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11–15 December 1978

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Preface

Sorghum is grown as a food, fodder, and feed crop on approximately 52 million hectares in tropical, subtropical, and temperate climates on six continents, producing an estimated 69 million tonnes of grain per annum. Yields of sorghum are low in most tropical less-developed countries, where the grain is needed for human food. Paradoxically, in countries where sorghum grain is used principally as animal feed, yields are much higher. Sorghum diseases are major contributors to this "yield gap," and if consistently higher sorghum yields are to be achieved in the tropical countries, stable disease resistance must be incorporated into cultivars with high-yield potential.

The International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), which has a world mandate for sorghum improvement, and the College of Agriculture, Texas A & M University, which has a long history in sorghum improvement research, recognize disease as one of the major factors limiting sorghum production. They jointly organized an International Workshop on Sorghum Diseases as a first step in the promotion of a worldwide collaborative effort on sorghum disease control. The workshop, hosted by ICRISAT at Hyderabad, India, 11–15 December 1978, received additional support from USAID, USDA, and IDRC, and was attended by 52 scientists from 20 countries in the Americas, Africa, Europe, Asia, and Australia. Fifty-six papers were presented in nine technical sessions, and considerable time was devoted to the discussion of research needs and priorities. Discussion groups prepared research recommendations that were finalized by the whole group at the final workshop session.

These proceedings, which contain the papers presented, edited transcripts of the discussions, and the final recommendations, represent the "state of the art" in sorghum pathology in December 1978, and we hope they will be a valuable source of information and inspiration to all involved in sorghum improvement.

The Editors

Opening Session

Chairman's Opening Remarks

R. J. Williams*

It gives me great pleasure to call to order this Opening Session of the International Workshop on Sorghum Diseases. I believe that this is the first time that so many sorghum pathologists, breeders, and other sorghum scientists from so many different countries of the Americas, Africa, Asia, and Australasia have gathered together to devote their efforts to the discussion of the "state-of-the-art" in sorghum diseases and breeding disease-resistant sorghums.

The seeds of the workshop were sown here in Hyderabad a little more than 3 years ago. A long vegetative period followed, and it was not until December 1977 that the workshop coordinators met to select the individuals who were to form the elite population assembled here today. Unfortunately, almost all the individuals selected were highly photoperiod sensitive and only began to produce their papers during the short days of November and December. However, as you will have judged from the weight of the bag collected in registration, the yields are high, and there appears to be no negative relationship between quantity and quality.

We now have 5 days to harvest all the results, the knowledge, the experience, and the ideas of this productive and experienced group of sorghum scientists. In order that we do not delay the harvest operations I must get this session underway, and I have great pleasure in calling upon Dr. Leslie D. Swindale, ICRISAT Director, to formally welcome you all to ICRISAT and to this workshop.

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* Principal Cereals Pathologist, (now Principal Pearl Millet Pathologist) Pearl Millet Improvement Program, ICRISAT.

Welcome Address

L. D. Swindale*

It is certainly a great pleasure for me to welcome you to Hyderabad, to ICRISAT, and to the International Workshop on Sorghum Diseases. It is a lovely time of the year to come to Hyderabad, and we hope that you all have a chance not only to enjoy the climate but also to see something of this extremely interesting and historical city in the middle of India.

I hope you will find that your deliberations are scientifically useful and interesting and even enjoyable, and that they will serve to advance the cause of improving the livelihood of the farmers and workers in the semi-arid tropics. We all know that each of you came here with your own particular purposes and interests. This is an international workshop, and you are not here solely to serve the purposes of ICRISAT or even the larger purposes of India. But the fact that the workshop is being held in India, where sorghum is such an important subsistence food, and under the partial sponsorship of ICRISAT with its mandate for the semi-arid tropics in general, will, I hope, allow you to give some special emphasis to improving sorghum as a food grain and particularly as a food grain of people who use the crop purely outside the cash economy.

Most of you know, of course, about ICRISAT and its mandate, or you would not be here. We have a global responsibility for the improvement of five crops — sorghum, pearl millet, pigeonpea, chickpea, and groundnut. We have a responsibility for improving the farming systems in the semi-arid tropics and the use of human and natural resources in these regions. We try to understand the socioeconomic constraints to improving agriculture in the semi-arid tropics and to evaluate alternative means to overcome these constraints. And, finally, our responsibility — our mandate — includes assisting national programs in research and extension through cooperation and support, hosting conferences such as this, and undertaking training programs. Within that large mandate, the Board of Governors of ICRISAT has decided we should focus immediate attention upon the target group of the small farmers of the semi-arid tropics, farming their lands without the benefit of regular regional irrigation. These are the poorest segments of the agricultural population in the semi-arid tropics and perhaps everywhere. Focusing our research efforts on them is very much in line with the requirements and wishes of our donors; this is what they want us to do, and they are very clear about this. I recently attended a meeting of all the donors of the Consultative Group on International Agricultural Research (CGIAR) held in Washington D.C., and though not all of them insisted that their funds be used for the poor segment of humanity and with a specific and clear social purpose, the great majority of them did. This applies not only to ICRISAT, but to the other International Agricultural Research Centers (IARCs) as well.

As regards our target group we do not serve it directly, that is not within our mandate. Rather we serve a client group of scientists and extension workers, particularly scientists such as you. It is our main responsibility: to do research and produce materials and information that are of use and value to people such as you, who in your turn serve the requirements of national purpose in the countries from which you come. Through you we serve the poor people of our target group.

I will mention briefly how we see ourselves serving you as a client group.

Firstly, in providing genetic resources, our international status allows us, we believe, a greater scope in collecting germplasm than is possible with individual national programs. Through this major advantage we can serve you by providing you, at your request, genetic material that suits your particular purpose.

Similarly, our internationalism allows us to work not only with each of your countries and

* Director General, ICRISAT.

your institutions individually, but also collectively, so that we have an advantage in helping to organize, coordinate, and carry out international programs — such as the international sorghum downy mildew nurseries — throughout the world, and to help collect and transfer the information from such international nurseries and activities from one contributing and participating institution and scientist to another.

Also, perhaps because we do not have anything like the same pressures and problems that you people have in national programs (although we certainly have our own), we are able to concentrate on trying to overcome some of the major blocks to research progress that individual scientists or institutions encounter. In much of our work we do try to explore large-size experiments, and particularly try to improve methodologies that will, we hope, advance the cause of science generally and the efficiency of your own work. We emphasize resistance to diseases and pests and tolerance to stress; these are tremendously important in ICRISAT's work because they relate to the target group that I mentioned, and by producing material that is stable over time and space, that is resistant, that is tolerant, we are aiming our work at this particular target group. To the extent that this material is suitable for what you are doing, it serves your purpose but sometimes perhaps it is not exactly what you want, because there are indeed other purposes for agricultural research. For example, the purpose that I've mentioned of producing sorghum mainly for food is not the major purpose for Texas A & M when it is working in Texas. Their purpose is different and quite properly and legitimately so. So the type of material that we produce may or may not be useful to them, but we can hope it will be.

One other point that I particularly want to mention, because it is not mentioned in your program, is that within our mandate we run training programs. Training is an important aspect of ICRISAT's work. I hope that some of you will be able to take advantage of our training programs, either directly or by encouraging younger scientists and younger workers in your programs and institutions to come to ICRISAT for training. These programs are primarily "dirty hands" programs; they are field-oriented and are not to give people academic background but teach them how to be efficient workers in the field and how to work efficiently with their colleagues in other programs.

These then are the things that I wanted to mention. They do not cover everything that ICRISAT does to serve its client group, but they include a good portion of it. Again let me welcome you to ICRISAT; I hope that you will take some time to understand our work and to see in what way ICRISAT can help you as well as the ways in which you can help ICRISAT discharge its responsibilities. Thank you very much.

The Importance of Sorghum in the Semi-Arid Tropics

J. C. Davies*

Sorghum is one of the five crops selected for research at ICRISAT and, considering its importance in the economics and very survival of the populations of many of the SAT countries, it is well chosen.

It is estimated that around 400 million people live in the 49 countries represented in the low rainfall, seasonally dry tropical areas of the world and 48 of these are classified as less developed countries (LDCs). The region with the largest population is India, with 56% of the total, followed by West Africa with 13% (Fig. 1).

The largest geographical area of the semi-arid tropics (SAT) in any one country is also in India, accounting for 10% of the total SAT area. On a regional scale the most extensive areas are in West Africa (24%), East Africa (22%), Southern Africa (20%), and the Americas (19%) (Fig. 2).

* Program Leader, Cereals Improvement Program, ICRISAT (now Director for International Cooperation, ICRISAT).

In terms of world cereal production, sorghum occupies just over 8% of the area sown to cereals and produces just less than 5% of the world production. In the semi-arid tropics sorghum occupies 15.6% of the cultivated cereal area and produces just under 11% of the cereal yield.

The countries of the semi-arid tropics account for 80% of the total world area sown to sorghum (about 43.3 million ha). India has the maximum area under the crop, but there is a very significant hectarage in West Africa. (It is worth emphasizing at this point that in the 49 countries in the semi-arid tropics an average of 33% of the land area in each has a SAT climate.) The SAT countries produce about 55% of the world's sorghum; India accounts for 66% of this total and West Africa, about 25%.

Various authorities quote various average yields of sorghum in the semi-arid tropics and most appear to be between 500 and 800 kg/ha. This is well below the 1771 kg/ha for the non-SAT areas of Africa and the 4194 kg/ha for the developed areas of Europe, which produce the

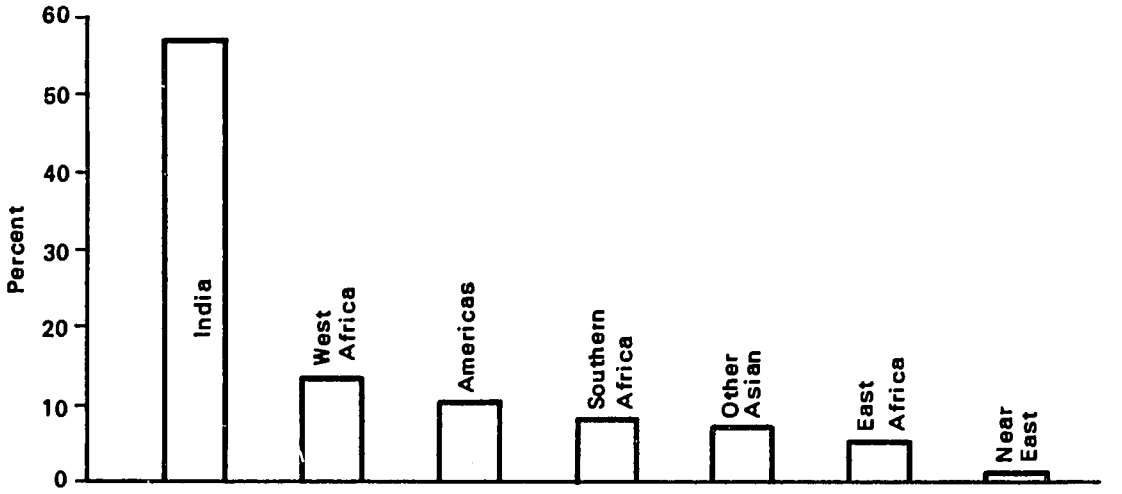

Figure 1. Population of the semi-arid tropics (SAT).

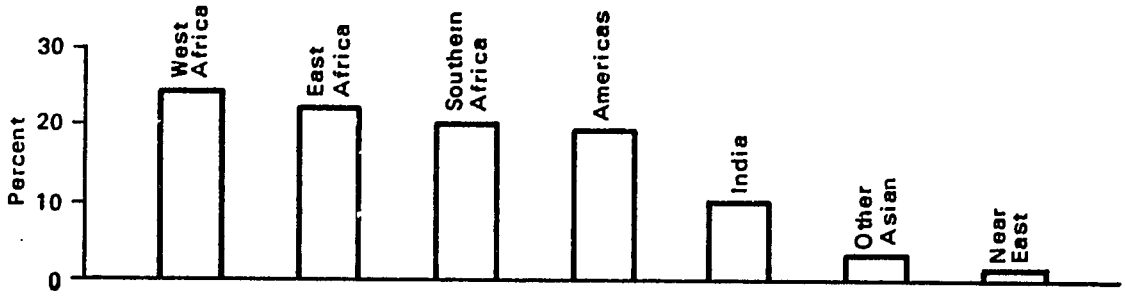

Figure 2. Geographical area of the semi-arid tropics (SAT).

highest yields in the world. Thus there is considerable potential for improvement in the semi-arid tropics.

The trend of area sown to sorghum in the less developed SAT areas is a rising one. An extra 235 000 ha annually have been brought into production since 1964. This increase has, however, been mainly in North, Central and South American countries, and to a lesser extent in Africa. Most of the increased production, from areas other than Africa, has gone into animal feed. Yields have also been rising, but only marginally (14 kg/ha per year), and the variability from this linear trend has been surprisingly low, only 26%.

Production has been increasing steadily since 1964, and is now estimated at about 59.1 million tonnes (29.1 of this is from the SAT). In spite of this, the FAO forecasts a deficit of coarse grains in West Africa for 1980 compared with a supply-demand balance in India. The world demand for coarse cereals will grow at 2.4% annually, and it is projected that there will be a substantial imbalance in supply and demand in the LDCs. There are several estimates that by the mid-1980s there will be deficits in India of 8 to 10%, while the situation is graver in East, West, and Central Africa, where a 28% deficit is predicted. The long-term coarse grains forecasts for the 1990s are that the LDCs will have deficits of about 37%.

Against the background of these data all of us can place in perspective the importance of this workshop. Diseases are an important element in yield reduction and in instability of yield throughout the SAT. It is unlikely, given the low cash value of sorghum and current yield levels on small farmer holdings, that inputs of expensive chemicals for disease control can be contemplated. It is up to you as pathologists to place on record the latest and most comprehensive review of the current state of the art in sorghum pathology research. I am particularly pleased to note that you have a session on the utilization of identified disease resistance. I am positive that this gathering will come up with some useful and realistic recommendations for future research and application that will greatly help to improve the lot of the small farmers of limited means in the semi-arid tropics.

I wish you a successful and useful week of reporting, deliberation, discussion, and recommendation.

Objectives of the Workshop on Sorghum Diseases

R. C. McGinnis*

I would also like to add my warm welcome to those of the previous speakers. We are certainly confident that the arrangements and preparations for the workshop that have been carried out by Drs. Williams and Frederiksen will certainly meet everyone's expectations. I might also say that we are extremely happy that Texas A & M are cosponsors of this workshop. We are looking forward to a very exciting and useful week of meetings at the end of which I believe we will all have advanced our knowledge of sorghum diseases internationally.

Our Institute has held a number of international workshops in the past, and in 1979 we plan to hold five more in various fields related to our major research programs. We find this mechanism to be invaluable in the forward planning of our research programs and in filling in gaps that might presently exist.

In this workshop, we will focus on the following objectives:

- to review the state of knowledge of the internationally important sorghum diseases with particular emphasis on identification and utilization of host plant resistance.
- to stimulate international cooperation in the control of sorghum diseases.
- to establish an international coordinated network for identification of stable resistance to the sorghum diseases.
- to explore the possibilities for integration of resistance with other control practices.

I am confident that these objectives will be fully achieved by the end of this week. We intend to produce a proceedings of the workshop that will contain all the papers presented and the discussions and recommendations of the workshop.

* Associate Director for Cooperative Programs and Training, ICRISAT (now Dean, University of Manitoba, Manitoba, Canada).

Country Reports
Central and South America, Africa, and Asia

Summary and Historical Review of Sorghum Diseases in Argentina

M. Frezzi and E. E. Teyssandier*

Research on sorghum diseases in Argentina began in the period 1939 to 1941, when Hirschhorn (1939, 1941, 1952) reported on 'cereal smuts.' Muntanola (1950) reported on a bacterial disease caused by *Pseudomonas andropogoni* (E. F. Smith) Stapp. She also studied the bacteria and fungi in the Tucuman province (Muntanola 1952, 1954). The following authors have also contributed to the knowledge of sorghum diseases: Tessi (1952), Tessi and Frecha (1952, 1953, 1956), Carrera (1964), Nider et al. (1969), Fresa (1969), Frezzi et al. (1970), Frezzi (1970, 1976), and Docampo and Laguna (1973).

By 1960 the main diseases had been known. These included kernel smut (*Sphacelotheca sorghi* [Link] Clinton), and bacterial diseases such as bacterial stripe (*Pseudomonas andropogoni* [E. F. Smith] Stapp), bacterial leaf spot (*P. syringae* van Hall), and bacterial leaf streak (*Xanthomonas holcicola* [Elliot] Starr and Burkholder). Diseases of lesser importance included leaf blight (*Helminthosporium turcicum* Pass.), rust (*Puccinia purpurea* Cooke), brown leaf spot (*Physoderma* sp), anthracnose (*Colletotrichum graminicola* [Cesati] Wilson), leaf spot (*Phoma insidiosa* Tassi), head smut (*Sphacelotheca reiliana* [Kuhn] Clinton), sooty stripe (*Ramulispora sorghi* [Ellis and Everhart] Olive and Lefebvre), zonate leaf spot (*Gloeocercospora sorghi* Bain and Edgerton), grey leaf spot (*Cercospora sorghi* Ellis and Everhart), leaf spot (*Phyllosticta* sp), head molds (various fungi) and red leaf spot (cause unknown, but apparently nonparasitic in origin — possibly caused by environmental or genetic factors).

Bacterial leaf stripe, leaf streak, and leaf spot are three bacterial diseases that can attack the leaves, sheaths, and stalks of affected plants. In years favorable to their development, they may completely destroy susceptible varieties, especially forage sorghums. At present, with the new cultivars, whether hybrid or not, these diseases do little or no harm in the central continental subzones. In the more humid and less temperate subtropical zones, however, bacterial diseases significantly affect susceptible sorghums. Disease incidence may also be severe in years with favorable weather conditions, as in 1976 and 1977, when intense and massive disease incidence was recorded, especially bacterial leaf stripe (*P. andropogoni*).

Since the development of seed treatment, kernel smut is no longer a problem for sorghum crops. In addition to smut control, seed treatment protects grain against mold fungi, thereby avoiding germination failure.

With the wide expansion of sorghum crops in the period following 1960, new diseases have appeared: root rot, basal stalk rot, and sorghum lodging in 1963–1964 (Frezzi 1976); downy mildew (*Peronosclerospora sorghi* [Weston and Uppal] Shaw) in 1968–1969 (Frezzi 1970), and virus diseases such as maize dwarf mosaic virus (MDMV) in 1972–1973.

The appearance of lodging and sorghum downy mildew, both serious and generalized, led to the elaboration and execution of two projects of the INTA Sorghum Program which deals with root rot and basal stalk rot — sorghum lodging and sorghum downy mildew — bioecology, infection, pathology, and sources of resistance.

As regards sorghum viruses, there is no existing work plan, but as the disease is reaching quite serious levels of diffusion and virulence, the phytopathology departments of Manfredi E.E.A. and Cordoba's National University,

* Head of the Plant Pathology Dept, Manfredi (Cordoba), E. E. A. (INTA), Argentina, and Plant Pathologist, Cargill (S. A.), Pergamino, Argentina.

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and the technicians of Cargill Company have focused their efforts on the study of damage to, and susceptibility and resistance of, the main grain and forage sorghum crops in the country, as well as typical manifestations of symptoms and pathogenicity tests.

Lodging

This is a summary of the work carried out within the country since sorghum lodging disease appeared (after Frezzi 1976). The following fungi were isolated from diseased plants: *Fusarium moniliforme* Sheldon, 84%; *Fusarium* spp, 33%; *Sclerotium bataticola* Taub., 40.5%; *Helminthosporium sativum* Pamm. King and Bakke, 17%; *Rhizoctonia solani* Kuhn, 15%; *Nigrospora sphaerica* [Sacc.] Mason, 10%; *Pythium* spp and *Tetraploa ellisii* Cooke, (percentage not given). Except for *Fusarium moniliforme*, which is easily isolated, the other fungi are always found in mixed colonies.

These fungi were tested for pathogenicity as follows:

1. Root infection, in seedlings approximately 10 days old without positive results.
2. Rathbun method, reproducing the tree nursery disease, or "damping-off." Almost all the inoculated seedlings, the resistant sorghum varieties, and the cultivars which were susceptible to lodging were diseased.
3. The hypodermic syringe inoculation technique.
4. Modified Young method (half toothpick infected with the fungus).

Large numbers of commercial cultivars, experimental cultivars, A lines, B lines, and restorer lines were evaluated following both natural and artificial infection on a 1 to 5 scale. The scale used was:

1. Immune: no lodged plants
2. Very resistant: 1–5% lodged plants
3. Resistant: 6–10% lodged plants
4. Susceptible: 11–20% lodged plants
5. Very susceptible: >20% lodged plants

After 8 years of observation with natural infection combined with artificial inoculation using the modified Young method, the following sorghums were classified as immune or very resistant.

Immune

1667A×R1126-1	Puntano A	384A×R978
Exp. 105	Norteno	Exp. 100
Amigazo	Tops	DA-42
DA-45	77	DA-46
DA-43	400 B	E-57-A
Exp. 7019	3026 B	Indiano
Jumbo C	MfRS 4021	Morgan 101
Morgan 103	Robusto	NK 233
Pampeano B	Y-101	Pioneer 866

Very resistant

399A×R1267-1-1	America	Baqueano
BR-64	BR-64-R	Chingolo
Corman	DA-41	DA-S-44
Dp 4	Traful	Savanna 2
Huerin	Enterriano	Granador
Mancor	H-62	Ibera
Pioneer 8440	MM 4883	Norteno A
R 978	RI 622	RI 678
3026×R 978	R 2544	124 OA
Total		

The very resistant sorghums listed above are immune under normal field conditions.

Since disease regularly appears from mid-February through March, a rainfall record over this period was useful in correlating soil humidity with intensities of disease attack. In general, from lodging data taken over various years, 1963 to 1964, 1969 to 1970, and 1974 to 1975 growing seasons were periods of more intense and widespread attacks (Table 1). These coincided with high rainfall recorded during the period. Although the disease existed in other crop cycles, damage (except in isolated cases of a few very susceptible sorghums) was unimportant even where rainfall equalled or exceeded that of high-disease years. Damage was light when rainfall was scarce during the observed period.

There are a number of environmental factors involved in the stalk rot complex. In the last few years, there has been a growing tendency to believe that lodging is favored by high temperatures and high soil humidity. Any influence of dry seasons upon disease would be due to a certain weakening of the plant which accelerates early senescence, favoring death. All these conditions relate to *Macrophomina phaseolina*, the charcoal rot fungus.

Although lodging may be found in all kinds of soils, under our conditions greater damage is

Table 1. Lodging in sorghum and rainfall from 15 February to 31 March at Manfredi Experiment Station (INTA) from 1963 to 1977.

Crop Year	Rains (mm) 15 Feb to 31 Mar	Lodging[a]
1963–64	153.0	**
1964–65	36.5	*
1965–66	250.0	*
1966–67	153.0	*
1967–68	135.0	*
1968–69	87.0	*
1969–70	142.0	**
1970–71	117.5	*
1971–72	335.5	*
1972–73	335.5	*
1973–74	88.5	*
1974–75	291.5	**
1975–76	160.5	*
1976–77	141.5	*

a. * little lodging; ** severe lodging.

recorded with low-lying soils where water accumulates and where cropping practices are recurrent.

Lodging has not been observed on young plants except during its first days of life ("damping-off"), nor has it been achieved with experimental inoculations when the plant is in full vigor, except on weakened and old tissues. Here the soluble solids play an important role, especially the carbohydrates and inorganic phosphates, which migrate to the head in order to form the grains, thereby weakening the tissues of the neck zone and basal internodes. This could be the main factor governing the disease, as it occurs in maize lodging and as it was shown in Argentina by Sarasola and Sarasola (1968).

Lodging is a typical senescence disease which appears during the last living stage of the plant, shortly before harvest. This is based on six observations:

1. While there is life within the cells and the plant regrows, lodging is not recorded.
2. Lodging is not observed in plants cultivated in pots, where the soil is kept moist at all times.
3. Grain-sorghum crops are more susceptible than those with a double purpose (i.e., grain and forage); forage types are practically immune, and maintain vitality until the first frosts.
4. The rainy or outward sorghum areas suffer less lodging damage than the central continental or semi-arid zones of the country.
5. The following types escape attack: (a) plants that form shoots and keep their vitality, (b) detasseled plants, and (c) plants with empty heads caused by, for example, insect damage.
6. In the seedling stage, or within the first 10 to 15 days of life, all sorghum roots are destroyed — both the very susceptible types and those immune to lodging.

Currently we recommend the following procedures for control of stalk rot:

1. Select lodging-resistant cultivars.
2. Avoid planting in fields with prior serious attacks.
3. Avoid excessively deep, profuse planting. This practice weakens the stalks, making them thinner.
4. Plant in fields with well-balanced fertilizers. Avoid excess nitrogen and insufficient potassium — factors that contribute to maize lodging.
5. Practice early harvest immediately after having observed the first lodged plants.

Sorghum Downy Mildew

Peronosclerospora sorghi (Weston and Uppal) Shaw is the causal agent of the "mildew" identified in Argentina at the present. This disease has spread to all Argentine sorghum-growing regions, with a wide range in susceptibility observed among popular hybrids. Unfortunately, the most widespread cultivars are usually susceptible to the systemic form during certain years. Forage sorghum cultivars appear generally more susceptible to "mildew" than do the grain sorghums. Infection develops in oospore-infested soil during the seedling stage, giving rise to the systemic manifestation of the disease, and secondary infection is caused by conidia. Seed treatments using common and systemic fungicides (Benlate, Vitavax) were totally unsuccessful in controlling downy mildew. Early planting carried out during the first 2 weeks in November can avoid or alleviate dis-

ease incidence, with remarkable results in susceptible sorghums and particularly in grain-sorghum cultivars. Late plantings, during the middle or end of December, develop maximum intensity of damage in the area around the Manfredi Experiment Station (INTA). We have examined the influence of planting depth on the incidence of mildew. This test involved planting at 4 and 8 cm of depth, on two planting dates (23 Nov 1976 and 3 Dec 1976), using Huerin sorghums (very susceptible) and Ibera cultivars (very resistant, almost immune). The results are presented in Table 2. In this test the normal shallower planting depth favored mildew.

Table 2. Influence of planting depth on incidence of mildew.

Cultivar	Depth (cm)	Damage (%)
Huerin	4	18.5
Ibera	4	1.5
Heurin	8	14.5
Ibera	8	0.0

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Sorghum in Brazil

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In Brazil sorghum is a relatively new crop that has increased significantly during the last decade. Grain sorghum accounts for about 75% of the area planted, and forage sorghum represents the remainder.

Data in Table 1 show that the area planted to grain sorghum increased rapidly during the first half of the 1970s, but has slowly declined since 1975. This decline has been caused principally by the lack of adequate drying and storage infrastructure, marketing problems, and an inadequate government policy for producing and exporting feed grains.

Table 1. Grain sorghum production in Brazil from 1971 to 1979.

Year	Area (1000 ha)	Production (1000 tonnes)
1971	80	170
1972	120	220
1973	210	400
1974	250	500
1975	230	483
1976	210	553
1977	178	435
1978	104	228
1979	200	450

Table 2 shows that grain-sorghum production is concentrated in the states of Rio Grande do Sul and Sao Paulo. In the semi-arid Northeast (Ceara, Rio Grande do Norte, and Pernambuco), the production is increasing. In this region sorghum is one of the best options for the farmer, as it is more drought-tolerant than maize and there is a large feed-grain deficit. In 1977 the average yield was 2.5 tonne/ha. In the state of Sao Paulo, where the sorghum area has been increasing rapidly, the average yield is 3 tonne/ha; while in the Northeast the average yield is much less. National trials of commercial and experimental hybrids and varieties have frequently produced more than double the state-average yield.

Table 2. Grain sorghum production in Brazil (statewise) in 1977.

State	Area (ha)	Production (tonnes)	Yield (kg/ha)
Ceará	2 000	1 600	800
Rio Grande do Norte	4 615	3 733	809
Pernambuco	106	152	1434
Minas Gerais	2 290	2 740	1200
Espirito Santo	205	615	3000
São Paulo	56 540	169 620	3000
Roraima	855	3 470	4058
Santa Catarina	450	1 320	2933
Rio Grande do Sul	91 000	214 000	2352
Mato Grosso	4 583	8 258	1802
Goiás	15 000	29 625	1975
Brazil	177 644	435 446	2444

Nearly all the grain-sorghum acreage is planted with hybrids except in the Northeast where some varieties are used. Until now sorghum seed is imported or produced by a few commercial companies, but soon all the seed will be produced in Brazil. Most of this seed will be produced by two companies with two or three other companies producing a small percentage.

