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## Progression and Impact of Laurel Wilt Disease within Redbay and Sassafras Populations in Southeast Georgia

R. Scott Cameron<sup>1</sup>, James Hanula<sup>2,\*</sup>, Stephen Fraedrich<sup>2</sup>, and Chip Bates<sup>1</sup>

**Abstract** - Laurel wilt disease (LWD), caused by the fungus *Raffaelea lauricola* and transmitted by *Xyleborus glabratus* (Redbay Ambrosia Beetle [RAB]), has killed millions of *Persea borbonia* (Redbay) trees throughout the southeastern Coastal Plain. Laurel wilt also has been detected in *Sassafras albidum* (Sassafras) in widely dispersed locations across the southeastern US. We established long-term laurel wilt disease-progression plots in Redbay and Sassafras stands in southeastern Georgia and monitored them through 4 years to document mortality rates and investigate long-term effects of LWD on Redbay and Sassafras survival and regeneration. Laurel wilt disease killed 87.3% of Redbay and 79.5% of Sassafras trees in the plots. The time from initial LWD detection to inactivity (no new mortality) in Redbay stands ranged from 1.1 to 3.6 years, with rate of disease progression positively related to host-tree size and abundance. Larger trees died at a higher rate in both Redbay and Sassafras stands, and mortality curves were similar for both species. All diseased Redbay trees died to the ground level, but the majority produced persistent below-ground basal sprouts, rapidly providing potential replacement stems. Few below-ground basal sprouts were observed on Sassafras trees killed by LWD, but over a quarter had epicormic shoots that survived up to several years after infection, and small trees remained alive on most sites, suggesting some level of tolerance to LWD. Substantial numbers of RAB were only captured in baited traps located adjacent to plots in an advanced-active stage of disease progression with abundant infested trees, both in Redbay and Sassafras stands. However, lingering presence of small numbers of RAB in post-epidemic areas and scattered LWD mortality in small-sized Redbay regeneration sprouts and seedlings suggest that secondary disease cycles may occur as Redbay trees there reach greater numbers and size in the future. Documentation of RAB and LWD spreading in Sassafras in the absence of Redbay supports concern that LWD will continue to spread into areas with abundant, large Sassafras trees, which would increase the probability that RAB and LWD will expand into extensive populations of other laurel species present in the western US and Central and South America.

### Introduction

Laurel wilt disease (LWD) is caused by the fungus *Raffaelea lauricola* T.C. Harrington, Fraedrich, & Aghayeva and vectored by *Xyleborus glabratus* Eichhoff (Coleoptera: Curculionidae: Scolytinae; Redbay Ambrosia Beetle [RAB]). Both were apparently introduced from Asia through the Port of Savannah, GA, sometime prior to 2002 when the first RAB was captured in a monitoring trap in Port Wentworth, GA (Fraedrich et al. 2008). Since that time, the disease has spread rapidly throughout the coastal plain forests in Georgia, South Carolina, and Florida, killing nearly all the large, and previously abundant, *Persea borbonia* (L.) Sprengel (Redbay) and

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*P. palustris* (Rafinesque) Sargent (Swampbay) trees (while some taxonomists distinguish *P. borbonia* and *P. palustris* as separate species, herein we consider these taxa as one species that we refer to as Redbay). More recently, LWD has spread into Redbay stands on the coastal plain of North Carolina and also has been documented in isolated locations in Alabama, Mississippi, and Texas (Bates et al. 2015). Laurel wilt was first observed in *Sassafras albidum* (Nuttall) Nees (Sassafras) in coastal Georgia (Fraedrich et al. 2008) and subsequently at numerous inland locations in Georgia (Cameron et al. 2008, 2014), Florida and South Carolina (Smith et al. 2009), Mississippi (Riggins et al. 2011), Alabama (Bates et al. 2013), and Louisiana (W. Johnson, USDA Forest Service, Pineville, LA, pers. comm.).

The spread of LWD through the southeastern United States has been mapped by state forestry organizations on a county-wide basis since 2005 (Bates et al. 2015). More-detailed systematic surveys conducted 2006–2010 documented the local spread of LWD in Georgia (Cameron et al. 2008, 2010) and illustrated how the disease advanced in surges and disconnected jumps followed by a more pervasive infection of most Redbay trees behind the advancing disease front. Koch and Smith (2008) developed a model predicting the temporal spread of *X. glabratus* based on climate, host density, and historical county spread, which provided some early guidance on how the disease could affect Redbay, but their model underestimated the importance of long-distance spread assisted by humans (Cameron et al. 2010, Riggins et al. 2011) and assumed that RAB would not spread in Sassafras in the absence of Redbay.

The short-term impacts of laurel wilt disease on Redbay and forest communities have been documented in 2 island maritime forests in northern Florida (Goldberg and Heine 2009) and southeast Georgia (Evans et al. 2014), and several coastal plain sites in Georgia (Spiegel and Leege 2013, Maner et al. 2014) and Florida (Fraedrich et al. 2008, Shields et al. 2011). Redbay trees over ~10 cm diameter at breast height (1.4 m above the ground; DBH) have been quickly eliminated, leaving only small-diameter trees and regeneration (Fraedrich et al. 2008, Shields et al. 2011). Evans et al. (2014), reported heavy mortality of Redbay basal sprouts and a lack of regeneration in an isolated maritime forest, and suggested that Redbay may become ecologically extinct from coastal forest ecosystems in the southeastern US. However, these studies have focused on impacts in forests with Redbay at one, often unspecified, stage of disease progression, and/or in unique areas that may not be representative of the broader distribution of Redbay.

The complete temporal stand-level spread and long-term effects of LWD on residual Redbay trees and Redbay regeneration over a broad geographical range have not been thoroughly investigated. Furthermore, relationships between RAB populations and stages of disease development, including post-epidemic sites, need further examination. Descriptions of symptoms, mortality rates, and spread of LWD in Sassafras stands are lacking, and little is known about disease and RAB spread in Sassafras communities in the absence of Redbay. Likewise, the long-term effects of this disease on residual Redbay and Sassafras trees and regeneration after the initial disease epidemic passes through an area are unknown.

We established a series of semi-permanent sample plots in southeast Georgia in front of, at, and behind the advancing laurel wilt disease front and revisited them semi-annually for at least 4 years to: (1) characterize the progression of mortality caused by LWD in Redbay and Sassafras, and document regeneration through time across a variety of geographical, site, and stand conditions; and (2) monitor RAB abundance associated with defined stages of LWD progression and quantity of infested host. Results from this investigation will provide a better understanding of LWD behavior in Redbay and Sassafras, the possibilities for Redbay recovery and LWD resurgence, as well as insight into the probability of continued spread of RAB and LWD into the extensive range of Sassafras in the eastern half of the US.

## Methods

### Laurel wilt disease impact and progression plots

*Field-site description and plant communities.* We conducted this field study over a broad area in southeastern Georgia between latitudes 31–33°N and longitudes 81–83°W, encompassing a range of host conditions, stages of disease development, and ecoregions (Griffith et al. 2001) from the Sea Islands/Coastal Marsh ecoregion, where the LWD epidemic originated, through the Sea Island Flatwoods and into the Atlantic Southern Loam Plains to the north and Bacon Terraces to the south (Fig. 1). We established standardized study plots in Redbay and Sassafras habitats with specific site selection based primarily on abundance of these species at set stages of laurel wilt development when the study was initiated. Redbay study sites roughly fit into 2 plant community types, bay forests and mixed hardwoods, some of which also included pine, with Redbay generally occupying a mid-story crown position. Four Sassafras study sites were located in dense open-grown thickets on deep sandy soils, and 2 others were in mixed hardwood/pine forests together with Redbay. Geographic location, ecoregion, soil series (USDA, Web Soil Survey), landscape position, plant community type, plot size, and host species for each of the study sites, along with common woody plant species associated with bay forests and mixed hardwood plant communities, are listed in Appendix 1.

*Study plot installation and data collection.* To document stand-level LWD development, we established 16 disease-progression plots during late winter through spring 2009 near the LWD advancing front: 10 with Redbay (R) only, 4 with Sassafras (S) only, and 2 with both (B) Redbay and Sassafras (Fig. 1). Nine of the plots—7 Redbay (Ra1–Ra6 and Ba1) and 2 Sassafras (Sa1 and Sa2), each separated from known diseased trees by 50 m to 15 km—were designated LWD-absent (a) when the study began. Seven plots—5 Redbay (Rd2–Rd5 and Bd1) and 2 Sassafras (Sd1 and Sd2)—were designated LWD-active with disease (d) in progress.

We demarcated 4 Redbay post-epidemic plots to document Redbay regeneration, residual disease, and RAB activity in the aftermath of the initial LWD epidemic. These plots were located where LWD had passed through ~7 years earlier, and all susceptible host trees were dead and in an advanced stage of decomposition. We utilized 4 additional Redbay sites (Ra7, Ra8, Ri5, and Ri6) and 2 Sassafras sites (Sa3 and Sd3) for monitoring RAB abundance without installing sample plots (Fig. 1).

