6. SORGHUM DOWNY MILDEW IN THE UNITED STATES

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Sorghum downy mildew (SDM) appeared in the Western Hemisphere in South Texas in 1961 (5). During the past decade SDM, caused by the fungus *Sclerospora sorghi,* has become a major disease of sorghum, corn and related species in Texas (2). In 1969 the disease caused an estimated $2,500,000 loss to those crops in the Gulf Coast area of Texas. The progressive spread of SDM throughout the region of the southern States and into several central States bordering the Corn Belt has aroused national concern over the potential threat of tropical downy mildews (2). The current distribution of SDM includes 65 Texas countries that produce corn and grain sorghum on about 2.6 million acres of crop land. Another 3 to 4 million acres are threatened. In addition to Texas, SDM has been found in 12 other States: Alabama, Arkansas, Georgia, Indiana,

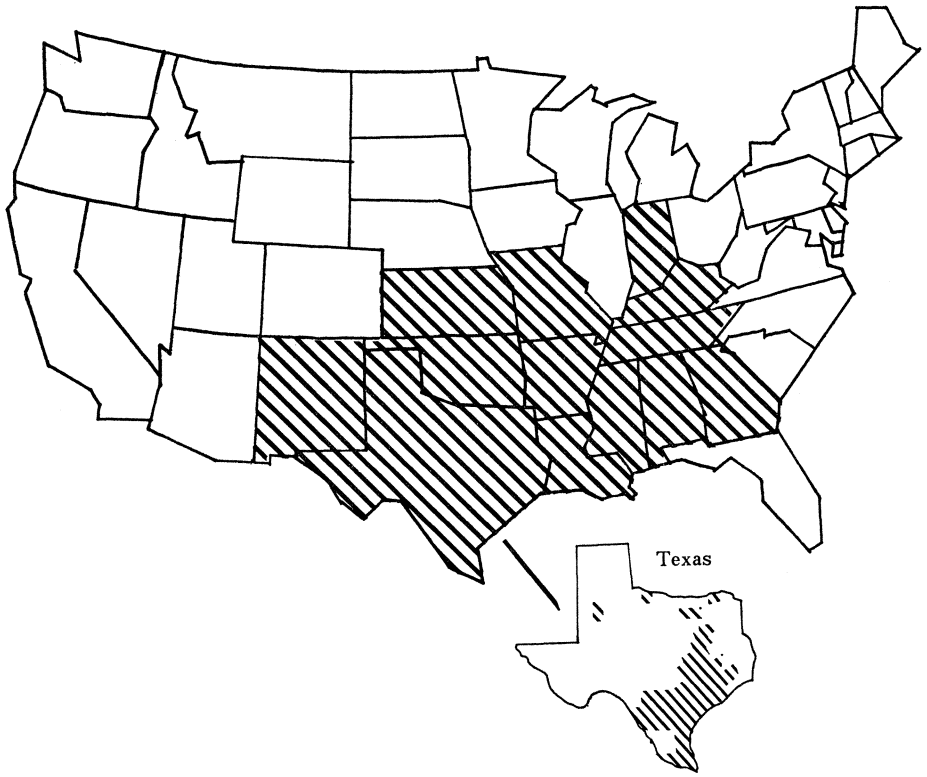

Fig. 1. Geographic distribution of *Sclerospora sorghi* in the United States, 1973; reported occurrence in States covered by shaded area; occurrence in Texas counties noted by shading on map insert

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Kansas, Kentucky, Louisiana, Missouri, Mississippi, New Mexico, Oklahoma and Tennessee (2, 3, 6) (Figure 1). Therefore the disease has reached the Corn Belt where conditions, such as those occurring during the 1970 Helminthosporum blight, may promote another disastrous epiphytotic. The value of corn and sorghum produced in 1973 was in excess of 12 billion dollars. Sorghum downy mildew, was reported from 13 states, producing 22 percent of the corn and 78% of the grain sorghum. Spread of the disease to areas distant from previously reported sites may however be more easily controlled than the Helminthosporum blight, since spread probably results from introduction of sorghum seed infested with oospores of *S. sorghi* (2), and not from windborne spores.

Although SDM has become widespread, its intensity still is greatest along the lower Gulf Coast. During the past decade research on the nature and control of SDM has centered in Texas (1). Laboratory work involving studies on infection processes and inoculation methods has been carried out at College Station and Weslaco while field work involving etiological studies and nurseries for screening, chemical and cultural methods of control has been conducted in South Texas near Beeville and Victoria. Because they are strategically unique, those nurseries are shared with cooperating plant pathologists and plant breeders located throughout the United States and Mexico.