During the first Brazilian Sorghum Symposium held in 1977, it was shown that sorghum was used in the feed industry at 8% of the ration. If sorghum continues to be used at this rate, the demand for sorghum in 1980 will be 1 million tonne, corresponding to 400 000 ha. This is more than double the amount of sor-

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ghum and nearly 5% of the maize produced in 1977/1978. The 1978/1979 sorghum forecast is approximately 200 000 ha, the limiting factor being the shortage of seed.

Grain sorghum is used principally for swine and poultry feed. The National Maize and Sorghum Research Center (CNPMS) is developing a sorghum for direct human consumption and in industrialized foods. Forage sorghum is principally used for silage for dairy cattle, but is also being used increasingly in beef production.

Preliminary experimental results and economic analysis indicate that sweet sorghum can be economically cultivated to produce alcohol for mixing with gasoline, and can be planted in areas near sugar mills and distilleries. In Brazil sweet sorghum can be harvested before or after the sugarcane harvest when the mills are normally idle.

Sorghum in Brazil is subject to damage by several diseases. Surveys conducted by us at the CNPMS showed that the principal diseases are foliar anthracnose, rust, sorghum downy mildew, and grain "weathering" (Table 3). Anthracnose and rust are quite severe in nearly all the regions where sorghum is produced, principally in central and southern Brazil.

Sorghum downy mildew is a potential problem and has been observed in many areas of the states of Rio Grande do Sul, Sao Paulo, and Santa Catarina. The importance of this disease increases when we consider that these states constitute the largest maize-producing region of the country. Results from downy mildew trials have demonstrated the existence of commercial and experimental material of both maize and sorghum with good levels of resistance.

Sorghum grain produced in central and southern Brazil is damaged both internally and externally by fungi that reduce the quality of the product. We have initiated selection for resistance to this damage but have not found any material without tannin that is resistant.

The economic importance of other diseases varies from region to region and from year to year.

All sorghum research in Brazil is coordinated by the National Maize and Sorghum Research Center of Brazilian Enterprise for Agriculture Research (EMBRAPA). In addition CNPMS also conducts research of national importance. For example two subprojects, disease-control, and sorghum disease survey are coordinated and executed by the pathologists at CNPMS. This group also forms part of the multidisciplinary research team in other subprojects such as sorghum productivity and quality improvement, germplasm bank activities, etc. These and other research subprojects, a total of 13, are summarized in "Research Program and Activities of the National Maize and Sorghum Research Center-1978," and will not be discussed here.

In addition to conducting and coordinating research in Brazil, CNPMS also participates in furnishing technical assistance to other research programs, prepares material to support other breeding programs in Brazil, principally in the state of Rio Grande do Sul and the Northeast, and prepares and distributes a network of grain, forage, and sweet sorghum trials covering the entire country in cooperation with private firms and official institutions. Breeding for resistance to disease is also conducted by private seed companies such as AGROCERES

Table 3. Principal sorghum diseases and the research priority in Brazil.

Disease	Pathogen	Research priority
Root and stalk diseases		
Red stalk rot, anthracnose	*Colletotrichum graminicola*	2
Charcoal rot	*Macrophomina phaseoli*	2
Foliage diseases		
Anthracnose	*Colletotrichum graminicola*	1
Rust	*Puccinia purpurea*	1
Downy mildew	*Sclerospora sorghi*	1
Leaf blight	*Helminthosporium turcicum*	2
Grey leaf spot	*Cercospora sorghi*	3
Zonate leaf spot	*Gloeocercospora sorghi*	3
Sooty stripe	*Ramulispora sorghi*	4
Head diseases		
Grain 'weathering'		1
Covered smut	*Sphacelotheca sorghi*	–
Head smut	*Sphacelotheca reiliana*	–
Virus diseases		
Sugarcane mosaic	virus	2

and CONTIBRASIL. Several thesis projects at various universities have focused on the selection of varieties for resistance to various diseases. Many of the postgraduate students working on these projects have been financed by EMBRAPA.

Most of the research in this area conducted at the National Center has been to obtain varieties resistant to anthracnose, rust, downy mildew, leaf blight, and grain "weathering." Selections are made in the field under natural epidemic conditions. Studies of the nature of inheritance for resistance to these diseases are also being initiated.

In 1977, EMBRAPA released two grain-sorghum hybrids, two forage-sorghum hybrids, and five sweet sorghum varieties with good agronomic characteristics and resistance to the principal diseases. Additional hybrids and breeding material will be released in 1979.

Summary

The area planted to grain sorghum in Brazil has increased since 1970. The major production areas are the states of Rio Grande do Sul and Sao Paulo. Even though the national average yield is 2.5 tonne/ha, results of national trials indicate that this yield can be doubled.

The use of sorghum grain in animal feed, at the rate of 8% of the ration, will create a demand of 1 million tonnes of sorghum grown on 400 000 ha by 1980. Recent problems in seed production have restricted the expansion of sorghum. Even though the potential for 1978/1979 is in excess of 1 million ha, the area planted probably will not exceed 200 000 ha due to seed shortages. Forage sorghum is being used in some regions, and sweet sorghum appears to be excellent for the production of alcohol used as a fuel mixture with gasoline.

The principal diseases are anthracnose, rust, sorghum downy mildew, and grain "weathering."

Sorghum research in Brazil is coordinated by the National Maize and Sorghum Research Center which is a part of the Brazilian Enterprise for Agricultural Research (EMBRAPA). This research is organized in 13 subprojects. In 1977, two grain hybrids, two forage hybrids, and five sweet sorghum varieties with good agronomic characteristics and disease resistance were released to the farmer. Additional hybrids and genetic material will be released in 1979.

The Present Status of Sorghum Diseases in El Salvador

George C. Wall*

El Salvador has a very high population density and growth rate, and there is an urgent need to maximize food production. Sorghum is its second most important cereal crop: annually 120 000 ha are planted producing around 155 million kg of grain at an average yield of about 1292 kg/ha. Sorghum has several uses in El Salvador: (1) grain is used for feeding livestock, either directly or in concentrates, (2) grain is made into tortillas for human consumption (although maize tortillas are preferred), and (3) the whole plant is used for forage, or as a dual-purpose (forage and grain) crop.

Sorghum is planted in nearly every part of the country, except in the higher altitude areas. Ninety percent of the sorghum planted is composed of "native varieties", which are actually neither native nor varieties, but can more properly be termed populations of African origin; they are very primitive and sensitive to photoperiod. The general practice is to plant these native varieties in association with maize. The sorghum grows practically unattended, and the effects of inadequate fertilization, insect pests, weeds, and plant diseases contribute to low average yield per hectare.

How Sorghum Is Grown

There are two distinct seasons in El Salvador — a dry season and a rainy season. Maize is planted at the start of the rainy season; 3 to 4 weeks later, as the maize is cultivated, sorghum is planted in association with it. Three months after planting, the maize stalk is bent in half to facilitate field storage of the partially dried ear until the dry season is established. It is not until this time that the sorghum plant begins to get full sunlight. From this point on, the sorghum plant develops without further competition from maize, but is generally completely unattended. Diseases and pests, as well as competition with maize and weeds, take a considerably high toll in terms of yield. Other factors include the growing of primitive varieties with low yield potential. However, considering the conditions under which these varieties grow, perhaps no other varieties could survive with such lack of attention.

The Sorghum Disease Panorama

Little plant pathology research on sorghum was carried out in El Salvador prior to the 1970s. The following account is based on the author's experiences and observations, since 1975, as a sorghum pathologist in El Salvador. The list of diseases is presented according to apparent order of prevalence. It should be pointed out that no specific disease is considered to be a major production-limiting factor on a nationwide basis; usually more than one disease is present in a given field, and it is in this way that they contribute to the low yield average obtained.

Grey Leaf Spot

Grey leaf spot (*Cercospora sorghi*) is the most common disease of sorghum in El Salvador and can be found anywhere sorghum is grown, on young as well as mature plants. Generally it is not sufficiently severe to defoliate or kill plants. On some sorghum varieties, it causes defoliation when plants approach maturity, and it will cause defoliation on young plants while these are still under the shade of the maize crop. The symptoms observed and microscopic observations of the fungus involved coincide with

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descriptions given by several authorities (Dickson 1956; Doggett 1970).

Rust

Rust (*Puccinia purpurea*) is most commonly found towards the end of the rainy season, and at the onset of the dry season it becomes more severe. It can defoliate some varieties. Generally it causes defoliation of lower leaves. A parasite of rust, *Darluca filum*, occurs on rust of maize (*P. sorghi*), and is known to parasitize *P. purpurea* as well (Contreras and Barahona 1974); this hyperparasite is apparently capable of reaching high percentage of infection on rust. Although rust may cause yield reduction, it is a latecomer in the sorghum plant cycle. It forms both uredospores and teliospores in El Salvador.

Zonate Leaf Spot

Zonate leaf spot (*Gloeocercospora sorghi*) is most commonly found on the lower leaves, but may also be seen on the upper leaves. Although it is widespread throughout the country, it does not appear to cause as much damage as the diseases previously mentioned. This conclusion is based on a quick estimate of the leaf area affected and may not be an adequate basis for comparison. Nevertheless, this disease is not considered very important at present.

Grain Molds

Most of the sorghum grown in El Salvador flowers and sets seed near the end of the rains. It is harvested in the first part of the dry season and, if not left in the field too long after it is ready for harvest, grain molds (*Fusarium, Curvularia* spp, and others) affect a relatively small proportion of the panicles. Several parameters can aggravate this situation, including the corn leaf aphid, excess humidity at harvest time, and delay in harvesting. The generalization previously mentioned refers specifically to native sorghum varieties, which are photoperiod sensitive. The new grain varieties are photoperiod insensitive and can flower at any time of the year. However, these, too, are planted so they can be harvested after the rains have stopped, with the purpose of escaping serious damage from grain molds.

Sugarcane Mosaic Virus, or Maize Dwarf Mosaic Virus

This virus disease is widespread on sorghum in El Salvador, particularly among the native varieties. It seems to affect seed setting, although this has not yet been demonstrated here, and an investigation is in progress. The corn leaf aphid is apparently the major vector of this virus in El Salvador, and it is present in most parts of the country, along with other insect species that are parasitic or predatory on it.

Leaf Blight

Leaf blight (*Exserohilum turcicum*) can be found wherever sorghum is grown in El Salvador, but normally the incidence is not so frequent as to make it a disease of prime importance. An outbreak was observed on experimental materials in San Andrés during 1976.

Sorghum Downy Mildew (SDM)

Sorghum downy mildew (*Peronosclerospora sorghi*) is not widespread in El Salvador. While not presently a limiting factor in nationwide sorghum production, it may become the most important maize and sorghum disease. Considerable attention is being devoted to SDM-resistant material in the government breeding programs. Surveys have revealed increasing numbers of locations where SDM occurs, either on sorghum or on maize. Although it seems to be spreading slowly, SDM causes great concern because of its ability to infect maize as well as sorghum, and because the close association of these two crops is common throughout the country. Several studies on SDM have been carried out (Wall 1977; Wall and Ortiz 1978), or are currently under way.

Maize and sorghum varieties are being screened for resistance to SDM. It is hoped that an epiphytotic can be avoided by the timely identification of susceptible and resistant varieties, and the consequent promotion of the resistant materials wherever necessary.

SDM has been observed on *Zea mays, Sorghum bicolor, S. bicolor* var. *dochna*, sorghum-sudan hybrids, *S. sudanense, S. halepense*, and *S. arundinaceum*. Fortunately, the grass hosts and wild sorghums are not widespread.

Loose Smut

Loose smut (*Sphacelotheca cruenta*) is most commonly seen on native varieties, but also appears on some experimental varieties. Its incidence is generally low, and therefore it is not considered to be of major importance. The seed of improved varieties is normally sown with Arasan treatment, which reduces spread of the disease.

Anthracnose

Stalk rot caused by this fungus (*Colletotrichum graminicola*) is occasionally observed in fields of experimental materials. It is not common and is not considered to be of great importance.

Bacterial Stripe

Sometimes seen on experimental varieties, bacterial stripe (*Pseudomonas andropogoni*) disease is apparently unimportant at present.

Bacterial Spot

One outbreak of bacterial spot (*Pseudomonas syringae*) was observed in 1977 at the Santa Cruz Porrillo Experiment Station. The outbreak occurred after a period of prolonged rain. However, the disease is seldom seen.

Summary of the Present Situation

In order to summarize the most common sorghum diseases presently found in El Salvador, it would be convenient to describe the typical sorghum plant found in the field. At the top of the panicle we find grain molds damaging the seed, and this could extend to some of the grain throughout the panicle; we see a few grey leaf spots on the top leaves, along with some aphid cast skins and mummies. The panicle could also show some midge damage. The next leaves, going from top to bottom, present more numerous grey leaf spots, and here we also find rust sori. Continuing below, we find more numerous grey spots and rust sori, and we may have (one or two) zonate leaf spots. The lower leaves will be dead and dry, and will bear large numbers of assorted fungal spots and sori. Some evidence of worm damage (*Spodoptera*) is likely in the middle or upper leaves, and the plant could be serving as support for some type of bindweed. The size of the grain would be small, and the size of the panicle would not be impressively large.

A Changing Situation

With the aim of increasing the average sorghum yield, the primitive so-called native varieties are slowly being replaced by high-yielding varieties and hybrids which, of course, demand more care. This change in turn increases the need to deal with sorghum pests and diseases.

Sorghum breeding programs are being carried out at CENTA (Centro Nacional de Technologia Agropecuaria). In the formation and selection of new sorghum varieties, major emphasis is on high-yield potential, adaptability, and resistance or tolerance to the major diseases. It is not economically sound for the farmer to invest much in sorghum pest control at present.

There are various methods employed by the National Sorghum Program in selecting for disease resistance and tolerance. In the case of most major diseases, this is done in field plots where yield, adaptability, or other traits are under observation. The different materials are graded as good, moderate, or poor, depending upon general plant health. Grading may be for a specific disease. In selecting materials with respect to any other parameter — such as yield or plant height, etc. — health grading is taken into account.

With certain diseases, such as SDM, a specific screening is carried out. Artificial inoculations are performed on test materials, which are then graded for percent infection. Wherever possible, responses to natural field infections are utilized to detect susceptibility.

Field inoculations with the major foliar pathogens were tried as a way of screening for resistance. At present, however, this method has been discontinued in preference to natural infection, which is less time-consuming and requires fewer personnel.

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Sorghum Diseases in Mexico

Alberto Betancourt Vallejo*

In the past 15 years, sorghum has become one of the most important crops in Mexico, due primarily to its wide range of adaptability to varied environmental conditions, its usefulness as a feed grain, ease of mechanization, drought resistance, and relative tolerance to disease and pest problems.

It is not possible to precisely say when sorghum was first introduced to Mexico, but it is believed that it took place at the end of the 19th century (Angeles 1968). Other evidence suggests that it was grown in small areas during the late 1940s and early 1950s, but production was very limited. Although there is no official report of sorghum production for this period (CENEINA 1967), many speculate that much of the reluctance of rural people to accept this new crop was probably due to the high preference for maize as a food grain.

It was not until 1958 that sorghum acreage started to expand in northern Mexico (Tamaulipas), displacing cotton. A major shift from cotton to maize and sorghum in this area has been occurring since 1965 (Futrell 1973). Sorghum also displaced maize in areas of low rainfall, where it outyields native open-pollinated maize varieties and where it withstands stress conditions better than most other cereals. Because of its excellent response to irrigation, sorghum is very popular in humid areas as well; large acreages are found in Tamaulipas, Guanajuato, Jalisco, Sinaloa, and in Michoacan where it is grown both under irrigation and as a rainfed crop.

Hybrids short in height are of U.S. origin and are harvested with a combine harvester, with the exception of El Bajio in the State of Guanajuato where 2% of the commercial hybrids are the result of a local breeding program carried out by INIA (The National Instituta of Agricultural Research [Frederiksen 1977]).

Sorghum-Producing Areas

In 1976 sorghum occupied approximately 1.25 million ha and is still expanding (Table 1). According to the available figures, not only have the areas and total production increased, but also the production per hectare, due mainly to better production practices such as fertilization, pest and diseases control, and better hybrids.

Sorghum in Mexico ranks third in acreage of the crops, exceeded only by maize and beans, and ranks second in production, exceeded only by maize. A large portion of the production is concentrated in the northern (Tamaulipas), west coast (Jalisco, Michoacan, and Sinaloa) and central (Guanajuato) areas. These states account for 82% of the total sorghum acreage. Other sorghum-growing states are: Chihuahua, Nuevo Leon, Morelos, Nayarit, Sonora, and Coahuila. In Mexico, sorghum is used primarily as a feed grain, and the expansion of this industry has had a substantial influence on the popularity of the cereal.

Table 1. Grain-sorghum area and production for 1960, 1970, and 1976 in Mexico.

Years	Area harvested (ha)	Production (m tonne)	Yield (kg/ha)
1960	116 000	209 000	1800
1970	971 000	2 747 000	2840
1976	1 250 000	4 026 864	3200

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Table 2. Climatic characteristics of the major sorghum-producing areas in Mexico (after Koeppen [Anon. 1976b, p. 21]).

Area	Climates[a]	Elevation (m)	Temperature[b] (°C)	Average annual precipitation (mm)
Temperate humid	H	1200–1800	18	800
Humid subtropical	Cfa	0–1200	24	500
Semi-arid area	BSh, BWh	0–1200	24	350

a. H – Highland climates.
BSh – Dry steppe climate.
BWh – Dry desert climate.
Cfa – Warm temperate climate.
b. Average monthly basis.

Adaptation

Sorghum is grown mostly in the temperate humid zone and in the humid subtropical and semi-arid regions of Mexico (Table 2).

In the temperate humid zone, 700 000 ha are now planted to sorghum annually. Rainfall varies from 700 to 900 mm and is generally well distributed. Guanajuato, Jalisco, South of Sinaloa, Michoacan, Nayarit, and Morelos are major areas of production. The El Bajio region in the State of Guanajuato produces yields up to 13 tonne/ha; while in Jalisco and Michoacan yields may vary from 5 to 8 tonne/ha. In the temperate humid zone, slightly more than 400 000 ha are grown under rainfed conditions.

An average of 430 000 ha are planted to sorghum annually in the humid subtropical and semi-arid areas. Tamaulipas, north and central regions of Sinaloa, Chihuahua, Nuevo Leon, Sonora, and Coahuila are representative states of this area. Sorghum is grown at elevations from 10 to 30 m (Los Mochis, Sinaloa, and Rio Bravo, Tamaulipas) up to 1200 m (some regions of Coahuila and Chihuahua); predominantly the climate is classified as BSh and BWh (Koeppen [Anon. 1976b, p. 21]). In the semi-arid area, sorghum is better adapted to rainfed farming than other grain crops such as maize, and also performs well under irrigation. Under dry conditions the average yield is about 2200 kg/ha; irrigated crops produce approximately 3300 kg/ha.

Tamaulipas leads in total acreage with 370 000 ha harvested in 1976; of this, more than 125 000 ha are grown under rainfed conditions. Slightly more than 40 000 ha were grown in Chihuahua and Nuevo Leon, while approximately 20 000 ha were planted in Sonora and Coahuila.

Important Sorghum Diseases in Mexico

As with many other crops in which cultivation has intensified, sorghum in Mexico has recently developed serious disease problems. The most important diseases are: downy mildew (*Peronosclerospora sorghi*), leaf blight (*Exserohilum turcicum*), stalk rot (*Fusarium moniliforme*), rust (*Puccinia purpurea*), and anthracnose (*Colletotrichum graminicola*). Zonate leaf spot (*Gloeocercospora sorghi*) and grey leaf spot (*Cercospora sorghi*), although common, have been causing only moderate damage. Diseases confirmed in the country are listed in Table 3.

Downy Mildew

By far the most important and damaging sorghum disease in Mexico is downy mildew (*Peronosclerospora sorghi*). This disease has caused severe losses in recent years, especially in northern Tamaulipas which is both humid and irrigated.

The disease was first observed in this region as early as 1964, but probably was present when it was first reported in Texas in 1961 to 1962 (Frederiksen and Renfro 1977). The average incidence of systemic infection in commer-

Table 3. Diseases of sorghum in Mexico

Diseases	Distribution in Mexico
Seed rot and seedling diseases	
Fusarium sp	Regions of high relative humidity and
Pythium sp	low temperatures in the soil[a]
Penicillium sp	in general
Helminthosporium sp	
Rot and stalk diseases	
Charcoal rot *(Macrophomina phaseolina)*	Tamaulipas, Jalisco[a, b, c]
Stalk rot *(Fusarium moniliforme)*	Guanajuato
Sorghum leaf diseases	
Leaf blight *(Exserohilum turcicum)*	Jalisco, Tamaulipas, Guanajuato[d, e, f, g]
Rust *(Puccinia purpurea)*	Guanajuato, Nayarit, Jalisco, Tamaulipas[f, g, h]
Grey leaf spot *(Cercospora sorghi)*	Tamaulipas, Jalisco, Guanajuato[f, g]
Anthracnose *(Colletotrichum graminicola)*	Tamaulipas, Guanajuato, Jalisco[g, i]
Zonate leaf spot *(Gloeocercospora sorghi)*	Yucatan, Tamaulipas[g, i]
Bacterial streak *(Xanthomonas holcicola)*	Sinaloa, Sonora, Yucatan, Guanajuato[g, j]
Bacterial stripe *(Pseudomonas andropogoni)*	Tamaulipas
Sooty stripe *(Ramulispora sorghi)*	Tamaulipas[k]
Maize dwarf mosaic virus	Tamaulipas[g]
Inflorescence diseases	
Covered kernel smut *(Sphacelotheca sorghi)*	Guanajuato, Coahuila, Sinaloa, Morelos[f, l]
Loose kernel smut *(Sphacelotheca cruenta)*	Sinaloa, Guanajuato[l, m]
Head smut *(Sphacelotheca reiliana)*	Sinaloa, Jalisco, Guanajuato, Nayarit, Tamaulipas, Estado de Mexico[f, g, k, l, m, n]
Sorghum downy mildew *(Peronosclerospora sorghi)*	Tamaulipas, Michoacan, Guanajuato, Jalisco, Guerrero, Veracruz, Puebla[d, f, g, k, o, p, q]
Crazy top *(Sclerophthora macrospora)*	Jalisco, Tamaulipas, Guanajuato[k, q]

a. Anon.1977a, p. 73
b. Critchfield 1960
c. Anon. 1975a
d. Castro 1974
e. Anon. 1975b, pp. 39–41
f. Frederiksen 1977
g. Giron 1977
h. Anon. 1976b, p. 21
i. Anon. 1977b, p. 18
j. Frederiksen and Renfro 1977
k. Valdivia B. and Betancourt 1973
l. Rodriguez S. 1972
m. Fuentes 1967
n. Anon. 1976c, pp. 49–51
o. Anon. 1976a
p. Garcia 1977
q. Leon 1974

cial fields in Tamaulipas shifted from the 10 to 15% common prior to 1973 to 40 to 60% in 1973 and in 1976. In 1978, some commercial hybrids showed up to 80% systemic infection, according to Rodolfo Giron (personal communication); sorghum downy mildew (SDM) is present in Jalisco, Michoacan, Guanajuato, Guerrero, Veracruz, and the lowlands of Puebla (Leon 1974), where large acreages of maize are grown every year.

The earliest report of downy mildew in the Jalisco area was in 1974. In this region 20 to 40% of systemic infection was observed in 1977 (Frederiksen 1977).

The increase of downy mildew in northern Mexico was mainly due to the introduction of high-yielding, yellow endosperm, grain sorghum hybrids highly susceptible to the disease. They have gradually been replaced by more resistant but lower yielding hybrids.

Leaf Blight

Leaf blight (*Exserohilum turcicum* Pass.) is characteristic of humid climates and moderate temperatures. It has become increasingly important in Jalisco, along the west coast, and El Bajio as well as Tamaulipas. The most severe damage has been reported in the Jalisco area. Little economic damage has been recorded in the less humid areas.

Leaf blight is probably the key disease at Jalisco. Sorghum cannot be grown profitably throughout this area until more resistant hybrids are obtained (Frederiksen 1977).

Rust

Rust (*Puccinia purpurea*) has been reported in several humid areas, such as Tampico, Rio Bravo, Tamaulipas, coastal area of Nayarit, Veracruz, and Jalisco (Garcia 1967; Anon. 1976b, p. 21; Rodriguez 1972; Frederiksen 1977; Valdivia and Betancourt 1973). In Jalisco severe losses were observed in all commercial hybrids during 1977, with disease intensities ranging from 7 to 9 on a 1 to 9 scale. In 1977, rust apparently developed late in the season, aggravating stress-related *Fusarium* stalk rot and head blight diseases (Frederiksen 1977).

At Rio Bravo Tamaulipas, some commercial hybrids in 1977 showed rust intensities of 3 to 4 on a 1 to 5 scale (Giron 1977). This disease has had an important impact on grain sorghum in humid areas, but the extent of damage has not been recorded, probably because sorghum is presently occupying these areas as a new crop.

Stalk Rot

Stalk rot (*Fusarium moniliforme*) has become increasingly common and in recent years is causing severe damage to most commercial hybrids in the State of Jalisco. All three phases — crown rot, sheath rot, and head blight — were observed in 1977 (Frederiksen 1977). It has also been observed at El Bajio, but its incidence has varied from year to year and no economically important damage has been recorded (Gabriel, personal communication).

Grey Leaf Spot, or Angular Leaf Spot

Grey leaf spot (*Cercospora sorghi*) has usually been a common disease at Rio Bravo Tamaulipas, where most commercial hybrids have shown ratings from 2 to 3 on a 1 to 5 scale during the last 3 years (Giron 1977). Grey leaf spot was also commonly observed at Jalisco and Guanajuato, where it has caused moderately severe damage. In most cases, the disease has been observed late in the growing season, after the grain-filling period is over; consequently, no severe damage was noted. One commercial hybrid was reported to be resistant in Jalisco during 1977 (Frederiksen 1977), although most commercial grain sorghums are susceptible.

Anthracnose

According to observations made at Rio Bravo, Tamaulipas, anthracnose (*Colletotrichum graminicola*) caused economic losses in most grain sorghum hybrids, with disease ratings varying from 2.5 to 3.0 on a 1 to 5 scale (Giron 1977). Its incidence, however, varies with environmental conditions. During 1977, for example, only a few hybrids were affected by this disease in Jalisco as well as in Guanajuato.

Zonate Leaf Spot

Zonate leaf spot (*Gloeocercospora sorghi*) has been ranked according to its incidence as the second most important disease in northern Mexico (Tamaulipas), with some commercial

hybrids ratings as high as 3 or 4 on a 1 to 5 scale (Giron 1977). In other areas, such as Yucatan in the southern part of Mexico, commercial hybrids have shown a moderate incidence of zonate leaf spot during the last 2 or 3 years (Anon. 1976b, p. 21).

Minor Diseases of Sorghum in Mexico

Head Smut

Head smut (*Sphacelotheca reiliana*) was a major problem in Rio Bravo, Tamaulipas, and the El Bajio regions in the late 1960s (Angeles 1968), but was partially controlled by resistant hybrids. It has also been reported in Jalisco, Sinaloa, Sonora, Nayarit, and Baja California (Angeles 1968; Frederiksen 1977; Garcia 1967; Anon. 1976b, p. 21; Rodriguez 1972). At present, head smut is found in most sorghum-growing areas of Mexico, particularly (though of moderate incidence) in northern Tamaulipas, Jalisco, and Guanajuato.

Other smuts, *Sphacelotheca sorghi* and *Sphacelotheca cruenta*, reported at El Valle de Culiacan in Sinaloa in 1967 (Fuentes 1967), and Coahuila and Guanajuato (Rodriguez 1972) were controlled by seed treatment and are now of low importance.

Charcoal Rot

Charcoal rot (*Macrophomina phaseolina*) has been found in northern Mexico (Giron 1977; Valdivia and Betancourt 1973), but apparently is not causing serious damage except under situations of severe stress late in the season.

Seed rot and seedling diseases caused by the fungi *Fusarium, Pythium, Penicillium*, and *Exserohilum* — formerly common in some regions in Mexico (Castro 1974) — are now of low importance due to fungicide treatment of most hybrid seed.

