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First Report of *Phytophthora ramorum* Infecting *Camellia* Flower Buds in North America

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Camellias are important nursery and landscape plants and are known to be highly susceptible hosts of *Phytophthora ramorum* Werres, De Cock & Man In't Veld, the pathogen that causes Sudden Oak Death. As part of a study investigating the seasonal disease progression of *P. ramorum* on camellias, ten leaves on each of 84 one-gallon *Camellia japonica* L. 'Kumasaka' plants were inoculated with V8 agar plugs from *P. ramorum* cultures on 18 July and 26 October 2005. The *P. ramorum* isolate used for inoculation was recovered locally from an infected rhododendron near Felton, CA. Beginning 20 December 2005, after several rain events, necrotic flower buds on many camellia plants were observed. The flower bud disease became widespread and severe by mid-January 2006 following another extensive rainy period. Infection began on the lowest sepals attached near the flower bud receptacle. Infected sepals contained relatively large areas of necrotic tissue and the lesions were often surrounded by diffuse necrosis (Fig. 1). The infection progressed from the sepals into the receptacle and then further into the flower bud. The petals eventually became completely necrotic within the flower bud. At this stage of disease development, the male flower parts initially appeared unaffected (Fig. 2). Most infected buds never opened, eventually abscised at the receptacle, and were shed. When flower buds fell to the soil, they often broke into many light parts that were observed moving readily with wind gusts. In some cases, the necrosis developed into adjacent vegetative and flower buds via stem tissue but the growth in stem tissue was generally very limited, and was only detected internally about 1 cm from the receptacle of an infected flower bud or vegetative bud (Fig. 3).

Fig. 1. Early flower bud infection by *P. ramorum* (left) and flower bud not infected (right).

Fig. 2. Internal symptoms of flower bud infection caused by *P. ramorum*. Male flower parts apparently are not infected at this stage.

Fig. 3. Symptoms of *P. ramorum* developing from flower bud into adjacent vegetative bud (left), and internal symptoms (right).

Before isolations, flower buds were microscopically examined and no hyphae, fruiting structures, or sclerotia were observed. Necrotic margins of the flower sepals, intact and abscised buds, and leaf spots, were rinsed with deionized water, and plated onto PARP media to determine if the necrosis was caused by *P. ramorum*. Additional samples of necrotic tissues were surface sterilized with a 0.6% sodium hypochlorite solution and plated on APDA media. In addition, immediately following several rain events, necrotic flower buds and leaves were separately rinsed with de-ionized water and the rinse-water was plated onto PARP media to assess sporangia production on infected tissues. Cultures were incubated at least 7 days at a mean temperature of 20°C in the dark. *P. ramorum* was identified morphologically by its coralloid hyphae, large chlamydospores, and sporangia.

P. ramorum was consistently isolated on PARP media from fully-developed flower buds, sepals, and occasionally from petals on opening flower buds. No plant pathogenic fungi were recovered from necrotic tissues plated on APDA media. Disease incidence was periodically assessed, and the apparent mean infection rate ranged from 5 to 23%, but individual plants could have an infection rate as high as 100%. Some leaves adjacent to infected flower buds became infected two to three weeks after flower buds showed symptoms (Fig 4). Mean propagule concentrations ranged from 1.1-10.0 $\times 10^4$ cfu/liter per washed flower bud and 0-2.6 $\times 10^4$ cfu/liter per washed leaf. Flower bud infections most likely arose from secondary inoculum produced by either previously artificially inoculated leaves or from secondary leaf infections arising from the inoculated leaves.

Fig. 4. Symptoms of secondary leaf infection arising from infected flower bud.

In an adjacent field experiment, similarly leaf-inoculated *Rhododendron* L. 'Cunningham's White' flower buds were also found infected with natural secondary infection of *P. ramorum*. The occurrence on rhododendron was much less extensive than on camellia, but with rhododendron, the infection could develop down the stem and into associated leaf petioles.

Camellia and *Rhododendron* are two of the most important ornamental host genera of *P. ramorum*, accounting for the majority of detections in infested commercial nurseries (4). Generally, camellia plants naturally infected with *P. ramorum* in the nursery show large necrotic leafspots. However, as leaf lesions enlarge, the leaves drop, sometimes resulting in total defoliation of the lower half of the plant canopy. Monitoring and detection of leaf infections has focused on symptoms of leaves and stems (3). Because infected leaves can defoliate rapidly, infected flower buds could be the only plant part with symptoms present during field monitoring. Consequently, infected flower buds may be an important plant part for detection of *P. ramorum* and source of inoculum and pathogen spread in nurseries or landscapes.

This is the first report of flower bud infection in the field with the North American genotype of *P. ramorum*. Previously, flower buds of the North American genotype were not tested for susceptibility to *P. ramorum* (2). In Britain, flower bud infection has rarely been seen with the European genotype (1). This may be because camellias are not frequently produced in Britain (P. A. Beales, *personal communication*), or environmental conditions that favor bud infection occur infrequently.

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