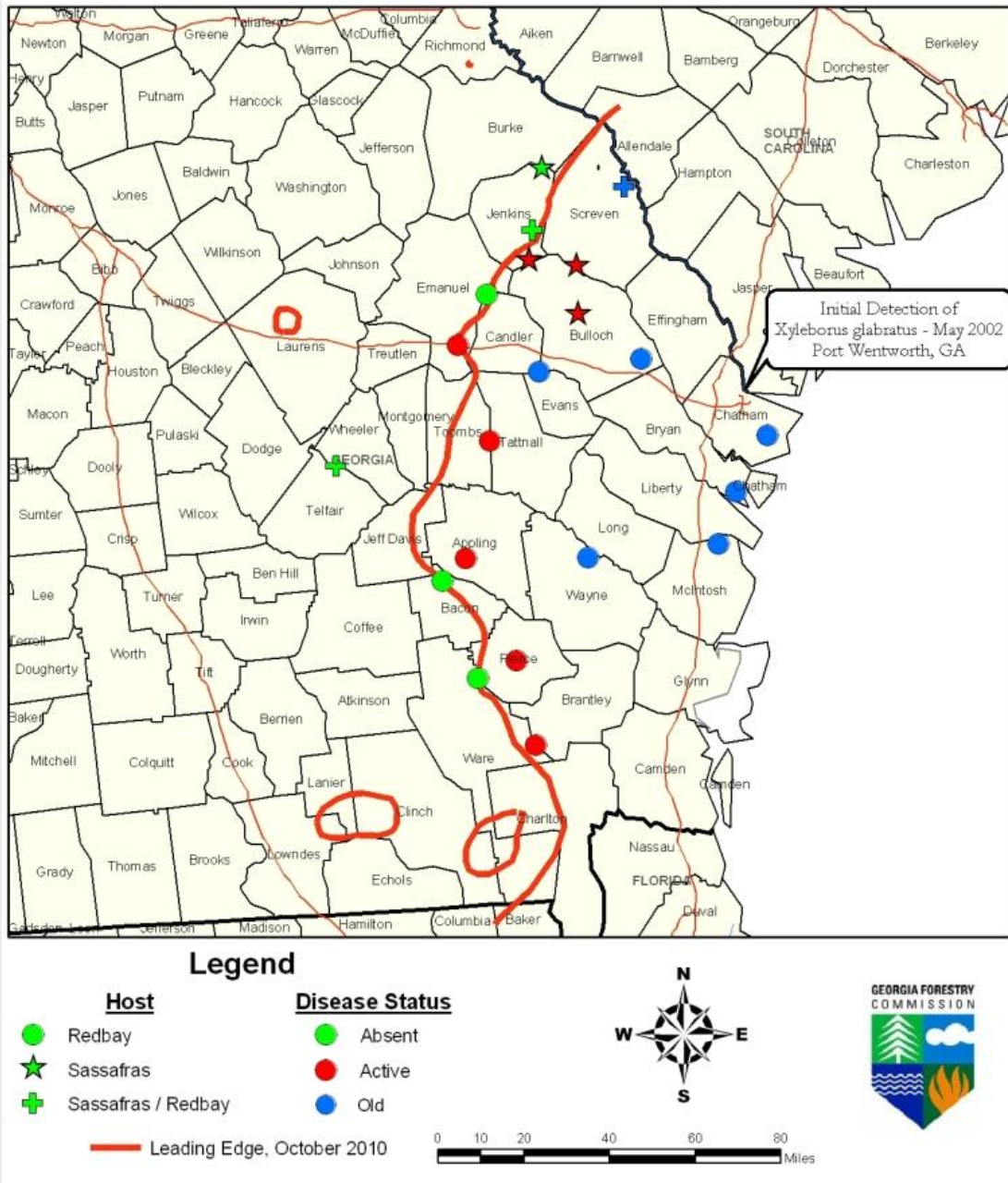


Figure 3. Georgia laurel wilt survey permanent plot locations and disease stage depicted by circles for redbay plots, stars for sassafras plots. Colors indicate stage of infection, which have been updated and color codes changed from those used in Figure 1 (green = absent, red = active and blue = old). The red line delineates the approximate disease front as of October 2010 and the irregular red outlines indicate isolated infection centers, possibly spread by human activities.

## LWD Long-term Monitoring Plots.

***Site and vegetation descriptions in LWD monitoring plots.*** LWD monitoring plots were established over a large area in southeastern GA encompassing diverse eco-regions and habitats. Redbay is found on a wide range of sites, but it prefers well drained soils with abundant water, such as, at the edges of ponds or small drains. It is not found in bottoms or ponds that flood and hold water for long periods of time. The sites located in this study with the highest densities of larger redbay trees were on the edges of small drainages or ponds on soils with organic surface layer overlaying deep sandy subsoil. All the sassafras thickets were located on upland sandy soils along old fence rows, edges of woods, agricultural fields, or road rights-of-way, while the scattered sassafras mixed with redbay trees on two sites, were on sandy soils at the edge of a drain and upland pond, on slightly higher ground than the redbay.

The eco-region classification for each monitoring plot is listed in Table 1. A key to the eco-region codes is listed at the bottom of the map (Fig. A-3) developed by Griffith et al. (2001). Three of the five initial “old” redbay plots are in the Sea Island (maritime forest) eco-region and the other two in the Sea Island Flatwoods (lower coastal plain). The “active” plots are on Sea Island Flatwoods, Bacon Terraces coastal plain sites, and Atlantic Southern Loamy Plains (upper coastal plain). The “absent” plots are located on the Bacon Terraces (southern coastal plain) and upper coastal plain. All sassafras plots are located on the upper coastal plain.

The plant species and percent cover in the understory and overstory vegetation varied by eco-region, position on the landscape (soil type), human land use patterns, and disease status. A compilation of the primary species in the overstory and understory on monitoring plots is listed in Table 2. Examples of LWD monitoring plot sites are illustrated in Figure 4. Most sites had a significant understory component of fetterbush, gallberry and/or titi, ranging from near 100 percent cover (“impenetrable”) to sparse, depending on the density of the overstory. The overstory was usually mixed hardwood/pine, except on three sites managed for pine production where redbay was growing under an overstory of pine. The sites with more abundant and larger redbay trees were “natural” mixed hardwood/pine type on soils with a thick organic layer over sandy subsoil (Figs. 4A, 4B, and 4D). The plots in sassafras thickets were in the open with nearly 100 percent sassafras due to abundant lateral root sprouts and apparent chemical inhibition by sassafras (allelopathy) that prevents the invasion of other plants (Fig. 4F). On the two mixed sassafras/redbay sites, sassafras was in the mid-story, generally below and smaller than the redbay and other hardwood species.

***Vegetation changes associated with LWD.*** After redbay trees are killed by LWD, they decay and begin falling apart within one to two years (Figs. 4E, 5A, and 5B). Naturally, on sites with a heavy component of redbay in overstory, there is a major, rapid change in the vegetation on the site caused by LWD. When the canopy is suddenly opened up, the ground cover and lower canopy vegetation proliferates in the sudden abundance of sun and other resources. Redbay regeneration (sprouts and seedlings) proliferated on several sites where redbay was a significant component in the overstory and there was little understory vegetation prior to the LWD epidemic (Cover Photo; Figs. 4E, 5A, and 5B). The impact of LWD on the habitat is far less on sites with small and/or sparse redbay in the overstory. If there is a dense overstory and/or understory of other species, redbay regeneration remains sparse after the passage of LWD (Fig. 5C). Open-grown sassafras thickets killed by LWD will likely be replaced by other pioneer species and little vegetation change will occur in mixed hardwood stands where sassafras is a minor component in the mid-story canopy.

Table 2. Common overstory and understory plant species on the laurel wilt disease monitoring plots; species present and densities vary greatly between sites.

Common name	Scientific name
<u>Overstory trees</u>	
Blackgum	<i>Nyssa sylvatica</i>
Live oak	<i>Quercus virginiana</i>
Loblolly bay	<i>Gordonia lasianthus</i>
Loblolly pine	<i>Pinus taeda</i>
Pond pine	<i>Pinus serotina</i>
Red maple	<i>Acer rubrum</i>
Redbay	<i>Persea borbonia</i>
Sassafras	<i>Sassafras albidum</i>
Slash pine	<i>Pinus elliotii</i>
Sweet bay	<i>Magnolia virginiana</i>
Sweetgum	<i>Liquidambar styraciflua</i>
Water oak	<i>Quercus nigra</i>
<u>Understory plants</u>	
Fetterbush	<i>Lyonia lucida</i>
Gallberry	<i>Ilex glabra</i>
Saw palmetto	<i>Serenoa repens</i>
Titi	<i>Cyrilla racemiflora</i>
Wax myrtle	<i>Myrica cerifera</i>
Wild grape	<i>Vitis rotundifolia</i>
Pondberry	<i>Lindera melissifolia</i>
Pond-spice	<i>Litsea aestivalis</i>

**Laurel wilt disease process in redbay.** As of September 2010, each of the LW monitoring plots had been reassessed three times at approximately 6-month intervals. Condition of tagged woody host stems, sprouts, and regeneration were noted at initiation and at each reassessment. Vegetation cover estimates were re-examined at each visit. Locations and color coded disease status as of September 2010 for each of the laurel wilt disease plots and special observation sites are illustrated in Figure 1. Additional details for each plot, including dates of initiation and assessments, disease stage over time, host species present, county locations, and eco-regions are listed in Table 1.

Considerable effort was made to establish plots in each of three disease stages (absent, active, and old). Sites selected for “absent” plots were intentionally located about 16 km ahead of areas known to have LWD, expecting that they would come under attack within a year. Yet, the disease has moved more slowly than anticipated and only one of five had an active disease episode 1 ½ years after initiation. The other four “absent” plots were still disease free 1 ½ years after initiation, but each had diseased redbay immediately adjacent or within a few kilometers. The disease stage designations served to spread plots over a variety of sites and disease conditions and disease stage classifications have been adjusted as needed based on the actual disease conditions within the plots.

