The Mode of Systemic Infection of Sorghum and Sudangrass by Conidia of Sclerospora sorghi

B. L. Jones

Associate Professor, Texas A & M Agricultural Research and Extension Center, Weslaco, TX 78596. Present address, Texas A & M Agricultural Research and Extension Center, Stephenville, TX 76401.

Accepted for publication 14 October 1977.

ABSTRACT

JONES, B. L. 1978. The mode of systemic infection of sorghum and sudangrass by conidia of Sclerospora sorghi. Phytopathology 68: 732-735.

Systemic infections with *Sclerospora sorghi* were obtained in sorghum and sudangrass by natural inoculation with conidia in the field and artificial inoculation with conidia in the greenhouse. Plants were most susceptible to systemic infection when inoculated the first night after seedling emergence. Sudangrass initially was more susceptible and it remained susceptible longer than sorghum. Penetrations by means of conidial germ tubes, were observed most often between epidermal cells of the stem at the soil surface. Hyphae advanced to the apical meristem of the plants and invaded developing leaves. It appeared that *S. sorghi* must enter leaves before they are fully developed for systemic symptoms to be expressed.

Additional key words: downy mildew, fungal penetration.

Sclerospora sorghi Weston and Uppal incites both systemic and local lesion infections in most of its gramineous host (2). The systemic phase of the disease is the most damaging on sorghum [*Sorghum bicolor* (L.) Moench]. In contrast, the local lesion phase is the most damaging to sudangrass [*Sorghum sudanense* (Piper) Stapf.] (2). Symptoms of systemic infection may appear either early or late in sorghum (1). Symptoms of early systemic infection usually appear within 1-2 wk after host emergence. Late systemic symptoms may not appear until plants are 30-60 days old. The lower portions of plants systemically infected at later stages of development often appear normal while the upper portions display systemic symptoms. Early systemic infection can be incited by oospores (2, 5, 6, 7) or conidia (4) of *S. sorghi*. The mode of late systemic infection is unknown.

This paper reports the mode of early systemic infection in sorghum and sudangrass by means of conidia.

00032-949X/78/000123$03.00/0
Copyright © 1978 The American Phytopathological Society, 3340 Pilot Knob Road, St. Paul, MN 55121. All rights reserved.

MATERIALS AND METHODS

Plants of grain sorghum cultivars DeKalb C48A and Pioneer 846, and sudangrass SA372, were grown in flats (45 × 60 cm) of soil that had been autoclaved for 2 hr at 120 C. The cultivars were planted at about 1-wk intervals for 3 wk and maintained in a greenhouse until seedling emergence occurred after the last planting. At this time plants of each cultivar were 1, 7, 13, and 17 days old. Plants of each age group were exposed to conidia of *S. sorghi* by placing flats in the field beneath the canopy of young sorghum plants systemically infected with *S. sorghi*. The flats were placed on boards to prevent contact with the soil. Abundant conidia were being produced on

TABLE 1. Systemically infected sorghum and sudangrass following inoculation with *Sclerospora sorghi* conidia in the field and in the greenhouse

Location and Age of plants when exposed (days)		Sorghum 846 Total exposed plants (no.)	Sorghum 846 Plants infected (%)	Sorghum C48A Total exposed plants (no.)	Sorghum C48A Plants infected (%)	Sudangrass SA372 Total exposed plants (no.)	Sudangrass SA372 Plants infected (%)
Field	1	189	12	239	4	397	17.0
	7	169	0	282	1	431	7.0
	13	253	0	193	0	243	1.5
	17	162	0	174	0	198	0.5
	Check	783	0	869	0	1,053	0.0
Greenhouse	1	173	47	199	34.2	294	73.0
	7	193	1.1	203	0.5	310	9.0
	13	141	0.0	146	0.0	192	2.1
	17	183	0.0	172	0.0	214	0.4
	Check	261	0.0	289	0.0	351	0.0

the leaves of these plants at night but oospore production had not commenced. After 5 days in the field, the plants were returned to the greenhouse and observed for 6 wk. Additional plants of each age group were inoculated in the greenhouse with *S. sorghi* conidia by use of the infected leaf technique (3, 4). The test was repeated in the greenhouse on cultivar C48A. Noninoculated seedlings served as checks.

