Stem Rot and Shattering of Easter Cactus Caused by *Drechslera cactivora*

A. R. CHASE, Assistant Professor of Plant Pathology, IFAS, University of Florida, Agricultural Research Center, Apopka, FL 32703

ABSTRACT

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Drechslera cactivora (*Helminthosporium cactivorum*) was isolated consistently from diseased Easter cactus showing symptoms of stem rot and cladophyll abscission (shattering). Inoculations with conidial suspensions of *D. cactivora* on artificially wounded or unwounded Easter cactus resulted in disease symptoms. Cellfree extracts of the pathogen also caused stem rot and shattering of injected Easter cactus. The related Thanksgiving (also called Christmas) cactus also was susceptible to *D. cactivora* although symptoms occurred infrequently. Isolates of the pathogen from *Cereus* sp. were pathogenic on Easter cactus.

Easter cactus (*Rhipsalidopsis gaertneri* (Regel) Moran) is a popular flowering plant sold during the spring holidays. Diseases of this plant may be the same as those of the related Thanksgiving (also called Christmas) cactus [*Schlumbergera truncata* (Haw.) Moran (= *Zygocactus truncactus*)] (3,4), but no data have been found to support this belief.

During the 1980-1981 winter and spring, Easter cactus with severe symptoms of stem rot and cladophyll abscission (Fig. 1) was collected from a nursery. Symptoms included water-soaked, black, mushy, irregularly shaped lesions above or below the soil line. Sporulation in lesions was profuse and gave lesions a furry appearance. Frequently, lesions as small as 2 mm were accompanied by abscission of the upper cladophylls, creating a condition that the grower referred to as "shattering." At the same time, Christmas cacti in the same nursery were found infected with *Fusarium oxysporum* Schlecht. (3,4) and showed the dry, tan lesions characteristic of that disease.

The possibility that *F. oxysporum* was involved in the Easter cactus disease was investigated. This research was conducted to determine the causal agent of Easter cactus stem rot and shattering, to investigate its host range, and to compare it with Fusarium stem rot of Christmas cactus.

MATERIALS AND METHODS

Organisms were isolated by using stem pieces from diseased Easter cactus, surface-disinfested in 0.52% sodium hypochlorite for 3 min, rinsed in sterile deionized water (SDW), and plated on potato-dextrose agar (infusion from 250 g of boiled potatoes, 20 g of agar, and 20 g of dextrose per liter), potato-dextrose agar amended with 100 μg/ml streptomycin sulfate, or V-8 juice agar medium (18% V-8 juice cleared with 4.5 g of $CaCO_3$ and 15 g of agar per liter). Culture plates were incubated at 24–26 C in approximately 2,100 lux fluorescent light (8 hr/day) for 5–7 days. These methods were used also to obtain isolates of fungi from Christmas cactus and *Cereus* sp.

Pathogenicity trials were performed with 10-cm-tall Easter cacti rooted from cladophyll sections in steam-sterilized potting medium consisting of Canadian peat, cypress shavings, and pine bark (2:1:1 by volume) amended with 6 kg of Osmocote (14:14:14 slow release fertilizer), 4 kg of dolomite, and 1 kg of Perk (micronutrient source, Estech Corporation, Chicago, IL) per cubic meter of medium.

Inoculum was prepared from single conidium isolates of the suspect pathogen grown on V-8 agar at 24–26 C in approximately 2,100 lux fluorescent light (8 hr/day) for 7–14 days. Conidial suspensions were adjusted to 1×10^4 conidia per milliliter. Ten Easter cacti each were inoculated with 1 ml of the conidial suspension by pipetting it onto a wound made with a sterile dissecting needle in the center of the basal cladophyll. Ten other plants were treated similarly but with SDW in place of the conidial suspension.

All plants were grown on raised benches in a glasshouse receiving approximately 10,700 lux of light, and symptoms were recorded twice each week for 2 wk. Organisms were reisolated by using the method described. The experiment was performed three times. A similar experiment was performed once by the same methods except that plants were not wounded before inoculation.

The pathogenicity of three isolates of the suspect pathogen (one from Easter cactus and two from *Cereus* sp.) and of *F. oxysporum* from Christmas cactus was tested on Easter and Christmas cacti by wound inoculation. In this case, five plants were each inoculated with one of the organisms or SDW. Each experiment was performed three or four times.

The host range of the suspect pathogen was tested by using the isolate from the Easter cactus. The plants tested were *Crassula argentea* Thunb. (jade plant), *Hylocereus* sp. (A. Berger) Britt & Rose, Easter cactus, *Sedum* sp. L. 'Weinbergii' (ghost plant), *Sedum morganianum* Walth. (burro's tail), and *Senecio herrianus* Dinter (string of pearls). Plants appearing healthy were obtained from growers or produced as described. Five plants of each species were wound-inoculated with either the conidial suspension or SDW. This test was

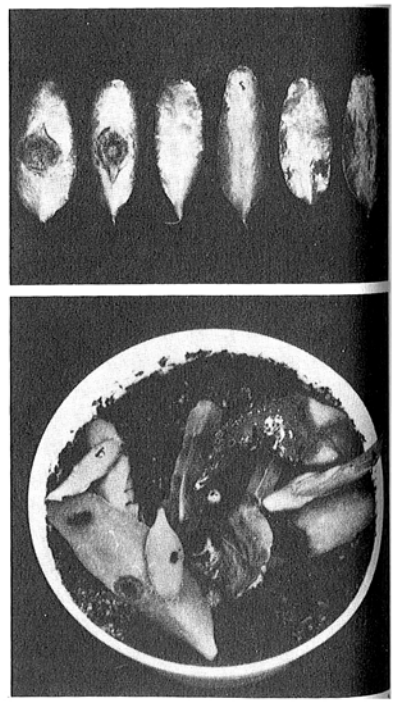

Fig. 1. Easter cactus (*Rhipsalidopsis gaertneri*) infected with *Drechslera cactivora*. **Top:** Cladophylls inoculated with mycelium (left two), cladophylls injected with cellfree extract from liquid culture of the pathogen (middle two), and cladophylls injected with sterile culture broth (right two). **Bottom:** Plant naturally infected with *D. cactivora*.