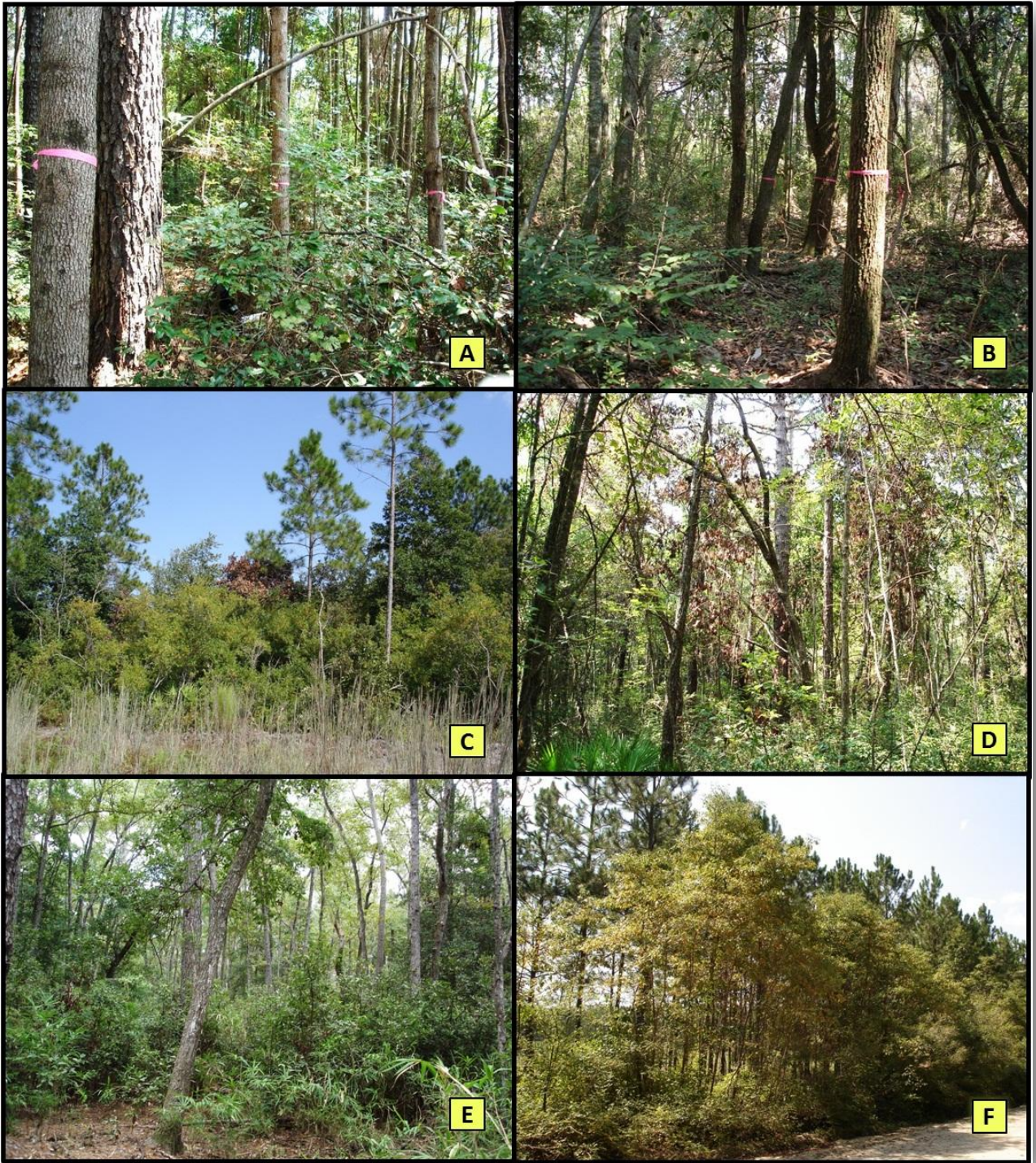


Figure 4. Selected laurel wilt monitoring plots in different disease stages as of September 2010: A) Redbay Plot #115 – LWD near but absent, B) Redbay Plot #114 - LWD recent very active, C) Redbay Plot #124 – LWD long time slowly active, D) Redbay Plot #126 – LWD recent very active, E) Redbay Plot #132 – LWD old, and F) Sassafras Plot #213- LWD absent.

A description of the LW disease process in redbay as observed in this project is summarized, roughly in chronological order, in the bullet items following below. Considerable additional detailed information is embedded in the data collected for this project, which would be more complete if extracted and summarized after the disease completes its process through more of the plots. Some of the observations presented here may be appropriately considered hypotheses and may be verified or discounted through additional observations in this project and scientific research done elsewhere.

- Leaf symptoms. Progression of leaf symptoms associated with LWD in redbay is illustrated in Figure 5. Leaves first lose turgor, droop, and turn an olive green color and remain on the tree for up to a year where they gradually change color to a red brown and eventually fade to a light chocolate brown.
- Disease progression into new areas. The LW disease process develops slowly in new areas, starting in one or a few individual trees, then accelerates rapidly in the presence of abundant host as the numbers (volume) of host trees involved (inoculum) and vectors (RAB) increase geometrically, and finally tapers off as the host is depleted. This is a typical polycyclic (sigmoid) disease process curve (Oregon State University Bot. 522). Observations:
  - First positive LWD in Pierce Co. was diagnosed in a redbay tree in May 2008; Plot #126 was established adjacent to this dead redbay in March 2009; the first symptomatic tree in the plot was not observed until February 2010; by September 2010, 60 percent of the redbays in the plot were symptomatic; this is a plot with abundant, large redbay trees.
  - First symptomatic trees in Plot #114 appeared about 1 year after a few scattered symptomatic redbay trees were first observed 100 m-300 m from the plot; nearly half of the redbay trees in the plot were symptomatic within 6 months after the first symptomatic tree was observed - this is a plot with many large redbay trees.
  - First isolated infected redbay (found and confirmed by Hanula/Fraedrich) ca. 200 m outside Plot #111 had a broken top, probably the source of attraction for the RAB vector. It filled in with unknown species of ambrosia beetles based on abundant frass, not necessarily all RAB. After six more months, no additional symptomatic redbay trees have been found in the area.
- Elimination of all large redbay. LWD killed nearly all redbay trees greater than 2.5 cm DBH in areas of high density, large trees. Observations:
  - No live redbay trees greater than 2.5 cm DBH were present in any of the five plots established in the “old” disease stage category.
  - There may be exceptions where some redbay trees larger than 2.5 cm escaped or survived LWD (see Special Redbay Survival Survey section below).
- Disease progression in areas with sparse host. The LW disease process appears to be slower in the presence of fewer, smaller, and more scattered hosts, such as that which is encountered at the western and northern advancing fronts of the disease episode in GA. This may be more of a monocyclic disease curve. Observations:
  - Plots #'s 123, 124, and 125 are located in areas of extensive commercial pine plantations with abundant small redbay under the pines, but large, dense redbay stands have not been observed.

- Redbay trees in these plots were relatively abundant, but small.
- LWD was present in each of these plots at establishment in March 2009, but substantial numbers of asymptomatic redbay trees remained in September 2010.
- When ambrosia beetles breed in redbay killed by LWD. Frass from ambrosia beetle attacks (RAB and/or other species) does not become abundant on infected redbay trees until months after initial crown symptoms appear and disappears within about a year after trees die.
- Breakup of dead redbay trees. Redbay trees killed by LWD start falling apart within a year and most branches and upper boles are on the ground within 1 ½ years after initial symptoms appear; breakdown of diseased trees is hastened by ambrosia beetle attacks and wood rot fungi (Figs. 5A and 5B).
- Fate of sprouts around trees killed by LWD. Sprouts start to proliferate around the base redbay trees killed by LWD within 6 months of initial disease symptoms and some remain alive for years (Fig. 5C). (Note some “sprouts” may be seedlings; no attempt was made to distinguish between sprouts and seedling regeneration in this study) Observations:
  - Epicormic sprouting occurs on some redbay trees infected by *R. lauricola*, but this is the exception and most die quickly as the fungus spreads rapidly through the tree all the way to the ground.
  - Root sprouts are not produced around all redbay trees killed by LWD and many that grow off the root flare on redbay trees are killed quickly by the disease.
  - Sprouts not obviously attached to the root flare die at a lower rate and new sprouts continue to emerge years after trees are killed by LWD.
  - Root sprouts under dense overstory canopy grow slowly (Fig. 5C).
  - Many redbay sprouts and seedlings are killed or die back from *Xylosandrus compactus* attacks.
  - Tender shoots on redbay sprouts and seedlings are commonly hollowed out and killed and patches of bark are removed from branches and stems by an unknown insect. This damage is possibly caused the adult avocado weevils, *Heilipus apiatus* (Hoffman 2003).

Redbay regeneration after LW. Observations:

- Redbay regeneration in the form of seedlings and/or root sprouts proliferates more on some sites than others. Regeneration appears to be more abundant on sites with dense redbay in the canopy, where the forest floor is open to full sun after redbay trees die and fall apart (Figs. 4E, 5A, and 5B).
- Occasional, scattered seedlings and/or root sprouts have been observed dying from LWD in areas where the disease passed through many year before (Fig. 5D), either from latent fungus in stumps or new beetle attacks (source of the fungus is not known, but a few RAB were trapped in these areas (see RAB Monitoring below).
- A very low percentage of the regeneration is killed by LWD in areas where the disease moved through many years ago, but these small trees may be poor host material for the RAB vector, and a second wave of disease could occur after regeneration reaches a more substantial size.



