Richard A. Frederiksen
Texas A&M University, College Station

Sorghum Downy Mildew in the United States: Overview and Outlook

Fig. 1. Striping of sorghum leaf with sorghum downy mildew.

Fig. 2. Maize seedling with sorghum downy mildew.

The downy mildews invariably cause considerable alarm and nearly always invoke quarantine measures. The following is from a letter dated 21 February 1916 and written by Walter T. Swindale, a physiologist with the Bureau of Plant Industry in Washington, DC:

"While in the Orient I had occasion to observe the action of a very dangerous corn disease, *Peronospora maydis*, which often destroys the crop entirely. Under rather unfavorable conditions for its spread I saw the disease causing great ravages and destroying in the fields I examined 77% of the plants. I learned last week that another corn disease of similar character, *Sclerospora sacchari*, causes very grave damage to corn in Formosa. I believe it is desirable to take steps immediately to prevent these diseases from reaching the United States and I would be glad to appear before the Federal Cultural Board and explain the situation in detail."

On 15 March 1916, a public hearing on corn diseases was announced. The notice stated: "In the Philippines a downy mildew attacks corn very destructively causing in some cases total loss of the crop. All of these downy mildews are favored by warm, moist weather such as is considered to be ideal corn growing weather in the Mississippi Valley. It is probable that if they succeed in entering the United States and got into the Mississippi Valley they would be able to cause immense damage." The outcome of this hearing was Corn Diseases Quarantine No. 24, reflecting the excellent insight of the early maize workers.

It was not until the summer of 1961, 45 years after Corn Diseases Quarantine No. 24 was initiated, that a tropical downy mildew disease similar to those noted in the initial quarantine statements was found in the United States. The disease is sorghum downy mildew (SDM), caused by *Peronosclerospora sorghi* (Weston & Uppal) C. G. Shaw (Figs. 1–3).

Following SDM's appearance during 1961 and 1963 in Texas, minor but important epiphytotics were observed periodically. In 1967 these became widespread along the Upper Coast and Coastal Bend regions of Texas. During the next few years, downy mildew was observed in several adjacent states and in sorghum-growing regions of the United States. By the early 1970s, downy mildew had reached the Corn Belt, appearing first in Kentucky and Tennessee and later in Indiana and Illinois. SDM did not thrive in the Corn Belt, as the early observers had suggested, however.

Distribution of SDM

Since its detection almost two decades ago, downy mildew has spread to include 16 states. The initial observations of SDM were on the Texas Agricultural Experiment Station farms at College Station, Chillicothe, Beeville, and Crystal City (7); from 1958 through the growing season in 1963, however, sorghum diseases were not routinely monitored in Texas. M. C. Futrell, a USDA small grains pathologist, and several Texas A&M University students made the initial identification of SDM during 1961–1962.

The appearance of SDM in forage sorghums in Panama as early as 1956 (9) added to the mystery of the original introduction of the pathogen to the Americas. SDM appeared to spread in waves—at first slowly, as only minor occurrences were noted (16). By 1967 the first major epiphytotic developed along the Texas Gulf Coast (10). During 1967–1978 the pathogen appeared in, or was reported from, nine other states: Alabama, Arkansas, Georgia, Kansas, Louisiana, Mississippi, New Mexico, Oklahoma, and Tennessee (11). The disease was nearly always reported in a sudangrass or sorghum-sudangrass hybrid. The appearance of SDM in Florida and Puerto Rico followed this pattern.

0191-2917/80/10090306/$03.00/0

Fig. 3. Maize tassel proliferation covered by *Peronosclerospora sorghi*.

During the early 1970s SDM was reported from Kentucky, Indiana, Illinois, Missouri, and Tennessee. In these states, SDM appeared in the Ohio River Valley and was associated with shattercane as a collateral host. Futrell demonstrated that infected shattercane produced abundant oospores that were the major source of inoculum for infection of maize. The 1978 outbreak in Nebraska completes the current distribution of SDM in the United States (Fig. 4). Shattercane appeared to be the source of inoculum in Nebraska, whereas the increased level of downy mildew in Kansas during 1978 was related more to the use of susceptible sorghum cultivars than to oospores produced by a collateral host (13,18).