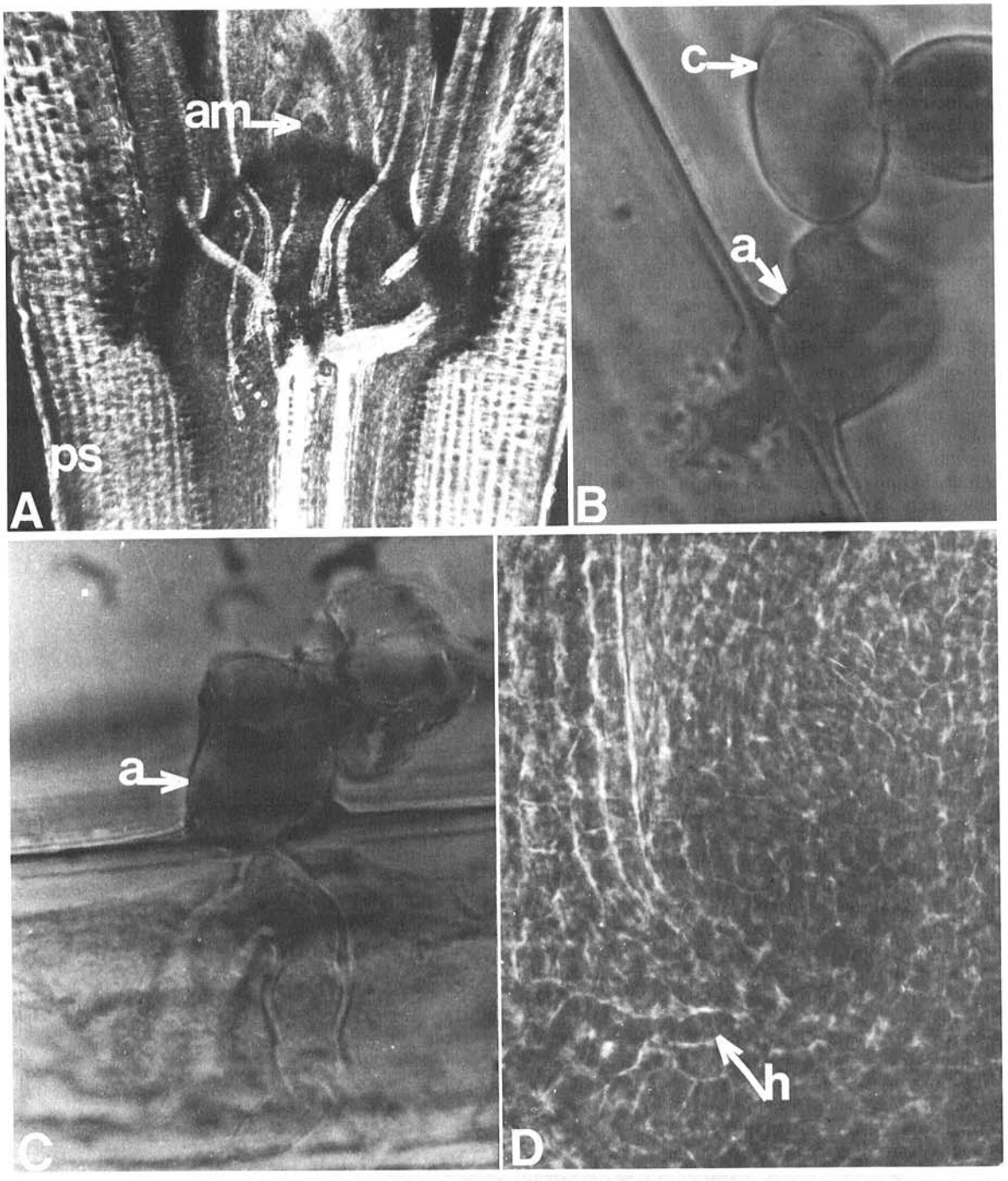

Fig. 1-(A to D). *Sclerospora sorghi* conidial germ tube penetration and infection of sorghum and sudangrass. **A)** Longitudinal section of a sorghum seedling immediately after emergence. Note the close proximity of the apical meristem (am) to the penetration site (ps) at the soil surface (×25). **B)** Penetration of a 1-day-old sudangrass seedling. The conidium (c) gives rise to an appressorium (a) which has forced apart the host epidermal cells at their anticlinal junction (×1,000). **C)** Penetration of a 1-day-old sorghum seedling at the soil surface (×1,000). **D)** Hypha (h) which advanced laterally, following penetration, toward the center of the stem (×600).

