Stem Rot and Wilt of *Exacum affine* Caused by *Nectria haematococca*

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ABSTRACT

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Nectria haematococca caused stem rot, wilt, and collapse of *Exacum affine*. The disease was severe at 27 and 32 C but not at 21 C. Treatment with mancozeb, captan, or chlorothalonil controlled the disease.

In January 1980, we received specimens of *Exacum affine* Balf., a flowering pot plant, with a basal stem rot. *Nectria haematococca* Beck & Br. was fruiting on the necrotic tissue; the imperfect stage, *Fusarium solani* (Mart.) Sacc., was also present. Research was undertaken to prove pathogenicity of the fungus, to determine the effects of temperature on the disease, and to develop control methods.

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E. affine is a relatively recent introduction to the ornamental industry. The only disease previously reported is a blight caused by *Botrytis cinerea* (4).

N. haematococca was first reported as a pathogen of trees (6). Only in 1961 was it found to cause disease of an herbaceous plant (pumpkin) (1). The imperfect stage of the fungus, *F. solani*, has long been known as a pathogen (5).

A disease of passion fruit in Uganda caused by *N. haematococca* was described by Emechebe and Mukiibi in 1976 (2). The vine exhibited a sudden, severe wilt. A collar rot developed, and the roots were also affected. The disease was more severe when wounds were present.

MATERIALS AND METHODS

Original isolates of the fungus were derived from diseased stem sections. Inoculum was a suspension of ascospores and conidia, $1–2.5 \times 10^6$ spores per milliliter, washed from 2- to 3-wk-old cultures of single-conidium isolates on potato-dextrose agar (PDA) plates.

Test plants originated as cuttings from mature, healthy plants. Cuttings were rooted in vermiculite under mist for 30 days and planted in a potting mixture consisting of 50% fine sand and 50% sphagnum peat.

Several inoculation methods were used in the pathogenicity tests. In method 1, a 6-mm incision was made at the base of the stem of each plant and foliage and stems were sprayed with the spore suspension. In method 2, the spore suspension was sprayed on the foliage and stems of unwounded plants. In method 3, the roots were dipped in the spore suspension for 30 sec before potting. Roots of each control plant were dipped in water before planting, the stems were wounded, and the foliage and stems were sprayed with water.

After inoculation, a polyethylene bag was placed over each plant for 15 hr, after which all pots of plants were removed to controlled-temperature rooms maintained at 21 C (range of 20–23 C), 27 C (range of 25–30 C), or 32 C (range of 27–34 C). There were 10 plants (replicates) for each inoculation method at each temperature, five of the cultivar Jill and five of the cultivar Lou.

To determine the effects of fungicides on growth of *N. haematococca* we added benomyl, captan, chlorothalonil, dicloran, mancozeb, or tribasic copper sulfate to the PDA at concentrations of 1,000 and 100 μg of active ingredient per milliliter. The control was unamended PDA. One milliliter of a spore suspension, 1–2.5 × 10^6 spores per milliliter, was evenly dispersed on each plate. There were four replicates per treatment. Plates were incubated at room temperature for 5 days and visually rated into categories of no growth, greatly inhibited growth, slightly inhibited growth, or normal growth (control).

For evaluations of fungicides for disease control, mature Jill plants were sprayed with the spore suspension, and cuttings were taken after the sprays had dried. Each cutting was dipped in a fungicide suspension for 30 sec; control plants were dipped in water. The base of each cutting was dipped in rooting hormone (Hormodin; indolebutyric acid 0.8%), inserted into individual pots containing vermiculite, and placed under intermittent mist for 30 days at 16–21 C. By this time, most plants had rooted and were moved to the controlled-temperature room at 32 C. There were six replicates (plants) per treatment.

Disease severity was based on the number of days that a plant survived after placement in the controlled-temperature room. After plants died, necrotic stem sections were placed on water agar with barley straw for isolation of the fungus. The plates were incubated at room temperature, with fluorescent lighting during the day.

RESULTS

Symptoms. The first observable symptom was wilting, which occurred above a brownish, soft decay of the stem. In less severely affected stems, the vascular system was discolored. When rot occurred near the crown, the entire plant wilted and died. When rot developed at the base of a branch, only that branch might die. Some infected plants lived for several weeks and lost only one branch; in others, the disease progressed downward on the stem until the plant was killed. Decay often began at an injury. Rooted cuttings were more likely to die than older plants, which often survived with the loss of a few branches. Infected, unrooted cuttings wilted and collapsed within 1 or 2 days of inoculation. Occasionally, adventitious roots formed at the first node above the rot, and the plant rerooted and survived.

Nectria haematococca was consistently reisolated from dead inoculated plants. Perithecia formed on rotted stems 2–3 wk after the plant died. Stems of plants left in the pot for several weeks after death often became orange red in color from masses of perithecia. Conidia of the imperfect stage, *F. solani*, developed on isolation plates within a week; perithecia formed in 2–3 wk.

Factors affecting disease development. Disease severity, temperature, and method of inoculation appeared to be related (Table 1). Using longevity of plants as a measure of disease severity, we see that the disease was most severe at 32 C in wounded plants and least severe at 21 C, regardless of inoculation method. All 10 wound-inoculated plants at 32 C died, while all controls lived. At 21 C, only 2 of the 10 wound-inoculated plants died; 1 control plant died, and *Botrytis cinerea* was isolated from it.

