Fusarium Collar Rot and Foliar Blight of *Aglaoe`nema* in Hawaii

J. Y. UCHIDA, Associate Professor, and M. ARAGAKI, Professor, Department of Plant Pathology, University of Hawaii, Honolulu 96822

**ABSTRACT**


A new and destructive collar rot and foliar blight of *Aglaoe`nema commutatum* in Hawaii was caused by *Fusarium subglutinans*. Collar rot was typified by chlorosis, wilting of older leaves, lodging of plants, and eventual plant death. Foliar blight started with elliptical dark leaf spots with chlorotic borders that expanded into petioles and stems. Roots were generally unaffected. *A. c. var. maculatum* was extremely susceptible to infection, whereas cultivars Emerald Beauty and Silver Queen were very tolerant.

Additional keywords: *Fusarium moniliforme*

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*Aglaoe`nema commutatum* Schott, *A. modestum* Schott ex Engl. (Chinese evergreen), and at least eight other species of *Aglaoe`nema* have been grown in Hawaii for decades without major disease problems. These popular, shade-tolerant plants, which are mostly native to the Philippines and tropical Asia, were virtually free of disease in Hawaii before the 1960s. In the 1970s, the rapid expansion of the foliage industry in the United States was accompanied by large, interstate shipments of new species and cultivars of *Aglaoe`nema*, *Syngonium*, and *Pothesis*.

*Aglaoe`nema* species introduced to Hawaii on newly imported aroids, such as *Dieffenbachia* cane rot caused by *Acremonium* and *Erwinia* species (2). Significant losses of *Syngonium* have also occurred due to *Ceratocystis* stem rot (10), *Acremonium* leaf spot (11), and *Xanthomonas* leaf rot (J. Y. Uchida, unpublished).

In 1987, a severe collar rot was discovered on new cultivars of *A. commutatum*. Diseased plants exhibited spotted and blighted leaves, rotted petioles, and dry cane lesions. Typically, plants were completely rotted at the collar, had yellow, wilted leaves, and frequently collapsed (Fig. 1A). Diseased stems were covered with white to cream-colored, crusty masses of *Fusarium* microconidia and macroconidia. Crop losses at commercial nurseries were significant, forcing some growers to abandon production of susceptible *Aglaoe`nema* cultivars.

This report describes the symptomatology and etiology of this new disease on *A. commutatum*. A preliminary report (3) and a popular account (12) have been published.

**MATERIALS AND METHODS**

Isolations and culture. Diseased *Aglaoe`nema* leaves, petioles, and canes were collected from several nurseries on the islands of Oahu and Hawaii. The specimens were washed under running tap water and blotted dry. Tissue sections were taken from the edge of each lesion, dipped briefly in 0.5% sodium hypochlorite, drained on clean tissue paper, plated immediately on 1.7% water agar (WA), and maintained at 24 C. Single hyphal tips were transferred aseptically to 10% vegetable juice agar (VJA) (10) after 2-4 days of growth. Single conidial cultures from 10 isolates representing specimens from four nurseries were established for this study. Two of these monokinetic cultures were deposited at the ATCC as voucher specimens (ATCC 90157 and 90158).

Morphological and pathological studies were conducted with cultures grown on VJA at 24 C under continuous cool-white fluorescent irradiation (approximately 2,700 lx).

Pathogenicity tests. Inoculum comprising microconidia and macroconidia of each isolate was prepared by adding 7 ml of sterile, deionized water to 6-day-old cultures grown on potato-dextrose agar (PDA) (oxygen-humidified atmosphere). The Petri dishes were removed, and spore suspensions were collected by scraping the agar surface with a sterile glass rod. The spore suspension was centrifuged at 5,000 rpm for 5 min at 4 C and washed with sterile deionized water. The spore concentration was adjusted to 10**6** spores/ml and stored at -20 C prior to use.

Plant sections were prepared by placing sections of 4-day-old leaves in the actinomycin D solution (2.5 mg/ml). Sections were flooded with 10 ml of spore suspensions at 10**6** spores/ml. Leaf sections were incubated for 3 days at 25 C.

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old VJA cultures, gently dislodging the conidia with a rubber spatula, combining suspensions from several plates in a beaker, and estimating the concentration with a hemacytometer. Inoculum concentrations were adjusted to 10^7 to 10^8 conidia per milliliter as needed. Pathogenicity tests were conducted with young seedlings or established cuttings of A. c. var. maculatum. Aglaonema cultivars Silver Queen and Emerald Beauty were propagated by stem cuttings. Plants were grown in a 1:1 mixture of Sunshine Blend No. 4 and vermiculite, and fertilized periodically with Osmocote slow-release fertilizer (14-14-14). Five plants (approximately 10–15 cm tall) were used for collar and root drenches, and three to five plants (20–24 cm tall) were used for foliar inoculations.

For foliar inoculations, plants were sprayed to runoff with a spore suspension of each of 10 isolates adjusted to 5 × 10^7 per milliliter, transferred immediately to a humidity chamber for 24 hr, and then maintained in a greenhouse (24–37 C) for 2–5 mo for symptom development. Controls in this and in subsequent tests received water only and were treated similarly.

For collar and root inoculations, 5 × 10^7 conidia were added per 5-cm cells. Inoculum of four isolates (1741, 1743, 1744, and 1745) was added at 5 × 10^7 spores per pot in initial tests and increased to 2 × 10^8 spores per pot in later tests. Water was withheld for 24 hr to retain inocula within pots.

