Brown Leaf Spot of Pearl Millet Caused by *Bipolaris urochloae* in Zimbabwe

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ABSTRACT

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A leaf spot disease of pearl millet (*Pennisetum glaucum*), characterized by the presence of extremely variable symptoms ranging from small dots or flecks to large, rectangular or spindle-shaped spots, was caused by *Bipolaris urochloae*. The spots have gray centers with brown margins. The disease, though not threatening at present, was observed at several locations in Zimbabwe during 1985–87. Limited observations suggest that the disease is potentially important. This appears to be the first report of this pathogen attacking pearl millet.

Pearl millet (*Pennisetum glaucum* [L.] R. Br.) is grown on considerable area in southern Africa, particularly in Tanzania and Zimbabwe. In Zimbabwe, the crop is grown mainly on rain-fed communal farm lands that are generally poor in soil fertility. Little research is reported on this crop in southern Africa. Surveys conducted during 1985–86 and 1986–87 on this crop in the region revealed a leaf spot disease at many locations on several genotypes grown in Zimbabwe and Zambia (W. A. J. de Milliano, *unpublished*).

Disease symptoms were characterized by the presence of leaf spots that were extremely variable in shape and size. Spots developed as small dots, brown flecks, fine streaks, small oval to large rectangular or spindle-shaped lesions measuring 2–15 mm in width and 2–25 mm in length. The spots had brown margins with gray centers. Under humid conditions, spots coalesced to cover large areas of the leaf and sheath, and the centers of the leaf spots were covered with a dense, grayish growth of conidia and conidiophores of the pathogen. At later stages, the affected tissues died. Severely infected plants failed to produce heads.

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MATERIALS AND METHODS

Isolation and identification of the pathogen. Leaves from the infected pearl millet plants collected from the Aisleby farm (near Bulawayo) and the Henderson Research Station (near Harare) in Zimbabwe were excised from the interface of the healthy and diseased tissues. The leaf pieces (3 mm × 3 mm) were surface-disinfected in 0.1% of $HgCl_2$ for 1 min, followed by several rinses in sterile, distilled water, transferred to freshly prepared potato-dextrose agar (PDA) in petri plates, and incubated at 20–25 C for 3 days. Stock cultures of the pathogen were grown on slants of PDA in test tubes and kept at 20–25 C. Conidia and conidiophores, obtained from the growth on infected leaves, were measured and used for taxonomic comparisons.

Pathogenicity test. Thirty-day-old plants of a susceptible landrace of pearl millet, IP 2696, grown in 30-cm-diameter plastic pots, were spray-inoculated. Inoculation was done with conidia (1×10^3 conidia per millileter) obtained from plates of PDA and suspended in sterile, distilled water. Each plant was inoculated with 50 ml of inoculum. Inoculated plants were kept in a humid chamber (RH 100%, 20–25 C) for 48 hr and then in a polyethylene house. Plants sprayed with water served as controls. The experiment was repeated twice with five to six plants each time.

RESULTS AND DISCUSSION

Isolation and identification of the pathogen. The organism isolated most frequently from both locations produced grayish mycelium on PDA. Sporulation occurred in 10–12 days at 20–25 C. Conidia were produced laterally on conidiophores and measured 12–18 μm × 30–105 μm. In some cases, a conidium was produced at the tip of the conidiophore and, just beneath the conidium, the conidiophore produced two branches that again bore conidia laterally. Conidia were septate (2–9 septa), brown, straight or curved, spindle-shaped, and occasionally branched. They were round at the tips, had a basal hilum and, upon germination, produced one to three germ tubes from the end cells. Conidiophores were single or branched, measured 12–15 μm × 30–110 μm, olivaceous, septate (2–5 septa), and emerged from stomata in groups of four to six. The fungus was identified as *Bipolaris urochloae* (Putterill) Shoemaker by the CAB Commonwealth Mycological Institute, Kew, England (Herb. IMI 317062). The pathogen has been described earlier (4).

Pathogenicity test. Inoculated plants developed symptoms typical of those observed in the field within 8 days after inoculation. Symptoms on younger and older leaves appeared simultaneously. Spots increased in size with time, and within 15 days of inoculation, younger leaves were fully covered with spots and died. Infection on older leaves, however, was less severe. When kept under a humid environment, a grayish fungal growth developed in the centers of the spots. Reisolations from these spots yielded *B. urochloae*.

This appears to be the first report of leaf spots caused by *B. urochloae* on pearl millet. Two related pathogens, *B. setariae* (Sawada) Shoemaker and *Drechslera setariae* (Sawada) Subramanian & P. C. Jain, have been reported on pearl millet in India (1,2), but their symptomatology and site of infection differ from those of *B. urochloae*. *B. setariae* produces brown eliptical spots with dark brown centers often surrounded by a pale yellow halo (2). *D. setariae*, on the other hand, infects only areas of the leaves infected by downy mildew (caused by *Sclerospora graminicola* [Sacc.] J. Schröt.) (1). The lesions have dark margins and ash-colored centers, vary in size, and sometimes

blight the leaves completely. In addition, three species of *Helminthosporium* have also been reported on pearl millet. *B. australiensis* (M. B. Ellis) Tsuda & Ueyama (syn. *H. australiensis* Bugnicourt) causes leaf spots in India (3), and *Exserohilum rostratum* (Drechs.) Leonard & Suggs (syn. *H. rostratum* Drechs.) causes severe leaf spotting and seedling death in the United States (6). *B. setariae* (syn. *H. setariae* Sawada) is reported to cause seed decay, seedling blight, and head molds, in addition to leaf spotting (5,6).

The severity of this disease appears to be greatly influenced by environmental factors. Under humid conditions during intermittent rains and at temperatures in the range of 20–25 C, the disease appears to spread rapidly. On the other hand, in dry, hot weather, the disease was restricted (lesions were fewer and smaller), although lesions expanded again with the increase in humidity during or after rains. Currently, the disease is not widely prevalent or severe probably because of the cultivation of resistant cultivars. However, the failure of a landrace (IP 2696) to produce panicles after severe natural infection at the Henderson Research Station in Harare clearly indicates that the disease is potentially important. Therefore, further surveys regarding distribution and severity of the disease are needed.

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