Blight of Prostrate Spurge and Cultivated Poinsettia Caused by *Amphobotrys ricini*

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ABSTRACT

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The fungus *Amphobotrys ricini* was isolated from blighted leaves and stems of prostrate spurge and cultivated poinsettia. Laboratory and greenhouse pathogenicity tests were positive on *Euphorbia supina* (prostrate spurge), *E. pulcherrima* (cultivated poinsettia), *E. heterophylla* (wild poinsettia), *E. hirta* (garden spurge), and *Ricinus cummunis* (castor bean). This is the first report of blight of prostrate spurge and cultivated poinsettia caused by *A. ricini* in the United States.

Additional keywords: *Botryotinia ricini*, fungal diseases of plants

Prostrate spurge (*Euphorbia supina* Raf. ex Boiss.) is a common annual weed in cultivated fields, gardens, and waste areas in the southeastern United States (8). A blight that resulted in plant death was observed on plants that had invaded bermudagrass test plots at the Hammond Research Station in October 1984. The blighted plants were noticed when dew formed on mycelial webs on infected plants. Blight and plant death of prostrate spurge occurred again in the fall of 1985 in turfgrass plots and also on spurge infesting container-grown ornamental plants. These blight outbreaks resulted in death of over 90% of prostrate spurge plants infesting the turfgrass plots and ornamental plant containers. A similar blight was observed also at this time on shoots and leaves of cultivated poinsettia (*E. pulcherrima* Willd. ex Klotzsch) growing in a greenhouse near the turfgrass plots and container yard. A literature search revealed no reports of natural occurrence of a similar blight on prostrate spurge or cultivated poinsettia. However, a report was found of a similar blight on Euphorbiaceae members, including cultivated poinsettia, which was produced by artificial inoculations with *Amphobotrys ricini* (Buchw.) Hennebert (teleomorph *Botryotinia ricini* (Godfrey) Whetzel) (4). We report here the identity of the causal organism and the results of pathogenicity tests. A preliminary report of this work has been published (6).

MATERIALS AND METHODS

Diseased plant materials were collected at the Hammond Research Station and returned to Baton Rouge for examination and pathogen isolations. Difco brand potato-dextrose agar (PDA) was acidified to pH 3.8–4.0 with 50% lactic acid and was used for making fungal isolations, and V-8 juice agar (200 ml of V-8 juice, 3 g of $CaCO_3$, 15 g of agar per 1 L of distilled water) was used to produce spores for inoculation tests. Turbid conidial suspensions were made by washing spores from agar plates with distilled water or distilled water plus 10% orange juice. Spore suspensions were misted on tested plants with an atomizer and inoculated plants were placed in a dew chamber (at 25 C) or in plastic bags for 24 hr. *E. hirta* L. and *E. supina* plants used in inoculation tests were collected in the field, transplanted to soil mix, and grown in the greenhouse. Detached leaves and stems of *E. heterophylla* L. and *E. pulcherrima* were also collected, mist-inoculated with spore suspensions, and held in plastic moisture boxes to observe disease development. Castor bean plants were grown from seed in the greenhouse and planted to the field for flowering. Developing floral parts were enclosed in plastic bags for 24 hr after inoculation.

RESULTS AND DISCUSSION

Early blight infection and death of *E. supina* is shown in Figure 1. Examination of diseased prostrate spurge and poinsettia revealed the presence of white to gray masses of fungal mycelia and conidia that were *Botrytis*-like, except that conidia were globose instead of elliptical. Small, black sclerotia were present on blighted poinsettia stems but were not seen on diseased prostrate spurge. Microscopic examination revealed the fungi on poinsettia and prostrate spurge to be identical. Axenic cultures of the fungus from prostrate spurge were established on PDA and grown in a laboratory without supplemental lighting. These cultures produced small, black sclerotia 1–5 mm long that frequently coalesced. Mycelia were septate (13–14 μm in diameter), white to gray in culture, with spore masses appearing tan to light brown on V-8 agar. Conidia were produced in compact clusters on dichotomously branched conidiophores.

