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Red Leaf Blotch of Soybeans

Red leaf blotch, or Pyrenochaeta leaf blotch, of soybeans (*Glycine max* (L.) Merr.) is caused by the fungus *Pyrenochaeta glycines* Stewart (= *Dactuliophora glycines* Leakey) (1,14). The incidence of the disease has increased concomitantly with increased production of soybeans in southern Africa, particularly in Zambia and Zimbabwe. In 1985, soybean production was approximately 26,000 t on 15,000 ha in Zambia and 84,000 t on 42,000 ha in Zimbabwe.

P. glycines was first described in 1957 by Stewart (14), and the sclerotial state, *D. glycines*, was described in 1964 by Leakey (7). Datnoff et al (1) showed that *D. glycines* is the sclerotial state of *P. glycines*. Schneider (12), in a review of the genus *Pyrenochaeta*, thought that the fungus should be placed in the genus *Phoma*. Stewart (14) reported that the fungus caused severe leaf blotching and up to 75% defoliation of soybeans grown at the Jimma Agriculture Research Station in Ethiopia. The disease has since been reported in countries from the central and eastern to the southern regions of Africa, including Cameroon, Malawi, Rwanda, Uganda, Zaire, Zambia, and Zimbabwe (5,7,8,13). The disease and its causal fungus have been reported on only one other host, *Neonotonia wightii* (Arnott) Lackey (6), a perennial relative of soybeans, in Ethiopia, Zambia, and Zimbabwe (1,7,11,14). The only known record of *P. glycines* on soybeans outside Africa is among mycological leaf collections from Bolivia made by Waller in 1982, now at the Commonwealth Mycological Institute Herbarium in Kew, Surrey, England.

In the mid-1970s, red leaf blotch was reported as a potentially serious disease of soybeans in Zambia when severe defoliation was recorded in field plots (5). In 1977, an estimated 50% reduction in yield caused by the disease was reported from Zambia (13), and in 1984, estimated yield losses there ranged from 7 to 37% in the medium- to late-maturing cultivars (2). In 1985, on the basis of fungicide trials, we estimated a 34% yield reduction over approximately 25% of the growing area of Zambia. In Zimbabwe in 1982, we found red leaf blotch throughout the soybean-growing area, with estimated yield losses ranging from 10 to 50% in Harare and Mashonaland provinces.

Research on red leaf blotch began in Zambia and Zimbabwe during the late 1970s. Studies have concentrated on the biology of the pathogen, disease epidemiology, germ plasm evaluation for disease resistance, quantifying yield losses, and testing chemical and cultural methods of control. We wish to acquaint plant pathologists and other agriculturists with this increasingly important disease of soybeans.

Symptomatology

P. glycines causes lesions on the foliage, petioles, pods, and stems of soybeans throughout the growing season (November to April) in Zambia and Zimbabwe. Lesions are often associated with the primary leaf veins. Initially, dark red to brown, circular to angular lesions 1–3 mm in diameter appear on the unifoliolate leaves. Soon after the trifoliolate leaves are fully expanded, dark red spots develop on the upper surface and reddish brown spots with dark borders develop on the lower surface (Fig. 1A–C). During periods of high humidity, a diffuse mycelial growth may surround the lesions. The lesions enlarge and may coalesce to form irregular blotches 3–10 mm in diameter with buff-colored centers and dark margins (Fig. 1D). Older blotches may be surrounded by chlorotic halos and cover over 50% of the leaf surface. The lesions merge to form large necrotic blotches up to 2 cm in diameter. Necrotic tissue frequently drops out, giving a shot-hole appearance to severely infected plants. Diseased plants defoliate prematurely and senesce 5–10 days before normal

maturation, and this early senescence probably contributes to yield losses. Lesions on the main stem, petioles, and pods are ovoid, 1 to 5 mm, and mauve to reddish purple (Fig. 2) and appear below the uppermost leaf with symptoms.

During lesion enlargement, sclerotia develop primarily on the lower leaf surface but occasionally on the upper leaf surface. Pycnidia are formed chiefly within the blotches on the upper leaf surface, although some form on the lower surface.

