# Leaf Sheath Blights of *Sorghum bicolor* Caused by *Sclerotium rolfsii* and *Gloeocercospora sorghi* in South Texas

G. N. Odvody and D. B. Madden

Assistant professor and research associate, respectively, Texas A&M Research and Extension Center, Route 2, Box 589, Corpus Christi 78410.

Texas Agricultural Experiment Station Article TA 18654.

Portions of this research were conducted under project TAM24/AID/DSAN/XII/G0149 from U.S. AID.

Appreciation is extended to R. A. Frederiksen and D. T. Rosenow for their considerable field observations and insight which led to a better understanding of the disease syndromes.

Accepted for publication 8 September 1983.

## ABSTRACT

Odvody, G. N., and Madden, D. B. 1984. Leaf sheath blights of *Sorghum bicolor* caused by *Sclerotium rolfsii* and *Gloeocercospora sorghi* in South Texas. Phytopathology 74:264-268.

*Sclerotium rolfsii* (causal agent of southern sclerotial rot) and *Gloeocercospora sorghi* (causal agent of zonate leaf spot) caused leaf sheath blights on grain sorghum (*Sorghum bicolor*) in fields and nurseries in South Texas. *S. rolfsii* infected and killed one to all leaf sheaths of susceptible sorghums growing in saturated soil under hot, humid conditions. Sclerotia of *S. rolfsii* were often present on the outside of necrotic leaf sheaths. *G. sorghi* also infected basal and underlying leaf sheaths on susceptible cultivars under hot, humid conditions. Sclerotia were produced in large quantities and gave a gray appearance. Infection of sheaths by *G. sorghi* either preceded or occurred simultaneously with infection of leaf blades. Free and residue-associated sclerotia of *G. sorghi* were isolated from field soil and germinated readily in culture. Three field soils had 4–17 germinable sclerotia per gram of air-dry soil. Occurrence of southern sclerotial rot was sporadic and predominantly in poorly drained areas, but zonate leaf spot on sheaths was present in most fields. Neither disease was economically important, but some cultivars were extremely susceptible to either one or both diseases in the field.

In South Texas during the high rainfall years of 1979, 1981, and early 1982, commercial hybrids and some experimental cultivars of grain sorghum (*Sorghum bicolor* [L.] Moench) consistently demonstrated yellow, wilted, and dead lower leaves when plants were observed at the boot stage or later. Lesions on leaf blades were not sufficient to account for expressed symptoms. Sheaths of affected leaves displayed symptoms of parasitism including pigmented lesions.

The two organisms consistently isolated from young and mature sheath lesions were either *Sclerotium rolfsii* Saccardo or *Gloeocercospora sorghi* Bain & Edgerton.

*S. rolfsii* is a soilborne pathogen that attacks a variety of plant hosts including *S. bicolor* in warm regions of the world (7). On sorghum it is reported to cause a seedling blight, stalk rot, and basal sheath rot. The basal sheath rot, referred to as southern sclerotial rot, was described from Georgia (5) and reported from other sorghum growing areas of the world, including the states of Louisiana and Texas (1), but was not considered economically important.

*G. sorghi* causes zonate leaf spot on sorghum, corn and other grasses (7) and is endemic in South Texas. The pathogen is reported to occur on both the leaf blade and sheath of sorghum (2), but symptomatology and disease development on these sites are not differentiated in the literature (2,7).

This paper describes the leaf sheath phases of the sorghum diseases caused by *S. rolfsii* and *G. sorghi* in South Texas and identifies sorghum cultivars susceptible and resistant to each pathogen in the field.

## MATERIALS AND METHODS

**Field locations and disease evaluations.** For both *S. rolfsii* and *G. sorghi*, sorghum hybrids in commercial fields and hybrids and other cultivars in experimental nurseries were evaluated for disease incidence and severity, symptom expression, disease progression, and environmental conditions associated with disease. Both diseases were observed in commercial fields and sorghum nurseries throughout the Coastal Bend region of South Texas, but primarily within a 125-km radius of Corpus Christi, and at the Texas Agricultural Experiment Station (TAES) in Weslaco.