Root inoculations were accomplished by carefully applying a spore suspension (10^7 conidia per pot) of ATCC 90157 near the edge of the pot. Vermiculite was then added to the surface of each pot to reduce subsequent movement of spores to petioles by splashing. Water was withheld as described above, and plants were maintained in the greenhouse.

All pathogenicity tests were repeated at least once.

RESULTS

Isolates. On WA, Fusarium colonies from all diseased specimens developed rapidly. Pure cultures were established on VJA, and Fusarium isolates producing polyphialides were common.

Pathogenicity tests. Foliar spots and blights (Fig. 1B) developed with all isolates following foliar inoculation. Within 3 wk, young leaves initially developed indistinct, generally elliptical, water-soaked areas on the abaxial surface (Fig. 1C). The adaxial surface was slightly chlorotic. As the spots enlarged, dark necrotic centers formed, with water-soaked borders on the abaxial surface and prominent, chlorotic borders on the adaxial surface. On older leaves, incipient lesions were characterized by a yellowing of the abaxial surface, which became olive green. Leaf spots expanded into dark brown or black blighted areas that frequently rotted adjoining petioles and stems. After 4 wk, severely diseased plants had deteriorated, with rotted apical tips and collapsed stems. Internally, diseased stems were light brown to pink with darkened vascular elements.

Although disease developed rapidly on leaves, severity after inoculation was very low. Inoculum levels of 10^7 conidia per milliliter resulted in one to 14 lesions per leaf. Commonly, five or fewer lesions formed on a leaf. Almost no lesions developed when the inoculum level was reduced to 10^6 conidia per milliliter.

Typical collar rot symptoms were reproduced with all 10 isolates, although virulence varied. For six isolates, chlorosis and wilting of older leaves developed 14–21 days following inoculation and collar rot was evident 1 wk later. The disease progressed rapidly thereafter, and severe rot at the stem base of all plants resulted in the plants falling over; 60–100% of the plants were killed in 6 wk. Four isolates that were collected in Oahu were less aggressive. Reproduction of collar rot symptoms was erratic when plants were inoculated with 5 × 10^7 spores per pot, resulting in plant kill of 0–60% in 6 wk. Increasing the inoculum level to 2 × 10^8 spores per pot resulted in 50–60% plant death in 6 wk. In these and other tests, the pathogen was readily reisolated by use of the procedures described previously.

Extensive rot of the root system was not observed. Root lesions and rots (independent of collar rots) occurred infrequently and were restricted in development. Careful inoculations of roots confirmed the low susceptibility of root tissue, since many plants remained healthy. A few plants developed small root lesions at the edge of the pot. Extensive rots developed only in association with collar rots after more than 8 wk.

Pathogen description. Fusarium isolates pathogenic to Aglaonema produced abundant, zero- to three-septate microconidia in false heads without chains. Macroconidia were abundant, nearly straight, and needlelike, with foot-shaped basal cells. Monophialides and polyphialides were present. Chlamydospores were absent.

The cultivars Silver Queen and Emerald Beauty were highly tolerant to F. subglutinans (Wollenweber & Reinking) P.E. Nelson, T.A. Tousson, & Marassas, since no symptoms developed on mature leaves and stems following inoculation. On Silver Queen, small lesions (2–10 mm long) occasionally developed on young, partially expanded leaves and emerging spear leaves. There was very little lesion expansion as leaves matured. Occasionally, larger lesions (10–20 mm) developed on unopened spear leaves of Silver Queen, but the typical large blights and stem rots characteristic of susceptible cultivars were not observed.
DISCUSSION

The *Fusarium* isolates pathogenic to *Aglonema* produced abundant microconidia only in false heads, with abundant, slightly sickle-shaped to nearly straight and needlelike macroconidia, polyphialides, and no chlamydoospores. These characteristics fit the description of *F. subglutinans*, recently elevated to species status (7). P. E. Nelson identified these isolates from *Aglonema* as *F. subglutinans*.

Although *F. subglutinans* has been distinguished from *F. moniliforme* J. Sheld., the reported host range of each is not clear, since most diseases caused by either species were attributed to the latter, and these diseases encompass numerous plant families (4). Both *Fusarium* species have been known in Hawaii for many years, occurring primarily on Poaceae and causing top rot of sugarcane and infecting maize (9). *F. moniliforme*, the causal organism of pineapple fruit rot core rot, was recently identified as *F. m. var. subglutinans* Wollenweber & Reinking (8). Heart rot caused by *F. m. subglutinans* has been recorded on banana (6). This report on *Aglonema* is apparently the first record of an aroid plant as a host of this pathogen.

Association of stem and root rots in *A. commutatum*, *A. simplex* Blume, and other *Aglonema* species with a *Fusarium* species has been reported from Florida (1). In addition, stem and root rots of *A. modestum* have been associated with *F. oxysporum* Schlechtend.; Fr. *F. solani* (Mart.) Sacc., the causal agent of Dieffenbachia stem rot, is reported to be pathogenic on *Aglonema* species but not to Silver Queen (5).

The origin of *F. subglutinans* on *Aglonema* in Hawaii is unknown. Despite the recorded presence of *F. subglutinans* many years ago, the outbreak of collar rot in *Aglonema* has occurred only recently, suggesting that strains pathogenic to *Aglonema* may have been newly introduced to Hawaii, possibly on recently imported novel cultivars of *Aglonema*. The host range of *Fusarium* isolates from *Aglonema* needs to be examined to determine the role of pineapple, sugarcane, maize, and other grasses in the epidemiology of this disease. In addition to cross-inoculations with isolates from sugarcane, maize, and pineapple, the susceptibility of other aroid species needs to be studied.

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LITERATURE CITED