Lesions on the leaves of the perennial host, *N. wightii*, are larger but not as distinct as those on soybean leaves (Fig. 3) and usually form later. The lesions are 5–10 mm in diameter, circular to irregular, and associated with or between the primary leaf veins. The lesions coalesce to form brown blotches up to 4 cm in diameter that may be surrounded by chlorotic halos. Infected leaves often become ragged, but defoliation does not occur as on soybeans (14). Pycnidia frequently develop in lesions on the upper leaf surface and may also occur on the lower surface. Sclerotia are less abundant than on soybeans and generally are scattered on the lower leaf surface, although occasionally forming on the upper surface.

The Pathogen

Stewart (14) described *P. glycines* by morphological characteristics, including the dimensions and shapes of conidia, pycnidia, and pycnidial setae. Leakey (7) described the sclerotia of *D. glycines* as dark brown with bristles externally and colorless and undifferentiated internally.

Sclerotia collected from leaf surfaces or screened from soil will germinate in 1–3 days on culture media, in a drop of water, or on moistened filter paper. The sclerotia germinate by directly producing hyphae, which later produce pycnidia, and also may produce pycnidia on their surfaces (Fig. 4).

The fungus produces pycnidia and can be cultured on common nutrient media such as cornmeal and malt agars. Isolates differ in colony pigmentation, which often varies according to the medium used (Fig. 5). On nutrient-deficient medium, such as water agar, mycelial growth is sparse and pycnidia production restricted. In observations of the fungus in culture (1), the pycnidia formed on mycelia were 101–284 μm long and 92–224 μm wide; those on sclerotia were 183–298 μm long and 133–247 μm wide. Pycnidia were brown to black with stiff, erect, dark brown setae scattered over the surface or clustered about the ostioles. Setae were 11–36 μ long and 2.5–5.5 μ wide. Conidia were oval to short cylindrical, straight to slightly curved, and 4.5–9 μm long and 1.4–2.2 μm wide. Sclerotia were 92–311 μm long and 50–298 μm wide, with the surfaces covered by setae 11–27 μm long and 2–5 μm wide (1).

On soybean leaves, pycnidia were similar in size and shape to those produced in culture, ranging from 110 to 160 μm (14). Sclerotia on soybean leaves averaged 125 × 250 μm and sclerotial setae, 15–24 × 4–5 μm (7). Comparisons of the morphological characteristics of isolates of *P. glycines* from *G. max* and *N. wightii* showed no significant differences in size of conidia, pycnidia, pycnidial setae, sclerotia, or sclerotial setae (1).

Disease Cycle and Epidemiology

Red leaf blotch frequently develops on soybeans planted into newly cleared land, but the source of primary inoculum for such epiphytotics is not known. Primary inoculum may be conidia or sclerotia from plants infected with *N. wightii*, which is widespread, or from other alternative hosts previously established on virgin land or growing adjacent to new soybean fields. A host range study has not been made.

The disease generally is distributed uniformly throughout an affected field, rather than in isolated pockets. The pathogen may be dispersed from field to field by sclerotia carried in contaminated soil during routine farm operations. We studied the microflora associated with seeds from infected soybean plants in Zambia and Zimbabwe and found that the fungus is not internally seedborne. An unidentified *Pyrenochaeta* sp. was isolated from soybean seeds sent to Brazil from Nigeria (15), however. Additional studies need to be made on seed transmission of this pathogen. Incidental transmission of the pathogen may be through seed lots contaminated with infected plant debris or soil peds carrying sclerotia of *P. glycines*.

The disease cycle has not been fully characterized on either soybeans or *N. wightii*. We propose a possible disease