Sorghum cultivars in a standard test nursery of two replications (30–60 plants each) of 70 entries (randomized block, ADIN = All Disease and Insect Nursery, TAES) were evaluated for leaf sheath reaction to *G. sorghi* and *S. rolfsii* in 1981 and 1982 at all nursery locations where either or both diseases occurred. Cultivar susceptibility and resistance was determined by rating disease under optimal conditions and by consistency of disease ranking over locations and years.

For each disease, a separate continuous rating system from 1.0 to 5.0 utilized a maximum number of diseased sheaths per plant (S) within specific incidence ranges (%) as follows: *S. rolfsii*—1.0 = no disease (resistant), 2.0 = 1 or 2 S at 25–50% (moderately resistant), 3.0 = 3 or more S at 25–50% (moderately susceptible), 4.0 = all S at 25–50% (susceptible), 5.0 = all S at 100%, plants killed (very susceptible); *G. sorghi*—1.0 = no disease (resistant), 2.0 = 1 or 2 S at 25–50% (moderately resistant), 3.0 = 1 or 2 S at 51–100% (moderately susceptible), 4.0 = 3 or more S at 25–50% (susceptible), 5.0 = all S at 100% (very susceptible). The resistance/susceptibility statements in parentheses refer to cultivar reaction under optimal disease conditions.

**Isolation and identification of pathogens.** Leaf sheath lesions were surface sterilized with 1% NaOCl for 1 min, rinsed with sterile distilled water, air-dried, and plated onto water agar (2% w/v) containing streptomycin sulfate (100 μg/ml) (WA + Str) and incubated for 1–3 days at 24–27 C. The plates were incubated in the dark for *S. rolfsii* and under longwave UV (blacklight, 20 W; General Electric Co., Lighting Business Group, Nela Park, Cleveland, OH 44112) for *G. sorghi*. Sclerotia from lesions were either surface sterilized or nontreated and incubated on WA + Str as above. *S. rolfsii* was identified on plates by typical mycelial growth emanating from lesions or sclerotia within 24–48 hr and by similar growth and sclerotial production on potato-dextrose agar (Difco) (PDA) (7). *G. sorghi* was identified by the presence of spore masses with typical conidia produced either on lesion tissue or from

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

sclerotia (2). Production of typical sclerotia (2) in infected sheath tissue before or during incubation was also used to identify *G. sorghi* and differentiate it from *S. rolfsii* occurring in the same or different sheaths.

**Pathogenicity.** Single spore isolates of *G. sorghi* were obtained from conidia of germinated sclerotia incubated on WA + Str. These isolates represented sclerotia derived from leaf blade and leaf sheath lesions of sorghum collected at Beeville, and from soil at three South Texas locations (Beeville, Corpus Christi, and Bishop) with a known history of zonate leaf spot caused by *G. sorghi*.

Inoculum was produced by streaking plates of modified V-8 juice agar (MV-8) (150 ml of V-8 juice + 3 g $CaCO_3$/L) with conidia of each isolate and incubating them under longwave UV at 27 C for 3–4 days. Conidial masses were the predominant fungal structures formed, and conidia from these plates were suspended in distilled water ($10^5$ conidia per milliliter) and either spray inoculated onto whole plants or injected behind leaf sheaths of 60-day-old sorghum plants of a hybrid highly susceptible to zonate leaf spot (RS 610). Inoculated plants were incubated at 100% RH at 25 C for 24 hr and removed to a greenhouse bench (27–30 C).

An isolate of *S. rolfsii* obtained from a single sclerotium on an infected leaf sheath was grown on PDA at 27 C in the dark for 1 wk until sclerotia formed and matured. Sclerotia were transferred individually with forceps and placed behind leaf sheaths of 60-day-old sorghum plants of RS 610. Inoculated plants were incubated and removed as with *G. sorghi*.

All inoculated plants were observed for symptoms every day for 2 wk, and lesions were periodically selected for reisolation.