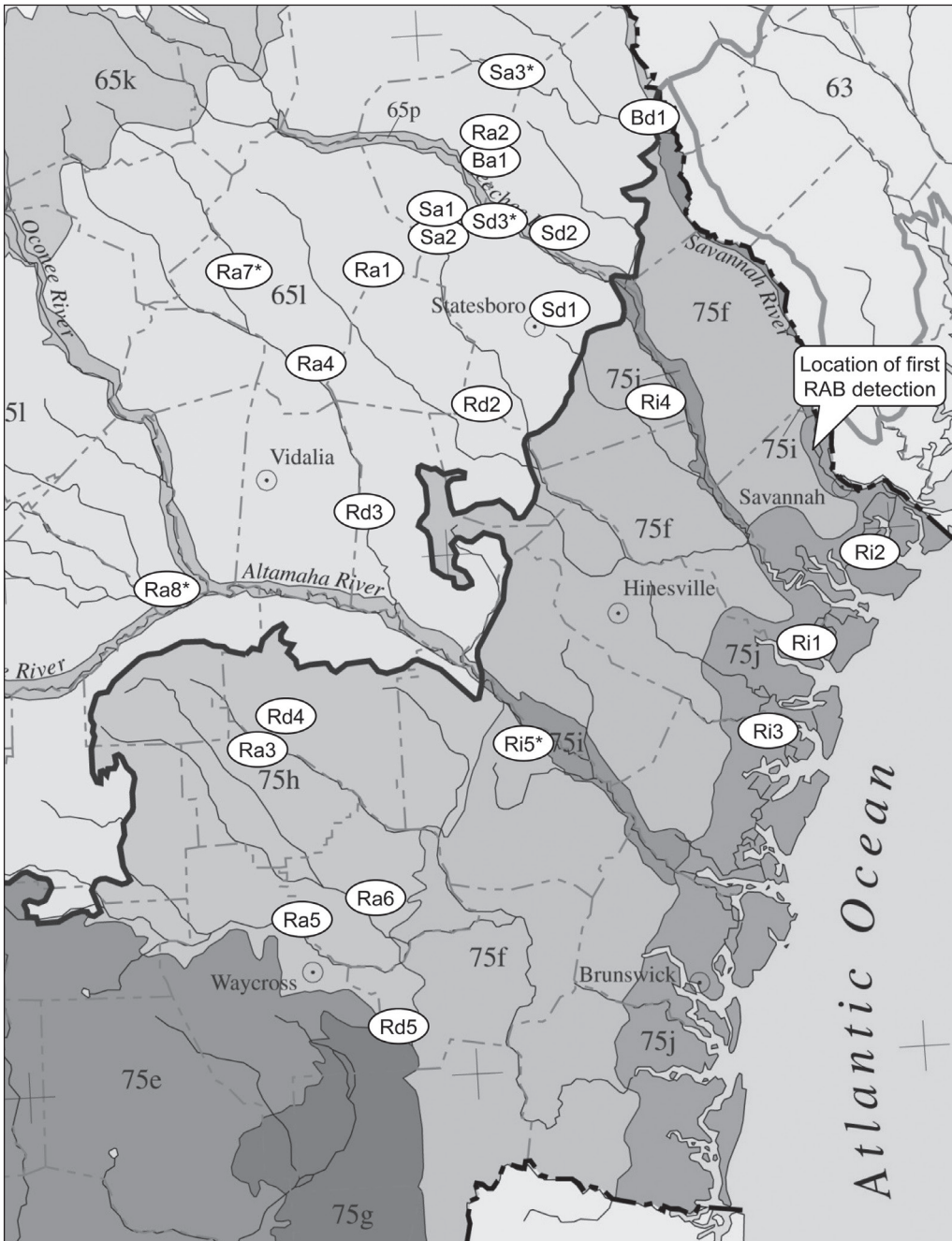


Figure 1. Location, host type, initial disease stage, and laurel wilt (LW)-monitoring plot numbers superimposed on the southeastern portion of the Ecoregions of Georgia map (Griffith et al. 2001) Key: RAB = Redbay Ambrosia Beetle, R = Redbay, S = Sassafras, B = both Redbay and Sassafras, a = LW disease absent at plot initiation, d = disease present, i = inactive, post-epidemic, \* = indicates RAB trapping sites without sample plots. Ecoregions represented: 75f = Sea Island Flatwoods, 75h = Bacon Terraces, 75j = Sea Islands/Coastal Marsh, and 65l = Atlantic Southern Loam Plains.



We laid out study plots using a simplified Carolina Vegetation Survey protocol (Peet et al. 1998, Wentworth et al. 2008) to facilitate relocation of specific host trees and document vegetation changes through at least 4 years, 2009–2013 (Fig. 2). Redbay disease-progression plots (including 2 with mixed Redbay and Sassafras) and post-epidemic plots consisted of 4 contiguous 10 m x 10 m modules (total = 400 m<sup>2</sup>) arranged in a square or line to include as many host trees as possible. Five modules were established on plot Bd1 to incorporate additional Sassafras trees. We marked the corners of each module with PVC pipe, placed an aluminum tag on a wire pin at the base of all Redbay and Sassafras trees >2.5 cm DBH in each plot, and recorded the location of those trees on a map. We documented initial tree diameter, health (live healthy; live with dieback; LWD wilting = drooping or off-color leaves; LWD dead = brown leaves or dead from other causes), and number of live basal sprouts emerging from below ground within 1 m of the base of each tree. We considered trunks forked below 1.4 m as separate trees. We assumed dead and fallen Redbay trees in post-epidemic plots were killed by LWD, and estimated their DBH based on the diameters of the remaining stumps and fallen trunks. We tallied stems of host regeneration (<2.5 cm DBH, including seedlings and sprouts) in 1 m x 10 m subplots on alternating edges of each module (Fig. 2). We revisited each plot at ~6-month intervals through 4–5 years to record tree condition, number of live basal sprouts, and presence of epicormic shoots over 1.4 m above ground level for each tagged host tree, and to count live regeneration stems in subplots.

We used similar procedures for plot installation and subsequent monitoring of 4 Sassafras plots. Due to the limited distribution or high density of Sassafras stems, we restricted plots on 2 sites to two 10 m x 10 m modules (total = 200 m<sup>2</sup>) and 2 others consisted of one rectangular module, one 90 m<sup>2</sup> and the other 50 m<sup>2</sup>. We tagged all Sassafras trees >2.5 cm DBH in plots and monitored them every 6 months, except in one plot where Sassafras occurred as a dense thicket, in which

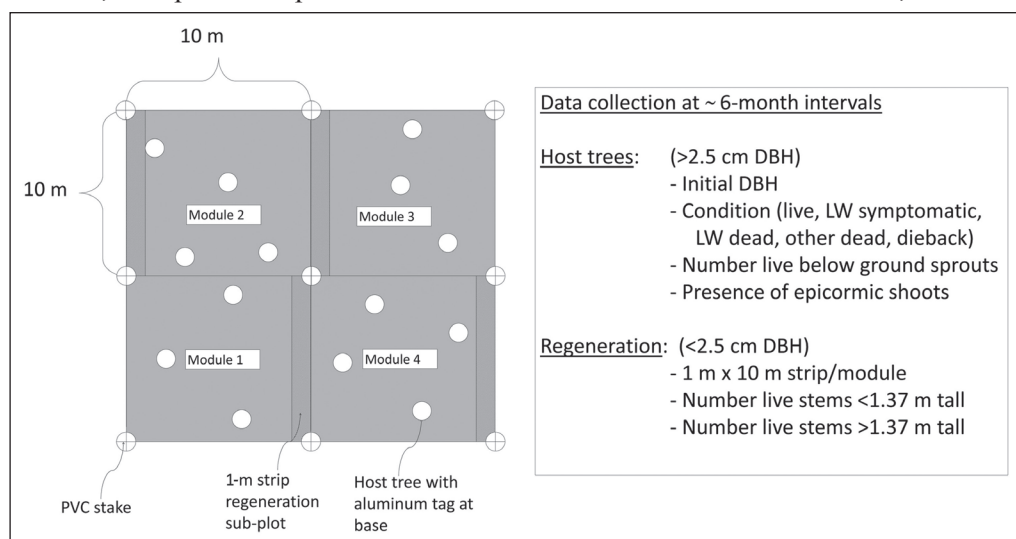


Figure 2. Schematic of LW disease-progression and post-epidemic plot layout and data-collection methods.

we therefore tallied trees by 2.5-cm-DBH classes without tagging or following the trees individually.

*Laurel wilt disease diagnosis.* Assigning LWD as the cause of mortality in Redbay and Sassafras was generally based on leaf symptoms (Cameron et al. 2010, NPDRS 2015). We considered gradual dieback from the apex of crowns and/or major branches to be not induced by LWD and attributed it to other undetermined causes, such as suppression, drought, or root disease. Mortality of small branchlets on Redbay, typical of damage caused by *Xylosandrus compactus* (Eichhoff) (Coleoptera: Scolytidae) (Black Twig Borer) also was not attributed to LWD (Dixon and Woodruff 1982). When the cause of mortality was in doubt, we removed small patches of bark and outer sapwood to look for the black streaking that is diagnostic of LWD (Fraedrich et al. 2008). If the cause of mortality was still in doubt, we collected samples of sapwood and then plated them on a selective agar medium to confirm presence of *R. lauricola* (Fraedrich et al. 2008).