Inoculated seedlings were removed at 1-day intervals for 6 days, beginning at 0800 hours the first day after inoculation, then at 1-wk intervals for 4 wk. The seedlings were sectioned longitudinally, cleared in hot lactophenol, and mounted in either lactophenol or lactophenol cotton blue. The sections were examined microscopically for penetration and hyphal invasion of tissue.

RESULTS

Systemic infection resulted in plants of all cultivars exposed to conidia in the field or inoculated in the greenhouse (Table 1, 2). Higher percentages of infected plants were obtained from artificial inoculations than from natural inoculations in the field. Susceptibility to systemic infection decreased with the age of plants in all cultivars. Sudangrass was more susceptible than sorghum and remained susceptible longer than sorghum. With the exception of one plant, sorghum plants 13 days old when inoculated either naturally or artificially failed to develop systemic symptoms (Table 2). None of the 17-day-old sorghum plants developed systemic symptoms. A few 17-day-old sudangrass plants were systemically infected. No systemic symptoms developed on noninoculated seedlings.

TABLE 2. Effect of age of plant at inoculation on systemic infection of sorghum C48A inoculated with conidia of *Sclerospora sorghi* at 1, 7, 8, 13, and 18 days after emergence

Age when inoculated (days)	Total plants exposed (no.)	Systemically infected (no.)	Plants infected (%)
1	213	94	42.7
7	120	17	14.2
8	75	5	6.6
13	162	1	0.6
18	144	0	0.0
Check	182	0	0.0

Penetrations by germ tubes of conidia were observed in both sorghum and sudangrass. Penetrations were found most often in plants inoculated when 1 day old. These plants were approximately 1-1.5 cm tall at the time of inoculation and true leaves were still enclosed by the coleoptile (Fig. 1-A). Most penetrations occurred at the level of the soil surface where conidia had landed either in contact with, or near, the young plants. Entry usually occurred at the junction of epidermal cells (Fig. 1-B, C), However some epidermal cell walls were penetrated directly. Penetration was observed at 0800 hours following spore release during the previous night. Intercellular hyphae developed laterally following entry (Fig. 1-D), then vertically toward the apical meristem