**Isolation and quantitation of sclerotia of *G. sorghi* in soil.** Soil from the upper 8 cm at the Beeville, Corpus Christi, and Bishop disease sites was evaluated for viable sclerotia of *G. sorghi* by using a flotation-sieving technique. Fifty grams of air-dry soil, sieved through a screen (850-$\mu$m pore size), was added to 100 ml of water in a 250-ml centrifuge bottle and mixed with a stirring bar on a magnetic stirrer at 1,100 rpm for 30 min. The suspension was centrifuged in a swinging-bucket rotor (International Equipment Co.) at 300 *g* for 10 min, and the supernatant decanted and saved. The pellet was resuspended in 100 ml of either saturated sucrose or corn syrup (Karo; Best Foods, CPC International Inc., Englewood Cliffs, NJ 07632), recentrifuged at 300 *g* for 30 min and the supernatant was decanted and saved. The suspending in sugar solution followed by centrifugation were repeated four more times and all supernatants were passed under negative pressure through a nylon fabric filter (Tetko, Inc.) with a pore size of 75 $\mu$m. Sclerotia of *G. sorghi* were retained on the fabric and washed with distilled water, either surface sterilized or left nontreated and incubated on WA + Str at 27 C under longwave UV for 72 hr. Germinated sclerotia of *G. sorghi* were counted under a binocular microscope (×14–60). The germination response of nontreated sclerotia isolated from soil (Bishop) was also evaluated either by submerging them in distilled water or by placing them on a cellulose membrane filter in contact with wet, nonsterile soil from the same field.

## RESULTS

**Field symptomatology and development of sheath blight caused by *S. rolfsii*.** Depending upon host pigmentation, initial symptoms appeared at boot stage or later as either bright red, purple, or brown water-soaked lesions predominantly at the base of the lowermost sheaths in contact with wet soil. Lesion development progressed from the point of soil contact in an upward and lateral manner to encircle and invade entire sheaths. Blades of infected


**Fig. 1.** Field symptoms of southern sclerotial rot on grain sorghum sheaths. **A,** Killed leaf sheath with limited development of *Sclerotium rolfsii* onto the leaf blade and fan-shaped mycelial mat on subsequent leaf sheath. **B,** Killed, overlapping leaf sheaths with dark, mature, surface sclerotia of *S. rolfsii* and easily shredded, deeply pigmented, inner leaf sheath surfaces.

sheaths yellowed, wilted, and died, but as described by Tarr (7), pathogen growth seldom extended onto the leaf blade more than 2–3 cm beyond the ligule (Fig. 1A). As the disease progressed, thick, fan-shaped, superficial mycelial mats were formed on the inner sheath surface, on the outer ligule area of necrotic sheaths, and sometimes at the point where the ligules contacted the subsequent sheath (Fig. 1A). Necrotic leaf sheaths shredded to reveal degraded internal tissue which was either pink-to-deep-rose or brown in color, depending upon host pigmentation (Fig. 1B). Areas of the necrotic inner and outer sheath surface not exposed to direct sunlight often displayed this pigmentation.

Under continued wet conditions, large, variously sized sclerotia and sclerotial aggregates were formed on the outside of necrotic sheaths (Fig. 1A and B), especially on areas with mycelial mats. These sclerotia (0.5–2.0 mm) were typical of *S. rolfsii* (5), and germinated readily when transferred directly to WA + Str. Some sclerotia germinated after being stored for 2 yr at 6–35 C on air-dried infected sheaths.

The sheath blight caused by *S. rolfsii* occurred sporadically and apparently was not economically important in commercial fields. When present, it usually progressed through one to three consecutive leaf sheaths, and disease incidence and severity were highest in the most poorly drained fields or areas of fields. Under optimal conditions, the disease rapidly progressed through all leaf sheaths of very susceptible cultivars and killed the plants. Culms were not parasitized and saprophytic colonization after plant death appeared minimal. No parasitism of roots or stalks was observed in South Texas.

**Field symptomatology and development of the sheath blight caused by *G. sorghi*.** Initial symptoms on sheaths infected by *G. sorghi* were, depending upon host pigmentation, either dark red, black-purple or brown variable lesions that irregularly encircled sheaths. Lesions usually appeared at boot stage or later anywhere on basal leaf sheaths but most commonly where they contacted soil (Fig. 2A) and frequently at or just below the ligules (Fig. 2B). Initial infection sites near the ligule area appeared to be on the inside surface of the leaf sheath. Basal and ligule lesions developed towards each other in a roughly cylindrical manner, but other midsheath lesions developed in a longitudinal, elliptical manner reminiscent of leaf blade lesions. The latter lesions became irregularly shaped blotches when coalesced.