### Disease-progression and post-epidemic plot data summary

*Laurel wilt disease impact in Redbay and Sassafras stands.* Variables assessed on each study plot included: (1) initial and final number and basal area (BA; m<sup>2</sup>) per ha of live host trees, (2) number and BA per ha of host trees killed by LWD, (3) percent of trees and BA killed by LWD, (4) percent of trees that died from other causes, and (5) initial and final mean DBH (cm) of live Redbay and Sassafras trees. For 2 plots with both Redbay and Sassafras, we determined host characteristics and mortality by species and treated them as separate plots. We computed mean mortality rates among all plots to characterize LWD impact and variation by species across the broad study area.

*Synchronized timelines.* We recorded individual host-tree condition, number of basal sprouts on each host tree, and host regeneration at ~6-month intervals in the spring (late winter–spring) and fall (late summer–fall) for each plot. Since disease infection in separate plots and individual trees started at varying calendar dates, we used synchronized time scales with 6-month intervals to standardize starting points for summarizing temporal progression variables, including: (1) cumulative mortality by species and diameter class, (2) number of sprouts per tree before and after the passage of LWD, and (3) number of host regeneration stems/m<sup>2</sup> (seedlings and sprouts) through time. We defined the zero point on timelines for disease progression plots and individual trees within each plot as 6 months (0.5 year) prior to the observation of the first LWD infected tree or initial symptoms on individual trees in that plot. For the 5 active plots, which had relatively recent disease episodes in progress, we estimated the zero point based on the number and condition of infected and dead trees in the plot at initiation.

In the 7 Redbay disease-progression plots in which LWD was initially absent, we positioned observations of basal sprouts and regeneration prior to LWD infection at corresponding negative 6-month intervals up to 1 year before the zero point on the timeline. We also placed number of sprouts per tree and regeneration per m<sup>2</sup> in post-epidemic plots on synchronized timelines starting at an estimated

7 years after initial disease infection (based on Redbay disease detection records [Bates et al. 2015] and decomposition condition of Redbay stumps).

We merged data summaries on synchronized timelines for each plot to calculate study-wide means for mortality rates by species, basal sprouts, and regeneration. Since individual plot data were positioned at varying locations on synchronized timelines, the number of observations for plot averages varied, generally with fewer replicates at both ends of the time scale for LWD-progression plots. We excluded from temporal data summaries the plot averages for time intervals with fewer than 4 observations for Redbay and 3 observations for Sassafras.

*Cumulative LWD mortality in Redbay and Sassafras.* We calculated mean cumulative percent LWD mortality at 6-month intervals (percent of initial live host trees) among 12 Redbay plots over a 3.5-year period and 5 Sassafras plots for a 2.5-year period. Host trees that died from other causes were deducted from the initial numbers of live trees for this analysis. We charted Redbay and Sassafras cumulative mortality curves and determined regression trend lines.

*Redbay and Sassafras mortality by diameter class.* We compared cumulative percent mortality caused by laurel wilt disease among 3 diameter classes for Redbay and Sassafras on separate synchronized timelines based on first LWD infection in each plot, as previously described. We combined individual tree data from the 8 Redbay plots that were observed for 4 years after initial LWD infection, or in which all Redbay trees were killed by LWD in less than 4 years. Individual tree mortality data from 4 Sassafras plots observed at least 2.5 years after initial LWD detection were also merged. We calculated percent mortality at each observation interval among all trees in three 11.5-cm-DBH classes for Redbay and three 4.6-cm-DBH classes for Sassafras, and plotted cumulative percent mortality by size class on separate charts for each species.

*Laurel wilt disease progression in Redbay stands.* We derived the number of years that LWD was active in 10 Redbay-only disease-progression plots by subtracting the date of the first observation of LWD in each plot from the date when all host trees in the plot were killed by the disease, or when disease progression ceased (defined as no new LWD infections for at least one year). Since one Redbay plot (Ra3) remained slightly active at the last field observation, we projected time to inactivity to 6 months after the final observation, based on the observed mortality trajectory through 3.5 years. We regressed years that LWD was active in each plot against initial mean Redbay DBH and total initial Redbay basal area ( $\text{m}^2/\text{ha}$ ) to determine if the episode duration was associated to host diameter or basal area. Too few Sassafras plots were monitored to determine disease progression rate with respect to DBH and BA for this species.

*Basal sprouts, epicormic shoots, and regeneration density in Redbay and Sassafras.* We recorded the numbers of live basal sprouts emerging from below ground around all tagged Redbay and Sassafras trees throughout the study. We computed mean basal sprouts per tree within and among plots from synchronized timelines as described above, excluding from this analysis basal sprouts around host trees that died from causes other than LWD and sprouts which could not be attributed to



particular individuals within clumps of trees. In 4 post-epidemic assessment plots, we synchronized the number of sprouts associated with stumps of Redbay trees assumed to have died from LWD as above. We determined percentages of Redbay and Sassafras trees with epicormic shoots >1.4 m above ground level after LWD infection on all plots. Regeneration density (stems/m<sup>2</sup>) in 2 size classes (<1.4 m tall and >1.4 m tall but less than 2.5 cm DBH) was computed for each plot assessment, arranged on synchronized timelines and summarized as described above.

### **Redbay Ambrosia Beetle (RAB) population monitoring.**

We deployed baited traps immediately outside the perimeter of most LWD-monitoring plots plus at 6 additional locations (Fig. 1) to track the relative numbers of RAB in areas at different stages of LWD progression in 2009 and 2010. One 8-funnel Lindgren trap baited with a manuka oil lure (P385-Lure M; Synergy Semiochemicals Co.) attached to the outside of the upper funnel was suspended on a rope between two non-host trees ~2 m above ground level and left in the field throughout August (30 days) each year. We monitored a total of 16 Redbay and 4 Sassafras sites in 2009 and 17 Redbay and 4 Sassafras sites in 2010.

We derived the LWD stage and cumulative DBH of infested host trees at each trapping site from the late summer/fall assessments for each respective year in adjacent LWD progression and post-epidemic study plots. Disease progression stages recognized for RAB trapping sites were: 1) “outside range” of LWD (~5 km to 30 km ahead of the advancing LWD front); 2) “absent-near” where trees were healthy in plots but LWD was present in trees within ~250 m of the plot; 3) “early-active” where there were recent infections in the plot, but less than 50% of host trees were wilting or dead from LWD; 4) “advanced-active” where more than 50% of host trees were wilting or dead; 5) “late-active” where all host trees were dead or the disease episode was ending, but some dead trees were still standing with major branches intact; and 6) “post-epidemic” where all host trees were dead with major limbs broken off and trunks in an advanced stage of decomposition. We combined data from both trapping years and computed mean numbers of RAB per day and mean cumulative DBH of infested Redbay trees for each Redbay disease stage, resulting in a range of 3 to 9 replicates per stage.

Numbers of Sassafras trapping sites were limited to 2 outside range, 2 early-active, 1 advanced-active, and 3 late-active. We determined the mean numbers of RAB per trap per day for Sassafras trapping sites and performed no further analyses due to limited replication within disease stages.

### **Statistical analyses**

We entered the data into Microsoft Excel spread sheets (Microsoft Corp., Redmond, WA) and generated sums, cumulative mortality, means, and standard errors (SE) using basic statistical functions and formulae for the variables to be assessed. Stand characteristics and impact data were summarized with Microsoft Excel PivotTable Tools. To determine if there was a size preference among initial Redbay infections, we used the paired 2-sample *t*-test in Microsoft Excel Data Analysis Tools to compare mean diameters (DBH) of the first symptomatic trees in

7 originally disease-free plots and 1 active plot (with only one initial wilting tree) to the mean diameters of all Redbay trees. Mean DBH and total BA in 10 Redbay-only plots were normally distributed according to the Kolmogorov-Smirnov test for normality in the Univariate procedure of SAS (SAS Institute 2000), and we regressed time to LWD inactivity for each plot against both mean DBH and BA using Excel Data Analysis Tools. We also regressed mean numbers of RAB per day in 6 LWD stages against cumulative DBH of infested host in adjacent plots. Graphs were produced with Microsoft Excel Chart Tools, and we determined best-fit regression equations to describe the relationships between disease episode duration and mean DBH, BA, and cumulative percent mortality over time caused by LWD.

Results

Laurel wilt disease impact and progression in Redbay and Sassafras

*Impact of laurel wilt disease in Redbay stands.* At plot initiation,  $4.2 \pm 1.83\%$  (mean  $\pm$  SE) of Redbay trees were dead from other causes. Laurel wilt disease killed 87.3% of Redbay trees (93.1% of the basal area) in disease-progression plots, 3.4% died of other causes, and only 9.3% remained alive at the end of the study (Table 1).