How the Pathogen Is Spread

SDM can be disseminated by: 1) oospores on seed or with debris, by wind, or in soil from infested areas; 2) conidia from infected plants; and 3) mycelium in seed or in living hosts.

Oospores. Oospores are thick-walled resting spores that are produced in systemically infected sorghum and, to a lesser extent, in maize (*Zea mays* L.). They commonly live for 3 years under a variety of conditions and usually are not controlled by seed-treatment fungicides.

Oospores infest the soil as free spores (15) and are wind disseminated. Unfortunately, there are no detailed studies on how many and how far spores are carried by the wind, although reports of virgin maize or sorghum fields with high levels of downy mildew adjacent to fields with infected plants are common. Generally, systemically infected plants are barren or produce very few seeds. Glumes are usually removed in the normal harvesting operation but occasionally remain on or with the seed. In Texas we observe oospores in glumes, not in seeds.

In experimental trials, a small percentage of seeds with attached glumes from systemically infected plants produced infected seedlings. In contrast, no infected seedlings were produced when glumes were removed (10). Very few other studies show the rate and extent of seed contamination with oospores of *P. sorghi*. This may be due in part to the low frequency of infected plants producing seed and the difficulty of obtaining downy mildew infection with oospores (19).

Conidia. Conidia are very short-lived (only a few hours under ideal conditions) and probably play no role in the long-range distribution of inoculum. In Texas, disease spread by conidia has not been observed between fields only 100 m apart. Within fields and between plants, however, these spores play a major role in distribution, particularly in susceptible sudangrasses.

Mycelium. The only other possible method of pathogen dissemination is by mycelium within the seed. Mycelium has been observed in the embryo region of normal-appearing seed of sorghum (1), millet (17), and maize (12). This method of spread is often suggested, but there is little evidence to indicate that mycelium in dry mature seed results in infected plants (19). Our work, as well as that of others studying downy mildew of maize, has consistently shown that infected seed of sorghum or maize dried below 20% moisture show no evidence for survival of the pathogen (12,19).

Generally, oospores must be the source of contamination with seed or debris and the primary means for distributing the pathogen. Because the majority of hybrid sorghum seed is produced in Texas and because seed production is in an area generally considered free from downy mildew, the Texas Department of Agriculture issues Phytosanitary Export Certificates after inspection confirms the production field is free from downy mildew (Fig. 5). Inspection reduces the possibility of exporting oospore-contaminated seed. In both policy and practice, the Texas Department of Agriculture issues certificates based only on inspection of individual fields (5).

Search for Host Resistance

SDM appeared to be a disease that could be controlled with host resistance (Fig. 6). However, the early searches for resistant sorghums among open-pedigree sorghums revealed the vulnerability of most of these lines (14). Fortunately, in 1963 a program was initiated to broaden the genetic base of sorghum by introducing African and Asian sorghums and converting them to agronomically acceptable temperate types. Numerous sources of resistance were detected in materials produced from this Sorghum Conversion Program (7,10). These apparently resistant sorghums, along with other lines developed by commercial firms, led to the initial deployment of SDM-resistant sorghum hybrids in 1971. By 1975 essentially all sorghum acreage in areas threatened by SDM was sown to resistant hybrids. Even sudangrasses resistant to downy mildew were released in 1978. For maize, downy mildew resistance (DMR) was observed in southern as well as Corn Belt inbreds (6,7). Under all but the most severe conditions, the level of resistance to SDM in U.S. DMR maize hybrids appears adequate.

Initially, all screening for SDM resistance was done in the field. Now, downy mildew nurseries are maintained by growing highly susceptible sorghums the year preceding testing to incorporate higher levels of oospores into the soil. Several such "SDM sick plots" are maintained in South Texas by commercial seed companies as well as by the Texas Agricultural Experiment Station.