Disease developed either a few centimeters or progressed through one to four sheaths on very susceptible cultivars and rarely through all sheaths. Culm tissue was not infected. As lesions became necrotic, numerous internal sclerotia (0.1–0.2 mm) were formed within and beyond the pigmented lesion borders where they were visible through the translucent outer and inner sheath epidermis (Fig. 2C and D) and produced an overall gray appearance. The sclerotia were formed on the internal surface of each epidermis and appressed to it especially near veins. Where appressed, sclerotia were flat but otherwise spherical and ellipsoid. Sclerotia from leaf blade lesions were described as being lenticular-spherical in shape (2,5) but we observed some from leaf blade lesions that were indistinguishable from those in sheath lesions. Sclerotial aggregates (0.3 mm in diameter) sometimes formed in sheath lesions. When blade and sheath lesions were shredded, sclerotia remained associated with the epidermal layers. Sclerotia from these lesions did not germinate if left nontreated and incubated on WA + Str, but many germinated if surface sterilized with 1% NaOCl prior to incubation. In leaf sheath lesions, sclerotia were produced more consistently than in leaf blade lesions, but both produced up to 500 sclerotia per square centimeter.

Leaf blades of sheaths infected with *G. sorghi* slowly yellowed, wilted, and died, but they were not infected from the sheath. Lesions from sheaths produced conidial masses when incubated in the laboratory under longwave UV, but no conidial production was observed on field-collected lesions prior to incubation.

Infection of sheaths by *G. sorghi* either preceded infection of leaf blades or occurred simultaneously on sorghum at the boot stage or later. The incidence of sheath blight caused by *G. sorghi* in most commercial sorghum fields was $\geq 25\%$, but severity was low and no direct losses were evident.

**Evaluation of cultivars for sheath blight reaction in the field.** Those cultivars from the ADIN that were susceptible to *S. rolfsii* under optimum conditions and had consistently high disease rankings were SC 326-6, B 35-6, SC 414-12, SC 748-5, and R 3338. Those resistant to moderately resistant under optimum conditions and with consistently low disease rankings were SC 103-12, SC 630-11E, B TX 378, TX 430, and IS 9530. Cultivar susceptibility to *G. sorghi* on sheaths was the same as previously reported from blade infection ratings (4), but disease incidence on these two sites on the same plant or plants within a row were often not equivalent. From the ADIN, cultivars susceptible to sheath infection by *G. sorghi* included TX 7078, SC 103-12, TX 2782, and B TX 2161. Resistant to moderately resistant cultivars included SC 326-6, 77CSI (IS 2930 × IS 3922), and TAM 428.

**Pathogenicity of *G. sorghi*.** All isolates of *G. sorghi* caused diffuse sheath reddening and initial red blade lesions by 48 hr. In infected leaf sheaths, lesions enlarged, coalesced, and became dark red with some necrosis after 72 hr, and leaves wilted. Many lower leaves were killed within 7 days and sclerotia of *G. sorghi* were evident in some necrotic lesions. Leaf sheath lesions collected at various developmental stages and examined at ×14–60 had no conidial masses of *G. sorghi*; however, conidial masses developed by 24–48 hr on either surface-sterilized or nontreated lesions incubated at 27 C under longwave UV. Many plants spray inoculated with conidia of *G. sorghi* developed both blade and sheath lesions similar to those observed in the field.

**Pathogenicity of *S. rolfsii*.** Reddish-pink circular lesions (5 mm) were observed 4 days after inoculation and some sheaths were killed by 6 days. Because of low humidity in the greenhouse, most lesions remained localized, but all were pink and similar to lesions on this cultivar in the field, and distinct from those caused by *G. sorghi*. *S. rolfsii* was reisolated from lesions that were four or more days old.