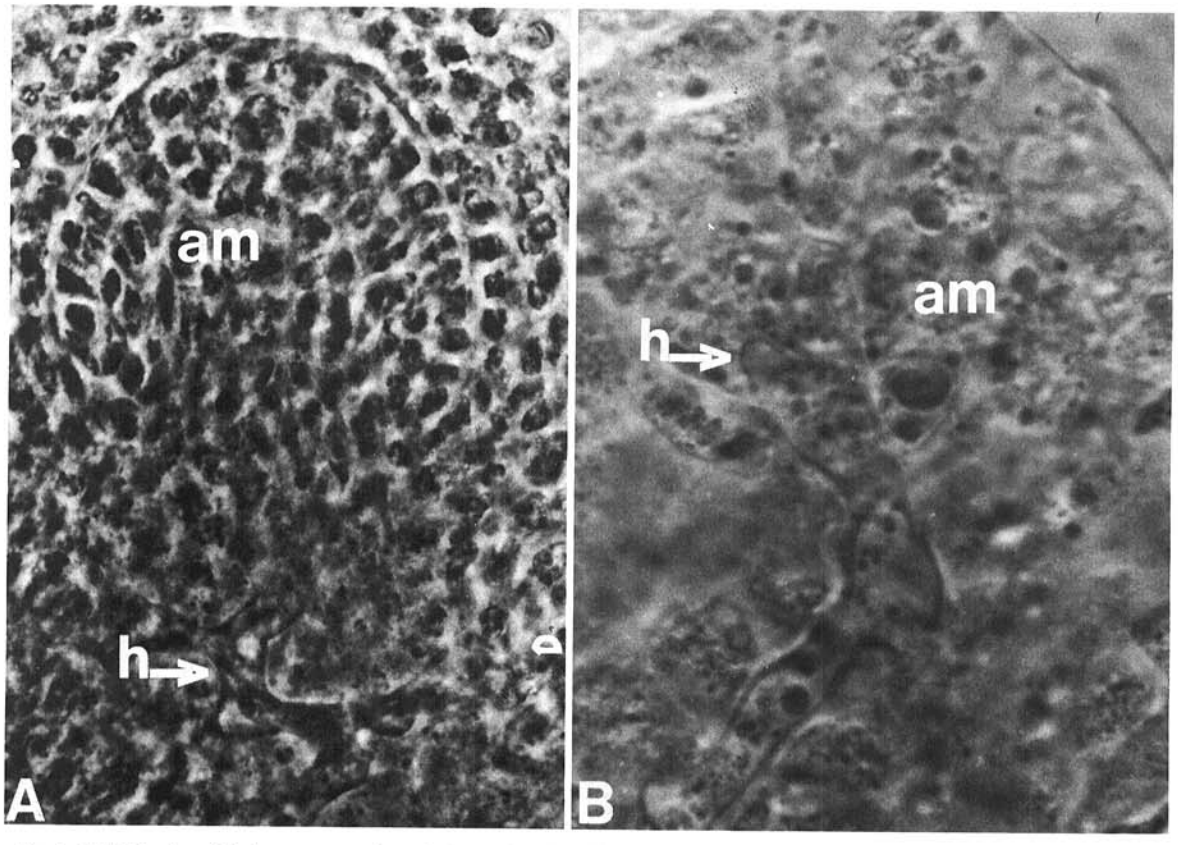

Fig. 2-(A,B) Hyphae of *Sclerospora sorghi* at the base of, and within the apical meristem of sorghum. **A)** Hypha (h) at the base of the apical meristem (am) 3 days after inoculation (×600). **B)** Hypha (h) within the apical meristem (am) (×1,000).

(Fig. 2-A). Hyphae were observed near the base of the apical meristem 3 days after inoculation. Hyphae invaded the apical meristem of several plants (Fig. 2-B), but in most, the fungus developed only to the base of the apical meristem. This was observed in plants even after systemic symptoms had developed in leaves.

Systemic symptoms appeared 7-9 days after inoculation. Several plants without systemic symptoms contained hyphae of *S. sorghi* within the stem, but no hyphae were found near the apical meristem of these plants. The fungus was not observed in the leaves of these plants. The sorghum plants appeared to be normal and grew as well as plants without the pathogen.

DISCUSSION

Local lesions generally are considered to be caused by conidial inoculum while systemic infections are believed to be caused by soilborne oospore inoculum (2, 5, 7). The results of this study indicate that conidia also are an important source of inoculum for systemic infections. Host plants that have just emerged are succulent and easily penetrated. The apical meristem is in close proximity to the soil and is protected only by the coleoptile. It is relatively easy for *S. sorghi* to penetrate, advance to the vicinity of the apical meristem, and invade the young leaves and leaf initials.

The fact that hyphae of *S. sorghi* were found in the stem but not in or near the apical meristem of normal-appearing plants indicates that *S. sorghi* can infect leaves systemically only by entering the leaves before they are fully formed. Infection apparently occurred in these plants when they were seedlings, but plant elongation prevented the pathogen from reaching the apical meristem. This could explain the occurrence of the late systemic phase of *S. sorghi* in sorghum. It is feasible that sorghum plants that display systemic symptoms late were actually infected early, either by means of conidia or by oospore inoculum. However, the apical meristem stayed ahead of the pathogen. Leaves that developed during this period were not infected. Eventually hyphae of *S. sorghi* reached the apical meristem, leaves not fully developed were invaded, and systemic symptoms were expressed.

LITERATURE CITED

1. FREDERIKSEN, R. A., and D. T. ROSENOW. 1967. Downy mildew of sorghum. Proc. 5th Bien. Grain Sorghum Research and Utilization Conf. 5:45-47.
2. KENNETH, R. 1970. Downy mildews of graminae in Israel. Indian Phytopathol. 23:371-377.
3. JONES, B. L. 1970. A simple technique of inoculating sorghum with Sclerospora sorghi using conidia as inoculum. Plant Dis. Rep. 54:603-604.
4. JONES, B. L., and R. A. FREDERIKSEN. 1971. Techniques for artifically inoculating sorghum with Sclerospora sorghi. Proc. 7th Bien. Grain Sorghum Research and Utilization Conf. 7:45-47.
5. SAFEEULA, K. M., and M. J. THIRUMALACHAR. 1955. Resistance to infection by Sclerospora sorghi of sorghum and maize varieties in Mysore, India. Phytopathology 45:128-131.
6. UPPAL, B. N., and M. K. DESAI. 1932. Two new hosts of the downy mildew of sorghum in Bombay. Phytopathology 22:587-594.
7. WESTON, W. H. JR., and B. N. UPPAL. 1932. The basis for Sclerospora sorghi as a species. Phytopathology 22:573-586.