During the past decade, a variety of laboratory techniques have been tested and evaluated for screening sorghum and maize lines for resistance to sorghum downy mildew (2). Laboratory tests include both oosporic and conidial inoculations. To date, the conidial inoculation techniques have proved more favorable because of consistency in reproducing the inoculation conditions. Subtle differences, however, in reaction of maize and sorghum lines to the different types of inoculum have encouraged considerable research into the survival and germinability of oospore inoculum (3). A major problem with these inoculation procedures has been the rather frequent appearance of escapes.

During the infection process, conidia form local lesions (Figs. 7 and 8). Some sorghum entries vary in reaction to the development of these lesions (Table 1). The reaction of sorghum lines to local lesions is closely related to their field

reaction to SDM. In crosses between some of these lines, recovery of parental types was observed in several F_2 families. Progeny from F_2 plants reacted similarly in the F_3 generation. This indicates that 1) there is an important relationship between the resistant lines to local lesions and their resistance to SDM in the field and 2) individual plants with local lesion reactions can be scored. Thus, one can identify individual plants possessing resistance in the F_2 and avoid the problem of escapes. Not all downy mildew resistance in sorghum or, for that matter, in maize is associated with resistance to local lesions. When combined with field evaluation, however, the resistance level observed with the local lesion technique is high enough to provide an acceptable method for evaluating in the early generations.

A Concept of DMR

Currently we base our concept of DMR on the incidence of systemic infection in a population of plants. Resistance and susceptibility are difficult to differentiate, however. For example, I know of no hybrid or inbred that is absolutely free from systemic downy mildew under conditions ideal for infection. Generally, plants become more resistant as they mature (20), ie, 1- and 2-day-old plants are more susceptible than 4- and 5-day-old plants. Plants known to be resistant in the field develop resistance to infection faster than those known to be susceptible. Types of inoculum and date of planting are also important. Oospores of *P. sorghi* are the principal inoculum in Texas, but abundant conidia also cause substantial infections, particularly in sorghum-sudangrass hybrids. Some cultivars are resistant to oospore infection but moderately susceptible to infection by conidia (3,20). Similarly, cultivars planted at different dates may have substantially different levels of downy mildew. Levels are lower in resistant hybrids than in susceptible ones.

Another subtle aspect of this concept relates to the genetic purity of the hybrids. One can almost always expect a low percentage of outcrosses or self-pollinated plants in seed. These could be susceptible to downy mildew. Fortunately, many grain sorghum seed parents are not nearly as susceptible to downy mildew as are the more susceptible pollinator lines.

Another factor that must be considered is the effect of downy mildew on production. We know that high levels of systemic infection (20%) are necessary in sorghum before losses are measurable. Plant densities must also be considered for maize (Table 2). The distribution of downy mildew reactions from a number of hybrids grown in South Texas over the past 3 years indicates that the incidence of downy mildew in the "resistant hybrids" generally is 6% or less under conditions of a severe epidemic.

Finally, the number of diseased individuals in a population of sorghum plants needed to maintain the pathogen in a field year after year is not known. In other words, how many oospores are needed to maintain a particular level of disease, and to what extent does the oospore population increase or decrease with a given level of systemic infection?

Consequently, SDM disease reactions can be placed into four convenient categories: highly resistant (R), moderately resistant (MR), moderately susceptible (MS), and susceptible (S). These reactions are based in part on our concept of host