Research Programs in the United States

This report reviews principally the research program of the Texas Agricultural Experiment Station, however much progress on asexual conidial production, inoculation techniques and disease development in corn and sorghum is being carried out by Dr. Chris Schmitt at the Plant Disease Research Laboratory, U.S. Department of Agriculture, Frederick, Maryland. The Texas program has been reviewed recently in two papers (1, 2).

Our current research objectives stress methods of control of SDM needed to reduce or eliminate losses from the disease in areas of the United States where it presently exists and preclusion of its occurrence in areas currently free of SDM.

We envision several possible approaches to control SDM, namely, development of resistant corn and sorghum cultivars and disease loss reducing or eliminating cultural practices and fungicides.

More specifically, the program can be divided into five separate projects:

I. Etiology of *S. sorghi*
II. Methods of inoculation with *S. sorghi*
III. Modes of inheritance of resistance to *S. sorghi* and extent of physiologic variability in *S. sorghi*
IV. Nature of host-parasite interactions
V. Biological and chemical control of sorghum downy mildew

I. Etiology of *S. sorghi*—Studies on identification, distribution, symptoms, damage, histopathology, spore germination, mode of infection, and cardinal temperatures have been reported (2, 5), but certain aspects of the infection process and survival of *S. sorghi* need further clarification. Currently, we are particularly anxious to broaden our knowledge of the production and fate of inoculum. We know that both oospores and conidia are etiologically important in SDM (4). Those spores, however, produced at different times in the disease cycle differ in their ability to cause plant infection. In Texas, we find that conidial inoculum results in higher incidences of infection and tends to negate resistance to infection by oospores. Changes in soil temperature affect the amount of SDM that develops in sorghum. Climatic conditions and date of planting influence incidence of SDM in corn more than in sorghum. Although the disease is of

tropical origin, oospores *S. sorghi* are capable of surviving winter temperatures as low as occur in the Corn belt (7). Information is needed, therefore, to establish the climatic limitations of oospore longevity and survival, independent of and interacting with host crop and cultural practices.

Cool moist nights and warm days common to temperate environments, and production of highly susceptible crops such as sorghum-sudan grass hybrids, shattercane (a feral sorghum), intensify the possibility of epiphytotics from conidial inoculum. The period of viability of conidia is short but so is the period required for conidial germination, host penetration and establishment of infection (2).

II. Methods of Inoculation with *S. sorghi*—We screen most sorghum and corn cultivars for resistance to SDM in field-grown nurseries. Results already obtained from such nurseries have been of great value, but these nurseries have serious limitations and faults. They can be conducted during selected times in the growing seasons; the incidence of disease at a given nursery site may vary widely from year to year, and within a given year. Furthermore inoculum potential within a site is usually sufficient to prevent detection of escapes among susceptible genotypes. The cost per test entry is much higher in such nurseries than in equivalent trials with seedlings in the greenhouse. A reliable method of greenhouse testing for host reaction to *S. sorghi* permits tests to be conducted during the whole year with more consistent results than is possible in field tests. Much progress has been made in developing greenhouse tests. Currently, conidia are used as inoculum in greenhouse trials because they can reliably produce high levels of infection. However, oospores represent the bulk of naturally occurring inoculum and comparisons between disease reactions produced by these forms of inoculum are at times striking (Table 1). Consequently, data from both inocula are necessary.

Table 1. Incidence of sorghum downy mildew following inoculation with conidia, oospores and in infested soil. (Selected data from unpublished study by Jarupong Boon-Long)

Corn Inbred	Inoculation techniques[1]		
	Oospore infested soil	Oospore coated seed	Conidial
Tx 601[2]	0	1.1	7.2
Tx 441	2.9	3.0	67.2
Tx 303	80.2	57.5	62.0

1) Techniques are described in Sorghum Downy Mildew Monograph (2)
2) Each test is based on about 80 inoculated plants.

Oospore inoculum frequently fails to produce infection and consequently further investigation on oospore survival and germination are planned.

III. Modes of Inheritance of Resistance to *S. sorghi* and Extent of Physiologic Variability in *S. sorghi*—Resistance in sorghum appears dominant, and the reaction to SDM in some sorghum cultivars involves more than one gene. Resistance in corn appears to be of at least 2 types, one operative against both soilborne and airborne inoculum (conidia), and another operative only against soilborne inoculum (2). One major locus is involved in the reaction of certain corn cultivars. Resistance is dominant in some crosses and recessive in others, but in such cases the probability is high that more than one pair of alleles is involved.