Other minor diseases reported in Mexico are maize dwarf mosaic virus (MDMV) in Rio Bravo, Tamaulipas (Giron 1977); sooty stripe (*Ramulispora sorghi*) in Tamaulipas (Valdivia and Betancourt 1973); bacterial streak (*Xanthomonas holcicola*) in Sinolca, Sonora, Yucatan, and Guanajuato (Valdivia and Betancourt 1973); crazy top (*Sclerophthora macrospora*) in Guanajuato, Jalisco, Tamaulipas (Castro 1974; Garcia 1967; Giron 1977; Leon 1974; Valdivia and Betancourt 1973); and rough spot (*Ascochyta sorghina*) in Puebla (Rodriguez 1972).

Location and Nature of Research on Sorghum Diseases in Mexico

The National Institute of Agricultural Research (INIA), carries out the sorghum-breeding program in Mexico. The goal of this program is to obtain high-yielding hybrids and varieties with good resistance or tolerance to pests and diseases prevalent in a wide range of climatic conditions.

In order to facilitate the breeding work, three working regions have been established. Each region has a multidisciplinary group of scientists in breeding, entomology, physiology, pathology, etc. The three work areas are:

1. Central plateau and high valleys with altitudes exceeding 1800 m above sea level.
2. Low lands (Bajio) and intermediate regions with altitudes of between 1200 and 1800 m.
3. Humid subtropical and semi-arid areas with altitudes of up to 1200 m.

As stated earlier, the major disease problem areas are located at the humid subtropical and intermediate regions with high relative humidity.

Most of the research for disease resistance is actually conducted at CIAGON (called CIAT before 1978) at Rio Bravo, Tamaulipas, which is an excellent location for screening lines for SDM resistance as well as a few foliar diseases, such as zonate leaf spot, anthracnose, and bacterial diseases. Screening for resistance to rust is done in the Tampico area.

A large number of parental lines for hybrids and materials from the world collection have been tested under field conditions at Rio Bravo for SDM resistance. Some work, however, is performed under greenhouse conditions whenever a reliable inoculation technique is available. We have recently introduced some converted lines and multiple disease-resistant populations developed at the Texas Agricultural Experiment Station (TAES), which promise to be very good sources of resistance for the most important diseases. Other materials, introduced from Kansas, Oklahoma, and Indiana

have also shown very good resistance to some important diseases, as well as adaptability to the Rio Bravo area.

The main aspects of this program are to (a) obtain disease ratings in commercial and experimental hybrids, (b) carry out inoculation techniques, (c) select for disease resistance, and (d) incorporate disease resistance into improved plant material.

Achievements, Problems, and Prospects of Sorghum Disease Research in Mexico

Control of sorghum diseases in Mexico has usually been accomplished through the use of local or introduced resistant hybrids and through cultural and production practices. For example, early plantings, especially in SDM problem areas (such as Rio Bravo), avoid infection.

Of 29 new sorghum hybrids recently released by INIA, several have shown resistance or tolerance to some important diseases. These diseases, however, are expanding rapidly in the sorghum-growing areas and represent limiting factors to production. These include SDM in northern Mexico, and leaf blight, stalk rot, and rust on the west coast. On the west coast, for example, not one commercial hybrid has been found to be resistant to all three diseases at the same time (Frederiksen 1977). Other diseases — such as anthracnose, head smut, and zonate leaf spot — are potentially destructive diseases that can be limiting in both humid and moderately humid areas.

A considerable effort must be made to reinforce the breeding program. The expansion of sorghum acreage and its monoculture have caused an increase of disease and pest problems. The following actions become important:

1. Reinforce training of personnel specialized in plant pathology, with emphasis on sorghum diseases.
2. Determine effect on yield that diseases, either individually or in combinations, are causing in Mexico.
3. Establish a breeding program in Jalisco for leaf disease resistance that will represent the temperate humid area problems.
4. Promote and establish international cooperation with other countries in terms of information, sources of resistance, etc.

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Sorghum and Sorghum Diseases in Venezuela

Mauricio Riccelli*

Venezuela is located in the northern part of South America. Its size is about that of Texas and Oklahoma combined. We have different geographical and ecological regions. Latitude is from about 0°50'S to about 11°N. There are three important sorghum-growing areas. One is located in the foothills of the Andes — a very humid region. The second is a drier region in the northern central part of the country, and it is the most important sorghum-growing area. A third region, with intermediate rainfall, is located in the eastern states of Anzoategui and Monagas.

Venezuela is known for its oil, and this is still the basis of the economy, but we now have some new developments and sorghum production is a good example of these. Sorghum first came to Venezuela about 15 years ago, and for some time it was only a curiosity at some experimental stations. Until 1972, only 10 000 ha were cultivated in the country. Since then, sorghum production has increased very rapidly and in 1978 there were about 300 000 ha planted to sorghum in Venezuela. No crop has increased so fast in Venezuela as grain sorghum.

We are still somewhat dependent on USA-produced hybrid seed for our sorghum production. These hybrids are very attractive, but they do not perform so well in the tropics. We had, therefore, to produce our own hybrids — brown-seeded with a lot of tannin, but well adapted and high yielding.

Sorghum is produced in areas where the climate is humid and warm during the rainy season, and these conditions promote diseases. There is a tremendous weed problem, and the short U.S. hybrids can be smothered by weeds if control measures are not taken in time. On the other hand, our tropical sorghums, which are taller, can grow faster than the weeds, and, even if the farmer is not careful with weeding, the weeds will not be as much of a problem as they are with the short hybrids. Another reason in favor of the taller hybrids is that the farmer needs what is left of the plants after harvest to feed animals during the dry season when other forage is not available. The taller hybrids present no problem for combine harvesting if the combine is adjusted correctly.

Our Venezuelan hybrids have open panicles, and brown seeds which mature in about 100 to 105 days. They are photosensitive and therefore take longer to mature in the more northern latitudes.

Local seed production is not sufficient, so we still import about 90% of our seed, mainly from the USA.

Birds are a major problem with grain sorghum in Venezuela, and sometimes even the brown-seeded sorghums are affected by them.

Diseases

Diseases are the main limiting factors for grain sorghum production in Venezuela. The occurrence of wild sorghums increases the disease problems in cultivated sorghum. There are at least three wild sorghums — *Sorghum halepense, S. verticilliflorum,* and *S. arundinaceum*. There are possibly other species and many natural hybrids among them. These wild sorghums are very early, they shatter, and they have become a very serious problem. They grow throughout the year and serve as a reservoir for the pathogens. It is impossible to think of eradication of these weeds which grow abundantly everywhere.

Another abundant and important weed is *Rotboellia exaltata*, which is a reservoir for maize dwarf mosaic virus (MDMV) or sugarcane mosaic virus (SCMV).

Viral diseases are very important in sorghum in Venezuela. There is a new and devastating strain of SCMV. SCMV can attack the plant at any age and destroy the young leaves in the

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whorl causing the plants to die. Some of the imported hybrids are so highly susceptible that they are wiped out by this disease.

Another very important disease is sorghum downy mildew (SDM). SDM was first observed in Venezuela about 1973, and in the following year it was found widespread in the country. We first found it in five states, and now it occurs throughout Venezuela. Fortunately, we have some hybrids resistant to SDM, so that this disease will probably become less important.

In the humid region at the foothills of the Andes, anthracnose is very important.

Cercospora leaf spot sometimes attacks the plants at an early stage, when it is damaging. Late infection probably has little effect on yield.

Zonate leaf spot can be damaging, completely blighting all the lower leaves of the plant.

Rust is present in Venezuela, but it generally occurs near maturity and is therefore not likely to cause significant yield reduction.

Head blight, charcoal rot, and *Fusarium* stalk rot also occur.

Some hybrids, when sprayed with insecticides, develop "nonparasitic" disease symptoms, which may resemble parasitic disease, and this can cause a lot of damage.

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Argentina

Craig:

Is the fungicide Ridomil being tested as a seed treatment for the control of SDM in Argentina?

Teyssandier:

Ridomil is being tested in Argentina primarily by Ciba-Geigy researchers in cooperation with other agencies. I have no information on the results.

Mathur:

When and where was sorghum downy mildew first observed in Argentina, and was it detected first on local cultivars or on introduced lines?

Teyssandier:

Sorghum downy mildew w[illegible] detected in Argentina in 1968 by Dr. Frezzi, on local cultivars at the Manfredi Experiment Station, Cordoba Province. Subsequently SDM was detected in all sorghum-growing regions of Argentina.

Brazil

(Dr. Fernandes was not present on the first day of the Workshop, due to air travel problems, and so there was no discussion on his paper.)

El Salvador

Frederiksen:

What percentage of the sorghum in El Salvador is used for human consumption and what percentage is used for animal feed?

Wall:

That is difficult to answer, and I will have to guess. Perhaps 70 to 75% is used as animal feed and the rest is used for human food. The importance of sorghum as a human food is increasing as more sorghum is grown, particularly in areas with unreliable rainfall.

Dange:

How important are the cross-infection problems from common pathogens in intercropped maize and sorghum in El Salvador?

Wall:

As 90% of the sorghum is grown in association with maize, these problems exist — particularly from sorghum downy mildew, leaf blight, and zonate leaf spot.

Balasubramanian:

Do you find an association between the occurrence of corn leaf aphids and more grain mold, particularly at low humidities?

Wall:

In El Salvador we rarely experience low humidities, but there is an association between grain molds and infestation with corn leaf aphid.

Mexico

Brhane:

Will you describe the environmental parameters and length of the growing season that allows grain yields of 14 tonne/ha from sorghum in Mexico?

Betancourt:

These yields come from high-yielding, USA-developed hybrids grown in a temperate climate — temperatures in the range 14 to 17°C, with 700 to 900 mm rainfall evenly distributed throughout a 5 to 6-month growing season (April to Sept). We use 140 kg N and 80 kg P_2O_5/ha.

Brhane:

How important are high altitude sorghums in Mexico and what are their major disease problems?

Betancourt:

There is a potential of about 4 million ha for high altitude sorghum production (1000 to 1900 m above sea level). We are developing cold-tolerant sorghums, based mainly on three cold-tolerant sorghum lines from Uganda, and will release three varieties in 1979. Disease problems are minor at the present time.

Murty:

Are grain molds on sorghum important in the lower-altitudes of Mexico?

Betancourt:

Grain molds do occur, but they are unimportant at the present time.

Venezuela

Malaguti:

Zonate leaf spot affects both sorghum and maize in Venezuela. The pathogen produces conidia and sclerotia. The sclerotia can persist for a long time in the soil. All hybrids, local and imported, are affected by zonate leaf spot. There is some confusion about the etiology of leaf spots and streaks resulting from infection by SCMV. In Venezuela, *Cercospora sorghi* produces catenulate chains of lesions and the more normal symptoms. The actual type of symptom depends on the environment, and vigor of the plant.

Balasubramanian:

Are the sclerotia of the zonate leaf spot fungus carried with the seed in Venezuela?

Malaguti:

The conidia are abundant on leaves and are the main source of inoculum. However, sclerotia can be found in the leaves. The sclerotia are soil- and debris-borne, and so are important epidemiologically — particularly if sorghum is planted on the same field year after year.

Sundaram:

I have observed abundant sclerotial formation in zonate leaf spot in Nigeria.

Malaguti:

The zonate leaf spot fungus was described originally by Bain, a long time ago. Sclerotia and conidia are commonly produced by this fungus.

Rao (N. G. P.):

How did the wild sorghums — the *S. verticilliflorum* and the *S. arundinaceum* — get into Venezuela?

Riccelli:

They were probably introduced in three ways:

1. Purposely for forage;
2. Along with sugarcane; and
3. With sorghum seed.

Malaguti:

Johnson grass introduction is somewhat complicated. I believe that johnson grass (*S. halepense*), was introduced with rice seed, for it appeared first in areas of rice production. Rice seed was widely introduced 20 years ago.

Sorghum Diseases in East Africa

H. Doggett*

The sorghum-improvement program for Kenya, Uganda, and Tanzania was a common program operated under the East African Community until the dissolution of that body in mid-1977. For much of the past 30 years, professional staff numbers were low; at times there was only one plant breeder. A good number of variety-trial centers was available, covering the full range of ecological conditions under which sorghum was being grown in the region.

Disease organisms are parasitic. Essentially, we are dealing with a system of two populations, each of which contains considerable genetic variability. During the course of evolution, there has been a constant selection pressure to favor resistance mechanisms in the host plants and a constant selection pressure on the pathogens to favor variants which can successfully break down such resistances. The host plants which could not develop resistance to serious diseases have been eliminated; the pathogens which could not succeed in breaking down the resistance mechanisms are no longer with us today. This statement is approximately true, because those organisms which were able to achieve some form of symbiosis with the host are probably not doing serious damage.

The question, "which sorghum diseases are important in East Africa?" is automatically asking about the disease resistances possessed by the cultivars grown there. Wild sorghums are indigenous to East Africa; the crop has been there for a long time, and there has always been some movement of sorghum into East Africa down the Nile from the Sudan. There is constant introgression between the wild sorghums and the cultivated crop, and we would therefore expect the indigenous cultivated sorghums to have accumulated sufficient resistances to ensure that diseases are not major causes of yield loss. This is essentially the picture, and lists of the names of diseases recorded are a function both of the cultivars grown and the placement of plant pathologists. There has never been a sorghum pathologist working in East Africa, and disease records were usually the work of government plant pathologists responsible for working on the whole range of cultivated crops. Conspicuous among these were George and Maud Wallace, working in what was formerly Tanganyika.

The third component of the system is the environment, the ecological conditions under which the interaction between pathogen and host plant is expressed. Certain diseases are greatly influenced by this. Among these in East Africa are grain (covered) smut, and downy mildew. A planting of an introduced cultivar as a strip down the catena in the Lake region of Tanzania showed a crescendo of incidence of grain smut increasing from negligible levels on the coarse sands near the hill top to a heavy incidence on the heavy black montmorillonitic clays in the valley. Similarly, cultivars which could be grown in the main growing seasons in Uganda with scarcely a sign of downy mildew could be quite severely affected by this disease, if grown during the cool period between seasons.

Diseases common on indigenous cultivars are few in number, but a glance at Table 1 shows that plenty of diseases occur. Some of these can show up in very severe forms on exotic germplasm; nobody knew that charcoal rot occurred on sorghum in Tanzania until a susceptible dwarf shallu was grown on light soil in 1952. All that I can usefully tell you is the names of those diseases which were recognized as common on the indigenous germplasm, and then to pick out a few which have shown up quite badly on exotic material. No doubt everything on the list will show up badly if the appropriate susceptible germplasm is introduced, and other pathogens not yet

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recognized could probably be found on appropriate exotic sorghum cultivars.

Diseases Common on Indigenous Cultivars

Covered smut (*Sphacelotheca sorghi*) is conspicuous and was considered sufficiently important to be worth recommending seed-treatment measures; sulphur dust was used in the early days. I am not aware of any estimate of yield loss, but Wallace (writing about Tanzania in 1953) commented that he had seen fields of grain smut, and also fields of downy mildew, where the loss was as great as 30%.

I have not seen this level of damage from downy mildew, on indigenous cultivars grown in the proper season, but there are times when grain smut incidence can be quite high. Perhaps the local people have not selected very strongly against it, since the young smutted heads are eaten, especially by children. Wallace estimated the damage caused by all sorghum diseases as being not less that 5%: but this eye judgment by an experienced plant pathologist is not based on any yield trials of which I am aware. I have been hoping to hear of yield trials which estimate actual grain loss due to individual diseases on cultivars with good general resistance levels. I question the extent to which leaf diseases reduce grain yields on cultivars which retain three largely disease-free upper leaves until the stage when dry-matter accumulation in the grain has been completed.

Very common leaf spots include sooty stripe (*Ramulispora*) and grey leaf spot (*Cercospora*). Rust (*Puccinia*) is also common.

Diseases on Introduced Cultivars

Helminthosporium turcicum can be very severe, both as a seedling blight and as a leaf blotch. *Colletotrichum graminicola* can also be very severe, mainly as an anthracnose, but also as a stem rot in some cultivars. The diseases already mentioned as being common on local material have been abundant on many introductions. *Macrophomina phaseolina* has been picked out by susceptible introductions.

A real nuisance has been ergot, *Sphacelia sorghi*, in the work involving male steriles for the hybrid program. Grain molds have been troublesome in many places where early-maturing material has been grown.

Table 1. Pathogens of sorghum in East Africa.

Seed rots and seedling blights
Ascochyta sorghina
Helminthosporium turcicum
Root and stalk rots
Macrophomina phaseoli
Gibberella fujikuroi var *subglutinans (Fusarium moniliforme)*
Leaf spots
Helminthosporium turcicum
H. sorghicola[a]
Colletotrichum graminicola
Cercospora sorghi
Gloeocercospora sorghi
Ramulispora sorghi
R. sorghicola
Phoma insidiosa (= Phyllosticta sorghi)
Ascochyta sorghina
Mycosphaerella holci
Puccinia purpurea (often parasitized by *Eudariuca australis*)
Sclerospora sorghi
Cephalosporium zonatum
Head blights and grain molds
Alternaria tenuis
Choanephora cucurbitarum
Curvularia lunata
Fusarium sp
Gibberella fujikuroi
Phoma insidiosa
Rhinotrichum cucumerinum
Trichothecium roseum
Cladosporium sp
Epicoccum purpurescens
Sphacelia sorghi (often associated with *Cerebella sorghi-vulgaris*)
Sphacelotheca sorghi
Sphacelotheca cruenta
Sphacelotheca reiliana
Tolyposporium ehrenbergii

a. not confirmed (to my knowledge), but was certainly on material at Serere. Bacterial diseases almost certainly occur, especially leaf stripe. Virus symptoms are also to be observed, possibly including sugarcane mosaic.

Disease Control

This topic belongs properly to the session on utilization of disease resistances. Seed dressing is so cheap and convenient that grain smut, and some of the other seedlings problems, can be controlled economically by its use. Once the farmer population has been converted to buying seed, this control method will be automatic. Until that time, however, it is important to check what happens when untreated seed is used in situations where smut and infection by seedling diseases is common. Apart from seed dressing, control measures in East Africa will be based on resistances for many years to come. As I have already indicated, there is no real possibility of building in resistances step by step, in the classical manner. Food quality and yields, and pests and diseases have all got to receive attention; national programs are not in a position to tackle leaf diseases one by one.

The crop improvement system used in East Africa involves a wide placement of variety-trial centers, where promising material was grown and evaluated primarily for yield. Rather broad observations were also taken for leaf-disease levels, which is one of the criteria for rejecting particular lines. This system is very effective, and every national program requires a scatter of small but numerous experimental plots. There may be disease objectives requiring specific attention, such as grain molds and *Sphacelia* resistance in short-term sorghums required to flower and ripen under conditions which are sometimes wet or humid. However, in general the levels of disease resistance in the breeding material will need to be maintained by screening for general levels of resistance. Effectively, in situations where disease levels are fairly high, this will amount to favoring the clean plants and discarding material at the susceptible end of the scale. A good scatter of experimental plots ensures that in every season there is some site, and usually several sites, where the susceptibles and the "cleaner" lines can be identified.

When the time comes to be thinking of yields of 14 000 kg/ha, then there may well be a place for "fine tuning" by breeding for specific disease resistances. Currently, we would all be delighted if the mean sorghum grain yield in East Africa could be raised to 2000 kg of grain per hectare. At these levels, I think that the plant breeder will find it impossible to give more priority to disease resistance work than that which I have outlined.

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Diseases of Sorghum in Ethiopia

Mengistu Hulluka and Brhane Gebrekidan*

Sorghum is perhaps the most widely distributed food cereal in Ethiopia. As an Ethiopian food crop, it is surpassed in importance only by tef (*Eragrostis tef*). Of the more than 1 million tonnes of sorghum grain produced on about 1.5 million ha, about 90% goes direct into human food and the rest into homemade beverages. Practically no sorghum is used as animal feed.

The sorghums grown in Ethiopia can be divided broadly into highland and lowland types. The highland types are generally adapted to altitudes above 1600 m, exceed 3 m in height, take about 9 months to mature, and are relatively cold-tolerant. The lowland sorghums, on the other hand, perform best below 1600 m altitude, have heights less than 3 m, mature in less than 4.5 months, and are best suited to the warmer and drier lowlands of the country.

In both groups, the range of genetic diversity is tremendous. Most farmers' fields in Ethiopia contain many different types of sorghum. Under these conditions, the development of diseases to epiphytotic levels is rare. The area in the country growing improved and uniform varieties is very small.

Even though sorghum is one of the most important food crops in Ethiopia, the overall national effort directed towards sorghum pathology work has been very insignificant. Depending mainly on the environment, certain diseases in some years become economically significant in some parts of Ethiopia. For the leading sorghum diseases, specific areas in the country are recognized as hot spots. Disease surveys in the major sorghum zones have shown specific diseases to be important in selected areas.

The Ethiopian national program on sorghum improvement, though concentrated on breeding, considers sorghum diseases as one of the major criteria of selection. The large Ethiopian sorghum germplasm holdings collected and maintained by the Ethiopian program have been visually evaluated for overall leaf-diseases reaction at the high-altitude conditions of Alemaya. A backcross program is under way to incorporate anthracnose resistance from local and exotic sources to selected elite highland sorghum varieties which are now susceptible to anthracnose. Segregating populations, advanced lines, and collections are grown in hot spots and resistant plants are identified for further advances. In general, in the breeding program, disease-resistant lines are a major group of parents used in the crossing block.

The major and minor sorghum diseases, with their general distribution in Ethiopia, are briefly listed below.

Grain Diseases

Smuts

Generally, four types of smuts have been confirmed in Ethiopia. Three of these, kernel smut (*Sphacelotheca sorghi*), loose kernel smut (*S. cruenta*), and head smut (*S. reiliana*), are widely distributed. Altitude and climate do not seem to be the factors limiting the distribution of these three types of smuts. They are reported in both lowland and highland sorghum-growing regions. Loose and covered kernel smuts are significant factors in reducing yield in some sorghum fields. On the other hand, head smut (*S. reiliana*) is probably more economically important on maize than on sorghum. This appears to be particularly true in many of the major maize-producing areas in the Rift Valley, even if sorghum is grown in adjacent fields. The fourth type of grain smut, long smut (*Tolyposporium ehrenbergii*, Kuhn), is usually limited to lowland regions, mainly in the administrative

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regions of Wollo, Tigrai, and Hararghe. In some areas and in some fields, the level of long smut infection could be quite significant.

Ergot

Ergot (*Sphacelia sorghi*) seems to be a relatively new disease in Ethiopia. It was first recognized in 1972 in experimental plots in Alemaya. For the last 3 years ergot has been quite bothersome at the Arsi Negele highland sorghum station. Infection may be initiated if the rainfall and the flowering time of sorghum coincide with the spore production of the fungus. There seem to be good sources of resistance against this disease in the breeding nursery. The Ethiopian sorghum program advances breeding lines at Arsi Negele only if they are free of ergot.

Grain Molds

If the sorghum grain matures under humid and rainy conditions, grain molds can become a serious problem. Early-maturing lines are generally affected by this disease. Several types of fungi have been known to cause grain mold, but the most common ones under our conditions are *Phoma insidiosum, Cladosporium* spp, *Stemphyllium* spp, and *Mycospharella* species. *Curvularia* spp are rare.

We have grown ICRISAT's International Sorghum Grain Mold Nursery over the last two seasons at our two highland stations, Alemaya and Arsi Negele; grain molds develop well at these stations. Almost all of the entries were lowland types, so they were poor agronomically and mostly sterile under our cool highland conditions. However, some lines have shown good tolerance to grain molds; some of these are being used as parents in our crossing program.

Leaf Diseases

Of the numerous leaf diseases of sorghum, several seem to be prevalent in many of the sorghum-growing areas of Ethiopia. In areas with high rainfall, anthracnose (*Colletotrichum graminicola*) seems to be the most common, and leaf rust (*Puccinia purpurea*) is perhaps second in importance. Leaf blight (*Trichometasphaeria turcica*) is important in many high-rainfall areas. Of the Ethiopian sorghum experimental stations, Arsi Negele and Alemaya (high altitude) frequently showed a much higher incidence of leaf diseases on sorghum than did the other stations. On the other hand, bacterial streak (*Xanthomonas holcicola*) is endemic in many of the fields from year to year at all altitudes. Its economic importance has not been assessed in Ethiopia. It appears to be of minor importance in its effect on sorghum production.

Other leaf diseases of sorghum are minimal in importance and sporadic in occurrence. These are *Ramulispora* leaf spot (*Ramulispora sorghicola*), sooty stripe (*Ramulispora sorghi*), bacterial leaf stripe (*Pseudomonas andropogoni*), downy mildew (*Sclerospora sorghi*), leaf spots caused by *Phoma sorghina, Mycosphaerella holci, Gloeocercospora sorghi*, and some others whose identity is not known. Of these diseases, downy mildew appears new to this area. It was first observed probably in 1972 in sorghum nurseries in the Dakatta and Alemaya research centers. It might have been first introduced with seeds from other countries.

Since anthracnose is the most important leaf disease in the country, the Ethiopian Sorghum Improvement Project (ESIP) has a backcross breeding program under way for introducing resistance to five of its high-yielding varieties. The recurrent parents are ETS-2111, ETS-2113, ETS-3235, Awash-1050, and WB-77. The resistance-donor parents are IS-2230, IS-158, Hafukagne × Hirna 305/547, NES-8827, and NES-8835. The recurrent parents are all well-adapted and high-yielding under cool highland conditions near 2000 m altitude. Donor parents have shown good resistance and excellent seed set in the Ethiopian highlands, though they are best suited to lowland areas. All possible combinations between the susceptible and the resistant varieties were made. One backcross to each of the 25 combinations has been completed to date. Crossing and identification of resistant segregates is done in the main season, while crossed seeds are grown for generation advance in the offseason.

Stalk Diseases

The major stalk disease observed in ESIP stations is charcoal rot. In the Asobot area, in years such as the 1977 crop season, the disease was

very severe and widespread. Charcoal rot has been observed at Asebot, Nazareth, and Alemaya. Drought and low soil-moisture levels during maturity of the crop appear very conducive to the development of charcoal rot.

One ICRISAT line, RS1 × VGC1, which looked excellent in several lowland Ethiopian locations, was very severely affected by the disease during the 1978 season.

Research on Sorghum Diseases

Location

Even though screening of sorghum germplasms and breeding materials for diseases is being undertaken in the field at many of the ESIP stations, most work on sorghum diseases has been undertaken at Alemaya College of Agriculture.

Nature of Research

As one of its objectives, ESIP has made various endeavors to upgrade research on crop protection. In relation to diseases of sorghum, several approaches have been tried to acquire knowledge on the situation of diseases of sorghum all over the country.

1. At different ESIP trial centers, germplasm screening for resistant lines has been going on since the establishment of the project. Advanced lines and varieties are evaluated each season for their reactions to diseases at various ecological zones. Resistant lines and/or varieties identified in such evaluations are either used as parents or advanced for their performance per se.
2. Annual survey for types, trend, and intensity of disease is being undertaken to assess any change in development of diseases of sorghum.
3. Some lines are being evaluated in a disease nursery to screen the superior materials with uniform infection pressure to verify levels of possible resistance.
4. Various seed-dressing chemicals have been tested for effectiveness to control some grain and leaf diseases.
5. In cooperation with international and national programs, various sorghum-disease nurseries are normally planted for evaluations under Ethiopian conditions.

In the future, further detailed studies of sorghum diseases are expected to be done if staff availability and facilities do not continue to be limiting factors.

Achievements, Problems, and Prospects for Sorghum-Disease Research in Ethiopia

So far our activity in sorghum-disease research has been centered mostly around survey work, assessment of the disease situation in the country, and evaluation of some of the collection of germplasms for their reaction to various diseases in the field. In this respect, we were able to categorize the major and minor diseases affecting sorghum production. Besides, in cooperation with breeders and other workers, we make periodic evaluations of all collected lines for their resistance to the known diseases occurring naturally in the field. Few detailed studies involving individual diseases have been undertaken. One could screen and select very good lines with good sources of resistance to many of the diseases listed earlier. Since most of the lines have been selected on the basis of their overall performance in other factors, some good sources of resistance could be rejected if selection pressure for other factors, such as yield and quality, were found to outweigh disease reaction. Shortages of personnel and funding have limited this aspect of research in the improvement of sorghum production.