Trees on the 7 Redbay plots initially classified as LWD-absent became infected with LWD, and disease progression was complete by the end of the study in all but 1 plot that was projected to be inactive in 1 year. All Redbay trees alive prior to the arrival of LWD in 6 of the 12 disease-progression plots died within 2 years after initial disease detection, and only a few small Redbay trees remained alive in 6 other plots. Initial mean DBH of live Redbay trees was  $11.6 \pm 1.57$  cm, and mean DBH of surviving Redbay trees was  $5.2 \pm 0.72$  cm, the largest of which was 13 cm. Among 232 Redbay trees that displayed LWD symptoms during the study, all died to ground level.

*Impact of laurel wilt disease in Sassafras stands.* At the beginning of the study, LWD was absent from 3 of the 6 Sassafras disease-progression plots and  $11.9 \pm 9.21\%$  of Sassafras trees were dead from other causes (primarily due to suppression beneath a dense hardwoods/pine overstory). Laurel wilt disease was present

Table 1. Initial mean number of live host trees and live basal area (BA), percent of trees and basal area killed by laurel wilt (LWD), percent mortality by other causes, percent final live trees, and initial and final mean DBH in Redbay and Sassafras disease-progression plots monitored from 2009–2013 in southeast Georgia.

Host, plot type	Initial # live host trees/ha	% LWD mort.	% mort. other causes	Final % live	Initial live host BA (m <sup>2</sup> /ha)	% BA LWD mort.	Initial live host DBH (cm)	Final live host DBH (cm)
Redbay, LWD-absent and LWD-active, <i>n</i> = 12								
Mean	573.3	87.3	3.4	9.3	6.8	93.1	11.6	5.2
SE	74.38	3.71	1.74	3.24	1.42	2.53	1.57	0.72
Sassafras, LWD-absent and LWD-active, <i>n</i> = 6								
Mean	2803.4	80.4	6.0	13.6	7.3	92.7	5.5	3.8
SE	1611.98	6.37	3.23	5.73	3.29	3.23	0.54	0.40

on all plots by the end of the study, and the disease killed an average of 80.4% of the Sassafras trees (92.7% of the basal area), while 6.0% died of other causes and 13.6% remained alive at the end of the study (Table 1). In the 2 plots with both Redbay and Sassafras, LWD killed all Sassafras trees in 1 plot, and only 1 Sassafras tree remained alive in the other. In 1 Sassafras plot, all initially tagged trees died, but 2 saplings grew to tree size and were alive at the end of the study. In the other 3 Sassafras plots, LWD killed 65.0% to 76.2% of host trees and only small trees remained alive. Mortality due to LWD ceased to expand in one initially active Sassafras thicket ~1.5 years after plot initiation and did not expand through 2.5 additional years of observation. However, the LWD pathogen remained viable in stumps in this plot, as confirmed by our ability to readily culture *R. lauricola* from wood chip samples taken at the bases of 2 trees that had died 2 years earlier but had epicormic shoots growing just above the soil level. Disease also lingered in other Sassafras stands, as indicated by slow continuous mortality at final assessments and the presence of wilted leaves on epicormic sprouts and black staining in the sapwood of a few remaining small trees.

*Cumulative LWD mortality in Redbay and Sassafras.* Cumulative mortality caused by LWD among initially healthy host trees in 12 Redbay and 5 Sassafras disease-progression plots followed very similar trajectories (Fig. 3). Host trees died

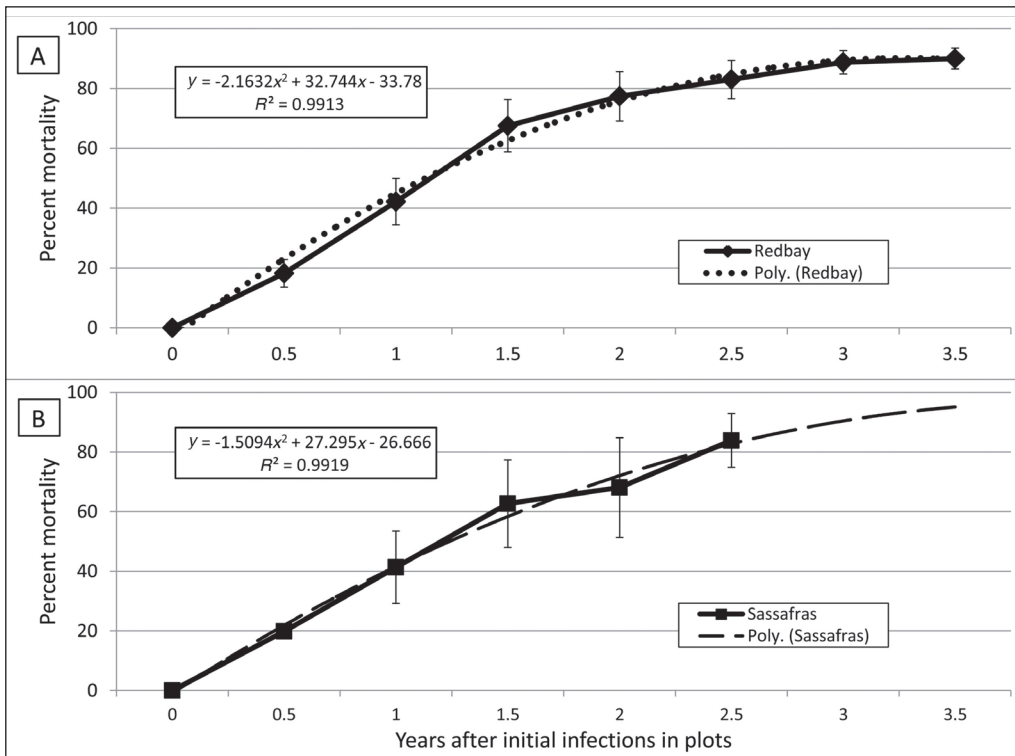


Figure 3. Mean cumulative percent mortality caused by laurel wilt disease in (A) Redbay ( $n = 12$ , mean DBH = 11.7 cm) and (B) Sassafras ( $n = 5$ , mean DBH = 6.0 cm) among trees alive at plot initiation (excluding mortality from other causes) monitored from 2009–2013 in southeast Georgia. Vertical error bars represent standard error of the mean.

at a rapid, steady rate up to ~60% mortality through 1.5 years after initial detection of LWD in plots, and then the rate of increase slowed among the remaining trees. Disease progression curves were best fit with similar second-order polynomial regression trend lines for both species.

*Redbay and Sassafras mortality rates by host-diameter class.* Mean DBH of the first infected Redbay trees in 8 plots (15.4 cm) did not differ significantly from the mean diameter of all Redbay trees (12.5 cm) in the plots ( $t = 1.38$ ,  $P = 0.210$ ). Similarly, incidence of LWD among Redbay trees, observed for at least 4 years and merged from 8 plots, appeared to affect the 3 diameter classes equally at first detection (0.5 years), with mortality ranging from 18% to 22%, but thereafter mortality rates increased rapidly for the 2 larger diameter classes (Fig. 4). All Redbay trees over 25.8 cm DBH were killed by LWD 1.5 years after detection, and all 14.2–25.7-cm-DBH-class trees were dead within 2.5 years. Mortality caused by LWD progressed more slowly in the smallest DBH-class trees, with 12.3% still alive after 4 years.

In contrast to Redbay, mortality in Sassafras trees was highest initially in the largest diameter class where 40% of trees were diseased 0.5 years after initial detection, while 10% or less were affected in the other diameter classes (Fig. 5). Sassafras mortality increased greatly in the intermediate and smallest diameter classes starting 0.5 and 1.5 year, respectively, after initial detection, and incidence of LWD among all Sassafras diameter classes was 80–100% after 2.5 years.

*Laurel wilt disease progression in Redbay stands.* The average time from initial LWD infection to inactivity in 10 Redbay-only disease-progression plots was 2.2 years (range = 1.1–3.7 years). The length of time LWD was active in plots was inversely related to the initial Redbay mean DBH ( $P = 0.019$ ) and BA ( $P = 0.040$ ) (Fig. 6). Redbay mortality progressed more rapidly and completely in plots with larger initial mean DBH and BA than in plots with smaller DBH and BA. The 5 plots with the fastest progression were all in “bay forest” plant communities and

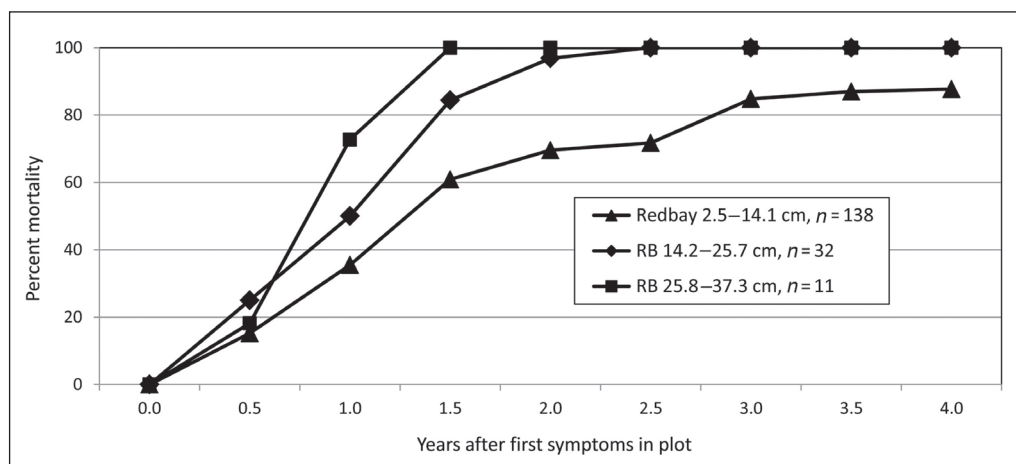


Figure 4. Cumulative percent mortality caused by laurel wilt disease in 3 DBH classes among all individual Redbay trees combined from 8 LWD-progression plots monitored for 4 years in southeast Georgia.

averaged  $1.4 \pm 0.12$  years to inactivity,  $15.7 \pm 2.06$  cm mean DBH, and  $10.8 \pm 2.23$  m<sup>2</sup>/ha BA, in contrast to the 5 plots with the slowest progression (4 in mixed hardwood forests and 1 in a pine plantation) that averaged  $2.9 \pm 0.30$  years to inactivity,  $6.8 \pm 0.87$  cm mean DBH, and  $3.7 \pm 1.17$  m<sup>2</sup>/ha BA. Also, all Redbay trees in the 5 plots in bay forests died from LWD, while a few Redbay trees remained alive ( $20.3 \pm 3.98\%$ ) in the 5 plots in mixed hardwood/pine plantation stands.