IV. Nature of Host-Parasite Interactions—The downy mildews have many phases of host-parasite interaction, (2). *S. sorghi* colonizes its host either or both locally and systemically, (4). Local infections follow initial invasion of leaf tissue from germinating conidia and result in necrotic local lesions. Systemic infections from germinating oospores may become evident in seedlings or somewhat older plants, or in leaves expanding after extensive local lesion development has occurred in older leaves. Extensive chloroisis, often limited by leaf veins, appears among seedlings. We have established criteria for 5 distinct phases of host parasite interaction, based on host maturity and pathogen reproduction and tissues affected (2).

These phases are more distinct in sorghum than in corn. To facilitate genetic analyses of resistance, we need to define host parasite interaction more clearly. While very subtle changes in seed quality (2) and host nutrition (2) affect SDM reactions, we are interested in the direct interaction of host and parasite.

V. Biological and Chemical Control of Sorghum Downy Mildew—Biological agents probably account for destruction or inactivation of tremendous quantities of inoculum. Oospores observed in the laboratory are readily invaded by bacteria and other miroorganisms. The parasitism inferred by such invasion may be a major factor in reducing viability of those oospores. Studies are being conducted to identify the invading organisms, and to determine the extent and effect that such invasion has on oospore survival and infection potential. Cultural control by promoting activity of naturally occurring predators, parasites, and antagonists of *S. sorghi* could reduce inoculum and consequently the incidence of disease.

Little research on chemical control of downy mildews of grasses has been conducted in the United States. In Texas, potassium azide effectively reduced soilborne inoculum of *S. sorghi,* but it was not effective as a foliar fungicide. In view of the progressive spread of SDM, additional efforts to evaluate chemical control of the disease is warranted. Selected chemicals would be evaluated for fungistatic or fungitoxic action against oospores in the soil and for protectant or disease eradicant properties when applied to the foliage. Time and method of application would be determined for all promising materials found.

Literature Cited

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Question and Answer

S. Y. Mah, Malaysia: Plants infected with downy mildew are either barren or produce nubbins only. Could it be that the plants infected at young stages become totally barren whereas those infected at later stages start producing ears that turn into nubbins when the disease sets in? What are your views on this?

Answer: It may be entirely possible that barrenness is correlated with early infection and the development of nubbins follows a somewhat later infection. This has not been established experimentally, however.

The formation of nubbins or failure thereof (barrenness) may well be related to massiveness of infection or the particular tissues invaded by the pathogen.

S. Lal, India: You have shown split maize stalks showing discoloration. Is it due to the downy mildew pathogen or due to some other?

Dr. Frederiksen has reported in his paper that potassium azide effectively reduced soil borne inoculum. What was his method and dose of application?

Answer: The discolored pith was apparently sterile—no fungus or bacterium could be isolated. The discoloration was assumed to be a result of infection by *Sclerospora sorghi.*

The potassium azide was applied and worked into the soil. For details, see; "Sorghum downy mildew—A disease of maize and sorghum". Texas Agr. Exp. Station Res. Monograph 2, 32 pp., illust. (R. Frederiksen ed.).

R. Kenneth, Israel: In Jubilee sweet corn, we found that when a stricken plant produces ears, they are usually on very long shanks coming out of the main stem. Have you seen symptoms such as these? Tassels may occasionally sprout at the distal end of the ear.

Answer: The abnormal extension of shanks is observed in both sorghum downy mildew and in crazy top. In the slides of symptoms observed in Indiana, the formation of tassels on the end of the ears was shown.

K. M. Safeeulla, India: It has been found in India that zinc deficient soils show a higher percentage of sorghum downy mildew on sorghum.

Answer: No comment.

J. Singh, India: Can we have some quarantine suggestion for checking the spread of downy mildew within the same country?

May I know something more about the role of zinc in controlling sorghum downy mildew, particularly the level and time of spray? Will soil application be good enough for control?

Answer: Most infected plants are barren. This in itself eliminates much of the chance of seed transmission. Secondly, nubbin ears on infected plants should not, or would not be used as a source of seed. Thirdly, when seed corn is dried at about 40°C, internally borne mycelium is killed.

These facts tend to minimize or obviate international spread of the pathogen.

Zinc applied to soils (not deficient in zinc) tended to reduce severity of sorghum downy mildew. For details, see "Sorghum downy mildew", Texas Agr. Expt. Sta. Res. Mono. No. 2, 1973.

T. Hino, Japan: I would like to know the situation of corn downy mildew in Central and South America. What kind of pathogen is the most prevalent?

Answer: The disease has been found in several South and Central American countries according to observers in those areas.