Advanced lines, by necessity, must pass through disease nurseries before they are released. Research on various approaches of disease studies, mainly on control measures and breeding for resistance, must continue along with the breeding program. Such research needs an integrated team approach where breeders and pathologists work together to find solutions to the sorghum disease problem.

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Sorghum Diseases in Malawi

B. D. A. Beck*

Sorghum is widely grown by smallholder farmers throughout Malawi, but is of economic significance only in the lowland, semi-arid areas of the Shire Valley.

Sorghum is grown in the warm, wet season as a food crop and as a source of grain for brewing beer. Epiphytotic outbreaks have not been reported, and no estimates of crop loss caused by diseases are available.

The following pathogens have been recorded on sorghum in Malawi:

Cercospora sorghi Ellis and Everhart — causing circular to elliptical lesions, which are grey with a red border, widespread and occasionally severe (in the Shire Valley).

Cercospora (?) vaginae Kruger — causing rotting of the leaf sheaths.

Cladosporium herbarum (Pers) Link ex Grey — causing mold of the inflorescence; severe in late rains in the Shire Valley.

Colletotrichum graminicola (Cesati) Wilson — causing a leaf spot, occasionally sufficiently severe to kill leaves.

Gibberella (?) *zeae* (Schweinitz) Petch — (conidial stage *Fusarium graminearum* Schwabe) causing long, elliptical leaf spots, grey with a red border and yellow halo.

Gloeocercospora sorghi Bain and Edgerton — causing small, oval, water-soaked leaf spots, with a red margin, which enlarge to elongate large zonate spots that may kill the leaf (widespread in the Shire Valley).

Helminthosporium maydis Nisikado and Miyake and *Cochliobolus heterostrophus* (Drechsler) Drechsler — causing large, elongate light grey lesions with a reddish margin and extensive leaf necrosis (in the Shire Valley).

Helminthosporium rostratum Drechsler — causing small brown spots, bounded initially by the leaf veins, but later coalescing to form large necrotic areas (occasionally severe in the Shire Valley).

Mycosphaerella spp and *Phoma insidiosa* Tassi — both causing grain spotting (widespread in the Shire Valley).

Puccinia purpurea Cooke — causing a typical rust, but generally attacking the crop too late to cause heavy yield loss (widespread and occasionally heavy in the Shire Valley).

Ramulispora sorghi (Ellis and Everhart) Olive and Lefebvre — causing elongate, straw-colored leaf lesions which later turn sooty black and cause large areas of leaf necrosis (widespread in the Shire Valley).

Ramulispora sorghicola Harris — causing small, water-soaked lesions which develop into small, elongate spots with a marked border and occasionally black sclerotia.

Sclerospora sorghi Weston and Uppal — causing blight, leaf shredding and stunting. When severe, it causes the death of young plants.

Sphacelotheca cruenta (Kuhn) Potter — stunting of the plant and distortion of the inflorescence to a "crazy top" effect. Infected spikelets form a typical loose smut (widespread in the Shire Valley and especially severe on ratoon crops).

Sphacelotheca reiliana (Kuhn) Clinton — causing a typical head smut, locally causing severe losses, especially in seasons when the rainfall is low (widespread in the Shire Valley).

Sphacelotheca sorghi (Link) Clinton — causing a typical covered smut with the grain being replaced by smut sori (widespread in the Shire Valley).

Tolyposporium ehrenbergii (Kuhn) Patouillard — causing occasional long spore sacs replacing a small number of grains in the head.

Maize Streak Virus (?) — causing symptoms in sorghum similar to those on maize: on the older leaves, necrosis occurs; buff or dark brown stripes appear and the vascular bundles turn red.

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Control of the major leaf diseases will be sought by the selection of resistant cultivars.

Control of covered smut and loose smut will initially be by the use of seed dressings of cultivars produced in the multiplication scheme on the project seed farm.

Acknowledgment

My thanks are due to the Malawi Government for permission to present this paper and to Dr. M. A. Siddiqi for checking disease identifications and for his advice in the preparation of the paper.

Principal Sorghum Diseases in Niger

O. Sidibe*

Sorghum is one of the major cereal crops in Niger. It is the most important cereal following pearl millet with an area of 732 480 ha and total production of 342 035 metric tons in 1977. In October 1978 the Directorate of the Agriculture Department estimated the area under sorghum at 799 995 ha with a yield of 361 031 metric tons. Disease is one of several factors that reduce sorghum yield in Niger.

Sorghum is grown on light dune soils and the heavy clay soils of the valleys. Sorghum is widely grown along waterways on receding moisture after floods (sorghum "de decru"). On dune soils sorghum is grown as the sole crop. It is also intercropped with millet or groundnut and sometimes with cowpea. It is found in the rainfall zones from 300 to 800 mm isohyets.

Major Sorghum Diseases

Observations on sorghum diseases in Niger have been reported by Jouan and Delassus (1971) and others.

Although several pathogens infect sorghum, only some are of great economic importance. Identified pathogens include *Ramulispora sorghicola* and *R. sorghi, Cercospora sorghi, Colletotrichum graminicola, Ascochyta sorghina, Helminthosporium turcicum* and *Gloeocercospora sorghi* (leaf diseases), and *Sphacelotheca cruenta*, which is rare. Grain molds are caused by a complex group of pathogens such as *Phoma insidiosa, Fusarium moniliforme, Curvularia lunata*, and species of *Aspergillus, Helminthosporium*, and *Colletotrichum.* These saprophytic and facultative parasites are very important when grain maturity coincides with late rains. Very little work has been done to study the role of these microorganisms in the propagation of molds, production of mycotoxins, and contamination of harvested sorghum.

Striga hermonthica is an important parasite of sorghum and pearl millet in Niger, causing considerable loss of yield.

Sorghum yield loss due to diseases has not been estimated experimentally, but from observations it is clear that serious infection leads to a considerable reduction in yield, and even to complete destruction of the plant.

The principal diseases are now discussed, in order of importance.

Leaf Diseases

Oval Leaf Spot

Oval leaf spot (*Ramulispora sorghicola* Harris) is the most important and most common disease. It was recorded for the first time in the neighboring country, Nigeria, by Harris (1960), and was observed in Niger by Jouan and Delassus (1971). This disease attacks all sorghum cultivars (local varieties as well as improved varieties) each year, regardless of ecological and weather conditions. Infection starts in June and the fungus can infect sorghum at any growth stage. The first symptoms are small elliptical water-soaked spots, 1.5 to 3 mm × 3 to 8 mm, surrounded by a thin reddish purple margin. These spots often cover the entire leaf surface. Black sclerotia are formed in the center of these spots on the lower side of the leaves, and are somewhat sunken within the tissues. Highly infected leaves dry rapidly.

Sooty Stripe

In sooty stripe (*Ramulispora sorghi* [Ellis and Everhart] Olive and Lefebvre) lesions, measuring 3 to 10 cm × 1 to 3 cm, develop on the leaves (particularly the lower and older leaves), and are elliptical, elongated, pale-colored and sur-

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rounded by a reddish purple margin which becomes light in color. Under humid conditions, especially early in the morning, a greyish brown fuzz of conidia and conidiophores can be observed in the center of the spots. Sooty stripe incidence was less in 1978 than in 1977. It seems that the intensity and frequency of this disease are promoted by heavy and continual rainfall from emergence to flowering.

Grey Leaf Spot

Grey leaf spot (*Cercospora sorghi* Ellis and Everhart) is also an important sorghum disease in Niger, and the symptoms can be confused with those of oval leaf spot particularly on young leaves. Several types of symptoms appear, depending on the cultivars. Generally, infection starts at an early stage (at the 1 to 4 leaf stage) but can appear at all growth stages. In cases of severe attack, infected leaves become completely dry. Grey leaf spot is found in all the Niger regions where sorghum is grown. It is common to observe greyish white fuzz covering the infected leaves early in the mornings. Yield losses due to grey leaf spot have not been estimated, but the leaf drying caused by the disease is accompanied by reduction in grain size and weight.

Anthracnose, or Red Leaf Spot

Anthracnose (*Colletotrichum graminicola* [Cesati] Wilson) can occur on sorghum during periods of heavy rainfall and high humidity. Several types of lesions are observed on the leaves. Infected stems become red to purple in color. In 1977 and 1978 it was found that early-flowering varieties were more susceptible to anthracnose, particularly on heavy soils.

Leaf Blight

Leaf blight (*Helminthosporium turcicum* Pass.) is not a very important disease in Niger and appears irregularly on sorghum at the 10-leaf stage. Humid climatic conditions increase intensity of the disease, especially in heavy soils. *H. turcicum* produces elongate and somewhat elliptical lesions over almost half the length of the leaf (1 to 5 cm wide, 4 to 15 cm long). Several yellowish brown or blackish brown lesions may appear. Humidity and heat promote production of fruiting bodies of the parasite, which can entirely cover the two surfaces of the infected parts and can be recognized by a deposit of blackish brown dust. Local varieties have better resistance to this disease than exotic varieties.

Zonate Leaf Spot

Zonate leaf spot (*Gloeocercospora sorghi* Bain and Edgerton) has been very severe in the Bengou region where rainfall is high. Large lesions, 7 to 10 cm in diameter of irregular shape, and yellowish brown to reddish purple in color, are observed on old and young plants. The central part dries and becomes silver grey.

Bacterial Leaf Streak

In bacterial leaf streak (*Xanthomonas holcicola* [Elliot] Starr and Burkholder) disease, water-soaked bacterial lesions appear on the leaves. At first these spots are light in color. They subsequently become red, reddish purple, and darker. This disease is characterized by the formation of exudate on the leaf. The disease becomes more important in regions where rainfall ranges between 450 mm and 800 mm.

Rough Leaf Spot

Rough leaf spot (*Ascochyta sorghina* Saccardo) is of secondary importance and can attack sorghum leaves from the 2 to 3 leaf stage up to just before flowering. The lesions contain minute black fruiting bodies. Apparently high humidity limits propagation of the disease.

Panicle Diseases

Among the diseases that affect the panicle, only the four types of smut and grain molds are of economic importance in Niger. Head smut and long smut were observed throughout Niger and on all existing varieties. Covered smut is less frequent.

Long Smut

Long smut (*Tolyposporium ehrenbergii* [Kuhn] Patouillard) is very common, especially when heavy rainfall accompanied by high temperatures coincides with flowering. Even when rainfall is low, infection by *T. ehrenbergii* becomes serious under very humid conditions in the morning. This is what happened to irrigated

sorghum and sorghum "de decru" grown along Lake Chad in 1976 and 1977. All cultivars are susceptible to this disease but intensity of infection varies with the region and year. For example, incidence of long smut was very high during the 1977 rainy season and low in 1978. Our observations in 1977 and 1978 have revealed that intensity and percent infection of long smut are related to the very favorable climatic conditions for the disease.

Head Smut

In head smut (*Sphacelotheca reiliana* [Kuhn] Clinton), grains are totally destroyed causing heavy loss. Intensity of the disease is high in shaded and slightly flat areas (for example under trees). Infected panicles are transformed into enormous bags (10 to 20 cm long, 5 to 15 cm wide) filled with spores. Once the spores have been liberated, all that remains of the bags are longfibers (remnants of the vascular system of the panicle).

Grain Molds

These are mainly caused by imperfect fungi of the following genera: *Phoma, Curvularia, Aspergillus, Helminthosporium, Fusarium*, and *Penicillium*. The problem of grain molds is closely related to heavy rainfall late in the season during or after grain maturity. In years of normal rainfall the problem is less important. Local varieties seem to have better resistance to this disease than introduced varieties, and late varieties are more resistant than early or semi-late varieties.

Loose Smut

Loose smut (*Sphacelotheca cruenta* [Kuhn] Potter) is often found in sorghum affecting secondary tillers. Main panicle infection by loose smut is rare and causes stunting of the plant. Low temperatures and high rainfall promote development and propagation of the disease.

Covered Smut

Covered smut (*Sphacelotheca sorghi* [Link] Clinton) is less common (few isolated cases in 1977 and 1978) than loose smut. At present, this disease has no economic importance in Niger.

Parasitic Weeds

Striga hermonthica Benth. is present in all millet fields and to a lesser extent in sorghum and cowpea fields. Sorghum parasitized by *Striga* is slightly stunted, the color of the leaves changes and becomes yellowish, similar to that produced by water deficit symptoms. In 1978, in a field at Tarna, we observed a millet field highly infected by *Striga*, but sorghum in the neighboring field escaped attack by this phanerogamous parasite. More to the south, in Bengou, on the other hand all sorghum fields were severely infected with *Striga* and neighboring millet fields escaped attack. These observations make us wonder if there are races in the *Striga hermonthica* species that prefer or are suited to particular species of cereals or types of soil.

Acknowledgment

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Sorghum Diseases in Nigeria

P. D. Tyagi*

Sorghum is one of the most important cereals grown in Nigeria. It is grown primarily as a grain crop for human consumption, but it is also used for making beer, medicines, and as livestock feed. From 1960 to 1975, it accounted for about 49% of the total production of cereals in Nigeria and, on average, 46% of the annual acreage devoted to cereals (Abalu 1978). The estimated total production of sorghum in Nigeria in 1977 was 3.5 million tonne, corresponding to a yield of about 610 kg/ha.

Four ecological zones are recognized within the sorghum-cultivation area (Table 1), and the prevalence and severity of sorghum diseases vary widely in these zones.

Research on all aspects of sorghum, including diseases, is the responsibility of the Institute for Agricultural Research, Ahmadu Bello University, Zaria. The Institute has its main research center at Samaru (11°03'N, 7°38'E, alt 685 m) and research stations located at Kano (12°03'N, 8°32'E, alt 472 m) and Mokwa (9°18'N, 5°4'E, alt 152 m). These three research stations, situated in different ecological zones, facilitate plant pathology trials.

Sorghum suffers from many diseases. The fungal diseases of sorghum reported from Nigeria are listed in Table 2. While diseases due to bacteria and viruses are not well documented, the importance of bacterial diseases was emphasized by Zummo (1975).

Table 1. Sorghum-growing ecological zones.

Ecological zone	Growing season (days)	Average rainfall (mm)
Northern Sudan savanna	90–110	600
Sudan savanna	120–150	750
Northern Guinea savanna	150–180	1000
Southern Guinea savanna	180–220	1100

Table 2. Fungal diseases of sorghum in Nigeria.

Disease	Causal organism
Grain molds	*Phoma, Fusarium, Curvularia etc.*
Foliage diseases	
Grey leaf spot	*Cercospora sorghi*
Sooty stripe	*Ramulispora sorghi*
Anthracnose	*Colletotrichum graminicola*
Leaf blight	*Helminthosporium turcicum*
Downy mildew	*Peronosclerospora sorghi*
Rough leaf spot	*Ascochyta sorghina*
Zonate leaf spot	*Gloeocercospora sorghi*
Oval leaf spot	*Ramulispora sorghicola*
Rust	*Puccinia purpurea*
Smuts	
Covered smut	*Sphacelotheca sorghi*
Loose smut	*Sphacelotheca cruenta*
Head smut	*Sphacelotheca reiliana*
Long smut	*Tolyposporium ehrenbergii*
Other diseases	
Ergot	*Sphacelia sorghi*
Top rot ("pokkah boeng") and twisted top	*Fusarium moniliforme var. subglutinans*
Charcoal rot	*Macrophomina phaseolina*
Stalk rot	*Fusarium moniliforme*
False smut	*Cerebella sorghi-vulgaris*

Grain Molds

In the drought years of the early 1970s, sorghum crops in the northern Sudan savanna and

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Sudan savanna zones failed, or their yields were much reduced, as the rains did not last long enough to allow for full growth and development of the crop. Consequently, it was realized that significant advantage, both in terms of drought avoidance and yield potential, could be gained by selecting shorter-duration photoinsensitive sorghums. However, short-season varieties set and ripen grain under humid conditions conducive to mold attack. Such sorghum varieties would therefore have to possess resistance to grain molds, which reduce the yield and quality of the grain and, if they produce mycotoxins, may also constitute a health hazard.

Grain Mold Organisms

The fungi isolated from field-collected sorghum grain in Nigeria include *Alternaria longissima, A. tenuis, Aspergillus flavus, A. niger, Cladosporium* sp, *Cochiliobolus* sp, *Colletotrichum* sp, *Curvularia lunata, Curvularia* sp, *Drechslera halodes, D. rostrata, Epicoccum* sp, *Fusarium equiseti, F. moniliforme, F. semitectum, Nigrospora* sp, *Penicillium* sp, and *Phoma sorghina*. The most common in relative order of frequency are *Phoma sorghina, Fusarium,* and *Curvularia*. To determine their frequency in grain at different stages of development and in different varieties, isolates were made on potato-dextrose agar medium from a 100-grain sample of each variety. The three fungi were detected from the "milk" stage of grain development and their numbers generally increased as the grain developed (Tables 3, 4). The predominance of *Phoma sorghina* over other fungi is interesting, in that in most other countries *Fusarium* and *Curvularia* are more common and *Phoma* occupies only a secondary position. However, the higher percentage of *Fusarium* over *Phoma sorghina* in variety GM-4 needs to be reconfirmed.

Fungi present in 31 market samples of sorghum collected during 1969 and 1970 from places situated in different ecological zones were identified by the Danish Government Institute of Seed Pathology, Denmark. Although these samples were not collected directly from the field, they consisted largely of *Phoma, Fusarium,* and *Curvularia*.

Mycotoxin Contamination of Grain

Recent work done in the Institute for Agricultural Research at Samaru (Salifu, personal communication) has shown the presence of some mycotoxins in field-collected sorghum grains of short-season varieties. For example, aflatoxins were detected in grains of varieties CK-60, Serena, IS-9289, and IS-5790. Another mycotoxin, Patulin, was found in grains of variety IS-5790. Zearalenone was detected in

Table 3. Percentage of *Phoma sorghina, Fusarium,* and *Curvularia* in four varieties of sorghum at different stages of grain development.

		Growth stage at sampling			
Variety	Fungus	Milk	Soft dough	Hard dough	Ripe
GM 1	*Phoma sorghina*	8	42	62	70
	Fusarium	6	14	25	30
	Curvularia	2	10	20	18
GM 6	*Phoma sorghina*	8	10	56	67
	Fusarium	7	12	38	35
	Curvularia	0	6	8	7
GM 4	*Phoma sorghina*	2	2	6	12
	Fusarium	5	18	70	90
	Curvularia	0	2	5	7
GM 8	*Phoma sorghina*	7	14	64	80
	Fusarium	5	7	5	12
	Curvularia	2	2	8	9

Table 4. Percentage of *Phoma sorghina*, *Fusarium*, and *Curvularia* in grains of different varieties of sorghum.[a]

Variety	*Phoma sorghina*	*Fusarium*	*Curvularia*
GM 1	70	30	18
GM 2	80	17	8
GM 3	30	43	6
GM 4	12	90	7
GM 5	81	11	14
GM 6	60	41	8
GM 7	32	9	2
GM 8	83	12	9
GM 9	48	7	16
GM 10	37	14	3
GM 11	59	23	4

a. Samples were collected from field-ripened grain.

grains of varieties CK-60 and Serena. However, grains of the long-season varieties Fara Fara and Short Kaura were free from mycotoxins.

Phoma is considered to be a nonmycotoxin-producing fungus. However, it has recently been found that various strains of *Phoma sorghina* produce a metabolite which is acutely toxic to rats and chickens and probably also to other mammals and birds (Boerema et al. 1977). These reports highlight the importance of grain mold resistance in short-duration varieties.

Screening for Grain Mold Resistance

Work on testing sorghum varieties against grain molds was initiated by King (1972) in Nigeria. It was further continued by Zummo (1974, 1975); Manzo (1976), and myself. Environmental conditions are normally favorable for the development of grain molds at Samaru. Screening of varieties was therefore done under natural conditions of infection, although testing under artificial conditions has its own utility. The following varieties/lines have shown good resistance to grain molds during 1973–1978:

Light-Colored Grain

SRN-374, SRN-401, SRN-412, SRN-414, SRN-415, SRN-416, SRN-418, SRN-420, SRN-1901, SRN-1902, SRN-1918, SRN-1923, SRN-2265, SRN-2657, SRN-2658, SRN-3667, SRN-4208, SRN-4209, SRN-4250, SRN-4698, SRN-5374, SRN-5695, SRN-6494, and SRN-6639.

Dark-Colored Grain

SRN-612, SRN-922, SRN-1956, SRN-2019, SRN-2111, SRN-2146, SRN-2152, SRN-2155, SRN-2230, SRN-2272, SRN-2303, SRN-2310, SRN-2586, SRN-2745, SRN-2775, SRN-2816, SRN-2826, SRN-2859, SRN-3175, SRN-3344, SRN-3604, SRN-3629, SRN-3660, SRN-3964, SRN-4327, SRN-5849, SRN-5982, and SRN-6076.

Although these lines have good resistance to grain molds, many of them are highly susceptible to grey leaf spot at Samaru where the tests were done.

Further work on grain molds at Samaru will include the following:

1. Isolating *Phoma sorghina*, *Fusarium*, and *Curvularia* from parts of the grain in order to know the depth of infection.
2. In vitro studies on antagonism and synergism among *Phoma*, *Fusarium*, and *Curvularia*.
3. Effect of these fungi (singly and in combination) on germination of different varieties.
4. Efficacy of various systemic and nonsystemic fungicides in eliminating infection of these fungi from grain.
5. Testing grains for mycotoxin contamination at different stages of development.
6. Finding new sources of resistance (particularly those which also have resistance to grey leaf spot and other foliar diseases).

Diseases of Foliage

A few years back, foliar diseases were considered of little economic importance in Nigeria (Anon. 1970, 1971). Local varieties grown for many years by farmers probably had some tolerance or resistance to a number of diseases. Low fertility, low plant populations, and intercropping characteristics of most sorghum production in Nigeria, were additional deterrents to the development of epiphytotics of foliar diseases in farmers' fields. However, with the introduction of new high-yielding varieties and intensified crop production, several leaf diseases have become important. Grey leaf spot is the most common foliar disease in Nigeria.

Although long-season varieties suffer from the disease, the short-duration varieties are most heavily attacked. Other foliar diseases that appear in severe forms are sooty stripe and anthracnose. High incidence of rough leaf spot and zonate leaf spot has been observed in some varieties. Leaf blight and downy mildew are more common on tall varieties like YG 5760-3-10, SFF-60, FFBL 3-1-6, C 7-4-2, ML-4, and FD-1.

Most of the earlier work on foliar diseases was confined to the testing of the world sorghum collection against different diseases using natural infection. Futrell and Webster (1966a) evaluated 2693 varieties/lines for resistance to sooty stripe; only 5% of the entries were resistant (Tables 5, 6). Among the resistant lines, 17% were from the Conspicuum race and 10% from the Caffrorum race. The highest percentage of resistant entries were from Upper Volta (47%) followed by Nigeria (10%).

Futrell and Webster (1966b) also tested a part of the world collection and the Nigerian collection of grain sorghums for resistance to SDM. Twenty-two lines in the Nigerian collection remained free of the disease. Fourteen percent of the entries in the world collection were resistant, the highest percentage (47%) being from the Caffrorum race. During 1977, 755 lines of the sorghum-breeding nursery were scored for their reaction to downy mildew (Table 7). Many of the lines showing resistance may be escapes. A sick plot is to be developed for screening sorghums against downy mildew.

King (1972) evaluated a part of the world sorghum collection for resistance to different foliar diseases (Table 8). During 1977, a sorghum breeding nursery consisting of 725 lines was tested for resistance to grey leaf spot, which appeared in epidemic proportions at Samaru and eighteen lines remained free from the disease (Table 9).

Table 5. Reaction to sooty stripe of a part of the world sorghum collection (by taxonomic group).

Race	Entries tested (No)	Entries resistant (%)
Conspicuum	544	17
Durra	510	2
Caudatum	381	1
Roxburghii	169	1
Dochna	142	3
Caffrorum	97	10
Nigricans	69	4
Caffrorum-Birdproof	50	8
Nervosum-Kaolang	35	3
Membranaceum	25	8
Caffrorum-Darso	22	5
All others	649	0
Total	2693	5

Table 6. Reaction to sooty stripe of a part of the world sorghum collection (by country of origin)

Country of origin	Entries tested (No)	Entries resistant (%)
India	855	1
USA	599	2
Nigeria	530	10
Sudan	194	1
Upper Volta	75	47
Japan	60	2
Uganda	53	0
Mali	47	6
Ethiopia	46	2
South Africa	42	2
Rhodesia	26	4
Mexico	21	0
Other countries	145	2
Total	2693	5

Table 7. Frequency of breeding lines with various incidences of downy mildew in the sorghum-breeding nursery at Samaru during 1977.

Incidence of downy mildew (%)	Number of lines (No)
0	652
up to 10	73
11–20	18
21–30	6
31–40	3
41–60	3
more than 60	0

Table 8. Reaction of the world sorghum collection to leaf diseases at Samaru during 1970, 1971, and 1972.

	Percent entries					
	Resistant or escape			Susceptible		
Disease	1970	1971	1972	1970	1971	1972
Grey leaf spot	8.0	34.2	12.8	92.0	65.8	87.2
Sooty stripe	31.6	15.5	6.5	68.4	84.5	93.5
Anthracnose	86.6	67.1	87.5	13.4	32.9	12.5
Rough leaf spot	64.1	50.8	51.2	35.9	49.2	48.8
Zonate leaf spot	78.0	23.0	84.6	22.0	77.0	15.6
Oval leaf spot	81.3	97.4	87.4	18.7	2.6	12.6

Number of entries: 1970 = 4000; 1971 = 3322; 1972 = 880

Table 9. Lines showing various grey leaf spot scores in sorghum-breeding nursery during 1977.

Rating[a]	Number of lines (No)
0	18
1	89
2	223
3	247
4	131
5	17

a. Based on a 0–5 scale, with 0 being free from infection

The field reaction of old and of recently recommended varieties for different ecological zones to some leaf diseases is given in Tables 10 to 13. Except for grey leaf spot, the new varieties have good resistance to most foliar diseases. One variety, B.E.S., is however highly susceptible to anthracnose, and will need to be replaced by some other variety.

Further work on foliar diseases is directed toward (a) estimating the losses in yield due to foliar diseases at different disease intensities, (b) studying the rate of increase of important foliar diseases on some varieties during different seasons, and (c) locating lines with stable resistance to as many diseases as possible.

Table 10. Field reaction[a] of varieties grown in the northern Sudan savanna to leaf diseases during 1977 and 1980.

Variety	Height (cm)	Days to heading	GLS		SS		Anth.		LB		OLS		SDM[b]	
			77	78	77	78	77	78	77	78	77	78	77	78
G-59	270	80	2	3	2	0	2	0	2	2	2	1	0	0
HP-3[c]	130	65	4	4	1	1	1	0	2	1	1	1	0	0
HP-8[c]	140	60	3	3	1	1	1	0	1	1	1	1	0	0
B.E.S.[c]	160	66	2	3	1	2	4	4	1	0	1	1	0	0

a. Based on a 0–5 scale, with 0 being free from the diseases
b. Percent downy mildew
c. New variety

Table 11. Field reaction[a] of varieties grown in the Sudan savanna to leaf diseases during 1977 and 1978.

Variety	Height (cm)	Days to heading	GLS		SS		Anth.		LB		OLS		SDM[b]	
			77	78	77	78	77	78	77	78	77	78	77	78
YG-5760	360	105	0	3	2	3	1	0	2	3	2	2	5	3
RZI[c]	190	103	0	4	1	0	1	0	1	2	1	2	0	0
KBL[c]	200	98	3	3	1	1	0	0	1	2	1	1	0	0

a. Based on a 0–5 scale with 0 being free from the diseases
b. Percent downy mildew
c. New variety

Table 12. Field reaction[a] of varieties grown in northern Guinea savanna to leaf diseases during 1977 and 1978.

Variety	Height (cm)	Days to heading	GLS		SS		Anth.		LB		OLS		SDM[b]	
			77	78	77	78	77	78	77	78	77	78	77	78
SFF-60	360	120	1	2	0	0	0	0	3	3	1	2	6	4
FF-B-L	360	120	1	2	0	0	1	0	4	3	1	2	9	5
SK-5912	260	130	4	3	1	1	1	0	1	1	1	2	0	0
SL-181[c]	190	127	4	2	1	1	0	0	1	1	2	3	0	0
SL-187[c]	180	126	4	3	1	1	0	0	1	1	2	2	0	0
SL-1499[c]	200	123	2	2	1	1	0	0	0	0	2	3	0	0

a. Based on a 0–5 scale with 0 being free from the diseases
b. Percent downy mildew
c. New variety

Table 13. Field reaction[a] of varieties grown in the southern Guinea savanna to leaf diseases during 1977 and 1978.