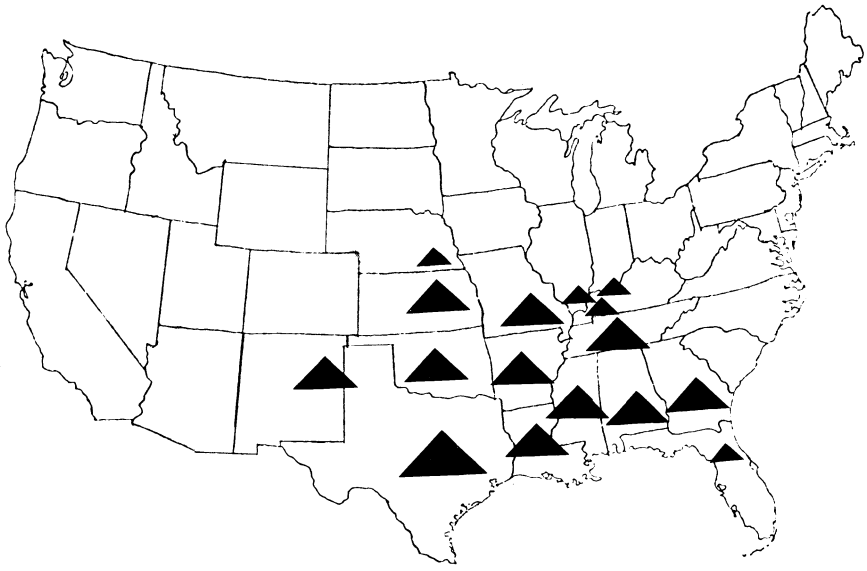

Fig. 4. Distribution and spread by state of sorghum downy mildew from 1961 to the present. Large triangle = 1961–1966; medium triangles = 1967–1970; small triangles = 1971 to present. Sorghum downy mildew was observed in Puerto Rico during this last period.

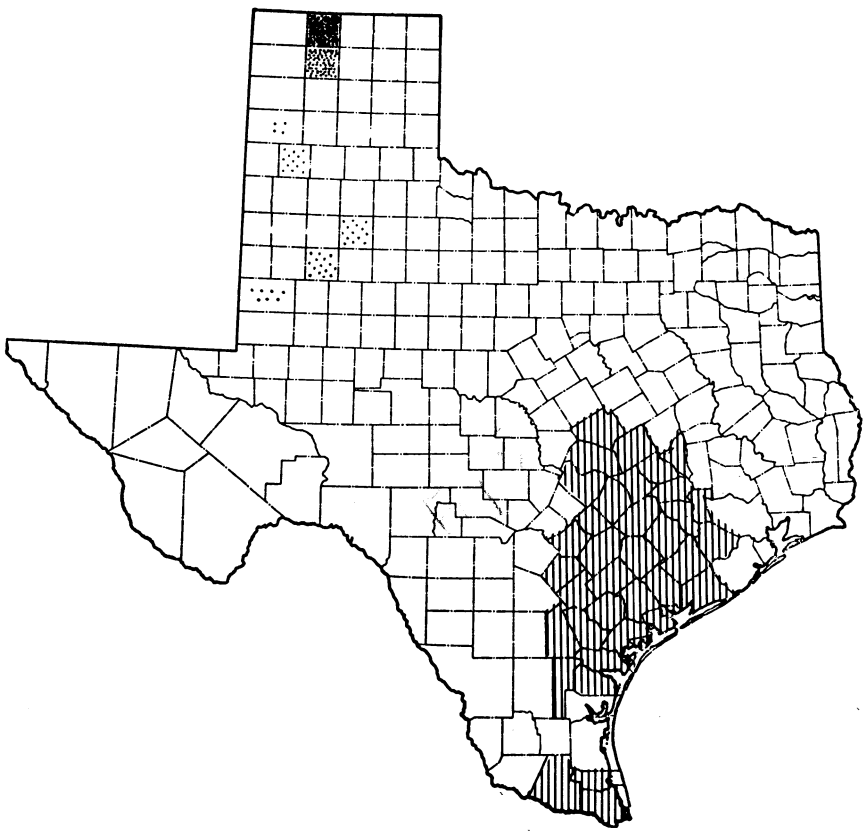

Fig. 5. Dots indicate areas of certified hybrid grain sorghum seed production in Texas during 1978 and 1979 (each dot represents 10 ha), and stripes indicate areas of endemic sorghum downy mildew. Occasionally, sorghum downy mildew occurs in nearly all sorghum-growing regions of Texas.

