
Cypress Canker of Leyland Cypress in Florida¹

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INTRODUCTION: Wagener (1928) first described a debilitating and sometimes lethal canker of planted Monterey cypress (*Cupressus macrocarpa* Gordon) and columnar Italian cypress (*C. sempervirens* L. var. *stricta* Ait.) in the coastal hinterland of central and southern California (Wagener 1939). Considered something of a local epidemic, “cypress canker” apparently destroyed large numbers of Monterey cypress in that area between the 1920s and 1940s (Panconesi 1990; Sinclair *et al.* 1987; Wagener 1939). Cypress canker and/or its causal pathogen(s) have subsequently been reported to occur throughout much of the world (presumably spread from California) on a variety of species in several genera belonging to the family Cupressaceae: *Chamaecyparis*, *Cupressus*, *Juniperus*, *Libocedrus*, *Thuja*, and the intergeneric hybrid genus *X Cuprocyparis* (formerly *X Cupressocyparis*) (Jones 1993; Sinclair 1987; Strouts 1973; Swart 1973, Tisserat *et al.* 1991; Wagener 1939; Windham *et al.* 1999). The disease is now particularly problematic on Italian cypress in the Mediterranean region and has been referred to as a pandemic and possible ecological disaster (Graniti 1993, 1998). In recent years, cypress canker has been increasingly recognized as a problem (Jones 1993; Sinclair *et al.* 1987; Tisserat *et al.* 1991; Windham *et al.* 1999) on Leyland cypress [*X Cuprocyparis leylandii* (Dallimore and A. B. Jackson) Farjon], a popular landscape screening and ornamental species in much of the southeastern United States. The disease was first recognized in Florida in a Christmas tree plantation in the state’s western panhandle in the spring of 2003 (Florida Division of Plant Industry, Plant Pathology Specimen Report P20030274).

THE PATHOGEN(S): Identifying the specific pathogen(s) causing cypress canker has been complicated by mycological reclassifications and uncertainties regarding whether the causal fungus or fungi is or are three distinct species or subspecific variants of a single species (Barnes *et al.* 2001; Graniti 1998; Guba 1961; Nag Raj 1993; Swart 1973). Contemporary thinking, however, leans toward three distinct species of *Seiridium* (Barnes *et al.* 2001; Boesewinkel 1983; Graniti 1998; Graniti and Frisullo 1990): 1) *S. cardinale* (Wagener) Sutton and Gibson [syn. *Coryneum cardinale* Wagener], 2) *S. cupressi* (Guba) Boesewinkel [syn. *Monochaetia unicornis* (Cooke and Ellis) emend. Ciccarone = *Cryptosticus cupressi* Guba and *Rynchosphaeria cupressi* Nattrass, Booth and Sutton], and 3) *S. unicorn* (Cooke and Ellis) Sutton [syn. *Pestalotia unicornis* Cooke and Ellis = *Monochaetia unicornis* (Cooke and Ellis) Sacc. and D. Sacc.]. Of these, *S. cardinale* is generally considered to be the most aggressive, *S. unicorn* the least aggressive, with *S. cupressi* being intermediate between the two (Graniti 1998; Graniti and Frisullo 1990). Although morphological differences among the three species are reported (Boesewinkel 1983; Graniti 1998; Graniti and Frisullo 1990), the near impossibility of separating *S. unicorn* and *S. cupressi* based on morphological traits has caused some (Barnes *et al.* 2001; Wingfield – personal communication) to advocate and employ molecular methodology to make

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or confirm specific identifications. Based on cultural and morphological characteristics, and supporting molecular evaluations (Dr. Irene Barnes, Department of Microbiology and Plant Pathology, Tree Pathology Cooperative Programme, Forestry and Agricultural Biotechnology Institute, University of Pretoria, South Africa), our Florida pathogen is apparently *S. cupressi*. If so, *sensu stricto*, this is a record for the State of Florida, and possibly the United States. It also raises questions regarding its occurrence in the United States, given its apparent Kenyan origin (Natrass *et al.* 1963). All of this should be considered with appropriate caution, however, given the natural variability of the organism(s) and the taxonomic uncertainties still surrounding *Seiridium* spp. associated with cypress canker. Artificial inoculations have demonstrated that our Florida isolate is pathogenic to Leyland cypress.

SYMPTOMS OF THE DISEASE: Leyland cypress trees infected with *Seiridium* spp. exhibit branch flagging; *i.e.*, dying of entire branches, characterized by reddening of the foliage thereon (Fig. 1). These branches may be infected by the pathogen, located on infected subtending stems, or both. Infected stems and branches display longitudinally elongated cankers that may appear “flattened, ridged, or otherwise contorted” (Wagener 1939). Bark on canker faces and margins often exhibits longitudinal fissuring and resin exudation as well (Fig. 1).



Fig. 1. Symptoms of cypress canker infection on Leyland cypress: A) Red-brown flagging of dead and dying branches; B) Sunken and ridged areas of infected stem with associated resin bleeding (resinosis); C) Infected stem showing bark fissuring and resinosis.

INFECTION BIOLOGY AND EPIDEMIOLOGY: *Seiridium* spp. are asexual fungi (Deuteromycotina, Coelomycetes) that reproduce via production of conidia produced in acervuli. New infections are initiated as conidia, spread by splashing water aided by wind, by beetles (*Phloeosinus* spp.; Coleoptera, Scolytidae), (Covassi *et al.* 1975), and possibly birds. These conidia lodge in, germinate on and infect abiotically or biotically wounded stems and/or branches. Direct infection through host epidermis and lenticels is also known (Birch 1933; Panconesi 1979). Rainfall and high humidity are important for conidial survival and germination,

and injured, freeze-damaged, and drought-stressed trees are more susceptible to infection than healthy trees (Panconesi 1990; Sandroock *et al.* 1999; Sinclair *et al.* 1987; Wagener 1939; Windham *et al.* 1999). Disease development is facilitated by phytotoxins produced by the pathogen(s) (Graniti 1993, 1998; Graniti and Sparapano 1990). The current, nearly worldwide geographical distribution of cypress canker is considered a function of the movement of infected or infested host material (Graniti 1993, 1998; Panconesi 1990).

CONTROL OF THE DISEASE: Control of cypress canker has not proven to be a simple task. Traditional methods employing surgery, sanitation and breeding for host resistance have demonstrated potential utility (Panconesi 1990), and fungicides may be useful in certain scenarios such as protecting high value ornamentals (McCain 1984; Panconesi 1990). Prevention is possible by avoiding movement of infected trees and planting and growing trees in suitable environments. (Panconesi 1990; Wagener 1939).

SURVEY AND DETECTION: Look for flagging dead and/or dying branches associated with flattened, ridged, or otherwise contorted branch and stem cankers. Cankers often show longitudinal bark fissuring and varying degrees of resin exudation. Diagnostic confirmation requires laboratory analysis as similar cankers may be associated with one or more species of *Botryosphaeria*.

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