### Regeneration after laurel wilt disease in Redbay and Sassafras

*Basal sprouts around Redbay and Sassafras trees killed by LWD.* Prior to LWD infection, we observed basal sprouts originating from below ground on only a few Redbay trees, primarily on smaller trees with crown dieback. However, basal sprouts around Redbay began increasing within 6 months of becoming symptomatic and continued increasing through 1.5 years after infection by LWD (Fig. 7). Among all individual Redbay trees, 67.2% had at least one sprout 1.5 years after initial LWD symptoms were recorded. Although, many below-ground basal sprouts apparently died between assessment periods, most either re-sprouted or were replaced by new sprouts, resulting in an increase in the number of sprouts to  $3.7 \pm 0.64$  per tree after 2 years, and sprout numbers remained constant over the next year. In the post-epidemic assessment plots,  $5.1 \pm 0.47$  Redbay sprouts were associated with stumps of trees killed by LWD ~7 years earlier. Numbers of sprouts per stump in post-epidemic plots remained relatively constant through 4 years of observation.

Sassafras did not respond to LWD by producing numerous below-ground basal sprouts. Only 10.9% of individual Sassafras stems killed by LWD had 1 or more below-ground sprouts, with a peak of  $0.4 \pm 0.19$  sprouts per tree 2 years after initial symptoms were detected (Fig. 7).

*Incidence of epicormic shoots in Redbay and Sassafras.* Epicormic shoots emerging on stems above 1.4 m were recorded for 6.6% (range = 0–23.3%) of

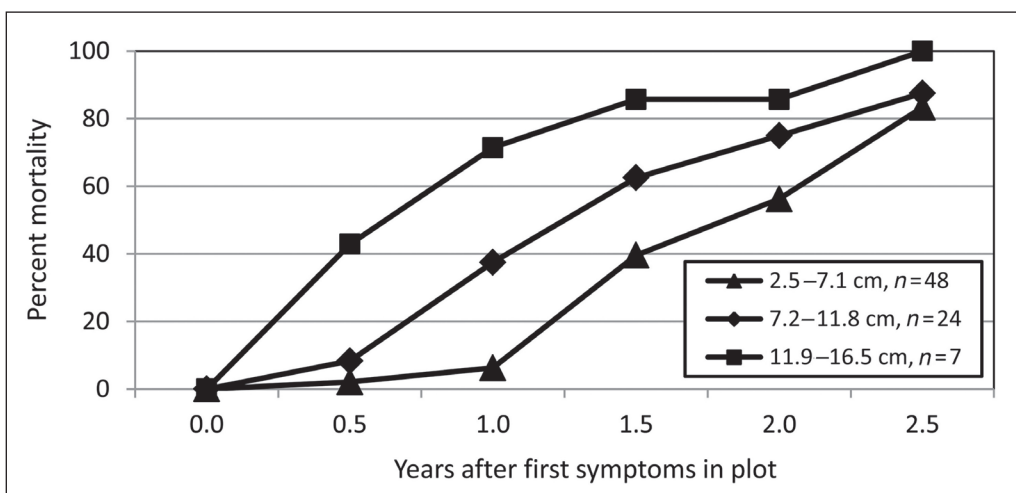


Figure 5. Cumulative percent mortality caused by laurel wilt disease in 3 DBH classes among all individual Sassafras trees combined from 4 LWD-progression plots monitored 2.5 years in southeast Georgia.



Redbay trees and 28.3% (range = 7.7–41.2%) of *Sassafras* trees killed by LWD in disease-progression plots. Epicormic shoots on Redbay trees infected with LWD died quickly as the disease spread rapidly through the crowns and trunks of trees during the year following the appearance of symptoms. In contrast, some *Sassafras* trees that appeared to have died from LWD continued to produce new shoots on trunks and portions of the crown several years after initial LWD detection in the tree. Cutting into the stems of these trees revealed black staining typical of LWD, often beneath 1 or 2 years of apparently healthy radial growth. However, many of these trees eventually succumbed to the disease and died.

*Regeneration density in Redbay and Sassafras disease-progressions plots.* The number of small Redbay (both seedlings and basal sprouts <1.4 m tall) was 0.3 stems/m<sup>2</sup> in plots at the time of the first LWD infections, but density of stems increased rapidly to 1.3 stems/m<sup>2</sup> from 0.5 to 2 years after LWD detection and remained relatively constant during the next 2 years (Fig. 8). Very few Redbay stems

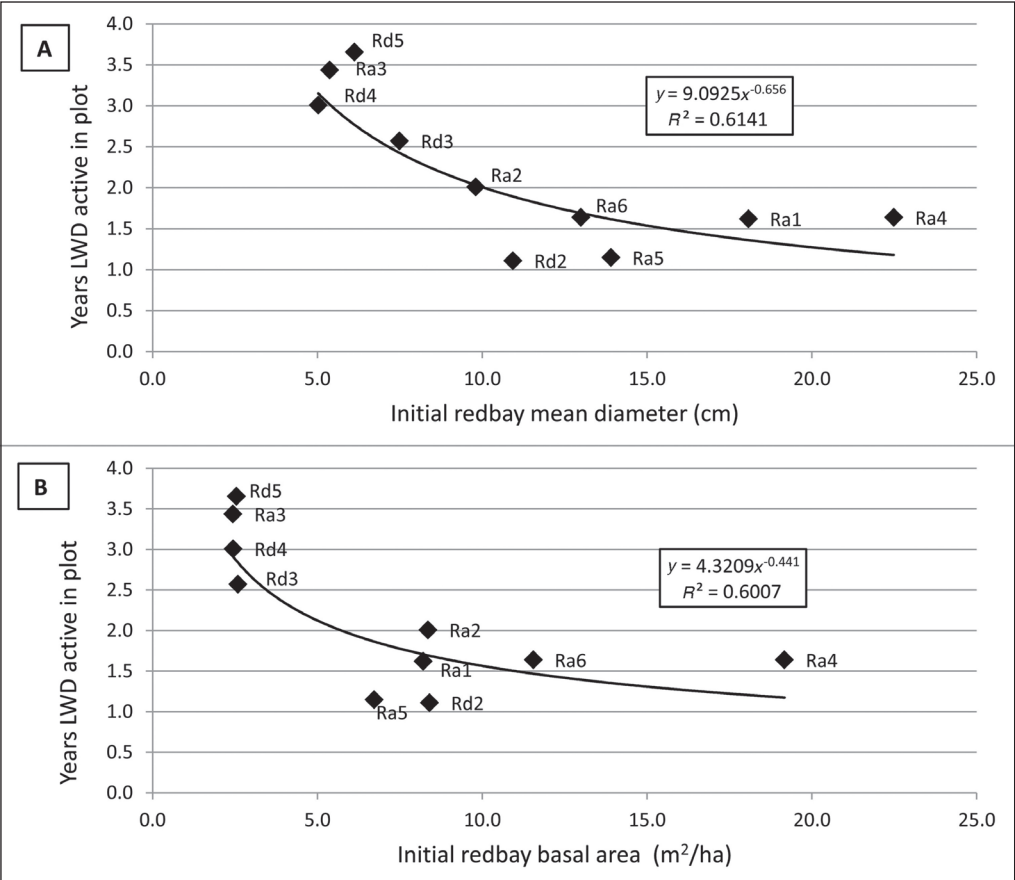


Figure 6. Relationship between years that laurel wilt disease was actively killing trees in plots (episode duration) and (A) initial Redbay mean diameter ( $P = 0.019$ ,  $n = 10$ ) and (B) initial Redbay basal area per hectare ( $P = 0.040$ ) in southeast Georgia, 2009–2013. Letters and numbers adjacent to each data point refer to plot identifications for which codes, locations, site characteristics, and plant community types are listed in Appendix 1.

<2.5 cm DBH but >1.4 m tall were present in the Redbay disease-progression plots (<0.1 stem/m<sup>2</sup>) through 4.5 years after initial LWD infection.