Canada has been an active cooperator from the onset and has maintained close ties through an officially appointed consultant to the committee.

IR-2's general objectives are to: 1) obtain apparently virus-free valuable cultivars and clones of deciduous fruit trees, verify their freedom from viruses, maintain them in isolated repositories, and distribute small amounts of propagating materials to research or regulatory scientists for research or release to industry; 2) develop virus-free individuals by any method from cultivars and clones with no known virus-free individuals from which horticultural propagations can be made; and 3) do research on techniques, viruses, and host plants with emphasis on improving repository performance. The nature of these objectives mandates a strong orientation toward plant pathology.

Procedures of IR-2

IR-2 normally receives requests to acquire specific clones from identified sources. Requests come from interested scientists, state regulatory personnel, and leading members of industry and sometimes from IR-2 personnel in anticipation of future requirements. The needs for each clone are evaluated, and propagation materials of the desirable ones are obtained. In this way, IR-2 acquires clones with immediate scientific, commercial, and/or practical interest.

All candidate clones are indexed to verify freedom from specific viruses universally infecting that species or infecting a certain species from particular area. A candidate clone found to be infected during this preliminary indexing is usually discarded, and an attempt is made to find a different, virus-free source. Sometimes the candidate is saved for thermotherapy.

Current IR-2 indexing procedures are based on graft-inoculated woody indicators. Herbaceous indicators, with one exception, have not been as accurate or conveniently handled as woody plant indicators. The exception is *Chenopodium quinoa* Willd., which will detect a few extremely mild strains of the apple chlorotic leaf spot and apple stem grooving viruses that cannot be detected

Fig. 1. Evolution of indexing for the apple chlorotic leaf spot virus using the apple virus indicator R 12740-7A: (Top) Original indexing methods required large, field-grown indicator trees for bud inoculations. Production was expensive and labor-intensive, and completed results usually required 2 years. (Middle) With the double-budding technique of field indexing, three inoculated indicators and one control per clone tested were used. Completed results required about 9 months. (Bottom) Current indexing methods use a greenhouse space 30.5 × 30.5 cm for 20 indicators. Completed results require less than 4 weeks.

season, with effective inoculum dropping off rapidly in the third growing season.

Periods of rainy weather after planting tend to reduce the incidence of systemic SDM in sorghum. The effectiveness of this as a management activity could be overestimated, however, particularly in dry land agriculture.

Chemical Control of SDM

During the past 4 or 5 years, metalaxyl [N-(2,6-dimethylphenyl)-N-(methoxyacetyl)-alanine methyl ester, CGA 48988, Ciba-Geigy Corp.] has controlled downy mildew diseases in many regions of the world. Metalaxyl significantly reduces the incidence of SDM in sorghum at rates as low as 0.1 gm a.i./kg of seed (8). These reductions are related to corresponding increases in performance (Table 2). In Texas, metalaxyl effectively controlled oosporic and conidial inoculum (8).

Table 1. Reaction of selected lines of sorghum to *Peronosclerospora sorghi*, pathotype 1

Line	Field reaction[a]	Reaction after conidial inoculation: Local lesions[b]	Reaction after conidial inoculation: Systemic infection (%)
IS12661 der. (SC170-6-17)	R[2]	1.5	0
IS2508 (SC414-12)	R	1.1	0
IS12612 (SC112-14)	R	1.1	0
CS3541	R	1.1	0
Tx430	R	1.5	0
Tx7078	S	4.5	50
IS12610 (SC110-14)	R	3.0	41
IS3757 der. (SC326-6)	MR	3.8	13
IS2930 × IS3992 (77CS1)	S	2.5	53

[a] Under conditions in South Texas. R = resistant (less than 6% infection); MR = moderately resistant (6–10% infection); S = susceptible (more than 20% infection).
[b] Based on scale of 1–5: 1 = essentially not visible, 2 = small and necrotic, 3 = larger and with some chlorosis, 4 and 5 = permit rapid colonization.