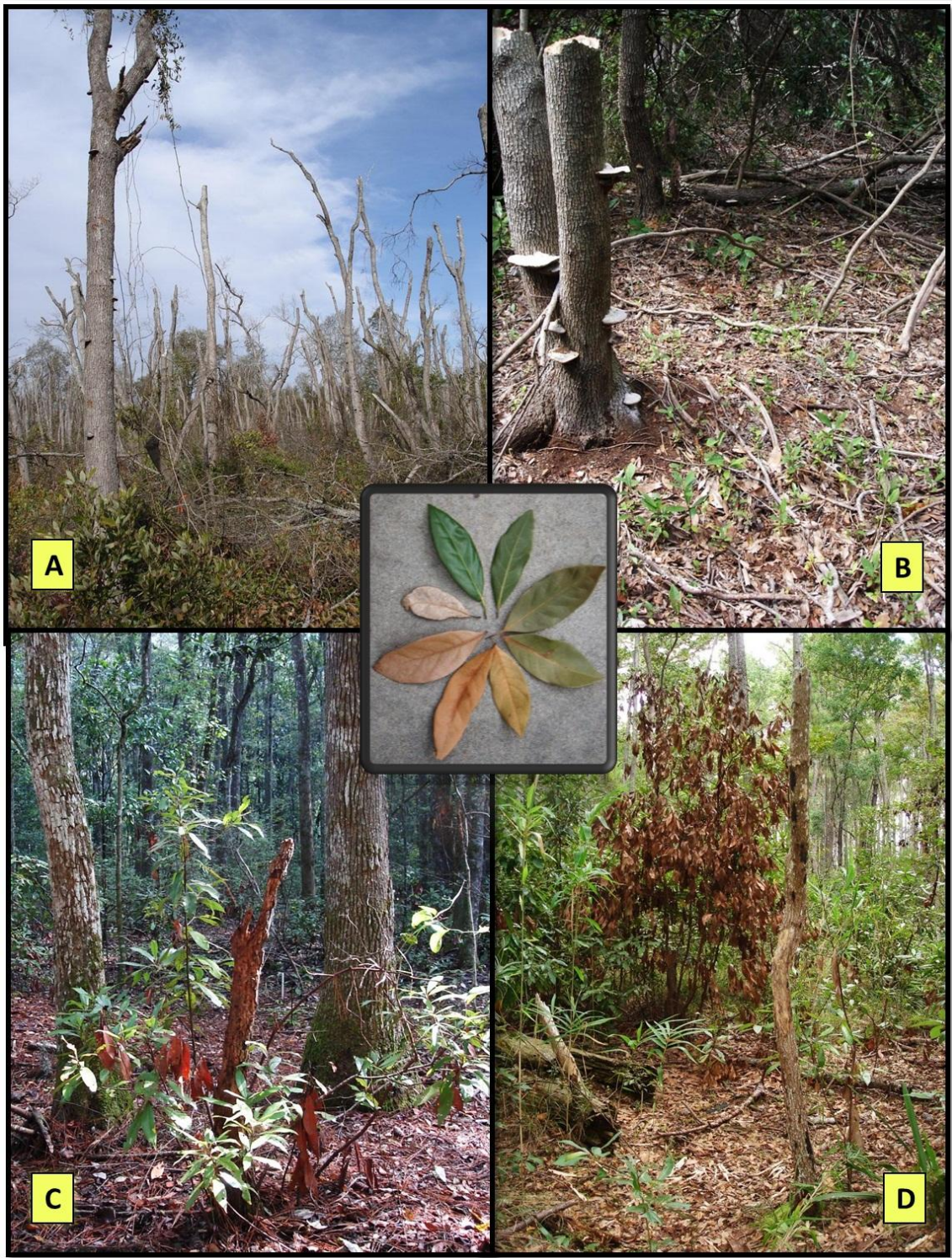


Figure 5. Laurel wilt in redbay with progression of leaf symptoms illustrated in the center: A) Total mortality and collapse in a dense stand of large redbay, B) regeneration after breakup, C) sprouts around stump, D) stump sprouts killed by laurel wilt after first wave of LWD.

**Laurel wilt disease process in sassafras.** Relatively little is known about the laurel wilt disease process in sassafras, but it may differ significantly from that in redbay due to inherent differences in site preferences, growth habits, and distribution on the landscape. Sassafras is generally intolerant of shade, often pioneering old fields, fence rows and edges of woods in dense thickets populated by sprouts (ramets) off underground runners from parent trees. Sassafras prefers well-drained sandy loam soils on dry ridges and upper slopes and is generally in pure stands and seldom grows with redbay on the same site, although two plots with mixed redbay/sassafras are included in this study.

Among the five long-term monitoring plots established in sassafras thickets, two already had active LWD at initiation in 2009, and the disease expanded and killed most of the trees in both stands over the year and a half period of observation. Two of the three sassafras plots without disease symptoms at initiation were located within 100 m of each other within the known area of LWD distribution. Two trees in one of these two plots and a few other sassafras trees outside plots were infected and died from LWD in 2010 (confirmed by Dr. Steve Fraedrich, USDA Forest Service). The third site was located about 16 km in advance of the disease and remained without symptoms through September 2010 (Fig. 4F). A sixth site with two groups of relatively large sassafras trees along an old fence row had active LWD in early 2009 and was observed at the time of each permanent plot assessment and photographed to document disease spread. The pattern of disease symptoms in numerous sassafras thickets and individual trees along roadways has been observed in Bullock, Jenkins and Screven counties while working on this project over the past several years.

To advance knowledge and encourage further research, preliminary descriptions of certain aspects of the LWD process in sassafras are outlined in the bullet items below. Many of the observations presented here are illustrated in Figures 6-10.

- **Area-wide disease distribution and spread.** The area in GA with the greatest amount of LWD in sassafras is in a band across the southern portion of Jenkins County, northern Bulloch and northern Screven counties where redbay is sparse and sassafras is relatively abundant in dense thickets and scattered trees, mostly along fence rows or the edge of openings. LWD has not moved through the area killing all sassafras trees in a contiguous front, as is often seen with scattered distributions of redbay. Rather it has infected sassafras in an apparent haphazard fashion, killing some individual trees and entire thickets, while others are untouched.
- **Leaf symptoms:** Sassafras is a deciduous tree, which results in very different leaf symptoms from those associated with LWD in redbay, which is an evergreen. Discoloration and wilting (drooping) of leaves are the first obvious symptoms of LWD disease in sassafras. Leaves fall shortly thereafter leaving branches bare (Figs. 6C and 6D). However, leaf symptoms on sassafras vary with leaf age and season. Trees succumbing to the disease in spring may have an aborted leaf-out, or new, tender leaves may emerge, wilt, become dark green, shrivel, turn brown, and fall from the tree (Fig. 6B). Fully developed leaves are more likely to droop and turn varying shades of fall colors (yellow, orange, red, and brown) and then fall (Fig. 6A).
- **Vascular staining.** Black staining and streaking in the vascular system of infected sassafras is similar to that in redbay (Fig. 9B), but sometimes it is not present in the outer growth ring on symptomatic trees. Rather, it may be in scattered points within the stem or in a solid band in a previous growth ring as illustrated in Fig. 9A. The solid dark stain in the middle of the stem

in Figure 9A may be a result of some other type of damage and unrelated to the LWD. The presence of internal staining and clear wood in the most recent growth ring may be a function of how the fungus moved in the vascular system or an indication of some resistance of the tree to the fungal invasion. Also, dark staining typical of LWD has been observed in apparently healthy trees in a diseased thicket of sassafras, indicating the presence of the fungus in the vascular system of these trees prior to becoming symptomatic.

- Disease initiation. RAB presumably is the vector that introduces *R. lauricola* into sassafras trees, as it is in redbay. Based on limited number of observations, it appears that the largest sassafras trees in a thicket or the largest scattered individual sassafras trees in an area are the first to die from the disease, although small individual trees are also killed by LWD. Individual beetle attacks have been found at the base of a few small sassafras trees, near the soil line, by removing the bark. This may be the point where the fungus was introduced, or an initial attack for colonization of an already diseased tree. In either case, it appears that beetles were attracted to the base of trees as opposed to higher locations on the stem.
- Spread within trees in thickets. Once the first symptoms of LWD are noted in a sassafras thicket, spread to other trees can be rapid, apparently moving from one tree (ramet) to another through interconnected lateral roots. Spread is often directional as it moves through thickets (Fig. 6C). From the time of initial symptoms, the disease can kill nearly all trees in a thicket within about a year or two. The dated photos in Figure 7 and Figure 8 illustrate the spread of LWD through two separate thickets. Most trees in Figure 7 progressed from symptomatic or green to dead in a three-month period. However, in all thickets monitored, individual trees, mostly small trees are still alive. These may be seedlings lacking root connections to diseased trees in the thicket or are otherwise unattractive to RAB.
- Spread through lateral roots. Debarking of several symptomatic sassafras trees revealed typical black staining in the outer surface of the wood in the stem, which continued into the root flare and roots below (Fig. 9B). Strong evidence that the LW fungus can move through lateral roots in sassafras thickets to infect adjacent trees was obtained by digging up and inspecting the roots of the most recent symptomatic tree (drooping, off color leaves) in a thicket with directional spread. The tree essentially had no roots of its own, but rather was a sprout off of a continuous lateral root. The root coming in from the last dead tree had abundant black staining that continued up one side of the stem of the symptomatic tree. The root on the other side of the tree that apparently was connected to adjacent uninfected live trees had no black staining.
- Preference between sassafras and redbay. The two plots with sassafras mixed stands with redbay afford an unusual opportunity to observe which species is preferred on a given site. This is a very small sample and only one site had LWD present as of September 2010. At that site (Plot #121), LW killed some of the redbay trees before sassafras started to die, but nearly all the numerous sassafras trees died rapidly within about a 6 month period, at the same time the last redbay trees died. Also, an important factor to consider is that the redbay trees on the site were larger (avg. 16.5 cm, ranging from 5.3 cm to 34.3 cm DBH) than the sassafras trees (avg. 6.9 cm, ranging from 3.0 cm to 15.0 cm DBH). Larger redbay trees have been reported to die more quickly than smaller ones in a given area (Fraedrich et al. 2008).
- Discontinuous spread. In contrast to the thickets mentioned above, groups of sassafras have been observed with some individual trees killed by LWD, while many other trees remain alive, apparently escaping or are resisting the disease. The thicket in which Plot 222 is located

had an aggressive disease episode spreading in two directions at plot initiation. Spread has continued in one direction in larger trees and slowed dramatically in the other direction as it moved into smaller trees. The sassafras trees in Plot 212a are more scattered and may have few root connections. The two largest trees in the plot died in 2009 and no other trees have become symptomatic yet.

- Root and epicormic sprouts. Root sprouts are common around redbay trees killed by LW, but few sprouts have been observed around sassafras trees killed by LWD, even though sassafras has the natural tendency to send out lateral roots and shoots. A lack of root sprouts in sassafras may indicate that the fungus spreads more effectively into, and kills, roots in sassafras than in redbay. On the other hand, epicormic sprouts on the stems of redbay trees killed by LWD are relatively uncommon, while epicormic sprouts have been observed on the lower stems on many sassafras trees killed by LWD. This is apparently a result of a distinct disease process in sassafras, perhaps involving some level of resistance. These observations are seemingly contradictory and need further investigation.
- Redbay ambrosia beetle (RAB). Given that 1) sassafras trees are dying from LWD in significant numbers and 2) the disease has continued to spread among sassafras trees in areas with little or no redbay, it is clear that RAB is attracted to and breeds in sassafras, at least to some extent. Frass accumulations, ostensibly from RAB attacks (although other ambrosia beetle species may be involved), have been observed primarily at the base of sassafras trees, mainly below 1 m, but occasionally up to 2 m (Fig. 10A). Long frass extrusions (“toothpicks”) have seldom been observed on sassafras. Numerous brood-adult RAB emerged from two bolts cut from the lower stems of sassafras trees killed by LW confirming that RAB can successfully breed in sassafras (Figs. 10B and 10C). However, more in-depth study is needed to determine what attracts RAB to sassafras and what portions of sassafras stems are utilized for brood production.
- Long distance spread in sassafras. One notable LWD incident indicates that RAB is capable of spreading the disease in sassafras to distant locations, probably aided by human transport of infested host material. In 2009, LWD was discovered along a 4-lane divided highway south of Augusta in Richmond County. Several small (<2.5 cm diameter), “off color” (symptomatic) sassafras trees were noticed at the edges of the rights-of-way on both sides of the road. Closer inspection revealed typical LWD staining of the wood of the small trees and a group of large (ca. 20 cm DBH) sassafras killed by LW (confirmed by Dr. Stephen Fraedrich) were found at the edge of an adjacent mature longleaf pine stand. RAB not only infected the larger trees, it apparently was attracted to and infected the small individual sassafras seedlings on either side of the road. Although a thorough reconnaissance of the area was not conducted, it appears that this is an isolated infection with the nearest known LWD about 65 km to the south.
- Break-up of trees killed by LWD. Sassafras apparently does not decay as quickly as redbay. If true, a possible explanation might be that the wood in sassafras dries out faster and/or is not utilized to the same extent by the variety of ambrosia beetles and wood rot fungi that colonize and hasten the decay of wood in standing redbay trees.