Variety	Height (cm)	Days to heading	GLS		SS		Anth.		LB		OLS		SDM[b]	
			77	78	77	78	77	78	77	78	77	78	77	78
C 7-4-2	400	150	1	1	0	0	0	0	2	3	1	1	7	3
ML-4	400	150	1	1	0	0	1	0	3	3	0	1	6	3
FD-1	400	150	1	1	1	0	0	0	3	3	1	1	4	2

a. Based on a 0-5 scale with 0 being free from diseases
b. Percent downy mildew

Smuts

Sorghum in Nigeria is attacked by the four smuts. Covered smut and loose smut are prevalent in all the sorghum-growing zones. Head smut is next in importance and distribution. Long smut is generally confined to the Northern Sudan savanna and Sudan savanna zones. In

years of drought, this smut appears in epidemic proportions. The restricted distribution of long smut is probably related to temperature and rainfall (Table 14). Minimum and maximum temperatures are higher in the Northern Sudan savanna and Sudan savanna zones as compared to the Northern Guinea savanna zone (based on data from Sokoto, Kano, and Samaru). Further, the amount of rainfall between heading and anthesis seems to be a critical factor in long smut infection. Soil temperature has also to be taken into account. However, to reach a definite conclusion as to why long smut is confined to the extreme north of Nigeria, careful experiments under controlled conditions of soil, air temperature, and humidity will be required. Losses in yield up to 10% have been reported (Anon. 1971). As a result of farmers practicing seed dressing during the past few years, losses from covered smut and loose smut have been substantially reduced. Fernasan D (25% thiram plus 20% Lindane) and Aldrex T (50% thiram plus 25% Aldrin) are generally used for seed dressing.

King (1972, 1973) and Manzo (1976) did considerable work on smuts in Nigeria. They standardized techniques for artificial inoculations with different smuts, particularly head smut and long smut. Manzo (1976) made a detailed study of the mode of infection by *Tolyposporium ehrenbergii* and found that infection resulted only when sorghum plants were inoculated with either sporidia or germinating teliospores from the boot stage to not later than anthesis. Nongerminated teliospores did not cause infection, while inoculations with germinating teliospores at the boot stage gave maximum infection.

Work on physiologic specialization showed that races 2 and 4 of covered smut were prevalent in the wet season. An unknown race also existed in the dry season around Lake Chad. A race resembling race 2 of loose smut was detected, and it is believed that another race of loose smut, not reported elsewhere, exists in Nigeria. The races of head smut prevalent in Nigeria differ from races reported so far.

Good sources of resistance to the four smuts are known (Table 15). It may be desirable to incorporate resistance against head smut and long smut, which as yet are not controlled satisfactorily by the use of fungicides.

Apart from grain molds, foliage diseases and smuts, little work has been done on other sorghum diseases in Nigeria.

Table 14. Mean rainfall in 10-day periods at Sokoto, Kano, Samaru, and Mokwa

	Sokoto	Kano	Samaru	Mokwa
	mm			
1–10 Jun	34.80	31.50	46.23	76.45
11–20 Jun	28.45	47.75	59.44	59.94
21–30 Jun	42.16	53.59	59.94	50.80
1–10 Jul	46.99	54.36	58.42	54.36
11–20 Jul	56.13	67.30	70.36	48.77
21–31 Jul	82.04	89.15	92.71	66.55
1–10 Aug	78.49	101.85	82.55	52.83
11–20 Aug	72.64	103.89	88.39	44.70
21–31 Aug	98.55	108.20	110.49	71.88
1–10 Sep	52.58	60.20	94.49	84.84
11–20 Sep	47.50	48.00	82.55	80.26
21–30 Sep	33.53	24.64	53.34	67.56
1–10 Oct	14.22	10.67	22.86	54.10
11–20 Oct	7.62	1.27	9.91	22.10
21–30 Oct	1.27	0.76	3.30	14.99

Table 15. Varieties/lines of sorghum resistant to smuts in Nigeria.

Smut	Variety/line
Covered smut	IS-9290
	CK × 299 b 6
	FFBL 3-1-6
	Line 453
	SKMDW-2347
	Durra selection
	Spur Feterita
	Kafir × Feterita
Loose smut	Kafir × Feterita
Head smut	Line 453
	U-10
	708
	H 37
	H 135-2
Long smut	Durra selection
	Pierce Kaferita
	Eup. Dwf. Broomcorn
	D. Dwf. Feterita
	Piper
	Desert Bishop
	AS-3749 STd. Feterita
	Dutch Boy

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Country Reports Discussion Session — Africa

Williams: I want to comment on Dr. Doggett's apparent view that there is no urgent need to work with disease resistance in breeding programs in East Africa. I agree totally with his balance concept — i.e., that the local materials have over many years come into balance with local pathogens. Thus if we are just trying to improve the local material by selecting within populations or crossing among the local types, these diseases may not become very much more important. But in most improvement programs exotic material is being introduced and is encountering new pathogen populations and new environments for the first time. When this happens, severe disease can develop. We are also vastly changing the characteristics of the cultivars grown. The introduction of early flowering and nonphotosensitivity is in effect changing the environment during flowering, and subjects the cultivars to stress pressure for which there has been no previous selection pressure for resistance — e.g., we see that most of our early improved cultivars are highly susceptible to grain molds. Also, in increasing yield potential, we increase sink size, and this creates a stress which does not occur in lower-yielding local sorghums. The sink-related stresses have a direct effect on susceptibility to such diseases as stalk rots.

Thus I do not agree that for improving sorghum in East Africa we need not worry about breeding disease resistance. As we introduce exotic types, as we change from late flowering to early flowering, as we increase yield potential, etc., we are going to encounter "new" serious disease problems which can only be tackled through incorporating disease resistance.

Doggett: I agree entirely with what Dr. Williams has said. I would not want to be misunderstood on this. I was saying that I cannot see at the present time that it is possible to think in terms of crossing programs for the purpose of introducing specific resistances. It is absolutely essential to screen for susceptibilities to all diseases in all material that is being developed in East Africa for East African conditions. But I do not think that at the present time deliberate crossing for particular resistances is really called for.

Sorghum Diseases in Bangladesh

M. I. H. Mian and A. Ahmed*

Sorghum is a minor crop in Bangladesh and ranks eighth among the cereals (Anon. 1978a). It is grown in scattered locations throughout the country. The important sorghum-growing districts are Noakhali, Comilla, and Rangpur. Sorghum is usually cultivated as a relay, mixed, or border crop in fields with other crops. It is rarely grown as a single crop.

The area of sorghum production is small (about 822 ha). The average yield is 744 kg/ha (Anon. 1978b). Growers of the Comilla and Noakhali districts, using improved methods of cultivation were, however, able to produce up to 2982 kg/ha with an average of 1492 kg/ha (Anon. 1978c). Sorghum can be grown throughout the year in Bangladesh, but mid-November to mid-December is reported to be the best time for planting to obtain high yields (Anon. 1978a).

Problems in Sorghum Cultivation

Limitations to sorghum production in Bangladesh include:

1. Lack of developmental programs and technical personnel.
2. Lack of adaptable, high-yielding, improved varieties with palatable grains.
3. Lack of knowledge by the growers of modern methods of growing and using sorghum.

Scope of Sorghum Cultivation

The Bangladesh Government has established programs to attain self-sufficiency in food production and attempts are being made to grow more crops around the year. It is attempting to include sorghum in the national development programs because agroclimatic conditions are favorable for sorghum cultivation (Salahuddin 1977). Farmers are being encouraged to cultivate sorghum: (a) on saline and light soil with less fertility where no other crop can be grown successfully, (b) in nonirrigated areas with scanty or no rainfall, (c) in flood-affected areas where other cereals cannot be grown, (d) following harvest of transplanted summer paddy (as a third crop), and (e) as a relay crop with winter vegetables (to get an additional crop).

Uses of Sorghum

Since the production of sorghum in Bangladesh is limited, its uses are also limited. Several preparations are made, including:

- Khoi — popped sorghum grains prepared by being placed on hot sand.
- Moa — popped sorghum shaped into small balls with molasses.
- Laru — slightly crushed popped grains shaped into balls with molasses.
- Paesh — porridge prepared from polished sorghum boiled in water and milk with sugar or molasses.
- Flour — prepared from polished grains mixed with wheat.

It is also used for poultry feed.

Present Status of Diseases

Sorghum is subject to many diseases. Symptoms include stunted growth, spotted or rotted parts, malformation of vegetative or reproductive organs, etc. Nine diseases have been recorded and studied to some extent (Table 1); others remain to be identified.

These diseases are also reported from many other countries, but in Bangladesh only grain smut and red leaf spot were included in Talukder's (1974) list of crop diseases. The other

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Table 1. Diseases of sorghum in Bangladesh.

Disease	Pathogens	Importance
Grain molds	*Fusarium* sp, *Curvularia* sp Aspergillus spp, *Helminthosporium* sp.	Major
Grain smut	*Sphacelotheca sorghi*	Minor
Bacterial Leaf streak	*Xanthomonas holcicola*	Major
Red leaf spot	*Colletotrichum graminicola*	Major
Leaf anthracnose	*Colletotrichum graminicola*	Minor
Leaf blight	*Helminthosporium turcicum*	Minor
Rough leaf spot	*Ascochyta sorghi*	Minor
Grey leaf spot	*Cercospora sorghi*	Minor
Curvularia leaf spot	*Curvularia lunata*	Minor

seven are additions to the list of sorghum diseases in the country.

Symptoms of these diseases have been described by many authors. Similarity amongst the early symptoms of sugarcane red stripe as noted by Edgerton (1958) and bacterial leaf streak of sorghum has been observed. Except for bacterial leaf streak and red leaf spot, disease incidence is conspicuous in summer (May to Nov). As in other countries this may be due to high temperature, high relative humidity, and high rainfall. Bacterial leaf streak appears on sorghum from the seedling to the booting stage (from Mar to May), whereas red leaf spot occurs in the period from September to December, on older leaves.

Though it has not yet been assessed experimentally, crop loss due to these diseases may not be considerable, except for the grain mold. Grain mold is noted as a severe constraint to the production of higher yield and good quality grains. However, other diseases may become a menace for sorghum cultivation in Bangladesh, as they are in many other countries.

Research Activities on Sorghum

Three organizations — the Bangladesh Agricultural Research Institute (BARI), Bangladesh Rice Research Institute (BRRI), and Mennonite Central Committee (MCC) — are conducting research on sorghum improvement in Bangladesh. Research programs are based mainly on adaptation trials and agronomic studies such as fertilizer trials, plant-spacing trials, planting-date trials, etc. Emphasis is also being placed on varietal improvement through breeding (Anon. 1978b, 1978c; Salahuddin 1977). The present important selected varieties are Martin Milo, Granador INTA, Early Hegari, and two breeding lines — 498002 and 498003.

Among the three organizations, the Plant Pathology division of BARI is responsible for plant pathological work. The division's research programs are confined to the identification of diseases, studies on their etiology, seasonal and regional variations, and varietal reactions. The test locations are BARI-Central Station at Joydebpur, Dacca, and the Regional Agricultural Research Stations at Jessore, Ishurdi, and Jamalpur.

Achievement, Problems, and Prospects for Sorghum Disease Research

Nine diseases have been identified in the sorghum fields of Bangladesh. Experiments on their control have not yet started. The effects of locations, growing seasons, and cultivated varieties on their incidence are still under study.

Problems facing sorghum research in Bangladesh include:

1. Shortage of specialists in sorghum diseases.
2. No capability of training for the development of skilled manpower.
3. Lack of modern laboratory facilities and literature on sorghum diseases.
4. Absence of proper developmental programs.

Diseases that are considered minor today may in time become major. Moreover, with the extension of sorghum cultivation, new disease problems may also appear. Hence, research on sorghum diseases and development of resistant varieties and other methods for controlling sorghum diseases are essential.

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Sorghum Diseases in India

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Importance of Sorghum

In 1966, sorghum was the fifth most important food crop of the world, preceded by wheat, rice, maize, and barley (Rachie 1970). During the past decade it has made spectacular progress in area planted; production and hectare yield have risen to the third position among the food crops. In India, it is next in importance to rice and wheat; it is planted on nearly 16 million ha and is of special importance to the country as a dual-purpose crop (Rao 1972). At present, its acreage is almost that of wheat.

Sorghum is grown in areas receiving 500 to 1000 mm annual precipitation and under the temperature requirement of 26 to 32°C. Plains and plateau areas up to 1000 m elevation offer excellent scope for successful cultivation. There are two seasons, kharif (June to Oct; rainfed) and rabi (winter, Oct to Feb; essentially rainless except in Tamil Nadu). More than 60% of the total crop is raised during the kharif. The rest is rabi, except for some grown during late kharif (maghi) in some parts of Andhra Pradesh. The principal areas of sorghum cultivation are Maharashtra, Andhra Pradesh, Madhya Pradesh, and Karnataka. Area, production, and hectare yield for 1976 to 1977 are presented in Table 1.

The importance and quality of sorghum as a dual-purpose crop is well recognized in India. Sorghum grain is a staple food for human beings in many regions, especially in Maharashtra, northern Karnataka, and parts of Madhya Pradesh. It is prepared in many forms by baking, cooking, or frying. Even though the majority of sorghum cultivated in India is for food grain, its stalks and fodder are utilized as cattle feed.

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Disease Situation and Priority of Disease Importance

As with any cultivated plant, a disease found

Table 1. Area, production, and yield in major sorghum-growing states in India, 1976–1977.

	Area (1000 ha)			Production (1000 MT)[a]			Yield (kg/ha)	
	Kharif	Rabi	Total	Kharif	Rabi	Total	Kharif	Rabi
Maharashtra	3 093.5	3345.8	6 439.3	2958.1	1762.1	4 710.2	956	527
Karnataka	689.5	1039.4	1 728.9	673.9	506.4	1 118.3	977	487
Andhra Pradesh	1 098.2	950.3	2 048.5	470.6	539.8	1 010.4	429	568
Madhya Pradesh	1 937.1	17.7	1 954.8	1292.5	11.7	1 304.2	667	661
Gujarat	869.9	173.8	1 043.7	436.2	144.5	580.7	501	831
Rajasthan	836.8	—	836.8	358.2	—	358.2	428	–
Uttar Pradesh	720.7	—	720.7	491.5	—	491.5	682	–
Tamil Nadu	627.6	143.1	770.7	557.1	143.1	700.2	888	1000
Others	234.9	—	234.9	—	—	—	255	60
All India	10 108.2	5670 3	15 778.5	7298.3	3097.7	10 396.0	722	546

Source: Agriculture Situation of India, Ministry of Agriculture and Irrigation, New Delhi, India, 1977.
a. MT = metric ton

associated with the crop can be classified in terms of importance according to its spread, nature of damage, and scope of control (Tables 2, 3).

Location and Nature of Research on Sorghum Diseases in India

The All-India Coordinated Sorghum Improvement Project (AICSIP) started functioning in 1970 as a part of the program initiated by the Indian Council of Agricultural Research (ICAR), in which all sorghum-growing states participate through their agricultural universities. It is a multidisciplinary, multilocational program with nine main centers in which pathologists are active workers. The entire project is guided by the Project Coordinator, AICSIP, Hyderabad.

The main emphasis of the program in pathology differs among centers, depending upon the

Table 2. Sorghum diseases of major importance in India.

Disease	Regions of spread	Remarks
Head mold	Widespread in Vidarbha, Marathwada of Maharashtra; parts of Andhra Pradesh; Coimbatore, North Arcot, and Salem districts of Tamil Nadu. Sporadic in Rajasthan.	A high potential for damage, especially of short-duration, of compact-headed strains whose grain-formation stage coincides with heavy rains.
Charcoal rot *Macrophomina phaseolina*	Widespread in Karnataka, Marathwada of Maharashtra, and Coimbatore region of Tamil Nadu.	High potential of loss, disease control difficult
Downy mildew *Peronosclerospora sorghi*	Endemic regions: northern Karnataka, southern Maharashtra and rainfed areas of Tamil Nadu. Sporadic regions: Hyderabad district, Delta regions of Andhra Pradesh; eastern Maharashtra (Vidarbha); Tirunelveli and Tiruchi districts of Tamil Nadu.	High potential of loss, disease control difficult.
Ergot *Sphacelia sorghi*	Severe in Vidarbha (eastern Maharashtra), southern and western Maharashtra; occasionally in Madhya Pradesh, Gujarat, and is on the increase in Telengana and Delta regions of Andhra Pradesh.	Some local and male steriles in seed-production plots are susceptible.
Rust *Puccinia purpurea*	Widespread in southern India and common throughout the country.	
Leaf blight *Trichometasphaeria turcica (Exserohilum turcicum)*	Widespread	High potential in rainfed and humid tracts.
Anthracnose *Colletotrichum graminicola*	Widespread. Severe in Madhya Pradesh, Andhra Pradesh, Rajasthan, Delhi, and Tamil Nadu.	High potential in rainfed and humid tracts.

Table 3. Sorghum diseases of minor importance in India.

Disease	Regions of spread	Remarks
Rough leaf spot *Ascochyta sorghi*	Widespread in Madhya Pradesh; sporadic in Uttar Pradesh, Andhra Pradesh, and Karnataka.	Serious in young plants, damage negligible.
Sooty stripe *Ramulispora sorghi* *Ramulispora sorghina*	Widespread in Marathwada of Maharashtra. Common in Andhra Pradesh and Madhya Pradesh.	Losses negligible. All strains, varieties/ hybrids are susceptible.
Zonate leaf spot *Gloeocercospora sorghi*	Widespread in Tamil Nadu, Uttar Pradesh, Andhra Pradesh; commonly found in other regions.	High-yielding strains are more susceptible than locals.
Grey leaf spot *Cercospora sorghi*	Widespread in Madhya Pradesh, Rajasthan, Delhi, Tamil Nadu; sporadic in the rest of the country.	Common on improved varieties and hybrids.
Grain smut *Sphacelotheca sorghi*	Very common all over	Though widespread and showing high potential of losses, it is easily controlled by seed dressings.
Loose smut *Sphacelotheca cruenta*	Very common all over	" " "
Head smut *Sphacelotheca reiliana*	Sporadic all over.	Low incidence, and as such disease control is not warranted.
Long smut *Tolyposporium ehrenbergii*	Rare occurrence in Tamil Nadu, Andhra Pradesh, Madhya Pradesh, and Karnataka.	No control is warranted.
Sorghum red stripe (Sugarcane mosaic virus)	Sporadic in Maharashtra.	
Crazy top *Sclerophthora macrospora*	Sporadic in Maharashtra	No control is Warranted.

regional importance of problems. All materials in yield trials and breeders materials are tested against all important diseases (Table 4).

Achievements of AICSIP

Emphasis has been placed on search for sources of resistance, breeding for resistance, fungicidal, and agronomical control of diseases. In some cases, fundamental studies have been carried out on pathogens and diseases.

Sources of Disease Resistance and Breeding for Resistance

The entire collection of breeding material, pre-released varieties, and hybrids is rigorously tested at all centers. Testing against sorghum downy mildew (SDM) in artificial as well as

Table 4. Important pathology problems identified with various centers of AICSIP.

Center	Average rainfall (mm)	Problems of major emphasis
Dharwar (Karnataka)	550–600	Sorghum downy mildew (SDM) testing and breeding in natural as well as artificial conditions; control measures. Testing against rust, ergot; breeding for resistance.
Parbhani (Maharashtra)	700–800	Charcoal rot. Head mold. Screening and breeding for resistance to charcoal rot; charcoal rot control (chemical as well as agronomical).
Indore (Madhya Pradesh)	1150–1200	Leaf spots, screening under artificial and natural conditions against individual as well as multiple-leaf spot diseases. Breeding for resistance against leaf spots and head molds.
Udaipur (Rajasthan)	550–600	Screening for leaf spots and breeding for resistance. Basic studies on head molds.
Coimbatore (Tamil Nadu)	650–700	SDM, screening of material, control of SDM and head molds.
Hyderabad (Andhra Pradesh)	900–1000	Head molds with special reference to yellow and white grain varieties. Testing for rust.
Akola (Maharashtra)	900–1000	Ergot, artificial as well as natural screening; breeding for resistance and control. Control of head molds.
Delhi	600–700	Screening for leaf spots
Navsari (Gujarat)	1300–1500	Leaf spots and head molds.

Note: In addition to these centers, Digraj (Maharashtra) is a testing location against SDM. It is an endemic area. Similarly, University of Mysore collaborates with AICSIP in detailed testing against SDM.

natural conditions is carried out at Dharwar, Digraj, and Coimbatore; for charcoal rot at Dharwar and Parbhani; for ergot at Dharwar and Akola; and for leaf spots at Indore. Testing against leaf spots and head molds at Delhi, Hyderabad, Udaipur, Parbhani, Coimbatore, and Navsari is done only under natural conditions.

Sorghum Downy Mildew

As a result of extensive testing for systemic as well as local infection, most of the hybrids and cultivars CSV-4 and CSV-5 are resistant. In addition the following 20 entries possess resistance:

CK60-B, 3660B, 1258-B, 2077-B, 2077-BE, 2219-B, 285, SPV-35, SPV-70, SPV-101, SPV-101A, SPV-102, SPV-102A, SPV-104, SPV-105, SPV-126, SPV-129, SPV-166, SPV-190, and SPV-191.

Inheritance studies of SDM resistance with cultivars CSV-4 and CSV-5 as resistant parents and cultivar SPV-3 as a susceptible parent are being conducted under artificial conditions at Dharwar.

Charcoal Rot and Head Mold

Testing of material in different seasons has indicated that cultivars 285, SPV-126, and SPV-193, along with released variety CSV-5 and CSH-7R (in which CSV-5 is a parent), are resistant. In breeding for resistance, a number of crosses have been found to be free from charcoal rot in which the parents involved are CS-3541 × 148, CS-3541 × 3942, CS-3541 × 3924, 36A × 148/168, CS-3541 × 92793, CS-3541 × 302, and PD 2-5 × 302. Similarly, CS-3541 × 148 and CS-3541 × 92793 have been

found to be resistant to both charcoal rot and head mold.

Ergot

Even though cultivar CSH-6 has field resistance to this disease, the problem of nonsynchronization and inactivity of the pollen during seed production must be dealt with. Apart from this, there are eight lines tolerant to this disease: CSH-5, SPH-1, SPV-34, SPV-61, SPV-126, SPV-129, SPV-162, and SPV-191.

Rust

Many tan plant types, including CSH-5 and CSH-6, have tolerance as well as a hypersensitive reaction to leaf rust. Eight additional lines are rust-resistant sources: 2077-B, 2077-BE, SPV-13, SPV-81, SPV-126, SPV-193, SPV-198, and SPV-199.

At Dharwar, for inheritance studies against rust, some crosses have been made between SPV-191 and SPV-34 (both resistant), and 36B and CSV-5, which are susceptible.

Rough Leaf Spot

Six lines show resistance to this disease: CK60-B, 2077-B, SPV-23, SPV-101, SPH-68, and MSH-33.

Anthracnose

SPV-99, SPV-101A, SPV-110, SPV-122, SPV-156, and SPV-191 have been found to possess some resistance.

For the other leaf spot diseases — such as *Helminthosporium* leaf blight, grey leaf spot, zonate leaf spot, and sooty stripe — there are a number of tolerant lines, but they require further testing since the number is large. Only progenies of CS-3541 × 148 have been found to be free from sooty stripe at Parbhani.

A glance at the above tolerant material indicates that varieties or hybrids that have multiple resistance to more than one disease are available. SPV-104, SPV-126, and SPV-178 have sources of resistance to six diseases, and SPV-193, CSH-6, CSV-4, and SPH-61 show resistance to five diseases (Table 5).

Disease Control

Downy Mildew

Satisfactory control has been achieved with four sprays of Dithane M 45 (0.3%) at 7-day intervals, beginning at germination. This has proved effective in many parts of Karnataka and is now recommended in the package of practices for that state. Irrigation of the crop to keep available moisture at 80% during the fortnight following sowing has been found useful in checking the disease.

Head Mold

Control of head mold has been attempted for many years, but so far we have had no success in spotting sources of resistance. Fungicidal application has been found to be quite effective in reduction of head mold and improvement of yields. A combination of Aureofungin (200 ppm) and Captan 0.2% is found to be effective and economical (Table 6). This control measure has been found to be essential in seed-production blocks. Control data has been collected at Coimbatore, Hyderabad, Navsari, and Udaipur.

Ergot

Two to three sprays of Benlate 0.1% are effective in controlling the disease if sprayed at 50% flowering. It is a good preventive spray in seed-production areas. Mechanical removal of sclerotia from seeds, by washing in 30% salt water followed by three rinsings in plain water before sowing, has been recommended by the Maharashtra Government.

Basic Work

Notable contributions in this aspect are as follows:

Downy Mildew

Oospores of *Peronosclerospora sorghi* germinate by producing 8 to 12 conidia which are round, hyaline, and thin-walled (Sundaram 1977).

Table 5. Sources of apparent multiple resistance[a,b] from noninoculated yield trials at AICSIP centers.

Entry	Downy mildew (%)	Zonate leaf spot	Rust	*Cercospora* leaf spot	Charcoal rot	Ergot	Anthracnose	Sooty stripe	Helminth blight	Rough leaf spot
SPV-104	Mys 0.0 Dha 0.0 Coi 0.7	Del 0 Dha 0 Uda 0 Ind 1	Del 0 Uda 1 Ind 1 Coi 1.7 Dha 3	Dha 0 Nav 0 Ind 0 Coi 1.7	Par[c] 0 Nav[d] 0 Dha 8.8	Dha 2 Ako B	– – – –	– – – –	– – – –	– – – –
SPV-126	Dha 0.0 Coi 2.3	Ind 0 Uda 0 Del 0	Ind 0 Uda 0 Coi 2 Dha 1	Ind 0 Nav 0 Dha 1	Nav 1.0% Par 4 Dha 7.2%	– – – –	– – – –	Ind 0 Del 0	– – – –	– – – –
SPV-178	Coi 0		Nav 0	Nav 0	Nav 0	Nav 0	Nav 0	– – – –	– – – –	– – – –
SPV-193	Coi 0 Dha 9.3	Dha 0 Del 0 Uda 0 Ind 1	Dha 0 Uda 0 Ind 1	Del 0 Dha 0 Nav 0 Uda 0 Ind 1	Par 0 Dha 4.3%	– – – –	– – – –	– – – –	– – – –	– – – –
SPH-61	– –	Dha 0 Ind 0 Ako 0 Uda 0	Ind 0 Uda 0 Coi 1.7 Dha 2	Dha 0 Ako 0 Ind 0 Uda 0 Del 1	Ako 0 Par 6.2 Dha 14.0%	Ako A Dha 2	– – – –	– – – –	– – – –	– – – –
SPH-80	Dha 0.7	Del 0 Dha 0 Ind 0 Ako 0 Uda 0	– – – –	Nav 0 Ind 0 Uda 0 Del 1 Dha 1	Par 0 Dha 14.9%	– – – –	– – – –	– – – –	Nav 0 Del 1	– – – –
CSV-4	Dha 1	Del 0 Dha 0 Uda 0 Ind 1	Ind 0 Uda 0 Del 2	Ind 0 Dha 0 Del 1	Par 0 Dha 6.0%	– – – –	– – – –	– – – –	– – – –	– – – –
CSV-5	Dha 5	Del 0 Dha 1 Uda 1 Ind 1	– –	Ako A Ind 0 Nav 2.0% Del 1 Dha 1 Coi 1.3	Par 0 Ako 0	– – – –	– – – –	Del 0 Ind 1	– – – –	– – – –

Table 5 *contd*

Entry	Downy mildew (%)	Zonate leaf spot	Rust	*Cercospora* leaf spot	Charcoal Rot	Ergot	Anthracnose	Sooty stripe	Helminth blight	Rough leaf spot
CSH-6	Dha 0 Mys 0 Coi 1.5	Dha 0 Ind 0 Uda 0 Del 1	Ind 0 Nav 0 Uda 0 Coi 1 Dha 2	Dha 0 Nav 0 Ind 0 Coi 0 Uda 0 Del 1	– – – –	Ako 0 Dha 1 Nav 3.7	– –	– –	– –	– –
SPV-70	Coi 0 Dha 14.9	Del 0 Uda 0 Ind 0 Coi 1	Ind 0 Uda 0 Dha 3	– –	– –	– –	– –	– –	– –	Del 0
SPV-23	– –	Ind 0 Del 0	Uda 0 Coi 1	– –	– –	– –	– –	– –	– –	Del 0 Ind 1
SPV-34	– –	Del 0 Dha 0 Ind 1 Uda 1	Dha 0 Uda 0 Nav 1.0% Ind 2	Del 0 Dha 0 Ind 0 Uda 1 Nav 2.0%	– –	– –	– –	– –	– –	– –
SPV-166	Dha 0 Coi 0	Del 0 Uda 0 Ind 1 Dha 1	– –	Dha 0 Ind 0 Uda 0 Del 1 Nav 2	– –	– –	– –	– –	– –	– –

a. Head mold – no source of resistance
b. Grades: 0 = disease free, 1 = resistant, 2 = moderately resistant, A = resistant (data from Akola)
c. Data on charcoal rot from Parbhani is in Arcsin values.
d. All data from Navsari is expressed in %

Mys = Mysore, Dha = Dharwar, Coi = Coimbatore, Del = Delhi, Uda = Udaipur, Ind = Indore, Nav = Navsari, Par = Parbhani, Ako = Akola

Continued.