Table 2. Effect of sorghum downy mildew on yield of a commercially developed grain sorghum hybrid at Beeville, TX, 1980

Treatment with metalaxyl[a] (gm a.i./kg seed)	Plant density (plants/m)	Downy mildew (%)	Yield: kg/ha	Yield: % of control
1	16.7	7.8	2,959	
0	15.6	63.8	1,569	53
1	23.0	3.0	3,475	
0	22.8	58.8	2,292	64,76[b]

[a] N-(2,6-dimethylphenyl)-N-(methoxyacetyl)-alanine methyl ester (CGA 48988), Ciba-Geigy Corp., Agricultural Division, Greensboro, NC.
[b] 64% with high and 76% with low plant density.

Progress Has Been Steady But Problems Remain

The appearance of SDM in the Western Hemisphere continues to attract considerable attention, and progress in containing and controlling the disease has been steady. SDM has been reported from locations where the oospores are known to overwinter or where a collateral or primary host produces oospores, but the principal maize-growing regions are safe from SDM because maize produces few oospores. Similarly, other tropical downy mildew fungi, such as *P. sacchari* and *P. philippinensis*, may not pose a threat to the Corn Belt because of the lack of collateral hosts capable of producing oospores. In tropical regions, these fungi appear to survive asexually—a most unlikely event during January in the Corn Belt states.

In the Americas, host resistance to SDM is sufficiently high in both maize and sorghum. Where a single source of resistance has been used for a number of years, variations in the pathotypes of the organism have been observed. Fortunately, numerous other sources of resistance to downy mildew are available. Local lesion types and field resistance can now be identified, permitting deployment of even higher levels of resistance to SDM and increasing the ease of incorporating resistance in breeding programs. Adoption of cultural control procedures and development of chemical controls will further reduce disease severity and losses.

The downy mildews are tropical in origin and nature and continue to be threatening in the tropical and semitropical areas of the world. The principles of disease control that have reduced the threat of SDM in the vulnerable regions of the United States can also be applied in the tropics, but much more in-depth understanding of the downy mildews is urgently needed.

Participants at a recent conference on graminaceous downy mildews addressed the following questions:

1. What are the major epidemiological constraints that need to be overcome during the next 5 to 10 years to reduce the severity of graminaceous downy mildew diseases?
2. What are the deficiencies in our knowledge of physiological processes of the host and/or pathogen whose understanding could help in developing controls for graminaceous downy mildew diseases?
3. What are the major limitations to the practical control of the graminaceous downy mildew diseases affecting maize, sorghum, and millet?

The conference identified 11 major problem areas needing solution in order to answer the three major questions:

1. Clarify the taxonomic confusion currently existing in graminaceous downy mildews.
2. Identify the sources of primary inoculum.
3. Determine the physiological and environmental factors controlling spore production and germination.
4. Determine the influence in inoculum type, quantity, and placement on disease reaction.
5. Identify critical factors affecting successive phases of the infection cycles of all spore types.
6. Determine the genetic basis for i) DMR in sorghum, maize, and millet and ii) variation in virulence in their pathogens.
7. Improve selection methods in breeding programs.
8. Determine those factors that contribute to and/or affect stability and durability of resistance.
9. Determine the mechanisms of susceptibility, tolerance, and resistance.
10. Develop hazard indices for specific geographical areas and formulate integrated control programs to accelerate and stabilize production in these designated regions.
11. Apply control methods to production systems by means of national and regional research programs.

Many of these questions are being investigated, and the answers will greatly enhance our knowledge of the downy mildews.

Acknowledgments

I wish to acknowledge the extensive contributions to downy mildew research in Texas made by J. Craig, M. Futrell, G. Odvody, R. Pratt, and B. Jones as well as the many enthusiastic graduate students and technicians whose work I have liberally utilized in this paper.