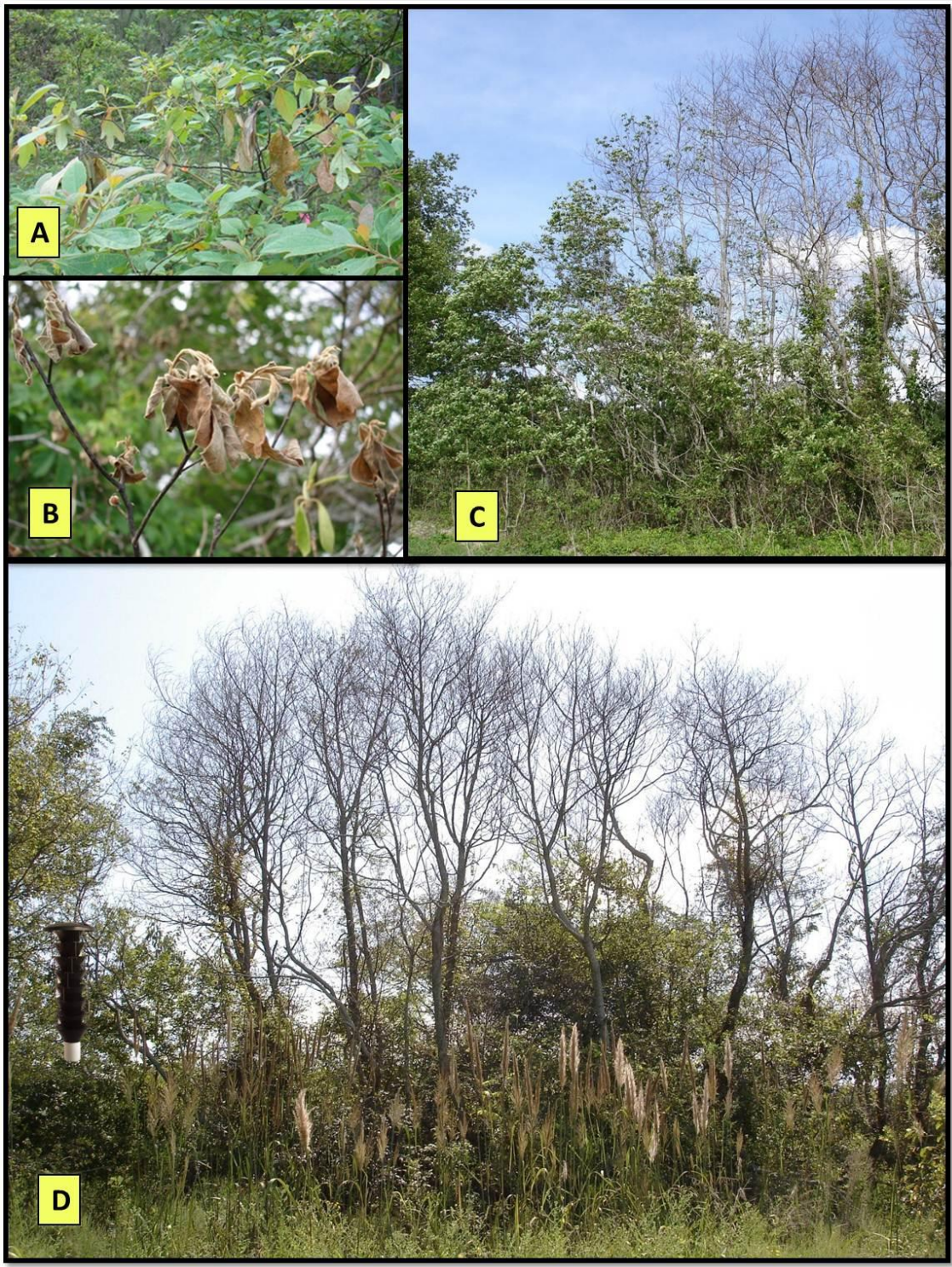


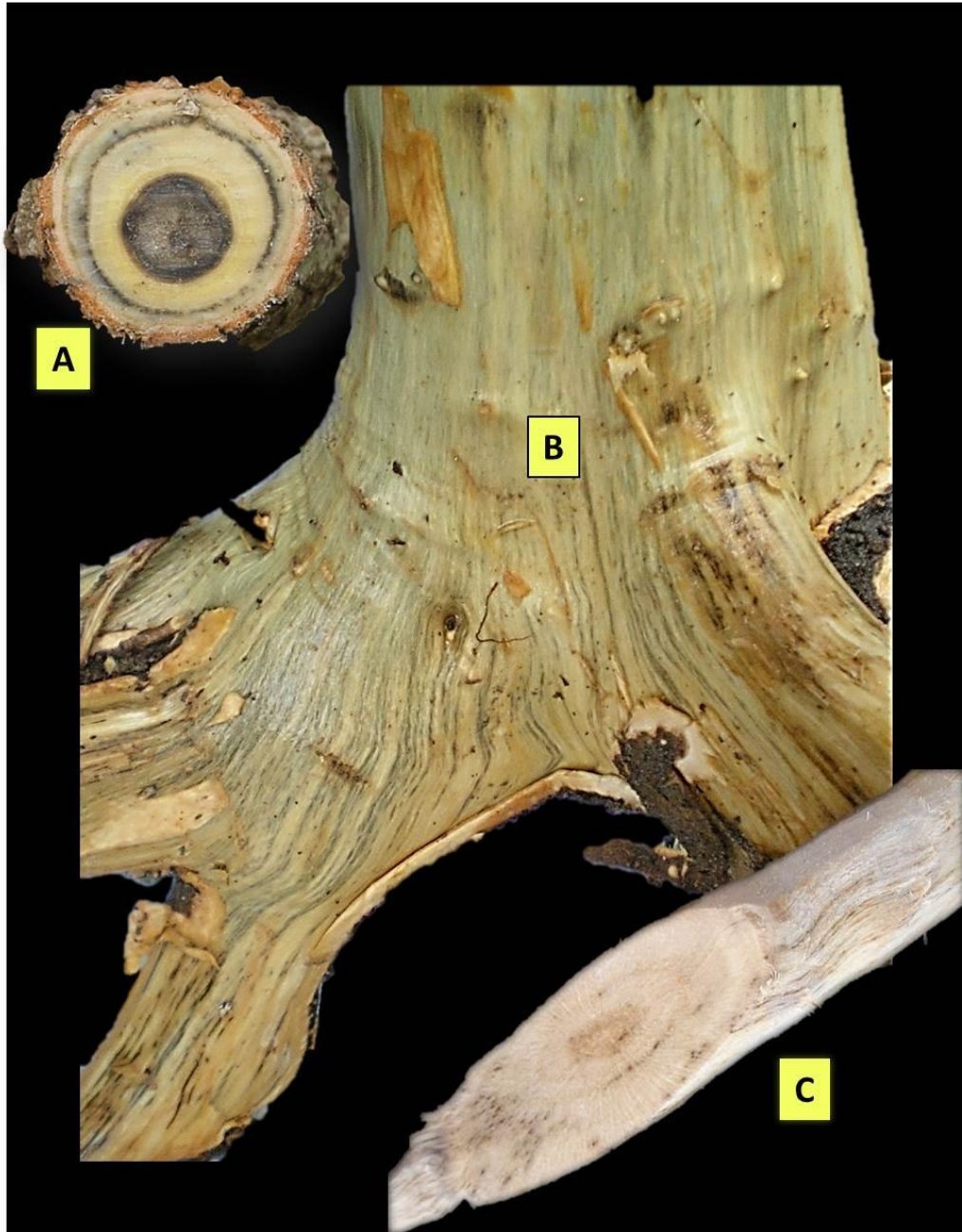
Figure 6. Progression of laurel wilt disease groups (thickets) of sassafras illustrating: A) Initial leaf symptoms, B) short-lived dead leaves, C) rapid spread through thicket, D) one year following rapid mortality and RAB trap position.



*Figure 7. Sassafras Plot 211 near Statesboro, GA showing progression of laurel wilt disease.*



*Figure 8. Sassafras Plot 224 in Bulloch County, GA showing progression of laurel wilt disease. The green plants in the middle and bottom photos are mostly vines (poison ivy and green briar) and other non-host plants.*



*Figure 9. Portions of three separate sassafras trees infected with the laurel wilt fungus, illustrating the black staining resulting from a reaction of the host to the presence of the fungus, (A) embedded within the growth ring of the previous growing season in the stem of a small tree, (B) visible on the outside of the wood exposed by removing the bark at the base of a tree, in the root flare and roots, and (C) in a long lateral root attached to a small recently symptomatic tree at the edge of an expanding disease center in a sassafras thicket .*