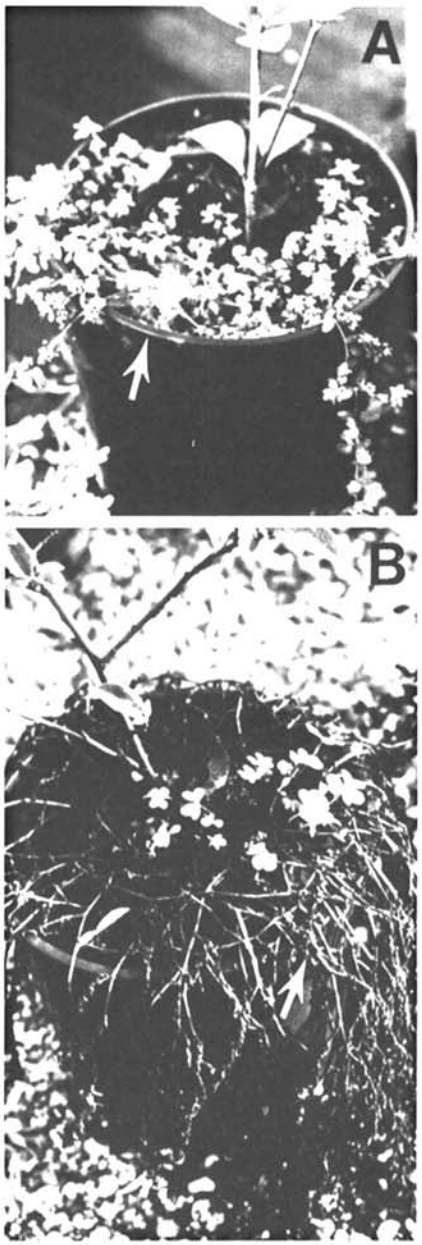

Fig. 1. Ornamental plant containers infested with the weed *Euphorbia supina* (prostrate spurge). **(A)** White mycelial mat (arrow) of the fungus *Amphobotrys ricini* infecting prostrate spurge. **(B)** Remnants of prostrate spurge (arrow) killed by *A. ricini*.

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Conidia were hyaline, globose, and 5.5–11 μm in diameter from diseased plant material, but were slightly smaller (5.5–8.5 μm in diameter) when produced on V-8 agar or PDA. Microconidia were produced on diseased plant material and in agar cultures and were 3.0–4.5 μm in diameter. Conidial production was sparse on PDA but abundant on V-8 agar. Sclerotia were produced more abundantly on PDA than on V-8 agar. The perfect stage of the fungus was not observed on diseased plant parts nor in culture.

Pathogenicity tests with the fungus isolated from prostrate spurge were positive on healthy plants of prostrate spurge and on detached leaves and shoots of cultivated poinsettia. Blight symptoms were often evident 24 hr after inoculations and continued to develop after plants were removed from the dew chamber or plastic bags. Symptoms also appeared within 24 hr on detached leaves and shoots placed in moist chambers. Other plants within the family Euphorbiaceae that also were found susceptible by artificial inoculations were *E. heterophylla* (wild poinsettia) and *E. hirta* (garden spurge). The fungus was also found to cause blight of floral parts and seed capsules of *Ricinus communis* L. (castor bean).

The fungal pathogen that caused blight on *E. supina* and *E. pulcherrima* was identified as *A. ricini*. Conidial dimensions (5.5–11 μm) from diseased plants fit very closely those published for *A. ricini* from castor bean (6–12 μm, mostly 7–10 μm) (3). Dimensions of microconidia (3–4.5 μm) from prostrate spurge and poinsettia were slightly larger than those cited for castor bean (2–3.5 μm). *B. ricini* was first described in 1919 as the cause of a blight of floral parts and seed capsules of castor bean in the southern United States and the teleomorph was named *Sclerotinia ricini* Godfrey (3). The genus name was changed by Whetzel (9) to *Botryotinia*, and Hennebert (5) erected the new genus *Amphobotrys* to accommodate the *Botrytis*-like conidial state of *B. ricini*.

Godfrey (4) observed natural infections by *A. ricini* only on castor bean in the United States and produced disease by artificial inoculations on the following Euphorbiaceae members: *Jatropha* sp., *Manihot utilissima* (cassava), and *E. pulcherrima* (cultivated poinsettia). The fungus has been reported pathogenic on species of *Acalypha*, *Euphorbia* (including *E. pulcherrima*), and *Phyllanthus* from Africa, Bulgaria, India, and South America, and the southern United States (1). This report and that of Whitney and Taber (10) are the first records of the natural occurrence of *A. ricini* on members of the Euphorbiaceae other than castor bean in the United States.

The family Euphorbiaceae contains a number of plant species that are important agricultural weeds. With biological control in mind, we submitted our isolates of *A. ricini* to a company interested in evaluating them for mycoherbicide potential. The results of these tests were negative, based on the failure of *A. ricini* to kill a high enough percentage of the *Euphorbia* species on which it was tested (A. D. Kern, *personal communication*).

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