

Stem Canker and Leaf Spot of Poinsettia Caused by *Pseudomonas viridiflava* in Florida

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ABSTRACT

Engelhard, A. W., and Jones, J. B. 1990. Stem canker and leaf spot of poinsettia caused by *Pseudomonas viridiflava* in Florida. *Plant Dis.* 74:528-529.

Stem cankers developed on outdoor poinsettia (*Euphorbia pulcherrima*) grown near Bradenton, FL, in December 1984. A fluorescent bacterium was isolated from the cankers. The bacterium was positive for tobacco hypersensitivity, grew on a minimal medium containing DL-lactate, D(-)-tartrate, or erythritol but not sucrose, and produced a negative reaction for arginine dihydrolase and oxidase and a positive reaction for potato soft rot and levan. The pathogen was identified as *Pseudomonas viridiflava*. It produced stem cankers on all seven cultivars of poinsettia inoculated on the stems and leaf spots on the two cultivars inoculated on the leaves. The disease was most severe on stems at 10 and 16 C; it was mild at 27 C and absent at 32 C.

During the 1984 production season for poinsettias (*Euphorbia pulcherrima* Willd. ex Klotzsch) in southwest Florida, disease symptoms occurred on plants growing outdoors that did not correspond to known diseases in Florida. During the warmer months of the summer and fall, plants of cultivar Gutbier V-14 Glory had spots on leaves, bracts, petioles, and ciathia and cankers on stems. These symptoms later were determined to be diagnostic for a new disease caused by *Alternaria euphorbiicola* Simmons & Engelhard (1,2).

In December, dark, wet stem cankers appeared. A fluorescent pseudomonad was isolated from the cankers. A stem canker and leaf spot caused by *Pseudomonas viridiflava* (Burkholder) Dowson had been identified previously in California (12). However, in that report, the optimum temperature range was 27-32 C, whereas the stem cankers observed in southwest Florida developed during a cool period in December.

The objectives of the study reported here were to describe the biochemical and physiological characteristics of the causal organism, investigate the effect of ambient temperature on stem canker development, and determine the susceptibility of nine cultivars to the pathogen.

MATERIALS AND METHODS

Tissue from cankers on Gutbier V-14 Glory was triturated in drops of sterile deionized water. Loopfuls of the resulting suspension were streaked onto petri plates containing medium B of King et al (KMB) (6) and Tween medium B (9). Two strains (designated 6436-3 and 6436-7) of a bacterium that produced a diffusible fluorescent pigment were collected and maintained on KMB for biochemical, pathological, and hypersensitivity tests. Composite suspensions of the two strains were used for stem inoculations, and strain 6436-3 was used to inoculate leaves.

Effect of temperature. Four replications of plants of cultivar Annette Hegg Dark Red were inoculated on the stems and kept at 10, 16, 21, 27, and 32 C. Each plant was grown in a 10-cm pot. Plants were inoculated when they were 15 cm tall.

The two strains (6436-3 and 6436-7) used for inoculum were grown for 48 hr on KMB at 28 C. A bacterial suspension at 10^8 cfu/ml was prepared in deionized water. A sterile wooden toothpick was submerged in the inoculum for 5 sec and then inserted through the stem about 3 cm from the terminal. Control plants were similarly wounded with sterile toothpicks that had been dipped in sterile water.

Immediately after inoculation, each plant was covered with an internally premoistened polyethylene bag and placed in a constant-temperature growth chamber lighted with fluorescent light (12 hr of light, 12 hr of darkness) (Percival Mfg. Co., Boone, IA). The temperature in the bags was 3 C higher than ambient

temperature when the lights were on. The length of each lesion was measured after 7 days. The experiment was repeated.

Cultivar susceptibility. Plants of the cultivars Gutbier V-14 Glory, Annette Hegg (AH) Brilliant Diamond, AH Top White, AH Dark Red, AH Hot Pink, and Eckespoint C-1 Red were inoculated as in the previous experiments. After the toothpick inoculation of the stems, the plants were placed in a lighted growth chamber at 17 C. The plants were rated 7 days later. The experiment was repeated with the same cultivars plus Gutbier V-10 Amy, and the plants were kept in a lighted (diurnal 12-hr light, 12-hr dark), constant-temperature Percival growth chamber at 16 C. Two and four control plants were used for the first and second experiments, respectively.