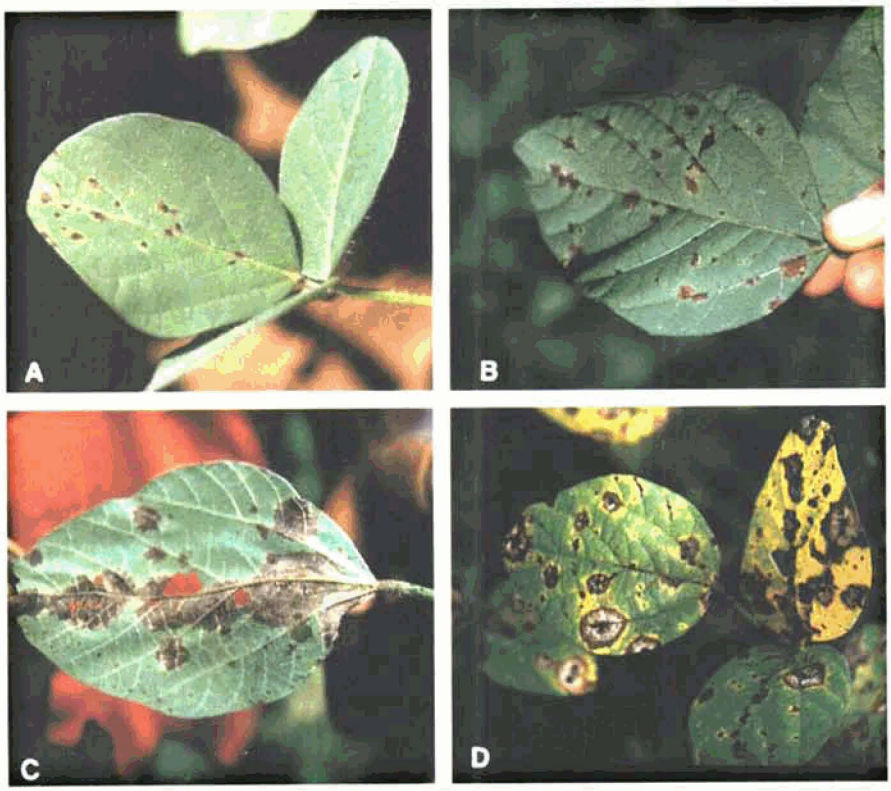

Fig. 1. Red leaf blotch (*Pyrenochaeta glycines*) lesions (A) on a young soybean leaf, (B) predominantly along the veins of an upper leaf surface, and (C) on a lower leaf surface. (D) An upper leaf surface showing advanced blotching.

Fig. 2. Red leaf blotch lesions on soybean petioles, pods, and stems.

cycle (Fig. 6). Soilborne sclerotia on or in soil particles may be rain-splashed onto leaf surfaces, where they germinate and infect the host. We have observed that sclerotia can give rise to pycnidia either on mycelium or on sclerotia in culture, and a similar process may take place in the soil or on the leaf surfaces, or both. We found that when conidia or sclerotia are placed on soybean leaves, infection occurs and symptoms appear 2–7 days after inoculation. Pycnidia and sclerotia develop within the leaf blotches, and both can be as numerous as 100 or more on a leaflet. Heavily infected leaves drop prematurely, releasing pycnidia and sclerotia back into the soil and allowing for secondary infection by conidia or sclerotia. The sclerotia and possibly the pycnidia overseason to provide primary inoculum for the next growing season, thus completing the disease cycle. How long sclerotia survive in the soil is not known.

No experimental data on the influence of environment on pathogen growth and disease development have been published. In Zimbabwe between 1982 and 1984 (drought years), disease severity in replicated trials was reduced by delayed planting. During the 1984–1985 growing season of normal to above-average rainfall, however, disease severity was not reduced by delayed planting. Our field observations indicate that abundant rainfall and high humidity promote disease development. The effect of canopy closure on disease development is not known.

Defoliation has been reported as the most damaging aspect of the disease (14). In one of our studies, the disease progressed from the bottom leaves to the top leaves, with more than 90% having lesions by 89 days after planting during

Fig. 3. Red leaf blotch lesions on a leaflet of *Neonotonia wightii*.