Table 5 *contd*

Entry	Downy mildew (%)	Zonate leaf spot	Rust	*Cercospora* leaf spot	Charcoal rot	Ergot	Anthracnose	Sooty stripe	Helminth blight	Rough leaf spot
CSV-2	– –	– –	– –	Ind 0 Del 1 Dha 1	Par 0	– –	– –	– –	Uda 0 Dha 0 Del 2	
2077B	Coi 0 Dha 0	– –	– –	Nav 0 Ind 0 Ude 0 Del 1 Dha 1	– –	– –	– –	– –	– –	
SPV-102A	Dha 0 Coi 2.2	Del 0 Dha 0 Ind 0 Uda 0	– –	– –	– –	– –	– –	– –	– –	
MSH-33	Dha 0	Dha 0 Ako 0 Uda 0 Ind 1 Del 1	Ind 0 Uda 0 Dha 2	– –	– –	– –	– –	– –	– –	– –
SPH-1	– –	– –	– –	– –	Nav 0 Par 0 Dha 19.0%	Ako 0	– –	– –	Uda 0 Del 1 Coi 1	– –

Table 6. Chemical control of head mold, Coimbatore centre variety CO-18.

Treatment	Head mold[a] (mean)	Yield (kg/ha)	Net profit or loss (Rs)
Aureofungin 200 ppm	35.54	2407	+429.12
Aureofungin 200 ppm + Captan 0.2%	29.79	2556	+468.48
Captan 0.2%	36.78	2370	+512.24
Thiram 0.2%	43.45	2074	+282.48
Thiram 0.2% + Ziram 0.2%	40.59	2259	+352.80
Ziram 0.2%	57.23	1593	−108.48
Dithan M.45, 0.2%	54.30	1741	+ 8.80
Dithane Z.78, 0.2%	50.39	1852	+107.04
Difolatan 0.2%	63.03	1370	−295.20
Control	62.75	1518	

	AVT Head mold			AVT yield	
Source	df	SS	Mean	SS	Mean
df	2	58.89	29.45	7.50	375
Treatment	9	3776.67	419.63**	468.08	5201
Error	18	125.01	6.95	34.5	192
SE of mean				1.522	80 (kg/ha)
LSD (0.05)				2.152	2375
CV (%)				6	7

a. Angular transformed value

Ergot

Pennisetum typhoides, Zea mays, Panicum maximum, and *Ischaemum pilosum* are collateral hosts of this disease in India. Of these, *Ischaemum pilosum* is very common in the Vidarbha region of Maharashtra and seems to be the chief source of the pathogen in that region. Spraying sorghum with Beniate 0.1% or 0.2% Captan two or three times at 50% grain-formation stage is effective, but collateral host plants on the field bunds must be sprayed also.

Germination of sclerotia, production of perithecia, asci and ascospores of this fungus have been observed at Akola.

Head Mold

Qualitative and quantitative studies of the fungi causing head mold have revealed 27 species, belonging to 13 genera, involved in the head mold complex. Quantitative and qualitative differences have been spotted in different grades of head molds (Ravindranath 1976).

Future Problems

In spite of multilocational testing under artificial and natural conditions in the kharif and rabi seasons, real immunity to charcoal rot, ergot, or (to a lesser extent) SDM is not yet available. Most of the hybrids and many of the varieties released are partially resistant. It appears that intensive regional testing and searching must be pursued to identify high resistance in future varieties and hybrids. Agronomical measures and nursery spraying seem to be working well against SDM in Karnataka. There is expectation of better control of SDM by new systemic fungicides, such as Ridomil.

The situation in terms of resistance to charcoal rot is not as encouraging as for SDM. Satisfactory resistance has not been found among existing materials; disease control, especially in seed-producing areas, must currently be by agronomic practices.

The situation with ergot is similar to that existing for charcoal rot: immunity is lacking. The role of collateral hosts makes the problem

more complex. Perhaps fungicidal control, coupled with seed certification and a drive for eradication of grass hosts, will provide control while the search for resistant material continues.

Fungicidal measures for several leaf spots may not be economically feasible; resistance breeding may be the only approach to further improvement. Fortunately, there are many sources of resistance.

The problem of head mold is restricted to the kharif season. With the fluctuating rainfall of September and October, when the majority of the materials have heads in the grain-formation stage, field testing is not dependable. Basic work on this aspect is needed. Even though other control measures for this disease are available, development of resistant cultivars will prove to be superior.

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Sorghum Diseases in Pakistan

S. J. Hamid*

Sorghum is the fourth most important cereal crop in Pakistan; in 1977, it occupied an area of about 450 000 ha with a production of 257 200 tonnes (Table 1). Due to its hardy nature, sorghum in Pakistan is grown mostly in the semi-arid rainfed areas. This crop is used mostly for fodder purposes, but its grain is utilized for human consumption in rural rainfed areas.

Little emphasis has been laid on the pathology of the sorghum crop, as attention was mainly focused on diseases of the more important staple food crops like wheat and rice. On the basis of the available information, the following diseases of sorghum in Pakistan are listed in order of their importance.

Table 1. Area and production of sorghum in Pakistan in 1977.

Province	Area (1000 ha)	Production (1000 tonnes)
Punjab	218.5	115.9
North West Frontier Province	22.5	11.1
Sind	153.3	102.0
Baluchistan	53.2	28.2
Total	447.5	257.2

Major Diseases

Grain, or Covered Kernel Smut

This disease, caused by *Sphacelotheca sorghi*, occurs in almost all major sorghum-growing areas of Pakistan. In favorable years, incidence as high as 30 to 50% has been recorded in individual fields. On an average its incidence is around 10%, with a range from trace to 50% from field to field.

Varieties Lyallpur Hybrid, 1616 X RRS-17, S.S.I, V-3, and Sorokartoho were resistant (with up to 10% head infection) in a trial comprising 25 varieties conducted in 1973. No studies on physiological specialization have been carried out so far.

Red Leaf Spot, or Anthracnose

Anthracnose (*Colletotrichum graminicola*) is widespread in Pakistan. In the high summer rainfall year of 1977, an average intensity of up to 30% was recorded in most areas. Almost all locally cultivated commercial varieties are susceptible to this disease.

Grain Mold

Fungal species of *Fusarium, Curvularia, Alternaria*, and *Ascochyta* have been isolated as primary grain molds from infected sorghum heads. These are widespread in occurrence, under wet summer weather. During an extensive survey carried out in 1977, up to 70% mold intensities were recorded in the farmers' fields at Sahiwal and D. I. Khan and up to 20% at Dadu, Sukkur, Mirpur khas, Islamabad, and Peshawar.

In an artificially inoculated National Cooperative Sorghum Field Trial in 1977, comprising commercially cultivated varieties and advanced lines, ten entries were found moderately resistant to head molds (with a score of 2 on a 0 to 5 scale, as proposed by ICRISAT pathologists) while two moderately susceptible lines (with a score of 3) were identified.

Long Smut

Long smut (*Tolyposporium ehrenbergii*) is encountered in all the major sorghum-growing areas of Pakistan, but its incidence is generally

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high in low-rainfall areas. Analysis of samples collected from 1967 to 1971 indicated an average incidence of 18, 14, 11, 9, 5, 5, 4, and 2% grain infection at Sibbi, D. I. Khan, Nawab Shah, Dadu, D. G. Khan, Multan, Larkhana, and Jacobabad, respectively.

In artificially inoculated varietal trials during the above period, varieties NK-125, NK-267, AUS-6, C-45, NK-404, Martin, and Caprock were resistant (0 to 0.2% grain infection) for 2 years while the first four varieties listed were resistant for 3 consecutive years.

Minor Diseases

Downy Mildew

Sorghum downy mildew (*Peronosclerospora sorghi*), not a major problem at present, is generally encountered in wet summer weather. During 1977, a wet year, its incidence ranged from trace to 40% at Yousafwala, Sahiwal Dadu, Sukkur, and D. I. Khan. The disease was not observed in 1978.

Other minor diseases recorded are rough leaf spot (*Ascochyta sorghina*), grey leaf spot (*Cercospora sorghi*), zonate leaf spot (*Gloeocercospora sorghi*), sooty stripe (*Ramulispora sorghi*), head smut (*Sphacelotheca reiliana*), bacterial stripe and streak (*Pseudomonas* and *Xanthomonas* spp). The relative prevalence, distribution, and intensity of these diseases has not been estimated, nor have sources of resistance been identified.

Location and Nature of Research on Sorghum Diseases in Pakistan

Pakistan's Cereal Diseases Research Institute has recently taken up research on sorghum diseases at the federal level, with special emphasis on locating sources of resistance, identification of physiologic races, and screening of national and international nurseries. Headquarters of this institute is located at Islamabad and substations with greenhouses are located at Muree and Karachi. Field trials are carried out at outstations in the "hot-spot" areas like Pirsabak (Nowshera) and D. I. Khan in the North West Frontier Province; MMRI, Yousafwala, and ARS, Bahawalpur, in the Punjab; Dokri, Dadu, and Tandojam in Sind; and Temple Dera and Naseerabad in Baluchistan. At the provincial level, some work on agronomy and breeding is being carried out at ARS, D. I. Khan in the North West Frontier Province; MMRI, Yousafwala, and ARS, Bahawalpur, in the Punjab; ARS, Dadu, and ARI, Tandojam, in Sind; and at Temple Dera in Baluchistan.

At the international level, the Cereal Disease Research Institute is collaborating with ICRISAT and with Texas A & M University, USA, in varietal screening against leaf diseases and grain molds. It supplies regular information on the reactions of their material in different ecological zones of Pakistan.

Achievements

1. The Cereal Diseases Research Institute, (CDRI) in an attempt to provide assistance to the National Sorghum Improvement Program, has initiated regular varietal evaluation of breeding material against major diseases. In 1978, 48 entries (present commercial varieties, parent lines, and advanced selections) were screened in a disease-screening nursery planted at various "hot-spot" locations. Results are being furnished to the cooperating breeding centers.
2. As a regular feature, the CDRI cooperates in planting the International Sorghum Grain Mold and Leaf Disease Nurseries coordinated by ICRISAT.
3. Preliminary work has been started on the race analysis of sorghum grain smut. Differential varieties have been multiplied this year and smut samples have been collected.
4. A National Sorghum Improvement Program has been developed, with the aim of evolving white, bold-grain types and sweet juicy, fast-growing varieties for taking two cuttings for fodder.
5. Varieties Pak-SS-II and J.S. 263 for the Punjab; Sorokartoho, Red Jampur, H-4-2, Depar for Sind; D.S. 75 (equal to Purdue-954125) for the North West Frontier Province; and Bagdar for Baluchistan have been recommended for commercial cultivation. Among promising material, Giza-3 proved good at D. I. Khan, but it is late-

maturing and attempts are under way to select early strains for that area. Likewise, variety Atlas is a sweet sorghum, and holds promise for good green fodder.

Problems

1. The breeding program for sorghum is not progressing satisfactorily for want of male-sterile lines. Crossing is primarily aimed at earliness and green stalks.
2. Breeding for disease resistance is yet to be established in the sorghum improvement program.
3. The importance of sorghum as a food crop deserves more serious consideration.

Sorghum Diseases in the Philippines

Samuel C. Dalmacio*

Sorghum is a relatively new crop in the Philippines, but is rapidly gaining popularity as an economical and efficient supplementary feed for poultry, swine, and cattle. As human food, however, Filipinos have yet to develop a taste for it.

Sorghum production in the Philippines is quite small. In 1977 the area planted to sorghum was 6100 ha with a total production of 10.2 million kg valued at 9.03 million pesos. Extensive cultivation is, however, anticipated in the next few years because of increasing support from the government in its attempts to reduce importation of feed grains. In early 1978 the area planted to sorghum was 8337 ha, an increase of 2237 ha (36.6%) over that of 1977. With a projected feed grain requirement of about 3.5 million tonnes by 1980, compared with the 805 307 tonnes of maize and sorghum utilization in 1977, maize and sorghum production must increase tremendously.

Diseases may be considered as one of the limiting factors in sorghum production in the Philippines. To date, 13 diseases are reported to occur on sorghum in the Philippines (Karganilla and Elazegui 1971–1973; Quebral and Gibe 1958). The most common and perhaps the most important are *Phyllachora* tar spot, *Exserohilum* leaf blight, grey leaf spot, head molds, rust, and *Rhizoctonia* banded leaf and sheath blight. The first four diseases are more destructive during the wet season planting; rust is more destructive during the dry season; and *Rhizoctonia* banded leaf and sheath blight is destructive in both planting seasons. Other diseases observed in the Philippines and ranked in descending order of importance are mosaic or red-leaf diseases (Benigno and Vergara 1977), charcoal stalk rot, bacterial stalk rot, zonate leaf spot, bacterial stripe, anthracnose, and an unknown virus disease.

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Research on sorghum diseases in the Philippines was initiated in late 1969 at the University of the Philippines at Los Baños (UPLB), a few years after the sorghum improvement program was started. It ceased to function temporarily in 1973 when the principal investigator left for further studies in the United States. It resumed in late 1977 with a research grant from the National Science Development Board of the Philippines. Presently only three institutions in the Philippines are doing research on sorghum diseases, although most of the work is done at the UPLB. The present staff consists of an assistant professor and two research assistants.

Early studies dealt mainly with the identification and survey of sorghum diseases in different sorghum-growing areas, assessment of yield losses and screening of fungicides for control of the major sorghum diseases. Some of the notable achievements on sorghum disease research include the identification of *Erwinia carotovora* var. *chrysanthemi* Dye as causing stalk rot, and sugarcane mosaic virus as causing the red leaf disease and mosaic on sorghum. Potential yield reductions of 22, 30, and 75% have been established for *Exserohilum* leaf blight, rust, and *Rhizoctonia* leaf and sheath blight (Elazegui 1971), respectively. Among several chemicals tested, Dithane M-45 and Benlate appeared effective against rust and leaf blight. More than 15 fungal genera have been observed associated with head molding, among which *Fusarium* sp, *Curvularia* sp, and *Phoma* sp are the most common, constituting about 72%.

Current emphasis is on the screening of sorghum germplasm for resistance to the major sorghum diseases in the country. Disease nurseries are now being established at UPLB where all breeding materials (local and introduced) can be subjected to high inoculum levels. Early this year, of the 355 lines and varieties evaluated against rust, 149 were resistant to rust, 13 of 236 entries were resistant to leaf blight, while none of 213 entries had adequate

levels of resistance to tar spot (Dalmacio 1978). There are some indications, however, that resistance to tar spot disease is expressed quantitatively, in that some lines showed fewer lesions, although resistance may be influenced by crop maturity.

As sorghum cultivation is intensified, vulnerability to disease is also increased. In anticipation of this increased disease problem, more basic and adaptive research is needed. To establish research priorities, the distribution and prevalence of sorghum diseases in different areas and their consequent effect on yield should be known. There is a pressing need to understand the epidemiology of certain diseases like tar spot to facilitate formulation of effective control measures and screening for resistance. Tar spot disease is a major concern in the Philippines that has received little attention in most sorghum-growing countries. In the meantime, efforts to test the efficacy of fungicides will be continued, as varieties resistant to most of these major diseases are not available. Lastly, the development of varieties and hybrids resistant to the major sorghum diseases will receive high priority.

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Sorghum Diseases in Thailand

Udom Pupipat*

Sorghum cultivation on a commercial scale is a rather recent development in Thai agriculture. Cultivars were first introduced from the USA in the early 1950s by the USAID program, and sorghum is currently grown as a cash crop destined for export and for local utilization as animal feed.

In 1965, the Thailand National Corn and Sorghum Improvement Program was initiated through the cooperation of the Thailand Department of Agriculture, Kasetsart University, and the Rockefeller Foundation. Emphasis was to be placed on sorghum breeding, agronomy, entomology, pathology, economics, and utilization. With the introduction of several hundred lines from the world sorghum collection, as well as from other sources, the breeding program started with the aim of combining disease, insect, and bird resistance and of improving agronomic characteristics and yield.

In most of the sorghum-growing area, the annual rainfall ranges from 900–1600 mm, with good distribution from May through October. Mean, maximum and minimum temperatures are 28 to 29, 40, and 10°C, respectively. Elevation of the area ranges between 100 and 150 m.

Data on the area, production, and export value for selected years are presented in Table 1.

Farmers are using the cultivars Early Hegari and Late Hegari. However, some new cultivars, e.g., IS-8719 and KU-357, were recently released.

Three institutions, Khon Kaen University in the Northeast, Thai Department of Agriculture, and Kasetsart University, are now conducting research in sorghum improvement for Thailand.

The major pests or sorghum in Thailand are birds and sorghum shoot fly (*Atherigona soccata*). Work on various aspects of controlling birds and shoot fly are in progress.

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Table 1. Sorghum production and export in Thailand.

Year	Area harvested (1000 ha)	Production (1000 tonnes)	Yield (kg/ha)	Export: Volume (1000 tonnes)	Export: Value (US $1000)
1964	20.0	30	350	13	650
1965	29.0	87	2506	54	3 050
1970	42.3	130	1926	80	5 150
1975	21.3	250	1378	200	24 084
1976	20.4	230	1308	181	18 697
1977	15.3	148	1156	135	14 936

Source: Ministry of Agriculture and Cooperatives

Sorghum Diseases

Up to the present, 15 diseases have been observed in Thailand. They are listed below, in order of importance.

Grain Molds

This complex disease is considered most damaging to sorghum grain in Thailand. It is prevalent on sorghum grown in the early season (Apr to July), and it is for this reason that most Thai farmers grow sorghum during the late rainy season (Aug to Nov). The fungi isolated from diseased grains are *Curvularia lunata, Fusarium* sp, and *Aspergillus* sp. All fungi isolated successfully produced head mold symptoms on sorghum heads within 7 to 8 days following artificial inoculation. The International Sorghum Grain Mold Nursery entries IS-2327, IS-2328, and IS-9225 have shown good promise in field and laboratory evaluations at Khon Kaen University.

Cercospora Leaf Spot

Cercospora leaf spot (*Cercospora sorghi*) is very severe in a number of fields in western Thai-

land, but is less prevalent in the other parts of the country.

Rust

Rust (*Puccinia purpurea*) is severe in some locations (especially in western Thailand), but is less prevalent in fields of central and northeastern Thailand.

A hyperparasite, *Darluca filum*, parasitizes this pathogen. Unfortunately, it comes in late in the season — after the rust fungus is established — and thus provides no significant control of the rust fungus.

Anthracnose and Red Rot

These diseases, caused by *Colletotrichum graminicola*, are quite widespread in the sorghum-growing areas. Anthracnose became severe on Thai Hegari in the rainy season. Cross-inoculation experiments indicated that the sorghum isolates in this country do not infect maize, nor are the maize isolates pathogenic to sorghum.

Zonate Leaf Spot

Zonate leaf spot (*Gloeocercospora sorghi*) is found occasionally associated with other leaf diseases in all regions. Damage is of no significance.

Helminthosporium Leaf Blight

Leaf blight (*Exserohilum turcicum*) is prevalent in the western provinces of the country, but is of no economic importance in other parts of Thailand.

Charcoal Rot

Charcoal rot (*Macrophomina phaseolina*) severe in the early years of the sorghum-improvement program, is gradually becoming less severe. This is mainly due to resistant selection. The pathogen can infect corn, sorghum, and other economic crops. Pycnidial and sclerotial stages occurred on both corn and sorghum.

Tar Spot

Tar spot (*Phyllachora sorghi*), scattered all over the country on *Sorghum bicolor* and other *Sorghum* spp, is of no economic importance.

Sorghum Downy Mildew

Sorghum downy mildew (*Peronosclerospora sorghi*) is found occasionally on experimental farms, but not to date in farmers' fields. The sorghum isolates can infect corn easily, but the corn isolate does not readily infect sorghum. So far, this disease is of no economic importance to sorghum in Thailand. On corn, the disease can be completely controlled by using a new systemic fungicide, Ridomil. Ridomil in different formulations can be effectively used as a foliar spray, seed dressing, soil banding, or broadcasting.

Pestalolia Leaf Spot

This disease, caused by *Pestalolia* sp, was found in 1967 on a plant in the Western Province, but we could not prove its pathogenicity on Hegari plants.

Root Knot

Root knot (*Meloidogyne naasi*) was first found in 1967 at the Thai National Corn and Sorghum Research Centre (Farm Suwan). The disease was confined to a single location, and was completely eliminated. Inoculation experiments indicated that this pathogen can attack 32 of 60 plant species tested. Besides sorghum, the most susceptible plants are members of the Cruciferae.

Mosaic (Maize Dwarf Mosaic Virus)

MDMV is found scattered on corn, sorghum, johnsongrass, and some other grasses. It is, however, of no economic importance.

Other Diseases

Other diseases of minor economic importance include brown spot (*Physoderma maydis*), *Phyllosticta* leaf spot (*Phyllosticta sorghina*), and *Cephalosporium* stalk rot (*Cephalosporium acremonium*).

It is interesting to note that none of the smuts or sugary disease (ergot) have been observed on sorghum in Thailand.

Country Reports Discussion Session — Asia

India

Frederiksen:
What is the percentage of hybrids currently planted in India?

N. G. P. Rao:
The distribution of hybrids in India is highly skewed. In Maharashtra, which is the number one sorghum-growing state, about 55% of the total area was grown to hybrids during the rainy season last year. In Karnataka, about 30 to 40% was under hybrids. In Madhya Pradesh, the hybrid area is negligible. So in some states hybrids account for 40 to 60% of the area, and in some areas within these states hybrid coverage is almost 100%. I would guess that on an all-India basis about 20% of the sorghum area is sown to hybrids in the rainy season. There have been no hybrids available for the postrainy (rabi) season until recently, and now seed production of two rabi hybrids is under way. In Andhra Pradesh, there is a rabi area where hybrid coverage is fairly high.

Tyagi:
Dr. Ravindranath said that two to three sprays of Benlate were effective in controlling ergot.
What was the effect of Benlate on head molds?

Ravindranath:
We have not tried Benlate on head molds. Trials on the control of ergot and head mold are done at different centers. At some centers, combination of fungicides with Aureofungin has controlled head molds.

Tyagi:
In Nigeria we have found that a combination of any benzimidazole fungicide with Dithane M-45 sprayed twice at the appropriate time gives almost complete control of head mold.

Williams:
What is the route of infection of the sorghum ergot pathogen? If it is principally through the stigma (as it is in pearl millet), it could be difficult to control by fungicide application.

Ravindranath:
Infection is through stigmas. In hybrid-production programs, if there is improper synchronization, the lines meant for seed production will be highly infected with ergot. Perhaps Dr. Sundaram can clarify further.

Sundaram:
The sorghum ergot fungus can enter the ovary either via the stigma or directly through the ovary wall. The entry of the fungus is restricted once the ovary wall becomes thickened and the stigmas wither.

Pakistan

Frederiksen:
What is the situation regarding hybrid sorghum in Pakistan?

Hamid:
In Pakistan farmers want tall sorghums with sweet juicy stalks for feeding cattle, and sorghums with white bold grains for making bread. At the moment hybrids are mainly restricted to Government farms and there is very little hybrid production by farmers. We are now urging our breeders to produce high-yielding, disease-resistant sorghum with sweet juicy stalks to meet farmers' demands.

Philippines

Balasubramanian:
Dr. Dalmacio mentioned the importance of tar spot (*Phyllachora*) in the Philippines. I

would like to know if this disease is worse on dwarf or on tall cultivars?

Dalmacio:
Tall and dwarf varieties and hybrids appear to be equally infected.

Malaguti:
In Venezuela, *Phyllachora* is not found in sorghum but is found only on maize in cool humid environments. What is your experience in the Philippines on sorghum and maize?

Dalmacio:
In the Philippines, *Phyllachora* is found throughout the year but is most predominant in the rainy season, and is not very common during the dry season. Of course, in the southern part of the Philippines it rains almost every afternoon.

Frederiksen:
I was wondering if you had some specific strategies in mind as to how you were attempting to control the serious leaf-disease problems in the Philippines?

Dalmacio:
The most that can be done at this time to control serious sorghum disease is to avoid ratooning and to discourage sorghum growing in the same area season after season. Also, adjusting the planting date may be helpful.

Right now we do not have varieties that are resistant to foliar diseases, but we hope that in the near future we can incorporate the resistance genes that we have identified in some materials into useful varieties. For *Cercospora* and *Phyllachora* we have no resistance at present, and while we are looking at possibilities of control with fungicides, we have no data on the economics of treatments.

Thailand

Balasubramanian:
Does SDM on maize in Thailand produce abundant conidia?

Pupipat:
Yes.

Wall:
How was the test material inoculated in the Ridomil tests?

Pupipat:
We inoculated by spraying or dipping the conidial suspension of the pathogen on the leaves and leaf-whorls, respectively. Usually we inoculated only the spreader rows, which were planted about 10 to 11 days earlier. In some cases, when the test plants did not get sufficient infection, we inoculated the whole plot. Tween-80 at 500 ppm was added to the conidial suspension in order to produce more infection.

Sundaram:
In some trials Ridomil has not given as much control of downy mildew in pearl millet as reported for sorghum and maize.

Williams:
In many trials which I have been associated with, Ridomil used as a seed dressing has given excellent control of downy mildew in pearl millet up until about 40 days after planting. We must remember that pearl millet is a highly tillering crop, unlike sorghum and maize, and when you have the trial growing in a disease-nursery situation, with sporangial inoculum continually provided by nontreated "infector rows," it is impossible to prevent infection on late tillers with seed-dressing treatments. The seed treatment will be much more effective on pearl millet in a farm situation where there are no infector rows to provide sporangial inoculum to the late tillers.

Sorghum
Grain Molds

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A Review of Sorghum Grain Mold

R. J. Williams and K. N. Rao*

In a recent phytopathology review article, R. R. Nelson states that review chapters should be speculative, probing, scientifically critical, and perhaps even provocative — rather than largely a review of what one author believes to be the relevant literature concerning a particular subject. He said authors should spend as much time or more looking forward as they do backwards, and that just as every generation of scientists is obligated to contribute to the existing body of knowledge, so is it obliged to critically assess the current knowledge to better guide its future scientific rationale (Nelson 1978). We agree with Nelson's concept of review chapters, and in this paper have attempted to live up to some of his criteria. We have not attempted to refer to and list all references relating to sorghum grain molds; instead we give a sprinkling of references to illustrate the points we try to make. Our listings will act as literature foci for those who want to study further the range of literature published on this topic. The omission of appropriate references should not be construed as an oversight or as a lack of recognition of significant contributions.

Sorghum is cultivated widely throughout the tropical, subtropical, and temperate regions within the latitudes 45°N and 45°S. It is grown largely in Africa, Asia, the Americas, and Australia, in regions that are generally too dry for consistent, reliable maize production. In North America and Australia, sorghum is grown commercially on large farms with grain yields averaging 3053 kg/ha and 2222 kg/ha, respectively (FAO 1977); the grain is used principally for animal feed. In the tropical less developed countries, sorghum is produced predominantly on peasant farms where grain yields are low (they average 544 kg/ha in India and 572 kg/ha in West Africa), and the grain is used primarily for human food. These figures indicate the major gap between yields in the tropical less developed countries, where paradoxically the sorghum is needed to directly feed people, and those in the more developed countries, where the sorghum is used to feed animals, which ultimately further embellish the diets of the populations of those countries. There is an urgent need to raise and stabillize sorghum yields in the tropical less developed countries, and major efforts to do this are under way in national, regional, and international programs.

