Etiology and Control of Alternaria Blight of Poinsettia

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ABSTRACT

Blight of poinsettia (Euphorbia pulcherrima) bracts, leaves, and stems was shown to be caused by Alternaria euphorbiaceae comb. nov. Bract lesions, circular to elliptical when small, then expanding to irregular shapes up to 40 mm across with a tan or light brown central area, were surrounded by dark brown zones with purplish black borders. Leaf lesions were irregular, elongate, or angular along veins up to 20 mm across, and dark brown. Lesions on green stems were elliptical, up to 3 × 20 mm, and tan to medium brown with dark borders. These lesions occasionally expanded to girdle stems, resulting in wilting of terminal parts. Ipodione or amitrazine provided excellent control in greenhouse studies.

Additional key words: Euphorbia marginata

Sporadic outbreaks of blight of poinsettia (Euphorbia pulcherrima Willd. ex. Klotzsch) caused by Phytophthora nicotianae B. de Haan var. parasitica (Dast.) Waterhouse or P. drechsleri Tucker have occurred during several Christmas seasons in Hawaii (7). In December 1981, a disease with some similarities to Phytophthora blight occurred on flowering poinsettia cultivar V-14 Glory at a single nursery in central Oahu. This disease reoccurred at the same nursery during late November to early December 1983. In March 1984, the disease was also seen on landscape poinsettia in a windward Oahu garden.

Examination of infected tissue revealed a large-spored, long-beaked species of Alternaria that was readily isolated and cultured. A poinsettia leaf spot associated with an unidentified Alternaria sp. (5) has been recorded, but there is no further information on the disease or the pathogen. A few species of Alternaria attack members of the Euphorbiaceae. These species are A. compacta (Cke.) McClellan (3) and A. ricini (Yoshii) Hansford (2) on castor bean (Ricinus communis L.), Macrosporum euphorbiaceae Barth. on E. marginata (E. Bartholomew, 1908 on specimen packet), and M. euphorbiaceae Rechert on E. prinfoliae (Jack.) J. Muller (4). Because the disease on poinsettia in Hawaii appeared to be new, we undertook this study to establish pathogenicity of the associated fungus, to identify and name the pathogen, and to develop control measures.

MATERIALS AND METHODS
Isolations were made by surface-disinfecting diseased tissue with sodium hypochlorite (0.1%) and plating on water agar. Single-conidium cultures were then established by picking up individual conidia from diseased tissue or developing colonies on agar and transferring to V-8 juice agar (VJA) (100 ml of V-8 juice, 2.0 g of CaCO₃, 17 g of agar, and 900 ml of deionized water). Reisolations after pathogenicity tests were performed in the same manner.

Single-conidium cultures, designated 1090 and 1091, were used throughout the study except when otherwise indicated. Cultures were grown at 28 C under cool-white fluorescent light (2,700 lux) for 4-6 days to allow conidiophore development, then placed in the dark for 24 hr to induce conidial production (1). Conidial suspensions were prepared by flooding these cultures with a 1:2,000 Tween 20 solution and by gently dislodging conidia with a rubber spatula. The spore concentrations were determined with a hemacytometer, then adjusted to 2 × 10⁵ conidia per milliliter. Freshly produced conidia (less than 24 hr old) were used in all studies.

Pathogenicity studies and chemical control studies were conducted on flowering plants of V-14 Glory. Plants were sprayed with conidial suspensions to runoff, placed in plastic bags at 24 C for a 24-hr moisture saturation period for

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initiation of infection, then returned to the greenhouse and maintained at ambient (24–32°C) conditions for 4 days to allow disease development. Observations on disease development and collection of quantitative data were made after this 5-day period.

In vitro tests were used to select chemicals with significant ability to inhibit conidium germination and vegetative growth of the pathogen. Anilazine (Dyrene 50W), bitertanol (Baycor 25W), chlorothalonil (Bravo 75W), iprodione (Rovral 50W), mancozeb (Manzate 200), prochloraz 50W, propiconazol (Tilt 3.6 EC), triadimefon (Bayleton 25W), and vinclozolin (Ronilan 50W) at 1, 10, and 100 g a.i./ml were incorporated into 2% V1A (20 ml V-8 juice and 1 L deionized water).