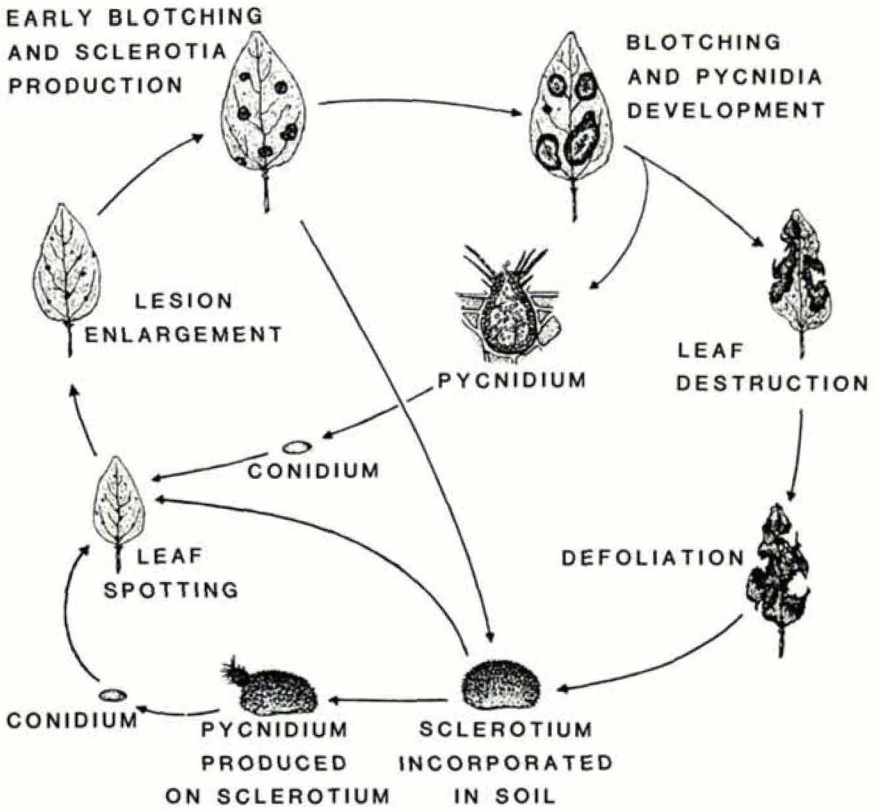

Fig. 6. Schematic disease cycle of red leaf blotch of soybeans.

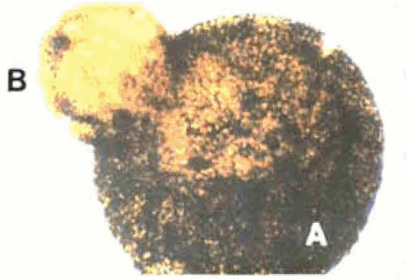

Fig. 4. Formation of a pycnidium (B) on a sclerotium (A) of *P. glycines* in culture.

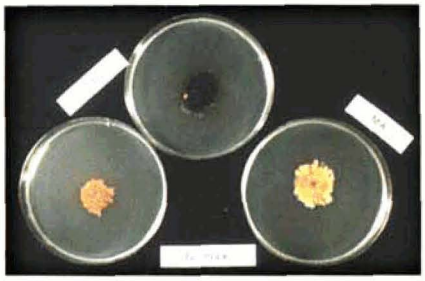

Fig. 5. Variations in color and characteristics of *P. glycines* isolated from soybeans and cultured on cornmeal agar (CMA) and malt agar (MA).

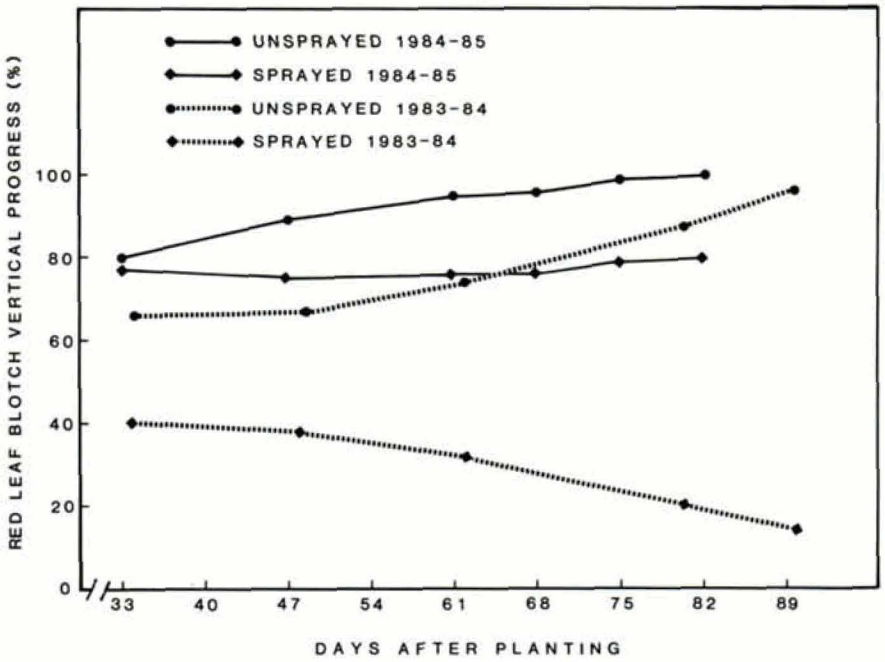

Fig. 7. Mean vertical disease progress of red leaf blotch on soybean plants unsprayed or sprayed with fentin acetate (0.9 and 0.6 kg a.i./ha during 1983–1984 and 1984–1985, respectively) in Zambia.

the 1983–1985 seasons (Fig. 7). Symptoms were always greatest on the lowest leaves and proportionately less on each leaf up the plant. Yield losses resulted from reduced seed size and weight.