Sorghums in the tropics have evolved in a hostile environment, where unreliable rainfall, poor soils, pests, diseases, and parasitic weeds all constantly exert a harsh selection pressure. The traditional cultivars may not be high-yielding under optimum conditions, but they have a high survival value when conditions are tough. One of the survival mechanisms is a flowering time linked with daylength, so that the grain generally matures in dry weather when pest and pathogen pressures on the grain are much reduced. This trait, however, is regarded as undesirable by most plant breeders, because photosensitive cultivars generally have bulky plants with a poor harvest index and are suited to cultivation at low plant densities to reduce the risk of drought stress. In years when the rains cease early, there is insufficient moisture for good grain filling of these photosensitive cultivars. In order to ensure good grain filling without the interference of drought stress, the production of relatively short-duration nonphotosensitive sorghums is a primary objective of almost all sorghum improvement programs. The resultant cultivars, however, lack the built-in mold-escape mechanism of the local sorghums, and the grains that mature during wet weather are vulnerable to infection by several fungi. Heavy selection pressure for resistance to these fungi has not been exerted on much of our working

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material, and thus most of our improved cultivars are highly susceptible to grain mold. The problem of sorghum grain mold has not been created by plant breeders, but has achieved considerably greater significance because of the efforts to improve the crop. Because of this, control of sorghum grain mold has also become a major activity in many sorghum improvement programs, and the time is ripe for a thorough review of the progress made and the problems that remain.

Head Mold, Grain Mold, Grain Weathering, and Head Blight

Terminology

In the various papers and reports on this topic, the terms *head mold, seed mold, grain mold, grain weathering,* and *grain deterioration* are all encountered.

In the early reports from ICRISAT Center you will find the term *head molds*, but now in ICRISAT Cereal Pathology reports you will find only the term *grain mold*. This reflects our recognition that the problem is specifically that of mold infection of the developing grain. In fact, evaluation of moldiness of the total head can be misleading, as some cultivars develop mold on the rachis and glumes but maintain clean seed — and vice versa. Our colleagues working with sorghum in the Francophone areas of Africa make a similar distinction (Denis and Girard 1977) and call the problem *"moisissures des grains"* which translates literally as *"molds of grains."*

The term *grain weathering* refers to the total physico-chemico deterioration of the grain, of which fungal infection and development are major components. Other factors contributing to weathering include alternate wetting and drying, prolonged exposure to sunlight, and insect and bird damage. Weathering symptoms include seed discoloration (*staining*), seed-coat splitting, and germination (*sprouting*) on the head. Castor (personal communication) believes that *Fusarium moniliforme* plays a role in sprouting and has experiments under way to test this hypothesis. Glueck et al. (1977) suggest that the term *"field deterioration"* is more appropriate to describe the complex weathering syndrome. We will hear more in this session about several aspects of grain weathering from other participants, and it is of course the whole complex with which sorghum improvers must be concerned. However, for the purpose of our review we will deal principally with the problem of grain mold, a problem that we believe is of the greatest importance in sorghum grain weathering.

The distinction between grain mold and head blight, and the possible relationship between the two, need discussion. Schroeder and Hein (1975) refer to head blight, but from their descriptions it appears that they were working with grain mold. Conversely Gray et al. (1971) in their discussion of losses due to head mold describe symptoms that are consistent with what we know as head blight. It may be useful if we record here our definitions of grain mold and head blight so that at least for this paper the distinction is clear.

Grain Mold

The term grain mold is used in this paper to describe the diseased appearance of sorghum grain resulting from the infection of the developing grain by one or more parasitic fungal species. The term is used in the literature to describe the diseased appearance of sorghum grain infected with fungi while developing in the field, while standing in the field after maturity, and while in storage.

The symptoms that develop as a result of field infection of the developing grain by parasitic fungi depend upon the fungus or fungi, and the time and severity of infection. Grains severely infected appear to be completely covered with pink and or black mold, and such grains disintegrate in the threshing process. Lightly infected grain may appear almost normal except for slight pink or black discoloration on a small portion of the surface; internally the grain appears normal.

It is our experience that grain showing no external symptoms may be infected by grain mold fungi, for we can consistently isolate *Fusarium* spp from surface-sterilized clean-appearing sorghum grain harvested during the rainy season.

A *Phoma* sp often produces small black pycnidia on otherwise clean sorghum grain. We feel this should be regarded as a part of the grain mold problem, as it falls within our de-

finition stated in the first sentence of this subsection.

Head Blight

Williams et al. (1978) describe head blight as the invasion of the tissues of the inflorescence by *Fusarium moniliforme*, with florets killed to various degrees up to complete destruction of the head. The internal tissues of the panicle develop a red to brown discoloration, which extends into inflorescence branches and even down into the upper portion of the stem. In severe cases the peduncles break over.

Is There a Relationship Between Head Blight and Grain Molds?

In a later section of this paper, we will show that *Fusarium moniliforme* is one of the major causal agents of sorghum grain mold. Head blight is also caused by *Fusarium moniliforme* when it attacks the structural tissues of the inflorescence. Is head blight merely the result of an early severe infection which, if it occurred later, would have been expressed as grain mold? Are the same strains of *F. moniliforme* capable of causing both diseases? Are sorghums resistant to head blight also resistant to grain molds?

We do not know the answers to these questions, but from our experience with grain molds and head inoculations at ICRISAT we believe that head blight is not just the result of an early severe infection by one of the grain-molding fungi. We would like the views of the participants on this.

Importance of Grain Mold

How Important is Grain Mold?

In his authoritative text on sorghum diseases, Tarr (1962) does not consider sorghum grain mold to be of great importance. He states in the introduction to the section on inflorescence and grain diseases that head molds may be of comparatively minor importance, though information is meager. Fifteen years later, in the questionnaires returned at the International Sorghum Workshop held at Hyderabad in 1977, sorghum breeders and pathologists from Africa, Asia and the Americas overwhelmingly placed grain molds as one of the major disease and research problems in the improvement of the sorghum crop.

It is significant that the 15-year period between Tarr's book and the International Workshop has been one of active sorghum improvement in many countries, particularly in India and in several African countries. It has also been the era of "miracle grains" and the "green revolution" in which, to begin with, it was thought that the production of short-statured, short-duration, fertilizer-responsive cultivars was the answer to the world's food problem. Pests and pathogens have shown this doctrine to be unsound. The wheat rusts, the rice viruses, and the grain molds of sorghum clearly demonstrate that if we ignore the pests and pathogens we may be able to produce breeding lines with exceedingly high-yield potential, but we will not be able to provide the farmers in the tropics with acceptable new stable high-yielding crop cultivars.

How is Grain Mold Important?

Grain mold has become a major and widespread sorghum disease problem because it significantly reduces yield and acceptability of the harvested grain, and because no high levels of grain mold resistance have been used in breeding most of the new short-duration sorghums for human consumption.

Actual Yield Losses

There are few reports of studies on the actual weight loss of grain resulting from infection by molds.

Bhatnagar (1971) reported a marked reduction in the size and weight of sorghum grain artificially infected with *Curvularia lunata*, but provided no quantitative data.

Gray et al. (1971), reporting from central Kentucky, mentioned sorghum yields of several head mold-susceptible cultivars of less than 30 bu/acre, when yields of 100 bu/acre or more were expected. The low yields were the result of the failure — believed to be due to head mold — of heads to produce mature seed. The

symptoms described by Gray et al. (1971) lead us to suspect that head blight may have been partially responsible.

Glueck and Rooney (1976) report variability in 1000-grain weight from 19.3 g to 33.5 g and of test weight from 47.3 lb/bu to 62.2 lb/bu under severe weathering conditions at College Station, Texas. They comment that although grain from all lines, with prolonged exposure to weathering, decreased in test weight, density, and germination, the reduction was minor for lines with superior weathering resistance.

In their paper for this Workshop, Denis and Girard report results of comparison of yield and mold infection at three sites in Senegal. They conclude that when there are few other limiting factors to plant development, grain mold has a clear negative effect on yield.

Castor (1977) also reports reduced yield, test weight, and 1000-kernel weight in sorghum due to grain mold caused by *Fusarium* spp.

Sundaram et al. (1972), in their report on a survey of sorghum and millet diseases in India, state that losses of up to 50% due to head mold were encountered in hybrid sorghum in experimental plots at Coimbatore. However, the table cited in support of their statement indicates that the proportion of plants with grain mold infection was up to 50%, and there is no actual data provided for yield reduction due to mold.

Glueck et al. (1977) report that significant losses due to grain mold occurred in Texas in 1974 and 1976.

Although we have not found definitive data on yield reduction due to grain mold, observations of several workers in different parts of the world suggest that they are significant. It is our experience that in highly susceptible cultivars the yield losses can reach 100%. Do we need more accurate information on the relationship between invasion of grain by fungi and the weight loss that results? It will require considerable time and effort to get meaningful data on the relationship between time of inoculation, degree of infection, and weight loss. Will this be time and effort well spent, or will it be more profitable to seek and develop resistance to the pathogens involved? As grain mold losses are not measured just in terms of weight loss on the head, but also in terms of loss in marketable grain and reduction in market value, we feel that determination of relationships between infection and weight loss is not at present of overriding importance.

Losses in Quality

Market Value

For grains produced for direct human consumption, the quality of the harvested product is of considerable importance. Grain mold discolors the grain, reducing its acceptability and thus its value. In surveys conducted in villages in central India, von Oppen and Jambunathan (1978) found market prices of sorghum grain to be negatively related to degree of mold infection. The price of the most moldy grain was about 20% less than that of the cleanest grain. The moldiest of the grain in these samples was, however, not nearly as severely molded as are typical grains from many of our improved sorghums in our grain mold screening nurseries.

So, in addition to actual weight losses caused by grain mold infection, losses in marketable yield occur due to a reduction in consumer acceptability, even with moderately molded grain.

Nutritional Value

Glueck et al. (1977) report that sorghum grain deterioration results from physical, physiological, and chemical changes. They state that starch granules of deteriorated grain have considerable pitting on the surface (resulting from enzyme degradation), and that the protein matrix is weakened and partially hydrolyzed. However, they also state that the chemical composition of deteriorated sorghum is not greatly different from that of undamaged grain, and that digestibility of the deteriorated grain may be slightly improved. In deteriorated grain, the content of soluble carbohydrates is usually reduced, because they are utilized to provide energy for the growth and development of the fungi. The proteins are hydrolyzed and partially utilized in the synthesis of fungal protein and remain in the grain. These findings of Glueck et al., appear, however, to be based on a relatively light degree of grain deterioration. Studies based on varying degrees of infection by differ-

ent weathering agents, including the different fungi involved, are needed.

A second major area of concern when considering nutritive value of moldy sorghum grain, is the possibility that the fungi will produce mammalio-toxic chemicals known as mycotoxins. The problem of mycotoxins in food has been recognized only in relatively recent times, and initially work was concentrated on the aflatoxin group of chemicals. The aflatoxins are powerful hepatic carcinogens, produced by *Aspergillus* spp. We now know that several genera of fungi — including *Fusarium, Penicillium, Stachybotrys,* and *Aspergillus* -- produce in food stuffs chemicals toxic to man and animals. The known mycotoxins, the fungi that produce them, and the nature of the diseases they cause are thoroughly reviewed by Mirocha and Christensen (1974) and Martin and Gilman (1976).

The aflatoxins, produced by the *Aspergillus flavus* group and *Aspergillus parasiticus*, are highly toxic to animals at very low concentrations. There are several reports of aflatoxins detected in sorghum grain and grain products. Ceady (1965) demonstrated aflatoxin in millet, sorghum grain, and sorghum flour in Ethiopia, and Alpert et al. (1971) found aflatoxin in sorghum in Uganda. In the USA, Shotwell et al. (1969) found aflatoxin in excess of 10 parts per billion in six of 533 sorghum samples.

In a survey of southern African foodstuffs, Martin et al. (1971) and Martin (1974) reported the presence of aflatoxin in excess of 10 parts per billion in three of 39 good quality sorghum samples.

Tripathi (1974) isolated aflatoxins B_1, B_2, G_1 and G_2 from moldy sorghum in northern India. The toxins were isolated from ears collected from the fields at least 7 days after rains and from ears collected immediately after the rains and stored at room temperature for 7 days or more before extraction. *Aspergillus*, with 13% of the colonies isolated, was the fourth most common genus detected in isolations from the moldy sorghum, following *Curvularia* (42%), *Fusarium* (19%), and *Phoma* (15%). This is significant, for it indicates that in certain environments aflatoxins can be important in sorghum grain right from the field. However, in terms of the sorghum field-grain mold problem, *Aspergillus* infection is not nearly so important as *Fusarium* infection.

Rukmini and Bhat (1978) isolated *Fusarium incarnatum* from naturally occurring moldy sorghum grain, and found a toxic metabolite, which they call T-2 toxin, associated with the presence of this fungus. The toxin was also recovered from rice artificially inoculated with *F. incarnatum.*

The fungi *Fusarium roseum, F. tricinctum, F. oxysporum,* and *F. moniliforme* produce the mycotoxin zearalenone, which has estrogenic properties and acutely affects some animals at very low concentrations. Martin (1974) demonstrated the presence of zearalenone in four samples of moldy sorghum and 27 pooled samples of "sour" fermented porridge and beer made from maize and sorghum in Swaziland. Martin and Gilman (1976) state that this study has been extended to Lesotho, where small quantities of zearalenone (up to 50 parts per billion) were found in 16 of 71 beer samples (probably sorghum-based), and they conclude that the regular ingestion of zearalenone by the African population could perhaps explain the high incidence of certain diseases (such as cervical cancer) in a way hitherto unsuspected.

Schroeder and Hein (1975) found zearalenone in grain sorghum heavily infected with *F. roseum* "Gibbosum" and *F. roseum* f. sp *semitectum*. Grains from selected infected heads contained twice as much zearalenone as grain from a combined bulk. They conclude that zearalenone contamination may be a significant factor when *Fusarium* head blight (grain mold) is severe in maturing grain sorghum during warm, highly humid weather.

Stipanovic and Schroeder (1975) investigated the zearalenone producing potential of *Fusarium roseum* cultures isolated from moldy sorghum. From one culture grown on grain sorghum they isolated zearalenone and two related compounds, zearalenol and 8'-hydroxyzearalenone. They conclude that since the synthetic zearalenol diastereomers have uterotropic activity, their presence in grain sorghum may present a potential health hazard.

From the above, it seems that the major mycotoxin danger in molded sorghum grain is zearalenone. Mirocha and Christensen (1974) state that a period of low temperature, or of alternating moderate and low temperature, is necessary for the production of zearalenone. However, low and moderate are not defined. Reports of zearalenone from several African

countries, including northern Nigeria (MacDonald, personal communication), indicate that perhaps the temperature requirements need not be low in absolute terms. We feel that much more work is needed on this potentially important problem throughout tropical sorghum-producing areas. Martin and Gilman (1976) raise an interesting point in their conclusions when they point out that the fungal estrogens may have important gynecological implications, and that zearalenone could perhaps be used in the manufacture of a chemical contraceptive.

Loss of Viability

Loss in viability of molded sorghum seed and reduction in seedling vigor is reported by many workers.

Arif and Ahmed (1969) found that all fungi isolated from sorghum grain reduced germination, and that *Fusarium* was the most inhibitive, followed by *Aspergillus*, *Penicillium*, and *Helminthosporium*.

Narasimhan and Rangaswamy (1969b) found viability to be reduced by 40 to 80% when healthy sorghum seeds were treated with mold isolates.

Tripathi (1974) obtained 56% germination with molded sorghum grain, whereas apparently clean grain (of the same cultivar?) gave 76% germination.

Castor (1977) reports 95 and 77% germination of grain harvested from water-sprayed and *Fusarium*-inoculated heads, respectively.

Mathur et al. (1975) reported that *Fusarium moniliforme* adversely affected germination and seedling growth in sorghum grain samples, and they frequently detected *F. moniliforme* in embryos of sorghum seed samples.

Rao and Williams (1977) recorded viability losses of up to 100% in sorghum grains with severe *Fusarium* and *Curvularia* infection.

Denis and Girard (1977) regard loss in viability to be so important a part of the grain mold syndrome that they recommend a germination test as part of the standard evaluation for identification of grain mold resistance.

It appears from these reports that the infection of sorghum grain by mold fungi, particularly *Fusarium* spp, can have a marked negative effect on seed viability. In addition, those seeds from infected grain that do germinate can produce blighted seedlings (Bhatnagar 1971; Lambat and Ram 1969), further reducing the value of the seed. In terms of marketable yield for food, this phenomenon alone probably has little significance. However, for the researcher, the seed producer, and the farmer, this aspect is particularly important.

Causal Agents, Time of Infection, and Predisposing Factors

Fungi Associated with Moldy Sorghum

In our search of the literature on sorghum grain molds, the most frequently encountered reports were those concerned with fungi associated with moldy sorghum grain. Many of these reports can be classified as seed mycoflora studies, and in many of them market samples or stored grains were used. Few workers have taken the extra step to determine the relative roles of the various fungal species in the etiology of the field problem of sorghum grain mold. We feel it will be of little use to attempt to list here all papers in which molds from sorghum grain have been described. We will just provide a reference from each of the main sorghum-growing regions so that the interested worker can follow up the other references listed in these papers.

In India, where there are many papers on this subject, Narasimhan and Rangaswamy (1969a) provide a detailed quantitative analysis of molds from sorghum cultivars from many locations. In Pakistan Junejo and Malik (1969) report a detailed study, and in Africa Denis and Girard (1977) provide a comprehensive list of fungi associated with moldy sorghum. There are also many papers on this subject from the USA: Hansing and Hartley (1962) report a large and detailed study and, more recently, Castor (1977) deals specifically with fungi causing grain molds on sorghum in Texas.

Fungi in the following genera are reported to have been detected from sorghum seed:

Acrothecium	*Curvularia*	*Pellicularia*
Alternaria	*Cylindrocarpon*	*Penicillium*
Aspergillus	*Drechslera*	*Phoma*
Bipolaris	*Fusarium*	*Pestalotia*

Chaetomium	*Gloeocercospora*	*Pycnidium*
Chaetopsis	*Gonatobotrytis*	*Ramularia*
Cladosporium	*Helicosporae*	*Rhizopus*
Cladotrichum	*Helminthosporium*	*Sordaria*
Cochliobolus	*Mucor*	*Thielavia*
Colletotrichum	*Nigrospora*	*Trichothecium*
Cunninghamelia	*Olpitrichum*	

Of these, the most frequently encountered genera were *Fusarium, Curvularia, Alternaria, Aspergillus,* and *Phoma.*

Fungi That Cause the Field Grain Mold Problem

The most detailed studies on the causal agents of sorghum grain mold have been made at ICRISAT Center (Rao and Williams 1977); in Texas, USA (Castor 1977, Castor unpublished); and in Bambey, Senegal (Girard 1976; Denis and Girard 1977).

Rao and Williams (1977) report the development of high levels of grain mold in sorghum heads inoculated at anthesis with conidial-mycelial suspensions of *Fusarium moniliforme, F. semitectum,* and *Curvularia lunata.*

Castor (1977) reports inoculation of sorghum heads in the field with isolates of *Fusarium moniliforme, F. semitectum, Curvularia lunata, C. protruberata, Alternaria* sp, and *Helminthosporium* sp at various times after flowering. The *Fusarium* and *Curvularia* isolates were the principal pathogens and caused discoloration and reduction in viability. *Curvularia* did not reduce germination as much as *Fusarium*, and seeds from heads inoculated with *Fusarium* at flowering had the highest proportion of split pericarps. *Curvularia* and *Fusarium* were easily recovered from surface-sterilized seed. In seed-dissection studies of sorghum lines inoculated with *Curvularia* and *Fusarium*, 16 and 23%, respectively, of the embryos were found to be infected. Castor observed that the scutellum was often partially degraded in seeds from *Fusarium*-inoculated heads, and suggests that the fungus can destroy the embryo indirectly by interfering with translocation from the endosperm to the embryo during germination. All of the grain from susceptible *Fusarium*-inoculated heads contained *Fusarium* in the endosperm, and 78% of grain from *Curvularia*-inoculated heads had that fungus in the endosperm.

In later studies, Castor (unpublished) found *F. moniliforme* to be more highly pathogenic than *F. semitectum.*

From these observations and studies in USA, Africa, and India, it is evident that *Fusarium moniliforme, Fusarium semitectum,* and *Curvularia lunata* are the primary pathogens in the field problem of sorghum grain mold. At this point we should note that, in the advanced lines developed with resistance to *Fusarium* and *Curvularia,* a *Phoma* sp caused considerable grain damage at ICRISAT Center during the 1978 rainy season. The general significance of infection by *Phoma* needs further investigation.

Time of Infection

The work of Rao and Williams (1977) and of Castor (1977, and personal communication) clearly shows that the principal grain mold fungi are pathogenic, and can invade the florets at anthesis. The grain mold problem is not a problem of saprophytic fungi invading a source of carbohydrate under moist conditions; rather, it is a problem of pathogenic fungi.

Undoubtedly if sorghum grain is left in the field long enough after maturity and the weather is conducive to the development of saprophytic molds, then they will develop on the grain. However, this would really come under the heading of storage problems, as under these circumstances the grain could be regarded as being stored on heads in the field.

Predisposing Factors

Tarr (1962) recognized that mold may develop in the sorghum inflorescence at any stage from the young inflorescence to the mature head, provided climatic conditions are suitably moist. It seems to be the general experience of sorghum workers that wet weather following flowering is necessary for grain mold development, and the longer the wet period the greater the mold development (Koteswara Rao and Poornachandrudu 1971; Gray et al. 1971; Rao and Williams 1977). Dry weather during flowering and grain development followed by wet weather near maturity will not promote such serious mold as wet weather that continues from the time of flowering onward.

It has been suggested to us that we should

determine the exact rainfall pattern, temperatures, and humidities that cause serious grain molds in sorghum. We have resisted these suggestions, for we feel that our time, energy, and money are better spent in efforts to locate resistance. Is there a need to define more accurately conditions of temperature, rainfall, and relative humidities associated with sorghum grain mold development? Is such information going to be useful?

The Control of Sorghum Grain Mold

The only two practically and economically feasible methods for the control of sorghum grain molds are avoidance and the use of grain mold resistant cultivars. The recommendation of the All-India Coordinated Sorghum Improvement Project to spray heads three times with a combination of 200 ppm Aureofungin and 0.2% Captan (Anon. 1978) are impracticable and uneconomic, except for small plots of valuable research material.

Control by Avoidance

Grain mold avoidance is the grain mold control method used by peasant farmers growing their traditional cultivars, because the daylength-related flowering period of these cultivars generally enables the developing grain to escape the most mold-conducive weather. In the semi-arid tropics the magnitude and duration of rainfall is highly variable and thus, even with traditional cultivars, molds are a problem in some years. Cultivars that have evolved represent a compromise between being early enough to fill the grain to a reasonable degree and late enough to avoid the severe mold and pest problems promoted by wet weather. Control through avoidance is not compatible with the requirements of a higher yielding phenotype, which must have a large number of heads/m^2, each of which needs to have a consistently high degree of grain filling. These requirements are met by the growing of improved early-maturing cultivars. The identification and/or development of resistant cultivars is the only solution to the grain mold problem, if improved high-yielding cultivars are to be promoted for widescale use.

Control with Host-plant Resistance

When we first started our resistance screening for sorghum grain mold, we were told that it was likely to be a long and futile effort. This opinion was based on the idea that the mold problem is caused by many weakly parasitic and purely saprophytic fungi. Based on our knowledge that the major problem is caused by two to three species of parasitic fungi, and our belief that, given the appropriate selection pressure on a genetically variable population, many unsuspected and unselected traits can be identified and strengthened, we were much more optimistic. Since 1974 we have made major efforts to identify and utilize grain mold resistance in sorghums with white, cream-colored, yellow, and light red grains. K. N. Rao, in his paper later in this session will give a more detailed account of our progress.

Other programs that have or have had a major component of sorghum grain mold resistance identification are the now-terminated JP-26 Project based in Nigeria (King 1974; Zummo 1975, 1976), the IDRC-ISRA program based at Bambey, Senegal; the program centered at Texas A & M University (Anon. 1976; Castor 1977); and the All-India Coordinated Sorghum Improvement Project based at Rajendranagar (Rana et al. 1978).

Since 1976, a cooperative international grain mold resistance screening program has been conducted annually (ICRISAT 1978b) with the objective of identifying mold resistance that is not location specific.

In all these programs, no entry has proved immune to grain mold, and no entry has shown a consistently high degree of resistance at all locations. However, certain lines consistently develop less mold than others at many locations and across seasons. More details are provided in the paper on the activities of the International Sorghum Grain Mold Nursery.

Identification and development of grain mold resistance in sorghum is a slow and difficult process. However, we believe that high levels of grain mold resistance can be assembled, and that in the past 4 years considerable progress has been made to this end.

It appears that the additive genes contributing to mold resistance in food grain sorghum are scattered in the germplasm. In the evolution of tropical sorghums, the development of the

avoidance mechanism was probably an easier and generally more advantageous process than the development of a high degree of resistance specifically to grain mold.

Let us now discuss in more detail important principles on screening for mold resistance, sources of resistance, and factors that may be associated with or responsible for mold resistance.

Identification, Nature, and Genetics of Grain Mold Resistance

Screening Techniques

Except for the ICRISAT and Texas A & M screening programs, the search for grain mold resistance in sorghum has been conducted mainly under natural incidence of mold in the field. While this method is satisfactory in years when the rains are frequent and prolonged throughout the flowering and grain-filling period of all the test entries, it is unsatisfactory when these conditions do not occur.

Strategies to reduce the chances of escape under natural moldincidence screening are to plant early (under irrigation) so that the flowering period of the test entries is more likely to coincide with prolonged wet weather, and/or to move the screening to wetter locations. The latter strategy has been successfully adopted by Denis and Girard (1977) in Senegal and by Frowd (personal communication) in Upper Volta. However, the semi-arid tropics are characterized by erratic rainfall (magnitude, duration, and distribution), and reliance on natural mold incidence for resistance screening will result in failures in some years.

At ICRISAT, we inoculate inflorescences at anthesis with conidial/mycelial suspensions of *Fusarium moniliforme, F. semitectum,* and *Curvularia lunata.* We attempt to provide the humidity required to promote mold development by bagging inoculated heads and (on rainless days) using sprinkler irrigation in the evening hours during the grain-filling period. This screening method works well, and is used on a large scale (3.5 ha) to screen thousands of breeding lines during each rainy season. The technique does not work in the extremely hot and dry part of the year.

Castor (1977) at Texas A & M uses a technique similar to that developed at ICRISAT Center.

The lack of a screening technique that can be used in the dry season slows down progress in the development of resistant cultivars, as only a single generation can be tested during any one year. Selections and progenies of crosses made in one year must wait until the next rainy season for evaluation. One way to overcome this problem is to develop cooperative links with scientists in areas where the calendar provides a rainy season during the dry season at one's own location. The rainy season in Tanzania is coincident with the dry season at ICRISAT Center. Now that ICRISAT has a sorghum breeder stationed there, we will attempt to exploit this strategy. However, phytosanitary regulations may well reduce, or perhaps even totally thwart, the effectiveness of such a system.

A second strategy to permit screening of more than one generation in a year would be to develop a laboratory-based technique, that would function independent of the weather. During the last 12 months at ICRISAT Center, we have attempted to develop a laboratory technique whereby meaningful selections can be made for degrees of grain mold resistance. By incubating grain harvested in the *rabi* (post-rainy) season on moist blotting paper at 25°C in an alternating 12 hr light and 12 hr dark regime, we are able to distinguish between cultivars in degree of mold development. Even when seed is maturing during the dry season, it seems that there is sufficient nighttime relative humidity (and dew formation) to support some grain infection. Differences among cultivars are not evident by visual examination of the grain, but become evident during the incubation described. If these results are well correlated with field reactions, the method will become an important tool in the screening process.