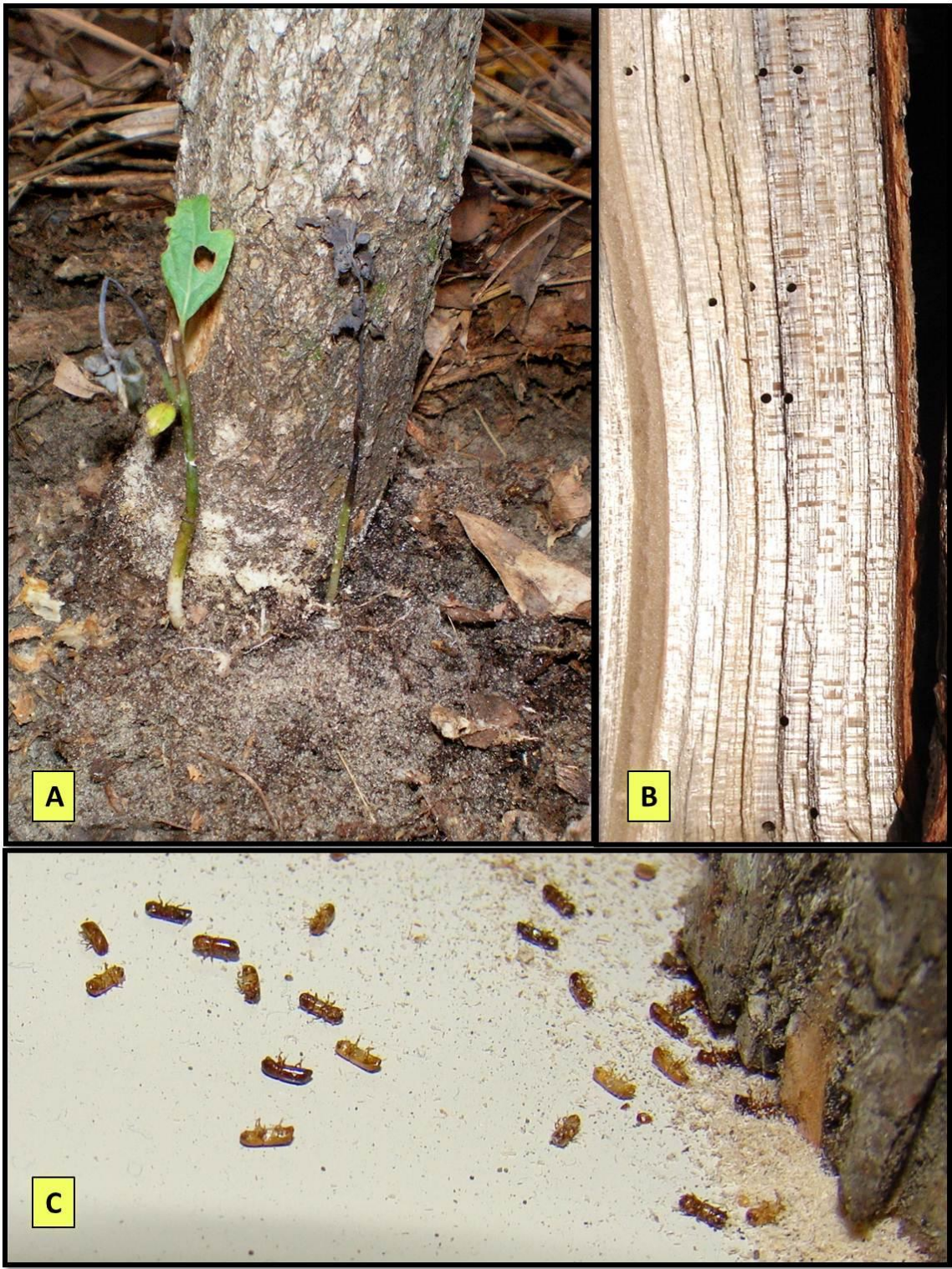


Figure 10. Redbay ambrosia beetle in sassafras: A) ambrosia beetle frass (presumably RAB) at the base of a sassafras tree with uncommon root flare sprouts, B) bolt from the lower stem of a sassafras tree split open to expose RAB galleries, C) boring dust and RAB brood adults (note callow beetles) that emerged from bolt in “B” above.

**Redbay Ambrosia Beetle (RAB) Monitoring.** RAB monitoring traps baited with Manuka oil were deployed throughout the month of August in 2009 and 2010 at 22 and 23 sites, respectively, most of which were monitoring plot sites. Traps were located in areas with no known LW disease (“absent”), areas of active disease development (“active”), and areas where the disease moved through at least several years before (“old”), most on redbay sites and a few on sassafras sites. The disease status designation for individual trapping sites was set for each year based on conditions at the time of trap deployment.

A total of 505 RAB’s were captured in 22 traps deployed for 30 days in August 2009 and 282 in 2010 (Table 2). Mean numbers of beetles captured in each of the three disease status categories and by host species are presented in Table 3 and Table 4 for 2009 and 2010, respectively, and the mean numbers for each disease stage and host species for 2009 and 2010 combined are illustrated in Figure 11.

*Table 2. Numbers of redbay ambrosia beetles captured in Lindgren funnel traps baited with commercial Manuka oil lures throughout the month of August 2009 and August 2010.*

Trap No	Host	August 2009			August 2010		
		Date	Status	# RAB	Date	Status	# RAB
131	Redbay	8/4-9/4	Old	0	8/2-9/2	Old	2
132	Redbay	8/4-9/5	Old	0	8/3-9/2	Old	0
133	Redbay	8/4-9/4	Old	2	8/1-8/31	Old	2
134	Redbay	8/5-9/5	Old	0	8/2-8/31	Old	1
135	Redbay	8/5-9/4	Active	5	8/2-8/31	Old	5
136	Redbay	8/4-9/4	Active	21	8/1-8/31	Old	1
114	Redbay	8/5-9/5	Active	2	8/2-9/1	Active	1
121	Redbay	8/6-9/5	Active	9	8/3-9/1	Active*	13
122	Redbay	8/5-9/5	Active	414	8/2-8/31	Active	15
123	Redbay	8/5-9/4	Active	14	8/2-8/31	Active	12
124	Redbay	8/4-9/4	Active	27	8/1-8/31	Active	4
126	Redbay	8/4-9/4	Active	1	8/1-8/31	Active	57
125	Redbay	NA	NA	NA	8/1-8/31	Active	55
113	Redbay	8/4-9/4	Absent	0	8/1-8/31	Absent	2
116	Redbay	8/7-9/5	Absent	0	8/2-9/1	Absent	0
117	Redbay	8/7-9/4	Absent	0	8/1-8/31	Absent	0
111	Redbay	8/6-9/5	Absent	0	8/2-9/1	Absent	0
112	Redbay	8/6-9/5	Absent	0	8/3-9/1	Absent	1
115	Redbay	8/4-9/4	Absent	0	8/1-8/31	Absent	0
212	Sassafras	8/6-9/5	Active	1	8/3-9/1	Active	2
222	Sassafras	8/6-9/5	Active	2	8/3-9/1	Active	0
224	Sassafras	8/6-9/5	Active	7	8/3-9/1	Active	109
213	Sassafras	8/6-9/5	Absent	0	8/3-9/1	Absent	0
			<b>Total</b>	<b>505</b>		<b>Total</b>	<b>282</b>
* Mixed redbay/sassafras plot - redbay old, probable emergence from sassafras							

Table 3. Numbers of redbay ambrosia beetles captured by disease stage in traps during August 2009.

Host	# Traps*	Disease Stage	# RAB	Range	Avg. # RAB
Redbay	4	old	2	(0-2)	0.5
Redbay	8	active	493	(1-414)	61.6
Redbay	6	absent	0	0	0
Sassafras	3	active	10	(1-7)	3.3
Sassafras	1	absent	0	0	0

Table 4. Numbers of redbay ambrosia beetles captured by disease stage in traps during August 2010.

Host	# Traps*	Disease Stage	# RAB	Range	Avg. # RAB
Redbay	6	old	11	(0-5)	1.8
Redbay	7	active	157	(1-57)	22.4
Redbay	6	absent	3	(0-2)	0.5
Sassafras	3	active	111	(0-109)	37.0
Sassafras	1	absent	0	0	0

\*Assignment of traps to disease stage categories was modified from the initial classification at plot initiation to reflect disease activity adjacent to trapping sites at the time of trap deployment.

Numbers of beetles captured varied greatly among sites and years, but were roughly correlated with the number and size (volume) of dying host trees in the vicinity of the trap. Many (414) RAB were captured at Plot 122 in 2009, which had a large number of dead and dying redbay trees with abundant boring dust (frass) during the trapping period. Fewer RAB were captured at several of the “active” sites than might have been expected based on host condition in the area, especially in 2010. Either there were few RAB flying during the trapping period at these sites or the baits are not highly attractive and did not compete well with other sources of attraction. No beetles were trapped at the “absent” sites in 2009, all of which were at least 17 km beyond the advancing disease front at that time. However, RAB were caught at two “absent” sites in 2010 (one beetle at one site and two at the other), both of which had dying host trees within a few kilometers from the traps. On the other hand, two other absent sites had dying redbay trees within a 200-300 m of the trap in 2010, but no RAB were captured at either of these sites. Only two beetles were captured at one of four “old” sites in 2009, but 1-5 RAB were caught in five of six “old” sites in 2010. Clearly, low populations of RAB remain for many years after LW moves through an area. This is not surprising since widely scattered redbay sprouts/seedlings have been observed dying from LW in these areas year after year.

RAB were captured at each of the three active sassafras sites in 2009 and at two of three sites in 2010, and no beetles were captured either year at the one absent site, which is located about 17 km from the nearest known LWD. Each of the active sassafras sites were in areas with substantial sassafras and little or no known redbay for many kilometers around. The largest catch in 2010 was from a trap located adjacent to an isolated group of large sassafras trees that died the previous year (Fig. 6D). In all probability, these RAB emerged from the adjacent sassafras, substantiating the conclusion that RAB can breed in sassafras and the disease can exist in the absence of redbay.

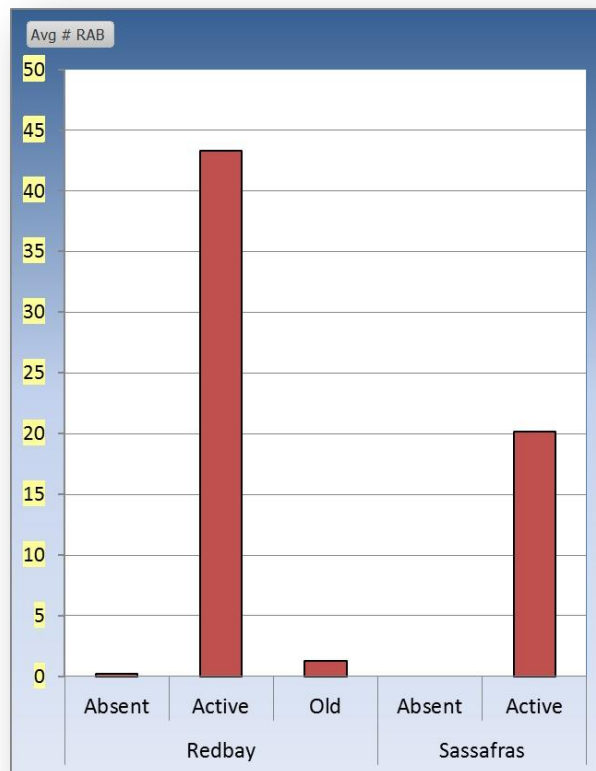


Figure 11. Mean numbers of redbay ambrosia beetles caught in August 2009 and 2010 by laurel wilt disease status and host species.