Numbers of Sassafras regeneration stems <1.4 m tall revealed no clear association with the progression of LWD. Three plots consistently had high numbers of Sassafras regeneration stems, root sprouts, and seedlings ( $3.8 \pm 0.27$  stems/m<sup>2</sup>), while 3 other plots consistently had low numbers ( $0.2 \pm 0.03$  stems/m<sup>2</sup>) throughout the monitoring period. Plots with high numbers of regeneration stems had open overstory canopies, while plots with little regeneration were covered by dense overstory canopies. As with Redbay, few regeneration stems <2.5 cm DBH and >1.4 m tall were present in Sassafras plots throughout the observation period.

*Redbay response to LWD in post-epidemic plots.* All original Redbay trees >2.5 cm DBH were dead to ground level (presumably killed by LWD) when we installed 4 post-epidemic assessment plots in 2009, but numerous basal sprouts and seedlings were present. In the 4 post-epidemic plots, density of Redbay <1.4 m tall at the first observation was 0.7 stems/m<sup>2</sup> and remained nearly constant throughout the 4-year monitoring period. Numbers of Redbay regeneration stems >1.4 m tall consistently averaged  $\sim 0.3$ /m<sup>2</sup> throughout the monitoring period (Fig. 8). Combining the numbers of regeneration stems in both size classes in post-epidemic plots brings averages to  $\sim 1$  stem/m<sup>2</sup>, which is comparable to the number in disease-progression plots 4 years after disease initiation.

At plot initiation, only 2 Redbay stems (both 3 cm DBH) had grown to tree size (>2.5 cm DBH) by  $\sim 7$  years after the epidemic passed through the area, 1 in each of 2 separate plots. During the 4-year study, an additional 41 Redbay stems grew to tree size (39 in plot Ri2 and 2 in Ri1), the largest of which was 3.6 cm DBH after 4 years. Among 43 small Redbay trees in these 2 post-epidemic plots, 4 (9.3%) died from LWD.

### RAB by stage of laurel wilt disease progression.

Redbay Ambrosia Beetles were not captured in areas outside the known distribution of LWD, and only 6 beetles were caught in areas adjacent to absent-near

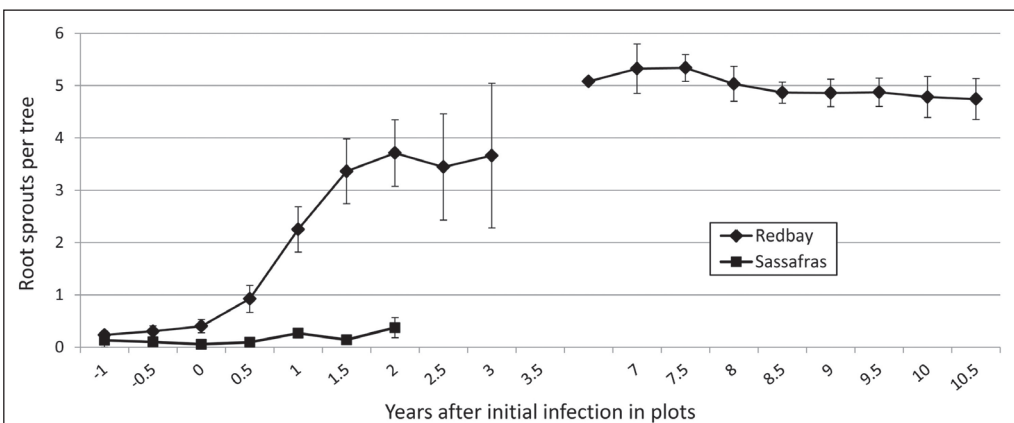


Figure 7. Mean numbers of below-ground basal sprouts around individual Redbay and Sassafras trees before and after infection in LWD-progression plots (Redbay:  $n = 4$ –11, Sassafras:  $n = 3$ –5); Redbay data for years 7–11 are from post-epidemic plots ( $n = 4$ ).

plots where RAB and LWD were within ~250 m. Relatively few RAB were caught at early-active and late-active sites (0.38 and 0.54 beetles per day, respectively). The largest numbers of RAB were trapped in areas where Redbay trees were in the advanced-active stage of LWD development (mean = 5.7/day; Table 2). A total of 8 RAB were trapped among 4 of 5 post-epidemic sites where the disease had passed through an estimated 5–10 years earlier.

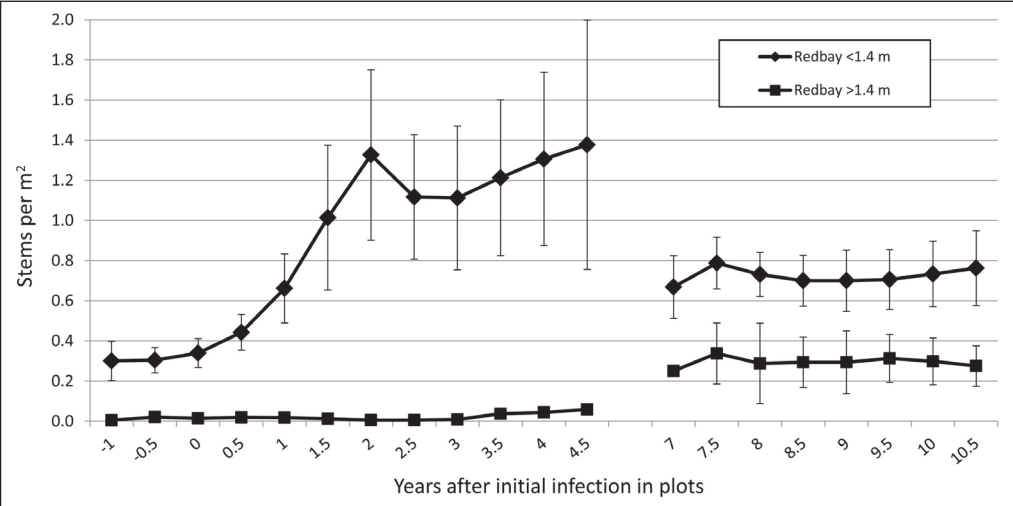


Figure 8. Mean numbers of Redbay regeneration stems (seedlings and sprouts <2.5 cm DBH) per m<sup>2</sup>, separated by size class and charted on a synchronized time scale (zero point on the x-axis is 6 months prior to first detection of laurel wilt in disease progression plots (*n* = 4–12). Data for years 7–10.5 years are from post-epidemic plots (*n* = 4)).

Table 2. Numbers of *Xyleborus glabratus* (Redbay Ambrosia Beetle) caught in traps baited with manuka oil located adjacent to Redbay and Sassafras monitoring plots in varying stages of laurel wilt disease progression, and mean cumulative DBH of infested Redbay trees in adjacent plots during August 2009 and 2010.

Host species/LW disease stage*	Trapping periods	Number/day (mean ± SE)	Cum. DBH (cm) of infested host (mean ± SE)
Redbay			
Outside range	8	0.00 ± 0.00	0.0 ± 0.0
Absent-near	6	0.03 ± 0.01	0.0 ± 0.0
Early-active	4	0.38 ± 0.19	47.8 ± 6.6
Advanced-active	3	5.70 ± 3.83	168.7 ± 53.0
Late-active	3	0.54 ± 0.08	27.3 ± 10.0
Post-epidemic	9	0.03 ± 0.01	0.0 ± 0.0
Sassafras			
Outside range	2	0.00 ± 0.00	-
Early-active	2	0.05 ± 0.02	-
Advanced-active	1	3.76	-
Late-active	3	0.10 ± 0.07	-

\*Disease stage at the time of trap deployment.

Numbers of beetles captured at individual Redbay trapping sites were strongly related to the amount of infested host in adjacent plots, expressed as total infested DBH at the time of trap deployment, excluding plots without infested host ( $n = 10$ ,  $r^2 = 0.80$ ,  $P < 0.0005$ ). Excluding 1 outlier for which 10 times more beetles were caught than in any other trap, a strong relationship remained between beetles caught and amount of infested host ( $n = 9$ ,  $r^2 = 0.50$ ,  $P = 0.03$ ).

In Sassafras, we caught the largest number of RAB (109 total, 3.76 beetles per day) adjacent to the only site in an advanced-active stage of LWD development. We trapped very few RAB at the early-active and late-active stages, and none in traps deployed outside the range of LWD (Table 2).

## Discussion

### **LWD impact, progression, and regeneration in Redbay**

Final Redbay mortality among 12 sites distributed over a broad area and a variety of coastal plain ecoregions in Georgia was 87% of trees and 93% of the BA, which is similar to that reported through portions of the disease process and/or at more restricted sites in Georgia (Maner et al. 2014, Spiegel and Legee 2013) and Florida (Fraedrich et al. 2008, Shields et al. 2011). However, this is the first report of LWD through the entire progression in individual stands over a variety of site/stand conditions. Disease progression and final mortality rate varied considerably among sites. All Redbay trees were killed by LWD within 2 years in 6 stands, and disease progression appeared to stop after larger trees were killed in 6 others. The diameter of the very first symptomatic trees in plots did not differ significantly from the average diameter of all trees in plots. However, starting 1 year after first infections, large Redbay trees were killed more rapidly than smaller trees, possibly because larger stem silhouettes are more attractive to RAB (Mayfield and Brownie 2013). There was no obvious pattern of spread through the stand, or indication that the disease spread through root connections from one tree to the next. Episode duration was inversely related to increasing initial mean diameter and basal area in stands. Stands with the fastest LWD progression and 100% mortality were all bay forest plant communities that had a greater abundance of large Redbay trees. Disease mortality curves and knowledge of factors affecting disease progression are essential for accurately predicting future spread of LWD.