Anilazine, bitertanol, iprodione, and prochloraz were the most inhibitory to the fungus in culture and were selected for greenhouse testing. Anilazine was applied at 0.3, 0.6, and 1.2 g a.i./L; bitertanol at 0.08, 0.15, and 0.3 g a.i./L; iprodione at 0.15, 0.3, and 0.6 g a.i./L; and prochloraz at 0.15, 0.3, and 0.6 g a.i./L. The fungicides were all suspended in 1:2,000 Tween 20. One week after fungicide application, plants were sprayed with a conidial suspension of isolate 1091, following procedures described earlier. Seven days after inoculation, the spots on four fully expanded leaves per plant were counted for three single-plant pots. The test was repeated once.

RESULTS AND DISCUSSION

The characteristic Alternaria sp. was always associated with the blight, was isolated in pure culture, reproduced the disease after inoculation with a pure culture, and was reisolated. Spots on bracts were initially circular, 0.5 mm in diameter, and purplish black, enlarging to elliptical lesions 2–4 × 4–7 mm and eventually becoming irregular, up to 80 mm across (Fig. 1). The larger lesions were brown with tan centers and purplish black borders, 0.5–3.0 mm wide. Lesions on leaves were dark brown, irregular, up to 20 mm across, and elongate or sharply angular across veins. Lesions on green stems were tan to medium brown with dark borders, elliptical up to 3 × 8 mm, and frequently girdled the stem. Lesions on cyathia were black, circular to elliptical, and initially small (0.5–2 mm) but expanded to rot the inflorescence.

Preliminary observations of spores from infected material revealed striking variation in conidial bead lengths from 30 to 400 μm (Fig. 2). Beaks were pale, olivaceous brown, 3–5 μm wide, and occasionally branched. For conidial measurements of the Alternaria sp., naturally infected bracts were washed, surface-disinfested with 0.1% sodium hypochlorite, and placed on water agar at 24°C under fluorescent light for 24 hr. The resulting colonies were allowed to sporulate at 24°C in darkness. Only dark, mature spores obtained from areas on the bracts with primarily solitary conidia were measured. Conidia were olivaceous brown, obelate to nearly ellipsoidal, solitary or in chains of two to five spores, and had light brown beaks. Means and
standard deviations of 50 conidia were as follows: 55.1 ± 9.1 μm (body length), 89.4 ± 22.0 μm (beak length), 144.6 ± 26.6 μm (total length), and 15.2 ± 1.3 μm (body diameter). The beak diameter measured at the narrowest part, usually near the middle, was 3.4 ± 0.3 μm.

Morphological characteristics of the poinsettia Alternaria fit the published description of M. euphorbiae Barth. very closely but not that of M. euphorbiae Reichert (4), a later homonym. According to Bartholomew, conidia were 40-75 × 12-20 μm with flexuous appendages that were as long or longer than the conidia. Upon examination of the exsiccatum specimens of leaf spots on E. marginata collected by Bartholomew, we concluded that the poinsettia Alternaria and M. euphorbiae Barth. are conspecific. The lectotype specimen, designated by M. Aragaki and J. Y. Uchida (6), fit the description given by Bartholomew, although conidial beaks in syntype specimens were short, usually shorter than the conidial body. Both long-beaked and short-beaked conidial types were present in monoconidial isolates made from poinsettia. In accordance with Wiltshire’s (6) recommendation to discard the genus name Macrosorum as nomina ambigua, Alternaria euphorbiae is proposed as a new combination.

**Alternaria euphorbiae** (Barth.) Aragaki & Uchida (comb. nov.) = **Macrosorum euphorbiae** Barth., *Fungi Columbani* #2633

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<th>Treatment (g a.i./L)</th>
<th>Control</th>
<th>Biteranol</th>
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*Mean number of spots per leaf, based on three single-plant replicates in two tests.