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repeated once for each plant species that appeared susceptible in the initial test.

Results from preliminary pathogenicity tests suggested a toxic action in advance of fungal hyphae during pathogenesis. Therefore, cellfree extracts of the three isolates mentioned earlier were produced by the method described by Van Etten and Daly (6). Fries medium (6) (50 ml/125-ml Erlenmeyer flask) was inoculated with mycelium of each isolate and grown on a shaker at room temperature for approximately 10 days. Then, excess broth was decanted and discarded, and the mycelial mat was ground in a Waring Blendor at high speed for 15–30 sec. The slurry was centrifuged at 5,000 rpm for 15 min and the supernatant decanted and filtered using disposable 0.2-μm filters (Acrodisc, Gelman). Three to five Easter cacti were injected with 0.1 ml of a culture extract or sterile, uninoculated broth using a sterile, disposable, 1-ml syringe (26 G 3/8). A similar number of plants was inoculated with mycelium (0.2 mm^2) of each isolate from the culture flask into a wound made with a sterile syringe. Plants were placed in a glasshouse for observation, and isolations were made after 10 days. This test was performed three times.

RESULTS

Drechslera cactivora (Petrak) M. B. Ellis (=*Helminthosporium cactivorum*) (2) was isolated consistently from Easter cactus with stem rot and shattering. Identification of the pathogen was confirmed by E. S. Luttrell of the University of Georgia. At no time was *F. oxysporum* isolated from Easter cactus with these symptoms, although occasionally it was isolated from Easter cactus with small (2-mm) tan lesions.

Easter cactus stem rot and shattering were reproduced when plants were inoculated with isolates of *D. cactivora* (Fig. 1). Plants that were wounded showed stem lesions (5–10 mm diameter) 4–7 days after inoculation, and cladophyll abscission occurred within 2 days of the appearance of the stem lesions. Unwounded Easter cacti inoculated with the pathogen also developed symptoms of the disease within 14 days. Some inoculated Easter cacti shattered before any stem lesion was noted above the soil line, but examination of the cladophyll base below revealed small (2–5 mm diameter) lesions. *D. cactivora* was reisolated from all stem lesions but not from any healthy, uninoculated plants.

Table 1. Effect of inoculation with mycelium or injection with a cellfree extract of three isolates of *Drechslera cactivora* or sterile culture broth on shattering of Easter cactus

Inoculum	Mycelium (M) Extract (E)	No. of plants shattered/total tested		
		Test 1	Test 2	Test 3
Isolate 135[a]	M	3/3	5/5	5/5
Isolate 135	E	0/3	0/5	0/5
Isolate 81-24[b]	M	3/3	5/5	5/5
Isolate 81-24	E	2/3	3/5	3/5
Isolate 81-70[a]	M	3/3	5/5	5/5
Isolate 81-70	E	2/3	3/5	1/5
Sterile broth		0/3	0/5	0/5

[a] Isolated from *Cereus* sp.
[b] Isolated from Easter cactus.

Inoculations of Easter cactus with *D. cactivora* and of Christmas cactus with *F. oxysporum* were the only two combinations consistently resulting in disease. *D. cactivora* from Easter cactus incited disease on Christmas cactus in only one of four tests, and *F. oxysporum* did not cause disease in Easter cactus in any test. Uninoculated plants did not develop stem rot, and no organisms were isolated from them. Inoculations of Easter and Christmas cacti with other isolates of *D. cactivora* gave similar results.

Cellfree extracts of two *D. cactivora* isolates incited stem lesions and shattering of Easter cactus (Table 1). One of the three isolates (135 from *Cereus* sp.) appeared to lack this ability or did not produce enough of the toxic substance to induce symptoms. Stem rot occurred within 1–4 days in plants inoculated with mycelium of the fungus but required 5–14 days in the plants injected with either extract. The pathogen was reisolated from plants inoculated with mycelium, and no contaminants were recovered from other treatments.

DISCUSSION

These tests demonstrate that Easter cactus stem rot and shattering is caused by *D. cactivora*. This organism causes stem rot of many cactus species (1,5), but this is the first report of Easter cactus as a host. It is also the first suggestion of the production of a toxic substance capable of inciting symptoms similar to those caused by conidia or mycelium of *D. cactivora*. Stem lesions were soft and water-soaked, and inoculation of a pectate base medium with either mycelium or the extract resulted in the formation of small depressions in the medium, indicating that the fungus produces pectolytic enzymes (unreported experiment). This may explain the rapid development of symptoms including the shattered condition (Fig. 1).

Although *D. cactivora* incited disease of Christmas cactus, it did so in only one test, and the disease has not been seen under natural conditions. In addition, *F. oxysporum*, a common pathogen of Christmas cactus, did not cause stem rot of Easter cactus in these tests. Both diseases are stem rots, and although lesion color and appearance differ greatly, diagnosis by growers has led to the unsuccessful use of benomyl for control of this disease. At present, the recommended control measures include discarding infected plants, using pathogen-free soil and pots, and isolating susceptible plants from one another. Chlorothalonil has been successful in controlling this disease but is not registered for use on this plant in Florida.

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