Control

Chemical and cultural methods for the control of red leaf blotch have been studied in Zambia and Zimbabwe. In addition, a concerted effort has been made to evaluate germ plasm and breeding lines for resistance to the pathogen. At present, no recommended commercial procedures for control of the disease are available, although field trials

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with fungicide sprays reduced vertical disease progress (VDP) up the plant, severity on foliage, and defoliation (Figs. 7–9). VDP was expressed as the percentage of plant height to which *P. glycines* had spread based on the following formula (10): VDP = (maximum height (nodes) at which red leaf blotch symptoms appeared divided by maximum height (nodes) of the plant) × 100.

In field trials in Zambia and Zimbabwe between 1982 and 1984, over 2,500 lines, cultivars, accessions, and breeding lines were evaluated for resistance (3). Most commercial cultivars now grown in the United States and included in these trials were susceptible. When symptoms on late- and early-maturing cultivars were compared at equal growth stages, both types were susceptible and had similar disease severity ratings. Stewart (14) reported that soybean cultivars with pale green foliage were more susceptible to *P. glycines* than those with darker green leaves; we found, however, that soybeans with dark or light green foliage were equally susceptible.

Research on cultural practices to control the disease, such as date of planting, long rotations, plant spacing, and varying tillages, must be continued before any can be recommended. A normal rotation for soybeans in Zambia and Zimbabwe is to grow soybeans and/or maize during the rainy summer months and wheat under irrigation during the dry winter months. Some farmers use longer maize rotations when red leaf blotch is a serious problem, but this practice does not always alleviate the disease. For example, a soybean grower in Zimbabwe planted his soybean fields to maize for four consecutive summer seasons after severe yield losses from red leaf blotch during the previous two years. In the fifth year, the fields were planted to soybeans and the disease was still severe.

In Zambia, fentin acetate effectively controlled disease, as reflected in greater yields than in unsprayed plots between 1982 and 1985 (2,9). During the 1982–1983 season, yields in field plots sprayed twice with either benomyl (Benlate 50WP) or fentin acetate (Brestan 60WP) at 0.5 kg a.i./ha were increased by 13 and 23%, respectively, over those in unsprayed plots (9). During the 1983–1984 and 1984–1985 growing seasons, soybeans in plots sprayed weekly with fentin acetate at 0.9 or 0.6 kg a.i./ha, respectively, had significantly ($P = 0.05$) higher total grain yields and seed weights and lower values for area under the disease progress curve, disease severity, and vertical disease progress than plants in unsprayed plots (Figs. 7 and 8, Table 1). Disease severity was calculated using the Horsfall-Barratt method (4).

Conclusions

On the basis of our observations and experience in Zambia and Zimbabwe, red leaf blotch can limit soybean production. The disease has been known in the region since 1956 but did not become economically important until the beginning of intensive soybean production during the early 1970s. Red leaf blotch is the most important disease of soybeans in the region. A similar pattern may develop in soybean-growing areas of Bolivia, since the pathogen has been collected from there. Red leaf blotch is not known to occur in the United States or Brazil but could become established in these countries if the pathogen is introduced and environmental conditions favor induction of the disease on

