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# Waterhyacinth Decline in Texas Caused by *Cercospora piaropi*

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## ABSTRACT

In June 1982, several disease foci were observed in a large mat of waterhyacinth in Lake Conroe, Texas. The disease foci were 10 to 20 m in diameter and occurred within the same general area of the 202 ha (500-acre) mat. Symptoms on the leaves were yellowing, with numerous small, sunken, brown lesions which coalesced. By October 1982, the disease was evident over the entire 202 ha and was most probably responsible for an early decline of the mat. A *Cercospora* sp. was isolated from leaf lesions and caused similar disease symptoms when inoculated onto healthy waterhyacinths. Based on symptomology, cultural and morphological characteristics, it was identified as *Cercospora piaropi* Tharp. This is believed to be the first report of this species causing large-scale, severe damage on waterhyacinths and a significant reduction in plant density.


*Key words:* biological control, remote sensing, infrared photography, *Eichhornia crassipes, Cercospora piaropi,* waterhyacinth, fungi, pathogen.

## INTRODUCTION

The floating waterhyacinth, *Eichhornia crassipes* (Mart.) Solms, is generally believed to have been introduced into the United States from South America in 1884 (5, 10). Since then its spread and colonization throughout the southern and western United States have been rapid. Lack of effective controls over the past century have demonstrated the extreme tenacity of this weed. Microbiological control agents, as well as insects, coupled with proper environmental conditions, offer some promise in controlling this pestiferous plant.

Although numerous fungi have been reported to cause disease on waterhyacinths (13), most do not cause sufficient damage to retard the plant's rapid growth. One such fungus is *Cercospora piaropi* Tharp, first isolated from diseased waterhyacinths in Palestine, Texas in 1914 (11). The pathogenicity of *C. piaropi* to waterhyacinth is well documented, but most reports indicate that it causes only minor to moderate damage to the plant (4, 9). Consequently, *C. piaropi,* has not been seriously considered as a biocontrol agent.

In June 1982, a natural epiphytotic of waterhyacinth was observed on Lake Conroe, in east Texas, that later spread to over 200 ha of waterhyacinth and was most likely responsible for an early decline of the mat. The purpose of this paper is to report *C. piaropi* can cause a severe disease on waterhyacinth which ultimately may reduce the plant population. An abstract of this report was published previously (6).

## MATERIALS AND METHODS

*Description of Disease.* Lake Conroe is a 8,100 ha (20,000 acre) manmade reservoir located near Conroe, Texas, approximately 72 km (45 miles) north of Houston.

Waterhyacinths have been a concern on the lake, primarily in the northern section, since it was filled in 1973. During an aerial photographic survey of the aquatic weeds in June 1982, several disease foci were observed in a large mat of waterhyacinth. The disease foci were 10 to 20 m in diameter and all occurred within the same general area of the mat (Figure 1a).

Symptoms on the plants were chlorosis and numerous small (1 to 3mm dia), sunken, tan to dark brown lesions on the leaves and petioles that eventually coalesced (Figure 2a). Water-soaking and rot of the bulbous petioles and leaftip dieback were common.

*Isolation of the Pathogen.* Symptomatic plants were collected on 1 July 1982 and transported to the laboratory in College Station. Leaves were excised from the petioles, surface disinfested in 5% Chlorox (0.26% sodium hypochlorite) for 5 minutes and then rinsed in sterile distilled water for 5 minutes. Explants (3 $mm^2$) were taken, transferred to potato dextrose agar (PDA, Difco) plates, and incubated at 25 C under fluorescent lights.

Pathogenicity of the isolates recovered was determined by inoculating healthy waterhyacinth leaves with a 0.75% water agar mycelia-conidia slurry as described by Martyn and Freeman (7). Inoculated and uninoculated control plants were maintained in 37.85 L (10 gal) glass aquaria half-filled with distilled water and fitted with plastic covers to maintain relative humidity near saturation. The water was aerated with air stones to prevent eutrophication. All plants were maintained in the greenhouse (24 to 35 C) and monitored 4 weeks for symptom development.