## Special Redbay Survival Survey

**Background.** Five LWD monitoring plots in an “old” disease status category were established in the spring of 2009 as part of this project. These five plots were located on the coastal plain in areas where the disease had swept through and killed most redbay trees several years prior to plot establishment. All redbay trees greater than 2.5 cm DBH (diameter breast height = 1.4 m) were dead in each of these plots, presumably killed by laurel wilt disease. Numerous live redbay trees of varying sizes ranging up to about 14 cm DBH were observed while walking along the Richmond Hill Trail in January 2011. These trees are situated on a dike bordered on both sides by tidal canals, which surround some long abandoned rice fields. This trail is near the origin of the laurel wilt disease epidemic that started near Savannah around 2002. Dead and dying redbay trees had been observed during occasional walks on this trail from 2005 through 2007. To investigate this surprising find, a simple redbay tally was conducted to document the numbers of living and recently killed redbay trees along a portion of this trail.

**Methods.** Redbay trees in a transect along a portion of the Richmond Hill Trail, approximately 1,592 m long and 22.9 m wide, encompassing an area of about 3.6 hectares, were surveyed on January 29, 2011. Numbers of healthy and dead redbay trees (with leaves attached) on either side of the trail were tallied in two size categories: greater than 2.5 cm DBH and less than 2.5 cm DBH.



Figure 12. Richmond Hill Trail located between Highway 17 and the railroad track adjacent to the Ogeechee River with the redbay survey traverse indicated in red.

**Results.** A total of 318 redbay plants were tallied, of which 78 (ca. 25%) were greater than 2.5 cm DBH, ranging up to about 14 cm for two trees, and 235 stems less than 2.5 cm DBH. Only five stems were dead, probably caused by laurel wilt (one was cut off and typical black staining was present), all of which were less than 2.5 cm DHB and in groups of sprouts (probably not seedlings) Only one redbay tree > 2.5 cm showed possible initial symptoms of LWD at the time of the survey.

**Discussion.** The presence of apparently healthy redbay trees greater than 2.5 cm DBH in this area and the lack of mortality among the largest trees was an unexpected finding. Whether these trees escaped the initial disease epidemic that swept through this area, resisted beetle attack and/or fungal infection, or have grown from a smaller size since the initial disease episode is unknown. Why numerous redbay trees greater than 2.5 cm are alive in this area, but not in any of the five “old” LWD monitoring plots is a mystery.

A search for similar surviving populations of redbay, a more thorough assessment of surviving redbay, and continued observations of this population may be warranted. Counting rings to age trees might shed light on whether or not these trees were small and unsuitable hosts during the original disease epidemic. Dissection of a few trees would reveal if typical black staining is present in the older rings, which might indicate the trees were infected by *R. lauricola* and overcame the disease. Whether or not these trees continue to survive and grow is yet to be seen.

## Summary

Much has been learned about the spread of LWD in GA and the LW disease processes in redbay and sassafras through disease distribution surveys and establishment and repeated assessments of long-term monitoring plots. Specific findings are summarized below.

### Distribution and spread:

LWD moved westward at about 17 km per year in 2009 and 2010, more slowly than in the past, but killed large numbers of redbay trees behind the front. Three notable exceptions include tree isolated disease incidents, all about 65 km ahead of any known LWD incidence and likely involved man-assisted dispersal of the RAB vector.

During the past 2-3 years, most new disease infections along the northern advancing front have occurred in scattered groups or thickets of sassafras trees, where redbay is relatively scarce or absent. While LWD has not spread rapidly through this area, it has steadily infected additional groups of sassafras. The disease is approaching other areas along the western front where redbay is scarce or absent, but there is a considerable population of sassafras. These areas are particularly important to follow, since sassafras could serve as a host for LWD over large areas in the eastern U. S. Along the southwestern front the disease is likely to kill large numbers of abundant redbay in this area during the next several years.

### Disease process in redbay:

The LW disease process develops slowly in new areas, starting in one or a few individual trees, then accelerates rapidly in the presence of abundant host as the numbers (volume) of host trees involved (inoculum) and vectors (RAB) increase geometrically, and finally tapers off as the host is depleted. The LW disease process appears to be slower in the presence of fewer, smaller, and more scattered hosts, which it has encountered at the western and northern advancing fronts of the disease episode in GA.

LWD has killed nearly all redbay trees greater than 2.5 cm DBH in areas of high density, large trees in the coastal maritime forest. One exception was found in which numerous redbay trees > 2.5 cm up to 12 cm DBH are still healthy. Abundant regeneration is present in areas where dense redbay stands were killed, fell apart, and the canopy was opening up. LWD and RAB are still active at low levels in redbay regeneration, many years after the initial epidemic. A second wave of disease could occur when regeneration reaches a more substantial size.

### Disease process in sassafras:

LWD has continued to spread in sassafras in areas where there is little or no redbay. LWD infects sassafras in an apparent 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 rapid in dense thickets, apparently through lateral roots. Leaves on sassafras trees with LWD droop, shrivel, or turn shades of yellow, orange or red and fall off shortly after symptoms are manifested leaving crowns bare within months after first symptoms appear. Characteristic black staining in the wood can be present on the surface of the outer sapwood, as well as embedded beneath a non-

symptomatic current year's growth ring. Epicormic shoots are common on the lower stems of larger sassafras trees, and ambrosia beetle attacks are most abundant at base of sassafras trees killed by LWD. RAB can introduce the LW fungus and produce brood in sassafras trees in the absence of redbay.

#### Redbay ambrosia beetle trapping:

Traps baited with Manuka oil and deployed throughout August 2009 and August 2010 proved effective in monitoring and comparing numbers of RAB in three stages of LWD. RAB were not detected at sites well in advance of the disease. Numbers of beetles captured varied greatly among sites and years, but were roughly correlated with the number and size (volume) of dying host trees in the vicinity of the trap. Small numbers of RAB were caught in traps deployed in areas where the disease killed all large redbay trees many years before indicating that low populations are being maintained in small redbay in the area. Substantial numbers of RAB were caught in traps adjacent to sassafras recently killed by LWD supporting observations that RAB brood can be produced in sassafras.

#### Future:

Some of the observations presented in this report are preliminary and need further study to prove their validity. Much additional information could be derived from continued plot assessments, for one or two more years, and in-depth evaluation of data sets collected in this project. Continued evaluation of the spread of LWD in sassafras will provide valuable insight into the potential for expansion of the disease well beyond the distribution of redbay. Research is encouraged to expand upon aspects of the LW disease process needing more in-depth investigation.

Long distance spread of LWD has continued to occur in GA and surrounding states, emphasizing the continuing need for more effective education aimed at limiting the movement of host material harboring RAB.

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## References

- Cameron, R.S., C. Bates, and J. Johnson. 2008. Distribution and Spread of Laurel Wilt Disease in Georgia: 2006-08 Survey and Field Observations. Georgia Forestry Commission report. September 2008. 28 p.
- Coder, K.D. 2006. Redbay wilt risk assessment map for Georgia. University of Georgia, Warnell School of Forestry and Natural Resources, Outreach Publication SFNR06-8.
- Fraedrich, S.W., T.C. Harrington, R.J. Rabaglia, M.D. Ulyshen, A.E. Mayfield, III, J.L. Hanula, J.M. Eickwort, and D.R. Miller. 2008. A fungal symbiont of the redbay ambrosia beetle causes a lethal wilt in redbay and other Lauraceae in the southeastern United States. *Plant Disease* 92:215-224.
- Griffith, G.E., J.M. Omernik, J.A. Cornstock, S. Lawrence, and T. Foster. 2001. Level III and IV Ecoregions of Georgia.
- Hanula, J.L., A.E. Mayfield, III, S.W. Fraedrich, and R.J. Rabaglia. 2008. Biology and host associations of the redbay ambrosia beetle, *Xyleborus glabratus* (Coleoptera: Curculionidae: Scolytinae), exotic vector of laurel wilt killing redbay (*Persea borbonia*) trees in the Southeastern United States. *J. Econ. Entomol.* 101(4): 1276-1286.
- Harrington, T.C., S.W. Fraedrich, and D.N. Aghayeva. 2008. *Raffaelea lauricola*, a new ambrosia beetle symbiont and pathogen on the Lauraceae. *Mycotaxon* 104:399-404.
- Hoffman, R.L. 2003. *Heilipus apiatus*, a striking large weevil new to the Virginia fauna (Coleoptera: Curculionidae). *Banisteria* 22: 58-59.
- Koch, F.H. and W.D. Smith. 2008. Spatio-temporal analysis of *Xyleborus glabratus* (Coleoptera: Curculionidae: Scolytinae) Invasion in Eastern U.S. Forests. *Environ. Entomol.*, V37(2): 442-452.
- Mayfield, A., E. Barnard, C. Bates, A. Boone, R. Bulluck, S. Cameron, F. Campbell, D. Duerr, S. Fraedrich, J. Hanula, T. Harrington, J. Johnson, J. Peña, R. Rabaglia, J. Smith, V. Vankus. 2009. [Recovery plan for laurel wilt on redbay and other forest species caused by \*Raffaelea lauricola\*, vector \*Xyleborus glabratus\*](#). National Plant Disease Recovery System, a cooperative project of The American Phytopathological Society and The United States Department of Agriculture, posted at <http://www.ars.usda.gov/research/npdrs>.
- Oregon State University. Bot 552 Plant Disease Management – 12/7/2011 online at: <http://www.science.oregonstate.edu/bpp/BOT552/10%20Lecture%20Principles%20of%20Disease%20Management.pdf>.
- Smith, J.A., T.J. Dreaden, A.E. Mayfield, III, A. Boone, S.W. Fraedrich, and C. Bates. 2009a. First report of laurel wilt disease caused by *Raffaelea lauricola* on sassafras in Florida and South Carolina. *Plant Disease* 93: 1079.