Once Redbay trees became symptomatic with LWD, their entire crowns generally died within months, and sprouts emerged at the base of the majority of those trees. Among 236 Redbay trees determined to be infected with LWD in this study, all stems and above-ground sprouts died within ~1 year, and no evidence of inherent post-infection resistance was observed in individual Redbay trees. All initially healthy Redbay trees died from LWD on 6 sites, but a few small trees up to 13 cm DBH remained alive on 6 other sites with smaller initial host size and density. Although putative resistance has been reported among surviving Redbay trees from 6 sites with high levels of LWD mortality (Hughes and Smith 2014), continued research is needed to determine whether Redbay trees surviving the LWD epidemic have escaped RAB attack or exhibit some form of host resistance.

Redbay regeneration (seedlings and sprouts) was abundant and reached a plateau about 2 years after first symptoms were observed in stands heavily impacted by LWD. Redbay seedlings were sparse in most disease-progression plots, but we observed abundant seedling regeneration in one Redbay disease-progression plot and one post-epidemic plot, suggesting it may be a relatively rare event. In contrast, below-ground basal sprouting, although variable among sites, was prevalent and persistent after the passage of LWD.

Heavy mortality of Redbay sprouts and seedlings after LWD has been reported in areas affected by LWD, and Redbay regeneration is predicted to be of little consequence in the replacement of Redbay stems after the passage of LWD (Evans et al. 2014, Spiegel and Legee 2013). Ultimately, this would have significant impacts on plant communities and on animals that utilize Redbay foliage such as *Papilio palamedes* (Drury) (Palamedes Swallowtail). Basal sprouts attached to the trunk of trees rely on these above-ground stem tissues for water and nutrients and thus generally are short lived as the main stems die to the ground level (Del Tredici 2001). Therefore, only basal sprouts originating from below-ground with potential access to live roots were inventoried in this study. Even though considerable mortality of below-ground sprouts was observed (often the result of Black Twig Borer attacks), the numbers of live sprouts increased rapidly and were maintained through 4 years in disease-progression plots and up to 11 years in post-epidemic plots. Differences in definitions of sprouts and inventory techniques or, in the case of St. Catherine's Island, a unique ecosystem not representative of most Redbay habitat (Evans et al. 2014) may have contributed to apparent contradictory results regarding Redbay regeneration.

Clumps of Redbay sprouts are commonly observed shortly after clear-cut logging or prescribed burns, and Redbay is common in the understory and mid-story of many pine plantations in the southeastern coastal plain. Thus, basal-sprouting appears to be a reliable and rapid means of regeneration and replacement of Redbay stems following disturbance, including LWD, and some herbivores may thrive on flushes of Redbay regeneration in the wake of LWD (Chupp and Battaglia 2014).

### **LWD impact, progression, and regeneration in Sassafras**

Laurel wilt disease killed 80.4% of Sassafras trees in disease-progression plots, and the Sassafras mortality curve was very similar to that of Redbay through 2.5 years. Large Sassafras trees were killed more rapidly than smaller trees starting with the first infections. Laurel wilt disease sometimes spreads rapidly from one tree to the next in dense Sassafras thickets, and black staining typical of LWD has been observed in Sassafras lateral and runner roots (R.S. Cameron, pers. observ.), suggesting disease transmission through interconnected roots. Additional studies are needed to characterize the spread of the LWD pathogen in Sassafras root systems.

Laurel wilt disease has spread intermittently among Sassafras stands in Georgia, affecting some thickets and individual trees, while others nearby remain apparently healthy. Disease progression in some Sassafras stands is incomplete. Mortality stopped in one plot after 90% of trees over 5 cm DBH were killed, and many small trees remained alive around the periphery of this and other diseased thickets.



Sassafras appears to have some degree of tolerance to LWD as many trees continue to produce new sapwood long after infection. Thus, some trees are not killed completely to the ground level and produce epicormic sprouts that grow into branches off the trunk or lower crown for at least several years after infection, despite the presence of the pathogen in the trees.

Sassafras proliferates rapidly through root suckers and produces seeds at a relatively young age, which are readily disseminated by wildlife. Sassafras is also known to produce both basal sprouts from stumps of young trees and abundant root suckers from long lateral runner roots, which are instrumental in the rapid colonization of openings in wooded areas and open fields, resulting in dense and relatively pure stands (Gant and Clebsch 1975, Griggs 1990). Regeneration by seedlings and/or root sprouts was abundant in half the Sassafras study sites, and numerous smaller trees on the periphery of most thickets remained apparently healthy many years after initial infection.

Sassafras is widely distributed throughout the eastern US, and our data suggest it will likely to be severely impacted by LWD, especially larger trees. However, through intermittent disease spread, host tolerance, and prolific seedling and root-sprout regeneration, Sassafras will likely continue to persist, at least as small trees, in the presence of laurel wilt disease.

### **RAB populations related to disease progression**

Redbay Ambrosia Beetles use olfactory cues in finding hosts and are attracted to volatiles emitted from cut Redbay (Hanula and Sullivan 2008; Hanula et al. 2008; Kendra et al. 2011, 2014). Although not definitively confirmed, circumstantial evidence suggests that at least some of the first trees that RAB attack and infect with *R. lauricola* in new areas are already damaged and emitting cues for attraction and boring. Among 4 sites in our study with no known LWD within at least several km, the very first tree confirmed with LWD in the area had broken limbs from storm damage (2 sites), dieback, or damage from human activity.

We captured no RAB in traps placed outside the known range of LWD and only a few in plots where LWD-infected trees were within ~250 m. Increasing numbers of RAB were caught during the early-active stage of disease progression. As RAB brood began emerging from the first infested trees, populations increased rapidly along with Redbay mortality, which reached 70% within 1.5 years after initial disease detection. During this rapid disease-progression phase, larger-diameter Redbay trees died at a higher rate than smaller ones, possibly reflecting RAB preference for larger silhouettes (Mayfield and Brownie 2013) at a time when the area is inundated with host volatiles, perhaps making point sources of chemical cues more difficult to locate. The largest numbers of RAB were captured in monitoring traps during the advanced-active stage, starting ~1.5 years after the first symptomatic trees were observed. This stage occurred after 50% of Redbay trees had died and RAB brood emergence was likely at its peak (Maner et al. 2013). Numbers of RAB decreased during the late-active disease-progression stage when most host trees had died and conditions for brood production were deteriorating within trees.

Low numbers of RAB captured at 4 of 5 post-epidemic trapping sites and observations of a few small-diameter Redbay trees killed by LWD adjacent to 3 of 4 post-epidemic plots indicate that low-level populations of RAB have survived in these areas up to 11 years after the passage of the original LWD epidemic. These findings are similar to those of Maner et al. (2014) confirming that RAB populations drop to very low levels after suitable hosts are eliminated by LWD, possibly allowing *Persea* spp. populations to recover after the epidemic moves through an area (Hanula et al. 2008). Over the longer term, the continued success of the RAB in southeastern forests will depend on its ability to find and utilize hosts that are on average much smaller in diameter and occur at lower densities than occurred when the beetle first arrived. As Redbay trees reach greater diameters and densities, however, LWD incidence may increase within localized areas.

Sassafras has been less attractive to RAB than Redbay or Swamp Bay in most trapping experiments (Hanula et al. 2008, Kendra et al. 2014, Mayfield and Hanula 2012), yet Sassafras has been shown to be a suitable host for RAB (Mayfield et al. 2013). Since RAB were trapped adjacent to an isolated advanced-active Sassafras stand in this study, and LWD has killed Sassafras trees in widely separated locations in the southeastern US where no Redbay are nearby (Bates et al. 2015), it is clear they can reproduce and sustain populations on Sassafras alone.

If RAB and *R. lauricola* can survive in colder climates to the west and north as Formby et al. (2013) suggest, the disease has a high potential to continue spreading and cause significant impacts in areas of abundant, large Sassafras trees. Perhaps more importantly, Sassafras may provide a reservoir of RAB and *R. lauricola* over a large area and long period of time, thus increasing the probability that the disease will be spread via human movement to other susceptible Lauraceae like *Umbellularia californica* (Hook. & Arn.) Nutt. (California Bay Laurel; Mayfield et al. 2013) and *Persea americana* Mill. (Avocado) in California.

### Disease management

Management of LWD in forested areas may be impractical, except possibly in high-value, isolated host populations, such as island communities, where prompt sanitation and removal of infected trees may slow the spread. The relatively slow initial progression of LWD observed in isolated Redbay stands in this study indicates there is a narrow window of opportunity to slow LWD through early detection and complete removal of the first infested trees.

