Influence of Host Condition on the Red Pine-*Sphaeropsis sapinea* Interaction in Wisconsin Plantations.

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Some of the most frequent and severe damage caused by *Sphaeropsis sapinea* (syn. *Diplodia pinea*) in North America has occurred in Wisconsin on red pine (*Pinus resinosa*) during the last 25 years. The influence of host condition, specifically stress associated with drought, on development of disease caused by *S. sapinea* has been examined in a series of studies (1, 2). The aggressiveness of *S. sapinea* isolates was compared on water-stressed and nonstressed red pine seedlings in greenhouse and growth chamber experiments and on established red pine trees in a 3-year plantation study. Predawn water potentials (ψ_{PD}) were manipulated by withholding water (greenhouse/growth chamber), and by removing competing vegetation and supplemental watering (plantation). In both situations, young shoots were wounded and then inoculated. The pathogen caused more severe symptoms and could be reisolated farther from the site of inoculation of water-stressed trees than nonstressed trees, in all experiments. In the plantation, the most severe disease development occurred in the driest year, and disease was least severe in the wettest year. Competing vegetation enhanced disease development by inducing water stress, even in the wettest year, even on these trees that were considered well established.

Damage associated with *S. sapinea*, in the absence of the more traditionally observed shoot blight symptoms, also has been investigated (3). Recently planted seedlings and established saplings frequently died during 1992 and 1991, respectively, in red pine plantations in Wisconsin. These epidemics appeared to be related to substantial rainfall deficits in each year, compared to 30-yr averages. Mortality of seedlings in 12 plantations ranged from 14 to 95%. Mortality of saplings in 16 plantations ranged from 0 to 30%. Symptoms included blackened cortical tissue and dark staining of the underlying xylem in the lower stems and root collars. Pycnidia of *S. sapinea* were observed in these areas on 17 to 97% of dead seedlings. Pycnidia of the pathogen were observed on the lower stems or root collars of 67 to 87% of dead saplings examined in four plantations. These observations comprise the first association of Sphaeropsis collar rot with high frequencies of red pine mortality in either established saplings or recently planted seedlings.

The role of water stress in the initiation of collar rot by *S. sapinea* in latently colonized red pine seedlings was investigated in two greenhouse experiments (unpublished data). Seedlings were not inoculated, but had been obtained from nurseries where the pathogen was present and where asymptomatic persistence previously has been demonstrated (4). In experiment 1, seedlings were either watered to maintain mean predawn needle water potential (ψ_{PD}) of -0.55 MPa, or not watered until mean $_{PD}$ of -1.1, -1.7, -2.0, -2.5, or -3.2 MPa were achieved. Seedlings developed symptoms, including mortality, resembling those of Sphaeropsis collar rot. Mortality ranged from 7.5% of repeatedly watered seedlings to 50% of those in the driest regime ($P < 0.001$). The pathogen was identified from 42% of the living seedlings and 92% of the dead seedlings, with the most frequent identification overall (for seedlings living or dead) in the driest regime (72%). In experiment 2, seedlings were either watered to maintain mean $_{PD}$ of -0.65 MPa, or not watered until mean $_{PD}$ of -2.8 MPa was achieved. Half of the seedlings in each watering regime also were treated with benomyl. Mortality was low ($\leq$ 4%) for watered seedlings, whether or not

fungicide was applied. Mortality of nonwatered seedlings, however, was greater among nontreated seedlings (61%) compared to benomyl-treated seedlings (37%) ($P < 0.001$). Results support the conclusion that physiological alteration can effect release from the quiescent condition to result in rapid disease development and confirm that *S. sapinea* can act as a latent pathogen. The ability of *S. sapinea* to persist on or in the asymptomatic host and its responsiveness to host condition help explain the repeated, sudden, and increasingly widespread episodes of disease experienced in Wisconsin.

Literature cited:

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