## RESULTS AND DISCUSSION

By October 1982, the disease was evident over the entire 200 ha mat of waterhyacinth and the entire mat appeared brown (Figure 1b). At this time numerous plants were in an advanced state of decline with dead leaves. Some areas of the mat were beginning to sink. By the end of November (prior to the first frost), the mat was beginning to break up and there were areas of open water. In spite of a mild winter, waterhyacinths did not return *en masse* the following spring and summer as in past years.

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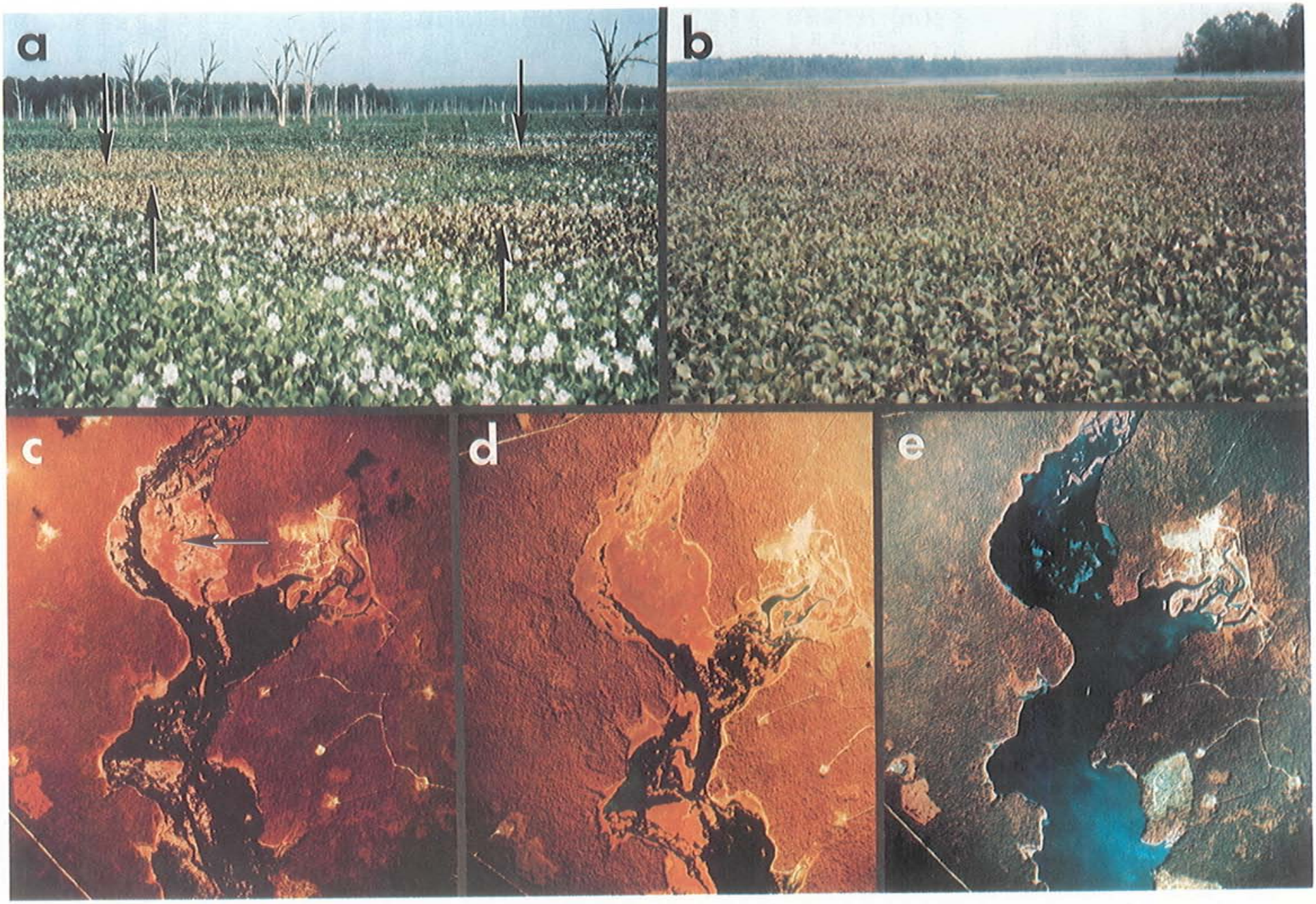