**Quantitation and viability of sclerotia of *G. sorghi* in soil.** When sclerotia of *G. sorghi* from soil were left nontreated and incubated on WA + Str at 27 C, under either longwave UV or darkness, a small percentage began germinating within 18 hr and produced immature conidia within 24 hr. Most sclerotia germinated and produced mature conidia within 36–48 hr, and little or no additional germination occurred after 72 hr. Sclerotia also germinated rapidly in distilled water or on a cellulose membrane in contact with wet, nonsterile field soil. Sclerotia produced predominantly one, but occasionally two or three, spore masses that progressed from clear at 24 hr to light pink and salmon at 72 hr with more rapid and greater pigmentation in light than dark. Free, nongerminated sclerotia of *G. sorghi* were not counted because of similarity to sclerotia of other fungi. Sclerotia of *G. sorghi* associated with lesion residues were coincidently isolated with free sclerotia and had a similar germination response.

The Beeville, Corpus Christi, and Bishop field soils had five, four, and 17 germinable sclerotia of *G. sorghi* per gram of air-dry soil, respectively, and some additional sclerotia associated with lesion residues.

## DISCUSSION

Although sheath blights caused by *S. rolfsii* and *G. sorghi* are not yet economically damaging, the disease reactions in the field of some susceptible cultivars suggested potential disease loss especially for southern sclerotial rot. The susceptibility of cultivars to zonate leaf spot on leaf blades and sheaths is similar and both may combine to cause extensive damage. The sheath lesions of zonate leaf spot may be most important in their contribution to soilborne inoculum of *G. sorghi* because they produce sclerotia more consistently than do blade lesions.

Rainsplashed conidia from soilborne sclerotia of *G. sorghi* are thought to be initial inoculum for infection (3) because lowermost sorghum leaves with adhering rainsplashed debris are the first to display symptoms of zonate leaf spot. The germination we observed with free and residue-associated sclerotia of *G. sorghi* isolated from soil are consistent with that hypothesis. Rainsplash probably also disseminates conidia to proximal infection sites on