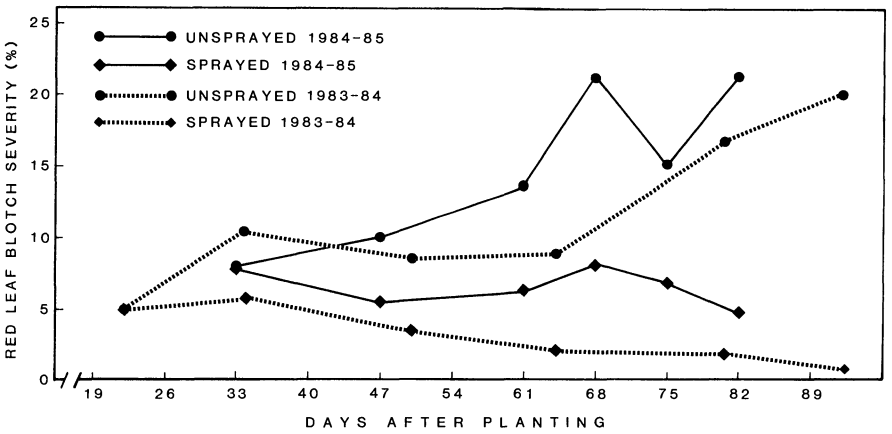

Fig. 8. Mean severity of red leaf blotch on soybean plants unsprayed or sprayed with fentin acetate (0.9 and 0.6 kg a.i./ha during 1983–1984 and 1984–1985, respectively) in Zambia.

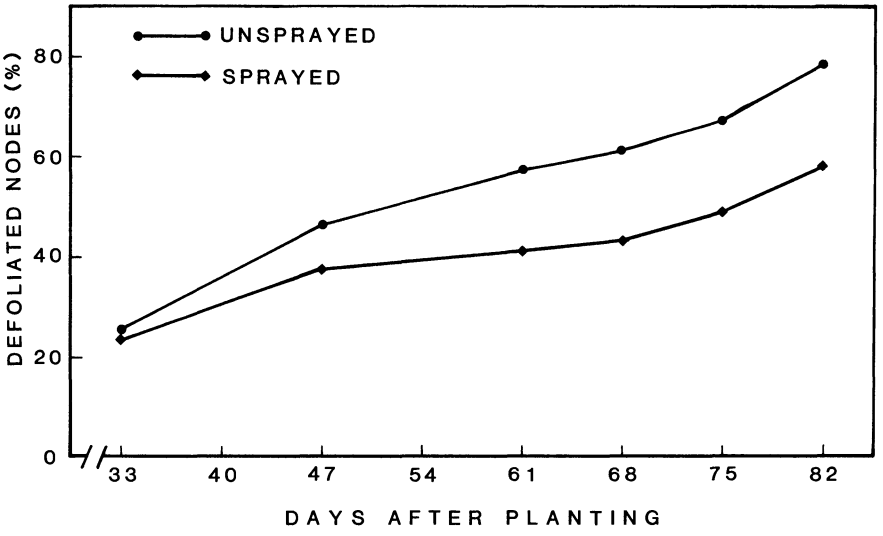

Fig. 9. Defoliation of soybeans by *P. glycines* on soybean plants unsprayed or sprayed with fentin acetate (0.6 kg a.i./ha) at Mpongwe, Zambia during 1985.

Table 1. Means of disease severity, area under the disease progress curve (AUDPC), yield, and seed weight for cultivars tested in Zambia during the 1983–1984 and 1984–1985 growing seasons

	1983–1984[v]		1984–1985[w]	
Parameter	**Unsprayed**	**Sprayed**	**Unsprayed**	**Sprayed**
Disease severity (%)[x]	24.7 a[y]	1.1 b	21.3 a	4.8 b
AUDPC[z]	839 a	282 b	665 a	313 b
Yield (kg/ha)	3.2 a	3.9 b	1.3 a	2.0 b
Seed weight (1,000 seeds/g)	176 a	226 b	135 a	190 b

[v] Cultivars Sable, Oribi, Tunia, Geduld, Jupiter, and Magoye; plots with and without weekly sprays of fentin acetate at 0.9 kg a.i./ha.
[w] Cultivars Oribi, Sable, Santa Rosa, and Tunia; plots with and without weekly sprays of fentin acetate at 0.6 kg a.i./ha.
[x] Percentage of total leaf area diseased at R-6 growth stage.
[y] Numbers followed by different letters are significantly different based on FLSD ($P = 0.05$).
[z] Based on percentage of leaf area infected.

susceptible cultivars. Since most U.S. and other germ plasm lines tested are susceptible, finding resistance in other accessions and exotic lines would provide soybean breeders with germ plasm to develop resistant cultivars. Continued research on this disease is important because control methods will benefit soybean production in Africa and other areas where the fungus and the disease may be found.

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