## Appendix A - Maps

### Laurel Wilt Disease Distribution Maps

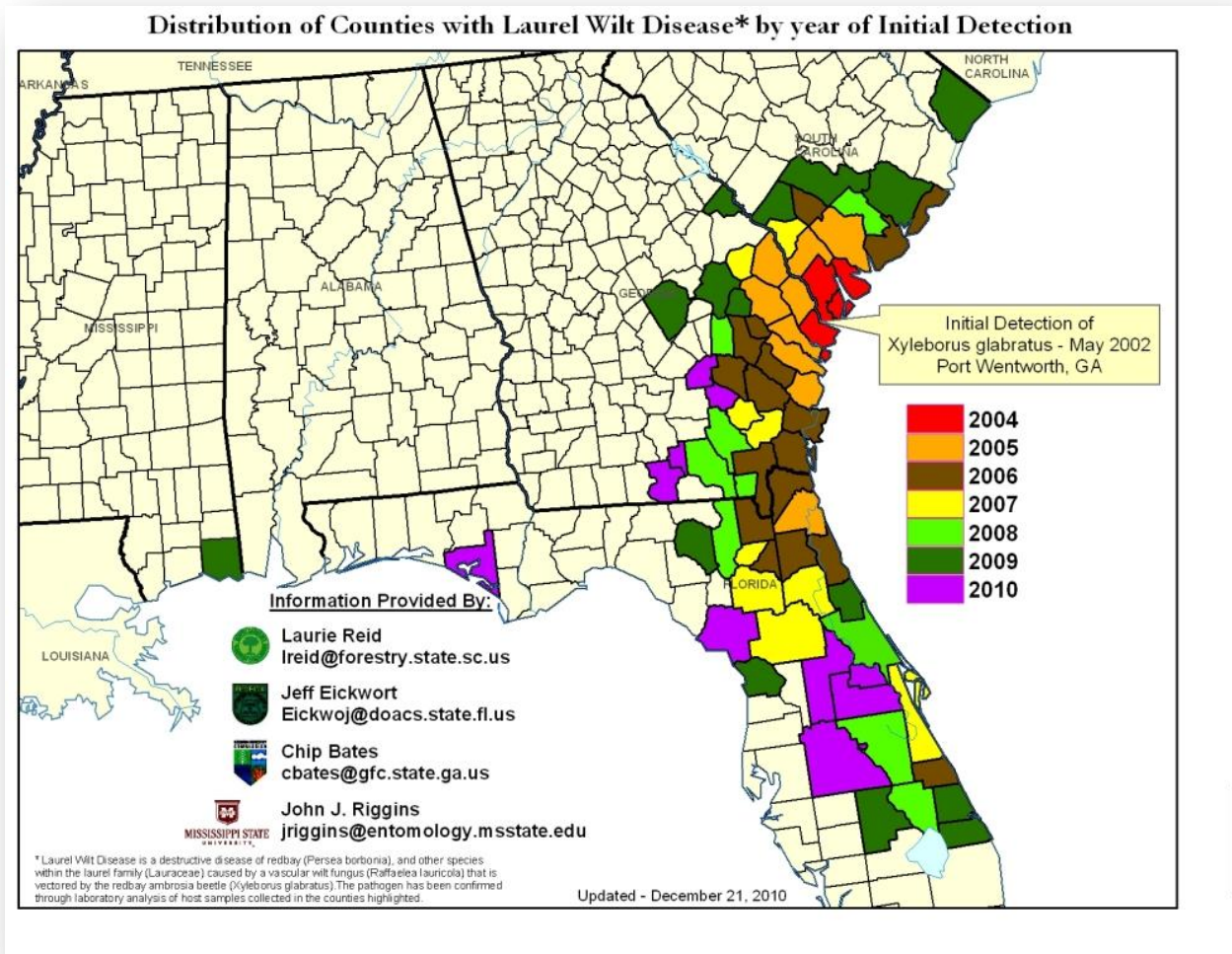


Figure A-1. Distribution map for laurel wilt disease by county and year of confirmation in the southeastern United States as of December 21, 2010.

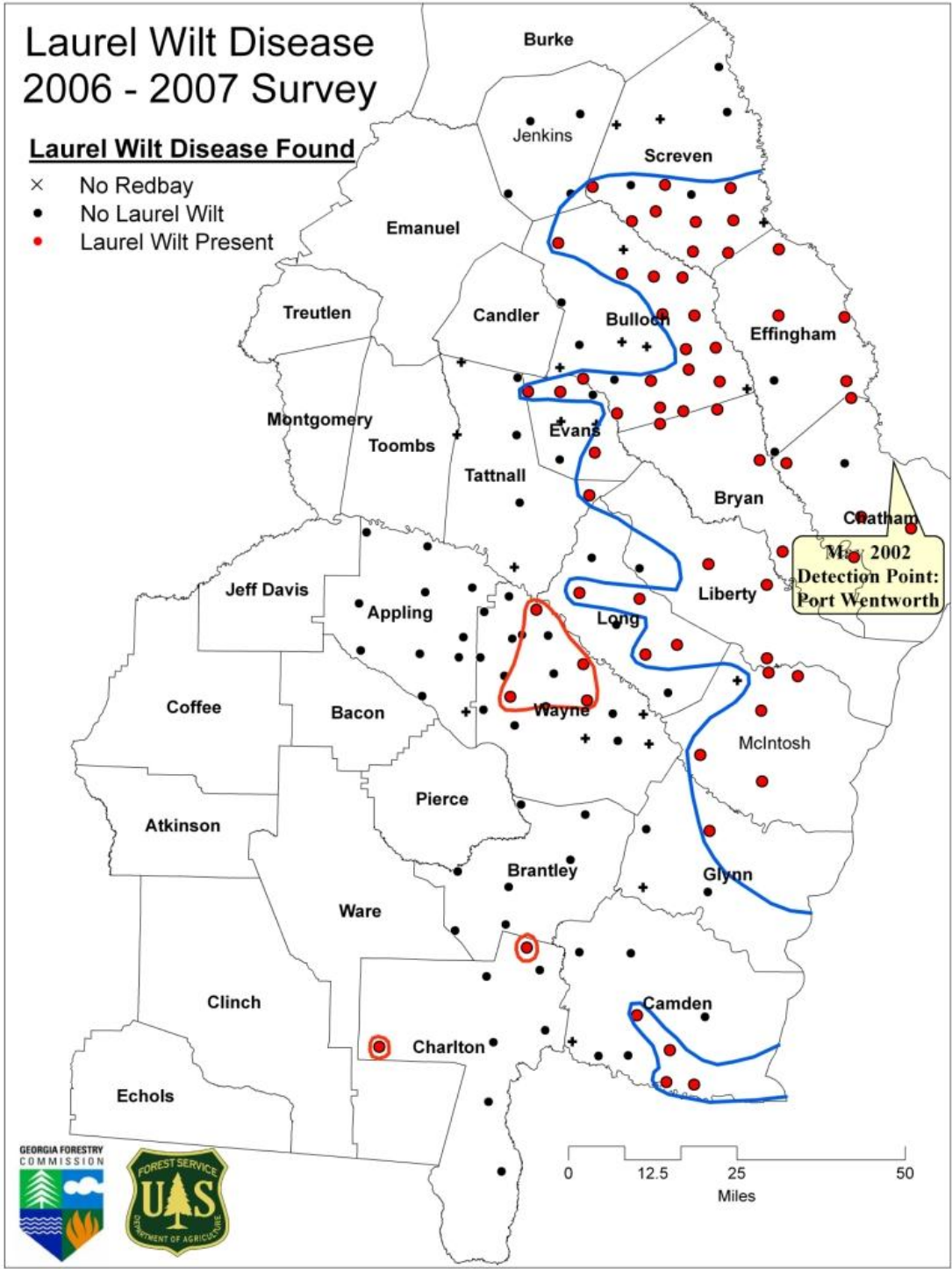


Figure A-2. 2006-2007 Georgia laurel wilt survey plot locations indicating presence (red dots), absence (black dots) and no red bay plots (plus sign “+”) as of early 2007. The blue line delineates the apparent advancing front of the disease with the “general area of infection” to the right. The red lines delineate outlying infection centers.

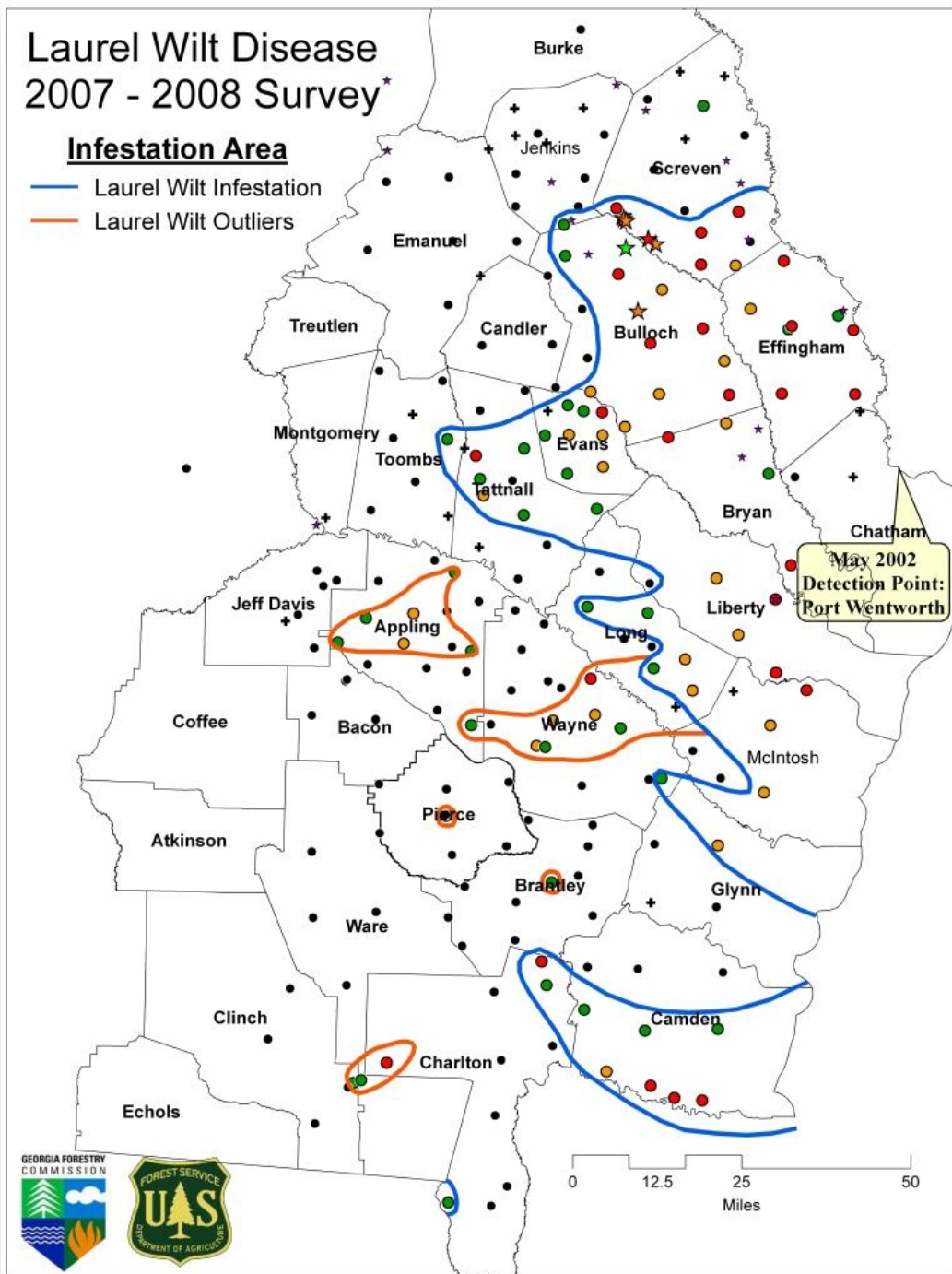


Figure A-3. 2007-2008 Georgia laurel wilt survey plot locations indicating four levels of infection (black = none, green = light, orange = moderate and red = severe) as of early 2008. Plus signs (“+”) indicate no red bay found. Stars indicate the locations of groups of sassafras trees. The blue lines delineate the apparent advancing front toward the west (left) or general area of infection of the disease and the orange lines delineate apparent outlying infection centers.