Inoculation method also had an effect on disease severity. Plants that were root-inoculated only had significantly less disease than wounded plants, except at 21 C. Roots were not affected by the disease. Plants showing wilt and stem rot had roots that appeared healthy.

An inoculated plant that was symptomless at 21 C sometimes became diseased if moved to a higher temperature. Surviving plants from the pathogenicity test at 21 C were moved to 32 C. All eight wound-inoculated plants died; five of eight unwounded, inoculated plants died; three of nine root-inoculated plants died. All nine controls survived.

Jill and Lou cultivars seemed to be equally susceptible to the disease. In the pathogenicity test, all wound-inoculated plants of both cultivars died at 32 C; four of five and one of five plants of each cultivar died at 27 and 21 C, respectively.

Control. *N. haematococca* did not grow on PDA containing captan, chlorothalonil, or mancozeb at 1,000 or 100 μg/ml. There was no growth on plates containing benomyl at 1,000 μg/ml, and growth was greatly inhibited at 100 μg/ml. Growth of the fungus was unaffected by dicloran and tribasic copper sulfate at either concentration.

Plants treated with captan, chlorothalonil, or mancozeb at 1,000 μg/ml survived longer than the controls, whereas those treated with tribasic copper sulfate at 1,000 μg/ml died sooner than the controls (Table 2). Disease severity in plants treated with benomyl or dicloran was similar to that of the controls.

DISCUSSION

Engelhard et al (3) described a disease of chrysanthemums caused by *F. solani* that was very similar to the stem rot of *Exacum*. Wilting was a major symptom, and roots were not affected. The

Table 1. Effect of time, temperature, and method of inoculation on Nectria stem rot of *Exacum affine*

Inoculation	21 C Days[a] (no. of plants alive)	27 C Days (no. of plants alive)	32 C Days (no. of plants alive)
None (control)[b]	74.4*[c] (9)	76.0* (10)	76.0* (10)
Method 1[d]	70.6* (8)	42.6 (2)	27.7 (0)
Method 2[e]	66.5* (8)	35.5 (1)	44.8 (1)
Method 3[f]	74.5* (9)	66.5* (4)	55.8* (5)

[a] Mean number of days plants survived. Experiment was terminated at 76 days, and plants alive at that time were considered as surviving for 76 days. Data are averages from 10 plants.
[b] Stems wounded, sprayed with water. Roots dipped in water.
[c] * indicates significant difference from method 1 at 32 C ($P = 0.01$, LSD = 19.14 days).
[d] Stems wounded, tops sprayed with spore suspension.
[e] No wounds, tops sprayed with spore suspension.
[f] No wounds, roots dipped in spore suspension.

Table 2. Effect of fungicide dips on Nectria stem rot of *Exacum affine*

Fungicide	Rate (μg/ml)	Plant survival[a] (mean no. of days)
Benomyl	100	57.3
	1,000	52.8
Captan	100	67.5
	1,000	74.0*[b]
Chlorothalonil	100	64.5
	1,000	74.0*
Dicloran	100	45.3
	1,000	65.7
Mancozeb	100	61.7
	1,000	74.0*
Tribasic copper sulfate	100	30.7
	1,000	25.0*
None (control)		48.0

[a] Experiment was terminated at 74 days; plants alive at this time were considered as surviving for 74 days. Data are averages from six plants.
[b] * indicates significant difference from control ($P = 0.01$, LSD = 21.25 days).

imperfect and perfect stages of the fungus were present. We inoculated Butterball chrysanthemums with the *Exacum* isolate of *N. haematococca*, but no disease resulted.

Although there were no significant differences in disease severity between wounded and unwounded plants in the pathogenicity test, other evidence indicated that injury might be important in stem rot of *Exacum*. Plants were repotted at the time of inoculation, which could have caused unintended injuries. In addition, a severe epiphytotic occurred in our greenhouse soon after some large plants were pruned back.

The fungal growth tests indicated that captan, chlorothalonil, mancozeb, and perhaps benomyl might be effective in controlling the disease, and good control was obtained with all except benomyl. The increase in disease severity in plants treated with tribasic copper sulfate may have been caused by a reduction in *Nectria* antagonists.

LITERATURE CITED

1. Dingley, J. M. 1961. New records of fungous diseases in New Zealand, 1960-1. N.Z. J. Agric. Res. 4:336-347.
2. Emechebe, A. M., and Mukiibi, J. 1976. Nectria collar and root rot of passion fruit in Uganda. Plant Dis. Rep. 60:227-231.
3. Engelhard, A. W., Crane, G. L., and Mellinger, H. C. 1976. Stem rot, a new disease on chrysanthemum incited by *Fusarium solani*. Plant Dis. Rep. 60:437-441.
4. Ploetz, R. C., and Engelhard, A. W. 1979. The Botrytis blight of *Exacum affine*. Proc. Fla. State Hortic. Soc. 92:353-355.
5. Snyder, W. C. 1934. Notes on Fusaria of the section Martiella. Zentralbl. Bakteriol. Parasitenk. Infektionkr. Hyg. Abt. 2:163-184.
6. van der Goot, P. 1935. Diseases and pests of cultivated crops in the Dutch East Indies in 1933. Meded. Inst. Plziekt. Buitenz. 84, vii. 97 pp.