Literature Cited

1. BAIN, D. C., and W. W. ALFORD. 1969. Evidence that downy mildew (*Sclerospora sorghi*) of sorghum is seed borne. Plant Dis. Rep. 53:802-803.
2. CRAIG, J. 1976. An inoculation technique for identifying resistance to sorghum

downy mildew. Plant Dis. Rep. 60:350-352.
3. CRAIG, J. 1980. Comparative reactions of corn inbreds to oospore and conidial inoculum of *Peronosclerospora sorghi*. Phytopathology 70:313-315.
4. CRAIG, J., and R. A. FREDERIKSEN. 1980. Pathotypes of *Peronosclerospora sorghi*. Plant Dis. 64:778-779.
5. FREDERIKSEN, R. A. 1980. Seed transmission of *Peronosclerospora sorghi* in grain sorghum: How can it be avoided? Tex. Agric. Exp. Stn. Misc. Publ. 1453.
6. FREDERIKSEN, R. A., A. J. BOCKHOLT, L. REYES, and A. J. ULLSTRUP. 1971. Reaction of selected midwestern corn inbred lines to *Sclerospora sorghi*. Plant Dis. Rep. 55:202-203.
7. FREDERIKSEN, R. A., A. J. BOCKHOLT, D. T. ROSENOW, and L. REYES. 1970. Problems and progress of sorghum downy mildew in the United States. Indian Phytopathol. 23:321-338.
8. FREDERIKSEN, R. A., and G. ODVODY. 1979. Chemical control of sorghum downy mildew. Sorghum Newsl. 22:129.
9. FREDERIKSEN, R. A., and B. L. RENFRO. 1977. Global status of maize downy mildew. Annu. Rev. Phytopathol. 15:249-275.
10. FREDERIKSEN, R. A., et al. 1973. Sorghum downy mildew, a disease of maize and sorghum. Tex. Agric. Exp. Stn. Res. Monogr. 2. 32 pp.
11. FUTRELL, M. C., and R. A. FREDERIKSEN. 1970. Distribution of sorghum downy mildew in corn and sorghum. Plant Dis. Rep. 54:311-314.
12. JONES, B. L., J. C. LEEPER, and R. A. FREDERIKSEN. 1972. *Sclerospora sorghi* in corn: Its location in carpellate flowers and mature seeds. Phytopathology 62:817-819.
13. LENGKEEK, V. H., and T. SIM. 1979. An outbreak of sorghum downy mildew in Kansas. Plant Dis. Rep. 63:905-907.
14. MILLER, F. R., R. A. FREDERIKSEN, S.T. ALI KAHN, and D.T. ROSENOW. 1968. Reaction of selected sorghum varieties and lines to downy mildew. Tex. Agric. Exp. Stn. Misc. Publ. 890.
15. PRATT, R. G. 1978. Germination of oospores of *Sclerospora sorghi* in the presence of growing roots of host and non-host plants. Phytopathology 68:1606-1613.
16. REYES, L., D. T. ROSENOW, R. W. BERRY, and M. C. FUTRELL. 1964. Downy mildew and head smut diseases of sorghum in Texas. Plant Dis. Rep. 48:249-253.
17. SAFEEULLA, K. M. 1976. Biology and Control of the Downy Mildew of Pearl Millet, Sorghum and Finger Millet. Wesley Press, Mysore, India. 304 pp.
18. SIM, T., IV. 1980. Sorghum downy mildew in Kansas in 1979. Plant Dis. 64:499.
19. WILLIAMS, R. J. 1979. Seed transmission of some graminaceous downy mildews. Mimeo Rep. International Crops Research Institute for the Semi-Arid Tropics.
20. YEH, Y., and R. A. FREDERIKSEN. 1980. Sorghum downy mildew: Biology of systemic infection by conidia and of a resistant response in sorghum. Phytopathology 70:372-376.

Richard A. Frederiksen

Dr. Frederiksen is a professor of plant pathology in the Department of Plant Sciences at Texas A&M University, College Station. He began his studies on sorghum downy mildew in 1964 and since then has traveled and worked extensively in endemic downy mildew environments throughout the world.