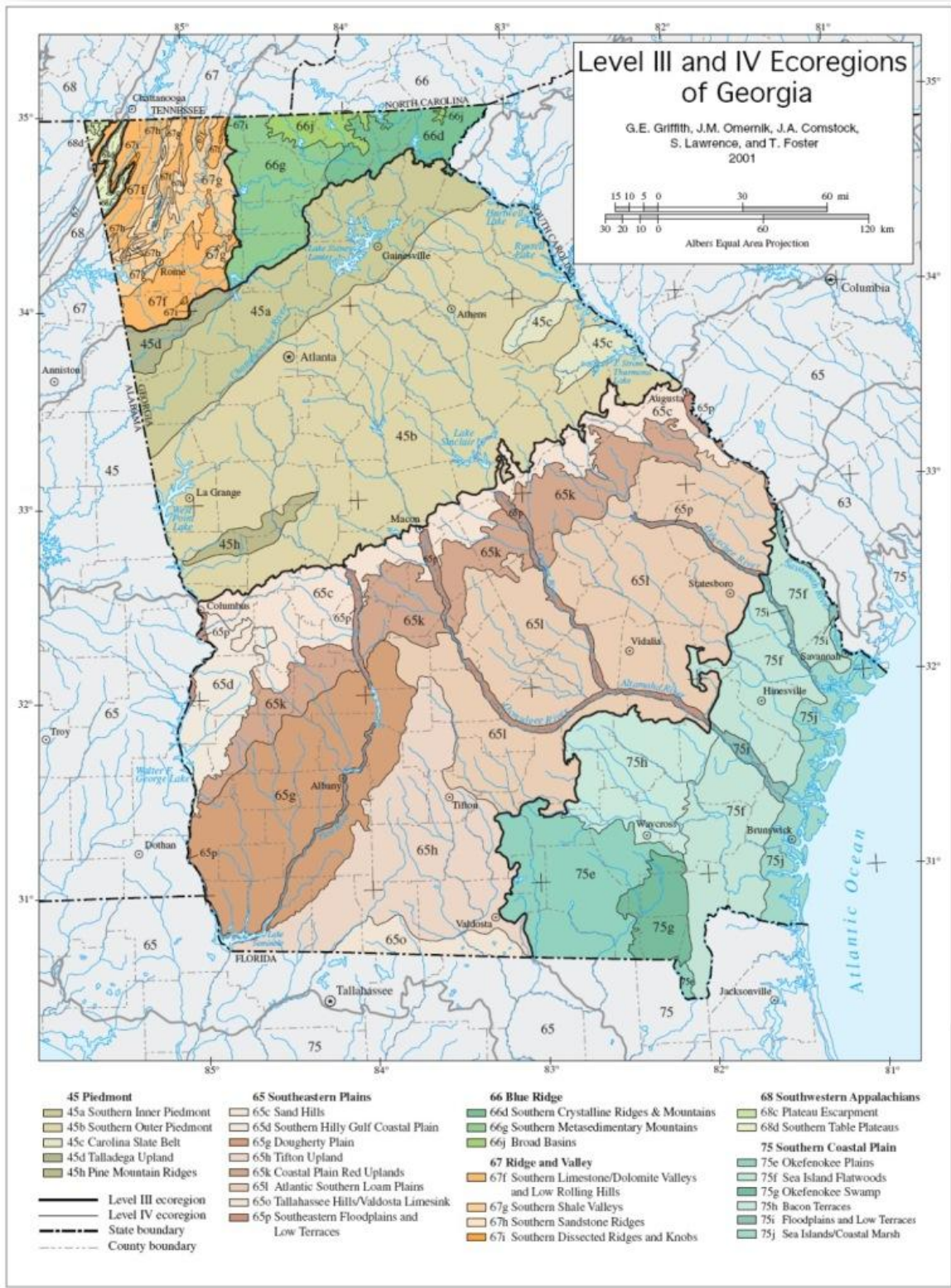


Figure A-4. Level II and IV Ecoregions of Georgia developed by Griffith et al. (2001).

## Redbay Wilt Risk Assessment Map For Georgia

Risk assessment for *Ophiostoma / Xyleborus glabratus* range expansion based upon combined climatic data (temperature, precipitation, and evaporation -- 30 year averages) and susceptible species present (Laurel family genera included were *Cinnamomum*, *Lindera* (3), *Litsea*, *Persea* (2), and *Sassafras*). The larger the number, the less the risk.



Figure A-5. Redbay wilt risk assessment map for Georgia (Coder 2006).

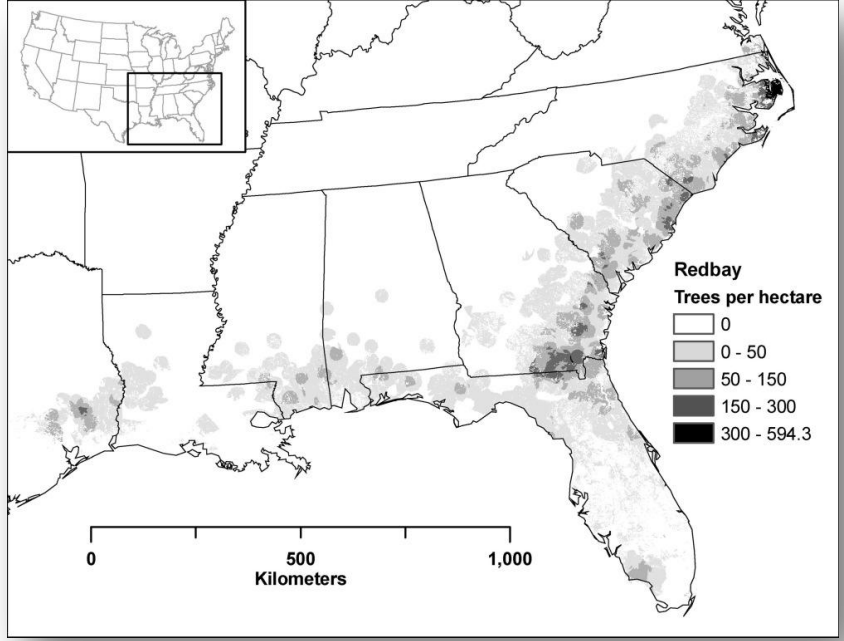


Figure A-6. Redbay density in trees per hectare copied from Koch and Smith (2008).

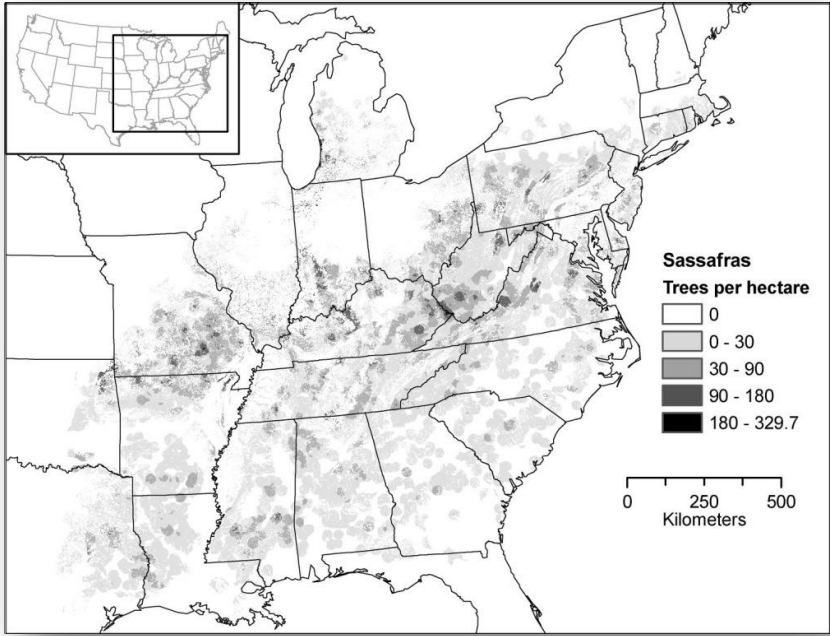


Figure A-7. Sassafras density in trees per hectare copied from Koch and Smith (2008).

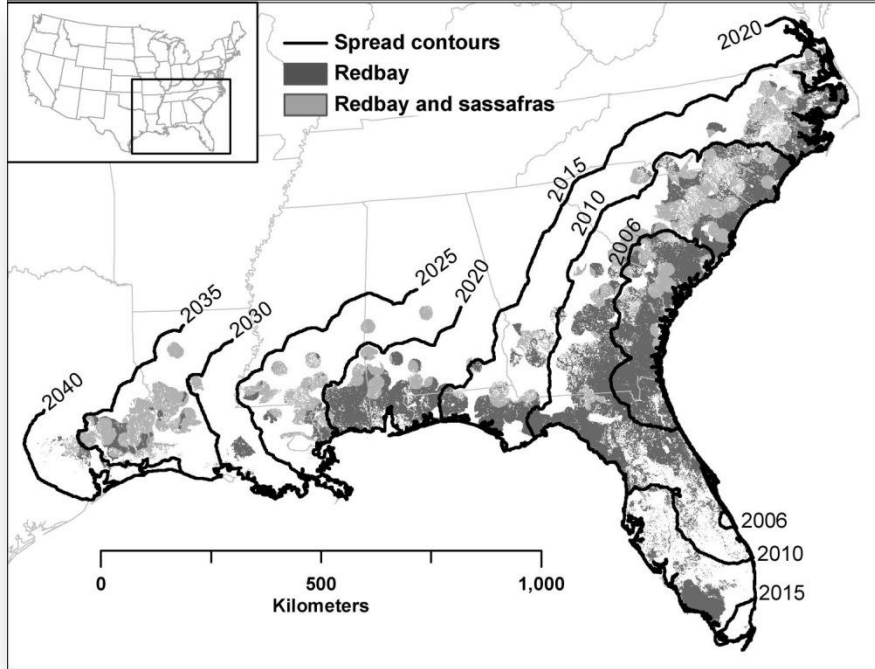


Figure A-8. Predicted extent of *X. glabratus* spread in the eastern U.S. through time based on cost-weighted distance modeling from three points of origin, and overlaid on a map of host density, copied from Koch and Smith (2008).