Figure 1. (a) Ektachrome photograph from ground level taken in July 1982 showing the yellow disease foci of *Cercospora piaropi* occuring within the waterhyacinth mat (arrows). Each focus is 10-20 m in diameter. (b) A portion of the same waterhyacinth mat as it appeared in October 1982. The entire mat has a burned or dried out appearance indicative of the severity of the disease. (c,d and e) Kodak Aerochrome infrared photographs of the northern end of Lake Conroe taken from 3.7 km (12,000 ft; 1:24,000 scale) showing the entire 200 ha waterhyacinth infestation. Image c was taken in June 1982 and the pink reflectance area (arrow) is indicative of healthy, green waterhyacinths. Image d was taken in October 1982 and the mustard-brown reflectance is indicative of the burned or dried out appearance depicted in Figure 1b. Image e was taken the following summer (June 1983) and shows a distinct lack of vegetation.



Figure 2. (a) Disease symptoms of *Cercospora piaropi* infection on waterhyacinth showing the small, discrete, circular leaf spots which coalesce and the associated tip die-back. (b) Photomicrograph of *C. piaropi* conidia (arrow) isolated from diseased waterhyacinths on Lake Conroe. Scale bar = 50 μm.

Color infrared photographic data documented the rapid spread of the pathogen. Figures 1 c-e are color infrared, time-sequenced photographs of the northern end of Lake Conroe taken from 3.7 km (12,000 ft) and depict the large area of waterhyacinth infestation. Figure 1c was taken in June 1982 and the pink reflectance represents healthy, green waterhyacinths.

By October 1982 (Figure 1d), the disease was widespread and the waterhyacinth mat appeared a mustard-brown color with color infrared film. Figure 1e was taken during the following summer (July 1983) and specifically shows the lack of any significant waterhyacinth infestation. A few plants are detectable as a pink fringe along the shore line.

Isolations from diseased waterhyacinth leaves yielded a *Cercospora* sp. (Figure 2a-b) that produced a distinct purple pigment on PDA. Leaf spot symptoms similar to those observed initially were reproduced on healthy waterhyacinth leaves 21 days postinoculation with the *Cercospora* sp.

Two species of *Cercospora* are reported to cause leaf spot diseases of waterhyacinth (3, 11). The first species, *C. piaropi,* has been reported from several locations (4, 11, 12) and, as indicated previously, generally does only minor damage to the plant (4, 9). A second species, *C. rodmanii* Conway, was isolated from declining waterhyacinth plants in Florida in 1973 (3). Primary differences from *C. piaropi* are longer conidial lengths (172 μm vs. 95 μm), an *Asteromella* pycnidial state, a well developed stromata, and a more rapid spread and subsequent severe disease (3). The Lake Conroe isolate was identified as *C. piaropi* based on the following: (a) conidia lengths averaged 138 μm, (b) the absence of an well developed stromata, (c) the absence of *Asteromella* pycnial state, and (d) a diffusable purple pigment, as opposed to the red pigment of *C. rodmanii,* was produced in both potato dextrose and V-8 juice agar culture.

Other factors which most probably contributed to the overall decline in waterhyacinths were the presence of waterhyacinth weevils (*Neochetina eichhorniae*) and the introduction of grass carp (*Ctenopharyngodon idella*) during 1982 for hydrilla control. Disease severity of both *C. rodmanii* and *Acremonium zonatum* on waterhyacinth has been shown to increase in association with *Neochetina* spp. feeding (1, 2). Examination of the waterhyacinths in Lake Conroe revealed some *Neochetina* feeding sites, but overall damage caused by the insect was minimal.

The impact of the grass carp was most likely on the regrowth of waterhyacinth the following season. At the time this disease was observed (June 1982) there were over 3,600 ha of hydrilla and other submerged weed species in Lake Conroe (8). Grass carp feeding would most probably be limited to these submerged species and not the waterhyacinth. By the following year; however, the grass carp had consumed virtually all the submerged weeds. At this time, the fish would be more likely to graze any newly formed waterhyacinth plants along the shoreline. This has probably been the primary reason for the lack of any substantial reinfestation of waterhyacinth on Lake Conroe.

*Cercospora piaropi* and *C. rodmanii* appear to be closely related, both in morphology and disease symptomology. The present data suggest that *C. piaropi* also can be highly aggressive towards waterhyacinth under certain conditions and should not be ruled out as a possible biological control agent.

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