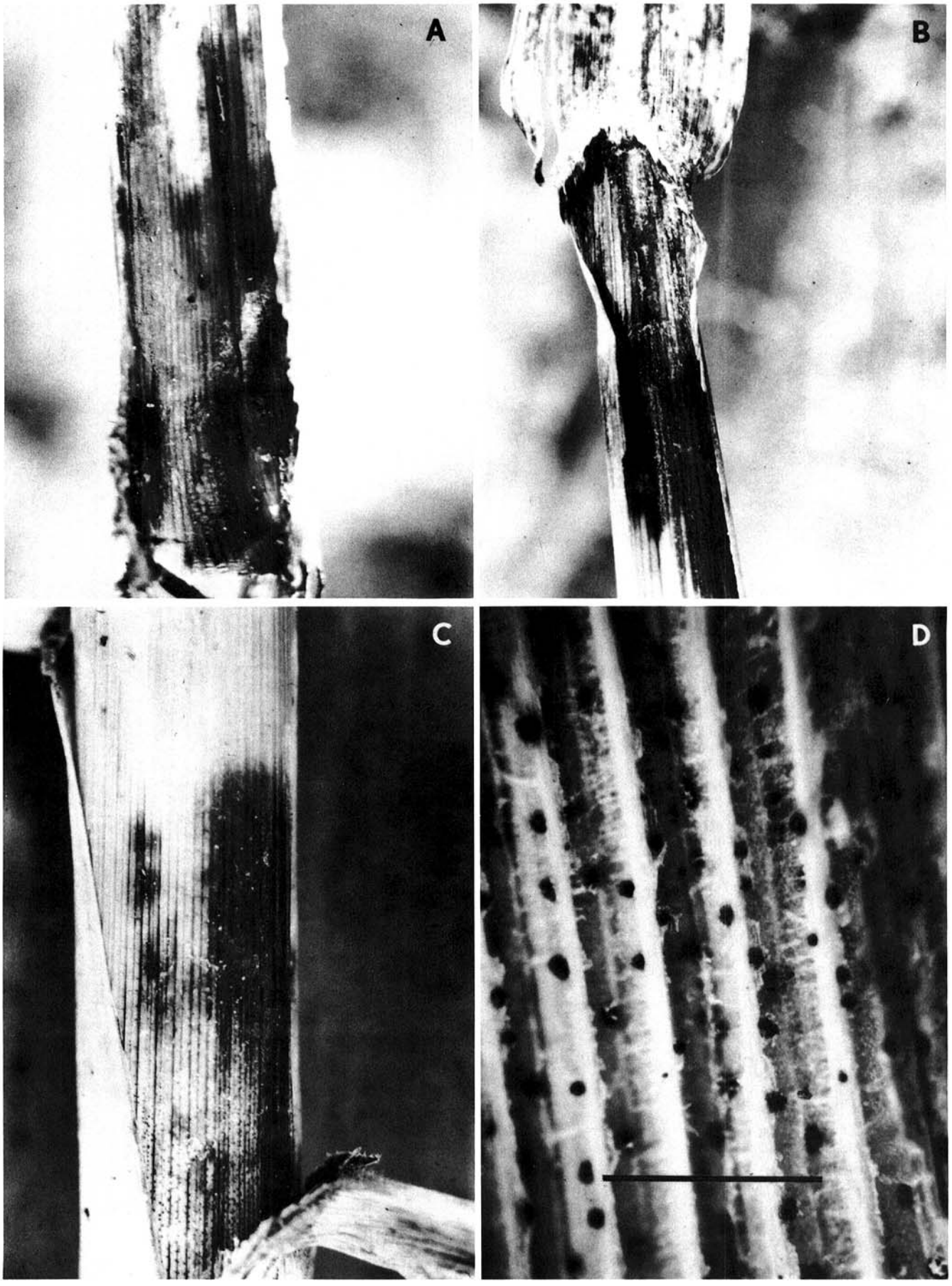


**Fig. 2.** Field symptoms of zonate leaf spot on grain sorghum sheaths. **A,** Basal and **B,** ligule area lesions occurring on sorghum leaf sheaths. **C,** Infected leaf sheath with sclerotia of *Gloeocercospora sorghi* visible through the outer epidermis. **D,** Sclerotia of *G. sorghi* visible through disintegrated inner epidermis of the leaf sheath. Bar represents 1.6 mm.

leaf sheaths, but some infection from conidia appeared to be initiated on leaf sheaths at or just below the soil surface. Ligule area infections may be associated with germinating sclerotia in soil that is often deposited there. Although sheath lesions do not appear to provide secondary inoculum (conidia) for leaf blade infections, it is possible that, conversely, the conidia from leaf blade lesions are washed down into leaf sheath areas where infection could occur.

Because soilborne sclerotia are the primary survival propagules of *G. sorghi*, crop rotation and burial of crop residue may decrease their survival.

Incidence of southern sclerotial rot may also be reduced by crop rotation and burial of residue (7) but the saprophytic ability of *S. rolfsii*, its wide host range, and its sclerotia make it difficult to control. Recent evidence from other hosts demonstrated that infection by *S. rolfsii* could occur from eruptively-germinating sclerotia of the pathogen (6). Thus, sclerotia may be the initial inoculum source for sorghum leaf sheath infections by *S. rolfsii*.

In disease-conducive areas, an avoidance of the cultivars most susceptible to each sheath blight pathogen should minimize the increase of soilborne inoculum of both pathogens and keep these diseases below economically-damaging levels.

## LITERATURE CITED


1. Anonymous. 1960. Index of Plant Diseases in the United States. Agric. Handb. 165. ARS, U.S. Dep. Agric., Washington, DC.
2. Bain, D. C., and Edgerton, W. 1943. The zonate leaf spot, a new disease of sorghum. Phytopathology 33:220-226.
3. Dean, J. L. 1968. Germination and overwintering of sclerotia of *Gloeocercospora sorghi*. Phytopathology 58:113-114.
4. Frederiksen, R. A., and Rosenow, D. T. 1979. Breeding for disease resistance in sorghum. Pages 137-167 in: Biology and Breeding for Resistance to Arthropods and Pathogens in Agricultural Plants. Misc. Publ. 1451 TAES, TAMU, and Univ. Calif.-AID Pest Management Project.
5. Luttrell, E. S. 1950. Grain sorghum diseases in Georgia, 1949. Plant Dis. Rep. 34:45-52.
6. Punja, Z. K., and Grogan, R. G. 1981. Mycelial growth and infection without a food base by eruptively germinating sclerotia of *Sclerotium rolfsii*. Phytopathology 71:1099-1103.
7. Tarr, S. A. J. 1962. Diseases of Sorghum, Sudan Grass, and Broomcorn. Commonw. Mycol. Inst., Kew, Surrey, England. 380 pp.