Poinsettia plants of cultivars Eckespoint Jingle Bells 3 and Gross Supjibi, planted one per 15-cm pot and 30 cm tall, were used in the leaf inoculation test. The plants were pretreated for 16 hr in a Percival dew chamber at 26 C. Five leaves per plant of each cultivar were dusted with powdered Carborundum and sprayed on the upper leaf surfaces with a cell suspension of strain 6436-3 at 10^8 cfu/ml. Five other plants with no Carborundum treatment were sprayed on the upper and lower leaf surfaces. The upper surfaces of dusted leaves were then rubbed lightly with a sterile cotton swab. Control plants were treated similarly, except they were sprayed with water. Each plant was covered with a premoistened polyethylene bag and placed in a fluorescent-lighted, walk-in growth chamber at 26 C. The bags were removed after 72 hr.

RESULTS

Both strains caused a positive reaction for tobacco hypersensitivity (7), fluoresced under ultraviolet light on KMB (3,6), and grew on a minimal medium containing DL-lactate, D(-)-tartrate, or erythritol but not sucrose. They produced a negative reaction for arginine dihydrolase and oxidase and a positive reaction for potato soft rot and levan (3-5,8,10,12,13). The strains were considered to be *P. viridiflava* (4,8,10).

Effect of temperature. In the regression of disease severity on temperature,



Fig. 1. Stem decay on cultivar Gutbier V-14 Glory poinsettia plant stem-inoculated with *Pseudomonas viridiflava* using the toothpick insertion method.

both the linear and the quadratic components were significant, and the slope was negative. Disease decreased as temperature increased. The mean length of the stem decay (two tests combined) was 22.4 mm at 10 C, 20.9 mm at 16 C, 6.2 mm at 21 C, 4.4 mm at 27 C, and 0 at 32 C. None of the control plants developed disease.

Cultivar susceptibility. All seven cultivars inoculated on the stems developed cankers (Fig. 1). The mean length of the decay ranged from 22 to 55 mm. The cultivars did not differ significantly when the two experiments were grouped. The stems in the control treatment in the first experiment were free of discoloration, whereas in the second test, four had slight, atypical lesions (1–3 mm long).

Spots appeared on inoculated leaves within 3 days. They were mostly round when they developed on the internal portions of the leaves. Infected spots on the leaf margins developed as necrotic areas along the margins or extended into the leaf lamina (Fig. 2). The spots were somewhat papery. They were light brown and sometimes turned dark with age. A chlorotic area always was associated with the spots.

DISCUSSION

The only report of *P. viridiflava* causing disease in poinsettia plants was

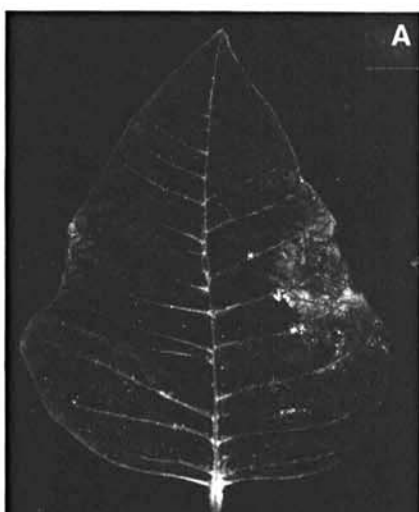


Fig. 2A,B. Leaf spots caused by *Pseudomonas viridiflava* on leaves of poinsettia cultivar Eckespoint Jingle Bells 3 are brown, papery, and surrounded by chlorotic tissue.

from California in 1981 (12). This is the first report of *P. viridiflava* causing disease on poinsettia plants in Florida. Three cultivars evaluated in the California study (12) were susceptible, and the seven evaluated in this study for stem decay and the two evaluated for leaf spots were susceptible.

The major difference between our results and those of Suslow and McCain (12) was the effect of temperature on disease. In Florida, a high level of disease developed on stems at 10–16 C, very little developed at 27 C, and none occurred at 32 C. In California, disease was most severe over the range of 27–32 C.

The canker disease in Florida was observed on outdoor, shade-grown poinsettia plants in December. The mean low and high temperatures for the first 17 days of December (the pathogen was isolated on 17 December) were 10 and 24 C, respectively (11). Therefore, the December temperatures were in the range for disease development.

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