Currently, it appears LWD will continue to spread and impact Redbay populations across the southeastern and south-central US. Laurel wilt disease was recently detected in Redbay in isolated counties in east Texas and it is likely to spread to the far western range of the species in south Texas. In addition, the disease is now spreading in Sassafras beyond the distribution of Redbay. The rate of spread has been much more rapid than originally predicted, in large part because of the human transport of infested host material and the efficiency of RAB as a vector. Thus, the most crucial management option would involve discovery of pathways of introductions into new areas and implementing means to prevent these long-distance

movements into disease-free areas in the southeastern US and other parts of the New World where there is an abundance of Lauraceae species of both ecological and economic importance.

### Conclusions

Laurel wilt disease progression in Redbay stands starts slowly with a few trees of varying sizes, but mortality increases rapidly, especially in larger trees, as RAB populations build to high levels. The rate of spread is greater within Redbay stands with higher densities of larger diameter trees, best represented in bay forests where nearly all trees >2.5 cm are killed within 2 years after the first symptomatic trees are observed. In stands with sparser and smaller-diameter trees, disease progression may last up to 4 years, sometimes becoming apparently inactive with a few small Redbay trees remaining alive. Abundant seedling regeneration appears to be infrequent after Redbay stands are decimated by LWD, but below-ground basal sprouts proliferate around most stumps within a year after trees are killed. This appears to be an important regeneration strategy for Redbay after LWD, and may lead to persistence and slow recovery for Redbay. However, low numbers of RAB and scattered LWD mortality in small Redbay trees continue on most sites up to 11 years after the initial epidemic, which suggests that the disease will persist at endemic levels and will continue to impede Redbay recovery.

Laurel wilt disease has spread out of the Redbay range and into Sassafras in parts of Georgia. The disease mortality curve and preference for larger trees are similar to that in Redbay. Progression in thickets can be rapid, apparently moving through clonal root systems, but the disease process slows or stops in some Sassafras stands for unknown reasons. Epicormic shoots and lingering decline in individual trees may be evidence of host resistance in Sassafras. Further investigation of the spread of LWD in Sassafras root systems and within stands in a wider variety of habitats and geographical locations is needed to evaluate the potential for spread over its extensive range.

Documentation of LWD progression in Redbay and Sassafras provides a better understanding of disease epidemiology and baseline data for modeling the spread of LWD. Observations of successful RAB development, disease behavior, and broadening distribution of LWD in Sassafras in the absence of Redbay, indicate a potential for disease spread through Sassafras populations in North America, increasing the likelihood it will eventually expand its range to the western US and Central and South America where members of the Lauraceae are more diverse and abundant.

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**Appendix 1.** Characterization of laurel wilt disease progression monitoring plots and *Xyleborus glabratus* (Redbay Ambrosia Beetle) trapping sites, with initial disease status, county location, ecoregion, soil series, landscape position, plant community type, plot size, and host species (R = Redbay, S = Sassafras, and B = both) present in southeastern Georgia.

Plot ID/ disease status <sup>A</sup>	County	Ecoregions of Georgia <sup>C</sup>	Soil series	Landscape position	Plant community type <sup>D</sup>	Plot size (m <sup>2</sup> )	Host
Ra1	Emanuel	Atlantic Southern Loam Plains	Kinston- Bibb	Upper stream terrace	Bay forest	400	R
Ra2	Jenkins	Atlantic Southern Loam Plains	Rains	Intermit. stream head	Mixed hardwood forest	400	R
Ra3	Bacon	Bacon Terraces	Surrency	Intermit. stream head	Mixed hardwoods, pond pine	400	R
Ra4	Emanuel	Atlantic Southern Loam Plains	Pickney	Upper stream terrace	Bay forest	400	R
Ra5	Ware	Bacon Terraces	Surrency	Bayhead	Bay forest, pond pine	400	R
Ra6	Pierce	Bacon Terraces	Chipley	Upper stream terrace	Bay forest, pond pine	400	R
Ba1	Jenkins	Atlantic Southern Loam Plains	Kershaw	Upland side slope	Mixed hardwoods, pine	400	B
Bd1	Screven	Atlantic Southern Loam Plains	Surrency	Bayhead	Mixed hardwoods, pine	500	B
Rd2	Bulloch	Atlantic Southern Loam Plains	Rutledge	Upper stream terrace	Bay forest	400	R
Rd3	Tattnall	Atlantic Southern Loam Plains	Osier	Upper stream terrace	Mixed hardwood, pine	400	R
Rd4	Appling	Bacon Terraces	Surrency	Bayhead	Mixed hardwood, pine	400	R
Rd5	Brantley	Okefenokee Plains	Mascotte	Upland flatwoods	Pine plantation, saw palmetto	400	R
Ri1	Bryan	Sea Islands, Coastal Marsh	Leon	Coastal flatwoods	Mixed hardwood, pond pine	400	R
Ri2	Chatham	Sea Islands, Coastal Marsh	Ellabelle	Coastal flatwoods	Mixed hardwoods	400	R
Ri3	McIntosh	Sea Islands, Coastal Marsh	Rutledge	Bayhead	Bay forest, oaks	400	R
Ri4	Bulloch	Sea Island Flatwoods	Rutledge	Stream head	Mature mixed hardwoods, pine	400	R
Sa1	Jenkins	Atlantic Southern Loam Plains	Troup	Sand ridge	Pine plantation, mixed hardwoods	200	S
Sa2	Jenkins	Atlantic Southern Loam Plains	Troup	Sand ridge	Pine plantation, agric. field edge	50	S
Sd1	Bulloch	Atlantic Southern Loam Plains	Cowarts	Upland side slope	Roadside, adjacent pine plantation	200	S
Sd2	Screven	Atlantic Southern Loam Plains	Fuquay	Sand ridge	Fence row between agricultural fields	90	S
Ra7 <sup>B</sup>	Emanuel	Atlantic Southern Loam Plains	Kinston- Bibb	Stream head	Mixed hardwoods	NA	R
Ra8 <sup>B</sup>	Wheeler	Atlantic Southern Loam Plains	Osier-Bibb	Stream terrace	Mixed hardwood forest	NA	R

Plot ID/ disease status <sup>A</sup>	County	Ecoregions of Georgia <sup>C</sup>	Soil series	Landscape position	Plant community type <sup>D</sup>	Plot size (m <sup>2</sup> )	Host
R15 <sup>B</sup>	Wayne	Sea Island Flatwoods	Kinston- Lynchburg	Bayhead	Mixed hardwoods, pine	400	R
R16 <sup>B</sup>	Evans	Atlantic Southern Loam Plains	Rutledge	Bay swamp	Bay forest	NA	R
Sa3 <sup>B</sup>	Jenkins	Atlantic Southern Loam Plains	Fuquay	Sand ridge	Roadside, adjacent pine plantation	140	S
Sd3 <sup>B</sup>	Bulloch	Atlantic Southern Loam Plains	Fuquay	Sand ridge	Fence row between agricultural fields	NA	S

<sup>A</sup>Plot ID and initial disease status key: R = Redbay, S = Sassafras, B = both Redbay and Sassafras, a = LWD absent at initiation, d = LW disease active at initiation, i = inactive, post-LW disease epidemic, NA = not applicable.

<sup>B</sup>RA B trapping sites only.

<sup>C</sup>Griffith et al. (2001) Ecoregions of Georgia.

<sup>D</sup>Redbay sites in this study generally fit into 2 plant community types, bay forests or mixed hardwoods, some of which also included *Pinus* spp. (pine). In addition to Redbay, the most common woody plant species on the bay forest sites were: *Gordonia lasianthus* (L.) Ellis (Loblolly Bay), *Magnolia virginiana* L. (Sweet Bay), *Cyrilla racemiflora* L.(Titi), *Ilex glabra* (L.) Gray (Gallberry), and *Lyonia lucida* (Lamarck) K. Koch (Fetterbush). Woody species present on the mixed hardwood/pine sites included *Acer rubrum* L. (Red Maple), *Liquidambar styraciflua* L. (Sweetgum), *Liriodendron tulipifera* L. (Yellow-poplar), Sweet Bay, *Nyssa sylvatica* Marshall (Blackgum), Redbay, *Quercus nigra* L. (Water Oak), other *Quercus* spp. (oaks), Gallberry, *Myrica cerifera* L. (Wax Myrtle), and Titi. *Smilax* sp. (greenbrier vines) were present on both bay and mixed-hardwood forest sites, but were especially prolific on bay forest sites. *Pinus serotina* Michaux (Pond Pine) was an important component on 3 bay forest and 3 mixed hardwood/pine sites. *Pinus elliotii* Engelm (Slash Pine) or *P. taeda* L. (Loblolly Pine), was present in several other mixed-hardwood/pine sites. Two plots were established in managed pine plantations, one Slash Pine and the other Loblolly, with Redbay in the mid-story. The Loblolly Pine plantation was an upland flatwoods site with *Serenoa repens* (Bartram) Small (Saw Palmetto) as a major understory component. Sassafras disease-progression plots were generally located on disturbed sites, adjacent to open fields, with thickets of nearly pure Sassafras. However, 2 plots with both Redbay and Sassafras present were classified as mixed-hardwood/pine plant communities.