Disease Note

Diseases Caused by Fungi and Fungus-Like Organisms

First Report of Laurel Wilt Caused by *Harringtonia lauricola* (Previously *Raffaelea lauricola*) on Northern Spicebush in Kentucky and Tennessee

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Laurel wilt (LW) is a vascular disease caused by the fungus *Harringtonia lauricola* (previously *Raffaelea lauricola*) and transmitted by its primary vector, the redbay ambrosia beetle (*Xyleborus glabratus*, RAB), both of which were first detected in the U.S.A. in 2002, likely introduced from their native range in Asia (Fraedrich et al. 2008; Harrington et al. 2008). LW has since spread across the southeastern U.S.A. causing the death of millions of native redbay, sassafras, silk bay, swamp bay, and other native Lauraceae species (Hughes et al. 2017). Detection of LW on the deciduous understory shrub northern spicebush (*Lindera benzoin*) was previously reported in South Carolina (Fraedrich et al. 2016) and Louisiana (Olatinwo et al. 2021) and is hereby confirmed in Kentucky and Tennessee. Spicebush plants displaying typical LW symptoms were observed in September 2020 on a property spanning the KY/TN border (Christian Co., KY, and Montgomery Co., TN). Several dense stands of spicebush exhibited leaf wilt, early fall leaf coloration, dead leaves on branches, and black streaks of discolored xylem. LW was already confirmed on sassafras in the area (Loyd et al. 2020), and there were abundant dead sassafras nearby. Ambrosia beetle boring dust was observed and callow female RABs emerged from containerized bolts of spicebush collected from the site, indicating that the vector used spicebush as a brood host. Samples of spicebush sapwood tissue collected from two symptomatic plants were plated onto CSMA (cycloheximide-streptomycin malt extract agar) medium. The cultures were grown at room temperature in ambient light, and a fungus was recovered and further transferred onto MEA (malt extract agar) and PDA (potato dextrose agar) media. The morphology of the two fungal isolates, referred to as LW415 and LW416, matched the typical white mucoid growth and ovoid conidia of *H. lauricola* (Harrington et al. 2008). DNA was extracted from conidia harvested from 2-week-old MEA cultures using a modified method of Dreaden et al. (2014). The identity of the fungus was confirmed by performing PCR with the *H. lauricola*-specific microsatellite primer sets IFW and CHK (Dreaden et al. 2014; Parra et al. 2020). A positive amplification product was obtained for LW415 and LW416 for both primer sets, validating their identification as *H. lauricola*. To confirm pathogenicity, four spicebush seedlings (mean height 22.5 cm; mean ground line diameter 3.3 mm) were inoculated: two with *H. lauricola* isolate LW415 grown on PDA for 2 weeks at room temperature in the dark, and two mock-inoculated with sterile PDA as a control. A scalpel was used to nick the spicebush stem at a bud about 5 cm above groundline, and a 3 mm^2 agar plug was placed in the wound and wrapped with Parafilm. The spicebush seedlings were maintained in a growth chamber with an average temperature of 24°C and a 15-h photoperiod. Wilt symptoms were evident on inoculated seedlings after 2 weeks, while the control plants remained healthy. Four weeks postinoculation, black staining of the vascular tissue was present in the symptomatic seedlings, and a fungus matching the morphology of *H. lauricola* was consistently recovered, while no fungus was isolated from the control plants. These results provide additional evidence that northern spicebush populations may be threatened by LW and could serve as a reservoir for the pathogen and vector (Gramling 2010). The spread of LW and RAB on spicebush may gain importance as preferred hosts (e.g., sassafras) are killed.

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