Rating Scales

The use of a meaningful rating scale or system is as important as the choice of an effective inoculation technique. Denis and Girard (1977) reviewed in depth the merits of and problems with grain mold-rating scales used in several programs. They concluded that a combination of several evaluation methods is useful in attempting to classify grain mold reactions. They

recommended including not only an estimation of the degree of molded grain surface, but also a germination test. At ICRISAT Center, we have found a visual on-table ranking of threshed grain from "least molded" to "most molded" a very simple and reproducible method for cultivar differentiation. A less detailed method can be employed in initially screening large numbers of source material, but will not be effective in attempting to distinguish between elite mold-resistant breeding progenies. The scoring method employed must allow meaningful differentiation of test entries, and should be reliable and reproducible. We recommend that the Denis and Girard (1977) review be studied, and that the appropriate combination of scoring methods be used to meet the requirements of the particular stage of the individual program.

Sources of Grain Mold Resistance

The acute problem of grain mold susceptibility and the subsequent search for resistance are relatively recent in origin. The problem is particularly difficult in the tropics, because in this situation factors that contribute to resistance must be consistent with acceptability of grain as a human food.

One of the earliest reports on grain mold resistance was made by Gray et al. (1971), who reported that Funks 814 was less susceptible than two other cultivars to grain molds, and that out of 16 Northrups-King sorghum lines, 3008 and 3016 had few or no head mold symptoms. These results were apparently obtained under natural field conditions, and no information is provided on the flowering time of these lines.

In the study of Koteswara Rao and Poornachandrudu (1971), the varieties IS-452, IS-455, IS-472, and IS-473 appeared to be fairly resistant to molds, though again no data on flowering dates are provided.

Other reports of sources of resistance (Table 1) include Zummo (1976), Glueck and Rooney (1976), ICRISAT (1978a, 1978b) and Rana et al. (1978).

Rao and Williams (1977) report that from a field-screening program of about 6000 sorghum lines in 1975 and 1976, they identified only 43 as relatively less susceptible.

Since 1976 an international cooperative grain mold screening program has been coordinated from ICRISAT (this is discussed in detail later in this session), and entries that have consistently shown up well across seasons and locations are E 35-1, IS-2328, IS-2327, IS-14332, IS-2261, and IS-9225. Flowering periods of these lines range from 68 days (IS-14332) to 80 days (E 35-1). These sources have been used intensively in the ICRISAT grain mold resistance breeding program, and some impressive advanced progenies from them were identified during 1978 at ICRISAT Center. In particular, E 35-1 progenies are very promising, with little mold development on large heads with plump white grain.

There is probably a need to look for further sources of grain mold resistance, with emphasis on germplasm from the wetter regions of sorghum production. However, much of this material is likely to be highly photosensitive and

Table 1. Reported sources of resistance to sorghum grain mold.

Authority	Resistance sources reported
Gray et al. 1971	Funks 814, Acc. No. 3008 and 3016
Koteswara Rao and Poornachandrudu 1971	IS-452, IS-455, IS-472, IS-473
Zummo 1975	IS-3555, IS-477, IS-473, IS-453
Glueck and Rooney 1976	SC-748, SC-279-14, SC-566-14, 74 PR 759, SC-103-12
ICRISAT 1978b	IS-2327, IS-2261, E 35-1, IS-9225, IS-2328
Glueck et al. 1977	SC-279-14, SC-748-5, 74 PR 759, SC-566-14, BTX-398, SC-103-12
Rana et al. 1978	CSV-4 (CS-3541), CSV-5 (148/168), SPV-81, SPV-141
ICRISAT 1978a	M-36008, M-36091, M-36284, M-35078, M-36188, M-35078, M-36188, M-35115, M-35194, M-35052

thus will not have been subjected to mold resistance selection pressures. We believe considerable progress can be made by intermating the present sources of low susceptibility to generate variability and to concentrate the scattered resistance genes.

Factors Responsible for or Associated with Resistance

Glueck et al. (1977) cite several sources in which grains with certain plant or kernel characteristics are reported to be relatively resistant to field deterioration — e.g., open heads with seeds completely enclosed in long papery glumes (Murty 1975), and brown seeds with high tannin content and the presence of a pigmented testa (Ellis 1972; Harris and Burnes 1973; Murty 1975). Some of these characteristics are undesirable from other standpoints, and we have concluded from our examination of several thousand diverse sorghum lines at ICRISAT that there is no apparent correlation between evident panicle and/or grain characters and the capacity to resist becoming severely molded. For example, we have seen severe mold develop on grain in very loose and in very tight panicles, in grain enclosed by long glumes and by normal glumes, and on dark red, white, and yellow grain. The low susceptible lines E35-1, IS-2327, and IS-2328 are all white-grained, with relatively small glumes, and E35-1 has a compact panicle.

Glueck et al. (1977) suggest several possible mechanisms for resistance to grain deterioration (a term that includes more than just grain mold), including rate of water absorption and conductivity of seed leachates. They suggest that water-absorption and leachate tests may be useful as preliminary screening methods. However, we believe that it is unwise to embark on screening for mold resistance by measuring a character that may or may not be always strongly correlated with mold resistance. As mold resistance is probably composed of a complex of characteristics, we believe that it is best to use the pathogens to integrate the many and various plant and grain characteristics that together result in resistance. It is better to use limited resources to look for additional sources of resistance and for further intermating and progeny testing of resistance sources, than to put effort into detailed studies of why a particular line or group of lines happens to be resistant. This is our personal bias and we will be interested to hear comments from participants on this issue.

The Genetics of Resistance

It is almost certain that grain mold resistance in sorghum is the result of the additive effects of many genes affecting several plant characteristics. This hypothesis is supported by the results of Murty et al. (1978) in studies with progeny of crosses between low-, moderate-, and high-susceptible parents. Rana et al. (1978) found that water absorption capacity and seed hardness (considered as probably important factors in susceptibility of grains to mold) are governed by additive genes.

We raise the question whether further studies on the genetics of resistance to grain mold are likely to be of any practical use or importance in the effort to further develop grain mold resistant cultivars.

Summary

The sorghum grain-deterioration problem has become acute with the development of early flowering sorghum cultivars, which frequently fill grain during wet weather. Grain mold, caused principally by *Fusarium* spp and *Curvularia* spp, is a major component of the grain-deterioration complex. Grain mold reduces the quality and quantity of marketable grain, though quantitative data on losses are not available. The possibility of the mold fungi producing mammalio-toxic chemicals in the grain needs further investigation. The only feasible way to control grain mold in early-flowering cultivars is through the use of host-plant resistance. It has been difficult to find a combination of good seed quality (from the consumer-preference aspect) and high level of grain mold resistance, and programs are under way to concentrate resistance factors in acceptable grain types. An international network of cooperators is testing elite products from these programs. Attempts have been made to identify factors responsible for or associated with resistance, but a complex of characteristics is likely to be operating against this complex problem. The genetic basis of resistance is likely to include the additive effects of many genes.

Questions

In order that grain mold control programs may progress in the most direct and sensible way, we would like the discussion on grain mold to include consideration of the following questions:

1. Is grain mold still considered a major problem for sorghum improvement in the tropics?
2. Are there situations, locations, regions, etc., in which grain mold is not a problem and for which we need not provide grain mold resistant cultivars? Keep in mind the evidence from dry-season sorghum grain production, in which grains of highly susceptible cultivars are infected particularly with *Fusarium* spp, even though the grains appear relatively clean.
3. Is there a need for quantitative studies on relationships between yield loss and mold incidence or (in view of the other important effects of grain mold on market value, viability, nutritive value, and mycotoxin potential) can we accept that the only good grain is a mold-free grain and use our resources on research to control grain mold?
4. What improvements can participants suggest in the screening and rating procedures described?
5. Is the international testing program worthwhile? (More questions on this in the paper on the International Sorghum Grain Mold Nursery.)
6. Is more emphasis needed on identification of factors responsible for or associated with resistance? If the answer is "yes," how is the information to be used?
7. Do we need to study further the genetic basis of resistance?
8. Is there a need to intensify research on the mycotoxin dangers of molded sorghum? If so, where can the work be done?

We hope that participants will help us answer these questions and raise others that they feel are important.

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Fusarium and *Curvularia* Grain Molds in Texas

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Grain sorghum is grown on 14.5 million acres (6 million ha) in the USA, producing annually about 800 million bu (22 million tonnes). Grain production in Texas accounts for about 40% of the USA total. Sorghum in Texas is grown under a wide range of climatic conditions — from the rainy humid southeastern coastal areas to the dryland agriculture areas of the northwest. Rainfall is evenly distributed throughout the year and is usually sufficient for crop growth, although in the dryland areas winter rainfall and summer irrigation are important for maximum crop yields. Typically, Texas sorghum hybrids are photoperiod-insensitive "red" plant types, and they are usually short-statured to facilitate combine harvesting. The grain is characterized by a pigmented (nonwhite) pericarp and the absence of a testa. Sorghum grain is used for feed, food, and industrial purposes in the USA, although in Texas the grain is used almost exclusively as animal feed.

Grain molds are a sporadic problem in Texas, usually associated with prolonged rain at maturity or during the grain-filling period. Rain favors fungal growth and delays harvest (which is by combine), in turn compounding the problem. Generally, grain molds are more often encountered in the southeastern areas; here unusually heavy rains, as the crop neared maturity in 1976, affected 400 000 ha and caused 46 million dollars loss (Texas Agricultural Experiment Station 1978). However, in 1974 grain mold and head blight fungi caused substantial losses on the High Plains, an area in northwestern Texas that is normally very dry. These experiences emphasize the importance of research on grain molds and the need to develop varieties with greater levels of resistance to grain mold fungi.

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Grain mold research in Texas has dealt with several basic and applied problems during the past 3 years. We have attempted to answer the following questions:

1. Which fungi cause grain molding?
2. What types of damage can grain mold fungi cause, and how can the damage be measured?
3. Which fungal species is/are the most damaging?
4. When and how does infection occur?
5. How can resistant sorghum lines be identified?
6. What is the most efficient and effective screening program?

These questions are interrelated to varying degrees. This paper will consider each separately. Speculation will be included, where appropriate, to stimulate thought and discussion. In addition, areas of thought important for future research are listed.

Causal Fungi

Numerous reports have listed the large number of fungi which can be isolated from sorghum grain, in storage as well as in the field. In Texas the predominant field fungi, in decreasing order of prevalence, belong to the genera *Alternaria*, *Fusarium*, and *Curvularia*. *Alternaria* spp are commonly found in 50 to 90% of the seeds from "normal" grain having high germination. A comparison of surface-sterilized with nonsurface-sterilized seeds indicated that the growth of *Alternaria* spp was relatively superficial; the percentage recovery was reduced with surface sterilization. In contrast, *Fusarium* and *Curvularia* spp were identified in high numbers from seeds of molded samples low in germination. Therefore *Fusarium* and *Curvularia* spp were identified as the important grain-molding fungi in Texas. The main species of *Fusarium* and *Curvularia* identified were

F. moniliforme, F. semitectum, C. lunata, C. protuberata and *C. trifolii.*

Types of Damage

Grain mold fungi may cause losses in yield and quality of harvested grain in five major ways. These types of damage are often related, and the importance of each depends on how the grain is used.

Grain mold fungi can produce discolorations, and the fruiting structures (e.g., pycnidia of *Phoma* spp) of fungi appear on the pericarp or outer endosperm regions of the kernel. These "blemishes" can reduce market value and cause an off color in grain processed for human food. This damage will be of less importance in areas of the world, such as Texas, where grain is used as an animal feed.

Fungi such as *F. moniliforme* and *C. lunata* secrete enzymes that can degrade endosperm (starch) and germ tissues; the process is often accompanied by visibly molded kernels (Wadje and Deshpande 1976). In addition, plant enzymes especially with *F. moniliforme*, may be stimulated, causing the initiation of germination and the subsequent breakdown of endosperm tissue. Regardless of the source, the enzymes reduce feed or food value per kernel. Generally, feeding trials have shown that there is little difference in energy value between degraded and nondegraded kernels (Texas Agricultural Experiment Station 1978).

F. moniliforme and *C. lunata* appear to interfere with carbohydrate translocation to developing kernels, causing a reduction in kernel size and/or weight (Bhatnagar 1971; Castor et al. 1978; Castor and Frederiksen 1977; Gray et al. 1971; Mathur et al. 1975). This possibly occurs when fungi colonize rachis branches or the hilar region. In this case, grain yield can be reduced without the occurrence of visibly molded kernels.

Fungus-infected seeds often exhibit a reduction in germination and emergence, which cause poor stands in farmers' fields (Bhatnagar 1971; Castor 1977). In addition, seedlings may be killed after emergence, or their growth may be reduced (Bhatnagar 1971). This is especially important in areas of Asia and Africa where farmers plant varieties (rather than hybrids) and save their seed for the next crop. *F. moniliforme*, in particular, seems to initially colonize the scutellar region. Such proximity to the embryo could easily explain the reduced germination of infected seeds (Castor 1977; Mathur et al. 1975). This damage also can occur without producing visibly molded kernels.

Finally, certain fungi can produce metabolites (mycotoxins) that are toxic or debilitating when fed to animals or humans. *F. moniliforme* and *F. semitectum* are known to produce zearalenone or its derivatives (Schroeder and Hein 1975; Stipanovic and Schroeder 1975). Fusarial mycotoxins, zearalenone and T-2, have been identified from molded sorghum in India and Texas (Rukmini and Bhat 1978; Schroeder and Hein 1975).

Measurement of Damage

Damage from grain mold fungi may take several forms and be measured in several ways. Molded and/or discolored kernels represent one of the most obvious and easily measured signs of damage by these fungi. In addition, molded grain usually has a reduced test weight when compared with nonmolded grain. Other types of damage may be determined by measuring such factors as germination, test weight, 100-kernel weight, preharvest sprouting, kernel hardness, conductivity of seed leachates, digestible nutrients, etc. Many of these are related; for example, test weight, 100-kernel weight, and kernel hardness would reflect the degree of fungal colonization (and digestion) of endosperm and germ tissues. Since most of these tests measure characteristics that depend to a large extent on variety, the true effect of grain mold fungi can be determined only by comparing molded or fungal inoculated grains with nonmolded or noninoculated grains. This is especially important when attempts are made to identify lines resistant to grain mold fungi.

Field ratings are universally used to identify resistant sorghum lines, although the exact rating scale may vary among researchers. Table 1 lists field and threshed-seed ratings of natural, noninoculated, and inoculated heads for 14 grain sorghum lines. The lines are ranked in order of increasing field ratings. The same order of the lines is maintained in subsequent tables for ease in comparison. The reactions of the lines range from resistant (SC-0103) to susceptible (Tx-2536), based on field ratings. When

Table 1. Comparison of 1977 grain mold field and threshed-seed ratings among 14 grain sorghum lines.[a]

Sorghum line	IS Number[b]	Field Rating[c]	Threshed-seed Rating[c]		
			N[d]	C[d]	F[d]
SC-0103	2403	1.0	2.0	2.8	2.2
SC-0719	7013	1.1	1.5	2.5	4.5
SC-0630	1269	1.5	2.7	2.3	2.5
SC-0748	3552	1.5	2.3	3.3	2.3
SC-0566	7254C	1.6	2.0	2.2	2.0
SC-0279	7419C	1.8	2.0	2.5	2.3
74 PR-759	–	1.9	1.4	1.7	4.5
SC-0097	12602C	2.0	2.5	3.0	4.5
CS-3541	–	2.1	1.5	1.5	1.7
SC-0599	17459	2.4	2.3	3.3	3.0
B-2219	–	2.5	1.7	1.7	2.2
BTx-398	412	2.9	2.3	3.5	2.3
TAM-428	12610	3.0	2.5	3.0	2.8
Tx-2536	10542	3.6	2.7	4.0	5.0

a. Heads inoculated at flowering; bagged for 1 week
b. Most entries are partially converted sorghums; those IS items followed by a "C" are fully converted
c. Rating on 1–5 scale where 1 = clean seed with no discoloration and no mold; 5 = molded seed
d. Treatments are N noninoculated, bagged control; C inoculation with a mixture of isolates from three *Curvularia* spp; F inoculation with a mixture of isolates from two *Fusarium* spp.

looking at the threshed-seed ratings, two things become apparent. First, field and threshed-seed ratings are not necessarily related. Three lines (SC-0719, 74 PR-759, and SC-0097) appear resistant, based on field ratings, but ratings on threshed seeds from *Fusarium*-inoculated heads show all three lines to be very susceptible. Field ratings on natural or *Fusarium*-inoculated heads would have permitted these lines to escape detection, since mold growth on kernels was predominantly hidden by the glumes and became visible only after threshing. Second, resistance to *Fusarium* spp is not related to resistance to *Curvularia* spp. Three lines (SC-0103, SC-0748, and BTx-398) appear resistant to *Fusarium*, based on threshed-seed ratings, but moderately susceptible to *Curvularia*. CS-3541, B-2219, and SC-0566 would be identified as the best or most resistant lines.

Test weight and germination are two commonly used means of measuring grain mold damage. Since 100-kernel weight relates reasonably well to test weight and is less variable, only kernel-wt data are considered. The percent reduction in 100-kernel weight and germination by *Curvularia* and *Fusarium* spp, compared with a noninoculated control, are presented in Table 2. In general, kernel weight and germination are reduced more by *Fusarium* than by *Curvularia* spp. Based on kernel weight and germination data, SC-0719, 74 PR-759, and SC-0097 again appear very susceptible, confirming the evaluation of threshed-seed ratings. SC-0103 and SC-0748 again appear more susceptible to *Curvularia* spp than to *Fusarium* spp, based on germination data. However, BTx-398 had little reduction in germination with either *Curvularia* or *Fusarium*, indicating that the discoloration/mold on the kernels was superficial; very little internal damage occurred. Two lines, SC-0103 and BTx-398, had little reduction in germination, but sizeable reductions in kernel weight. Apparently, reduction in kernel weight need not be related to a reduction in germination or to visibly molded or discolored kernels. Fungal

Table 2. Comparison of the percent reduction in 100-kernel weight and germination by *Curvularia* (C) and *Fusarium* (F) species among 14 grain sorghum lines.

Sorghum line	Percent reduction[a]			
	100-kernel weight		Germination	
	C	F	C	F
SC-0103	12	16	7	1
SC-0719	4	16	6	36
SC-0630	0	0	6	2
SC-0748	1	13	13	6
SC-0566	2	3	12	13
SC-0279	0	3	15	4
74-PR-759	12	23	24	47
SC-0097	16	25	16	45
CS-3541	9	5	18	17
SC-0599	5	1	4	16
B-2219	5	13	2	10
BTx-398	11	11	2	1
TAM-428	0	15	4	34
Tx-2536	1	25	60	92

a. Percent reduction = $\frac{100\,(N-C)}{N}$ and $\frac{100\,(N-F)}{N}$

Table 3. Percentage preharvest sprouting and *Fusarium* sprouting index for 14 grain sorghum lines in 1977.

Sorghum line	Preharvest sprouting[a]			*Fusarium* sprouting index[c]
	N[b]	C[b]	F[b]	
SC-0103	.9	.9	1.8	1.8
SC-0719	.3	2.0	10.3	3 429.9
SC-0630	0	0	0	0
SC-0748	0	0	.5	0
SC-0566	1.2	4.7	5.5	82.5
SC-0279	.4	.8	1.3	2.6
74-PR-759	.9	7.3	28.3	23 602.2
SC-0097	2.3	2.0	12.7	596.9
CS-3541	.5	1.3	1.2	1.2
SC-0599	.4	.3	4.9	250.0
B-2219	.3	1.7	8.3	1 356.0
BTx-398	.5	.2	2.7	27.0
TAM-428	.1	1.3	1.3	18.2
Tx-2536	2.4	4.3	15.9	1 228.4

a. Measured by observing ruptured pericarp above germ

b. Treatments are: N noninoculated control; C inoculation with three *Curvularia* spp; F inoculation with two *Fusarium* spp.

c. Sprouting index = $\frac{F\,(N-F)^2}{N}$

treated heads often had noticeably smaller kernels when compared with noninoculated controls. The three best lines based on threshed-seed ratings appeared relatively susceptible; CS-3541 and SC-0566 had reduced germination, while B-2219 had reduced kernel weight. Only one line, SC-0630, had little or no reduction in kernel weight and germination.

Preharvest sprouting can occur under conditions of excessive moisture from the time sorghum kernels approach physiological maturity until harvest. Sprouting can be measured in the field as the shoots become visible, or in harvested seed by observing the ruptured pericarp above the germ — often with an elongated epicotyl or hypocotyl present. Preharvest sprouting for this study was measured by the latter method. One point emerging from these studies is that, with certain varieties, *Fusarium* spp increase preharvest sprouting when inoculated into heads at flowering (Tables 3, 4, 5).

Seitz et al. (1975) measured sprouted kernels, incidence of *Fusarium*, and germination of samples of weathered sorghum in Kansas. Although not discussed in their paper, their data (Table 3, p. 1262) show an inverse relationship between germination and incidence of *Fusarium*. In addition, the incidence of *Fusarium* was positively related to the amount of sprouting. Since *Curvularia* spp have little or no effect on preharvest sprouting, a "*Fusarium* sprouting index" was devised to measure the relative resistance of lines to *Fusarium*-induced sprouting. A large sprouting index denotes a susceptible line, and is obtained when there is a large difference in sprouting between *Fusarium* and noninoculated treatments. SC-0719, 74 PR-759, B-2219, and Tx-2536 were among the lines more susceptible to *Fusarium*-induced sprouting under the conditions of this study. SC-0630, SC-0748, CS-3541, and SC-0103 were the lines more resistant to *Fusarium*-induced sprouting. *Fusarium*-induced preharvest sprouting was negatively correlated with germination, but not with any of the other variables

Table 4. Comparison of average values among treatments applied to eight grain sorghum lines in 1976.

	Treatments[a]				
	NAT	N	NW	C	F
Field rating[b]	2.0	1.7	1.7	1.9	1.8
Moisture[c] (%)	18.5	16.5	16.4	15.2	13.0
Yield[d] (g)	604.0	448.0	497.0	422.0	376.0
100-kernel wt (g)	3.02	2.76	2.89	2.77	2.55
Germination[e] (%)	92.0	97.0	95.0	94.0	77.0
Sprouting (%)	1.0	6.0	6.0	8.0	16.0
Curvularia (%)	0	8.0	11.0	80.0	0
Fusarium (%)	4.0	28.0	29.0	36.0	100.0

a. Heads inoculated at flowering and bagged until harvest. Treatments are: NAT noninoculated, nonbagged control; N noninoculated, bagged control; NW water-sprayed, bagged control; C inoculated with *Curvularia* spp, bagged; F inoculated with *Fusarium* spp, bagged.
b. Ratings on 1–5 scale where 1 = clean seeds; 5 = molded seeds.
c. Dry-weight basis.
d. 10 heads/treatment/line.
e. Rolled-towel method.

Table 5. Comparison of seed rating, kernel weight, germination, and preharvest sprouting among treatments applied to 14 grain sorghum lines in 1977.

	Treatments[a]			
	NAT	N	C	F
Threshed-seed rating[b]	2.4	2.1	2.7	3.0
100-kernel wt (g)	2.62	2.68	2.53	2.34
Germination[c] (%)	84.0	90.0	79.0	71.0
Sprouting[d] (%)	1.1	0.7	2.0	6.6

a. Heads inoculated at flowering; bagged for 1 week. Treatments are: NAT noninoculated, nonbagged; N noninoculated; C inoculated with three *Curvularia* spp; F inoculated with two *Fusarium* spp.
b. Ratings on 1–5 scale, where 1 = clean seeds; 5 = mold seeds.
c. Rolled-towel method.

measured. However, a reduction in germination could occur independently of sprouting.

Identification of Resistant Lines

Two major points emerge from the preceding studies. First, lines resistant to *Fusarium* are not necessarily resistant to *Curvularia*. Second, lines resistant to one type of damage (and one mode of fungal attack) are not necessarily resistant to other types of damage (and possibly other modes of attack). Since particular tests (field rating, threshed-seed rating, germination, etc.) measure particular types of damage, no one test has yet been found that can reliably identify sorghum lines resistant to grain mold fungi. In fact, reductions in kernel weight or kernel size could easily be overlooked or attributed to other causes, unless a specific effort was made to compare fungus-inoculated heads with controls. Several tests would offer a much better chance of finding lines with resistance. In an effort to integrate the results of several tests, lines were ranked from least-damaged to most-damaged for each of eight tests. Rank values for each line were summed to obtain an overall acceptability rating (Table 6). SC-0630, SC-0297, and SC-0566 are the three lines most resistant to *Curvularia* and *Fusarium* spp. Actually, SC-0630 may show a slight reduction in germination with *Curvularia*. SC-0279 may show a sizeable reduction in germination with *Curvularia*. SC-0566 may show reduced germination with both *Curvularia* and *Fusarium* and a slight tendency to sprout. Interestingly, some of

Table 6. Relative acceptability[a] of 14 grain sorghum lines for resistance to *Fusarium* and *Curvularia* grain mold fungi, based on rankings for eight criteria.

Sorghum line	Field rating	Seed rating		100-kernel weight		Germination		Sprouting index	Acceptability value
		C	F	C	F	C	F		
SC-0630	3	5	8	1	1	5	3	1	27
SC-0279	6	6	5	1	3	10	4	5	40
SC-0566	5	4	2	6	3	8	7	8	43
SC-0748	3	11	5	4	7	9	5	1	45
SC-0103	1	8	3	12	10	7	1	4	46
B-2219	11	2	3	8	7	1	6	12	50
CS-3541	9	1	1	10	5	12	9	3	50
BTx-398	12	13	5	11	6	1	1	7	56
TAM-428	13	9	9	1	9	3	10	6	60
SC-0599	10	11	10	8	2	3	8	9	61
SC-0719	2	6	11	7	10	5	11	14	66
74-PR-759	7	2	11	12	12	13	13	13	83
SC-0097	8	9	11	14	13	11	12	10	88
Tx-2536	14	14	14	4	13	14	14	11	98

a. Determined by ranking lines from least-damaged to most-damaged, and summing rank values for each line. Minimum score = 8; maximum score = 112.

the same lines — SC-0630, SC-0279, SC-0566, SC-0748, and BTx-398 — have been identified as being relatively resistant to field deterioration (weathering). Glueck and Rooney (1976), and Glueck (1979) have measured various physical characteristics of the grain from these lines in relation to their resistance. Rooney summarizes their findings in this session (see "Chemistry and structure of grain in relation to grain mold resistance").

Importance of Fungal Species

Results from studies comparing *Curvularia* and *Fusarium* spp are summarized in Tables 4 and 5. *Fusarium* spp are generally more damaging than *Curvularia* spp, although particular lines (SC-0103 and SC-0748) may be damaged more by *Curvularia* spp. *Fusarium* spp reduce yield, moisture content, 100-kernel weight, and germination more than *Curvularia* spp, when compared with noninoculated controls. The reduced moisture content at harvest may indicate that *Fusarium* can cause premature senescence of the kernels. Maturity earlier than normal could easily explain the reductions in kernel weight, size, and yield with the *Fusarium* treatments.

A comparison was made between the two *Fusarium* spp to determine their relative importance (Table 7). *F. moniliforme* was found to be much more damaging than *F. semitectum; F. moniliforme* reduced yield, 100-kernel weight, and germination to a much greater extent than *F. semitectum* compared with the noninoculated controls. *F. moniliforme* appears to be responsible for the increased preharvest sprouting. Inoculations with a mixture of isolates of both *Fusarium* species resulted in less reduction in yield and 100-kernel weight than when *F. moniliforme* alone was inoculated. This indicates that single-species inoculation would give a more reliable measure of damage. This is supported by data from experiments conducted at ICRISAT (Castor et al. 1978). Mixtures of *Fusarium* spp or mixtures of *Fusarium* and *Curvularia* spp produced less damage than inoculation with single species. Additionally, *C. lunata* and *F. moniliforme* both reduced moisture content at harvest under ICRISAT Center conditions; *F. semitectum* did not reduce moisture content.

F. moniliforme is generally more damaging

Table 7. Comparison of four characteristics among five treatments applied to three grain sorghum lines in 1977.

	Treatments[a]				
	NAT	N	F	FS	FM
Yield[b] (g)	385	344	284	291	269
100-kernel wt (g)	2.30	2.39	2.31	2.35	2.23
Germination[c] (%)	81	92	76	85	78
Sprouting[d] (%)	1.6	2.0	9.1	3.6	6.9

a. Inoculated at flowering; bagged for 1 week. Treatments include: NAT noninoculated, nonbagged control; N noninoculated control; F inoculation with two *Fusarium* spp; FS inoculation with *F. semitectum* isolates; and FM inoculation with *F. moniliforme* isolates.
b. Based on 10 heads/treatment/variety
c. Rolled-towel method
d. Based on split pericarp above germ

than *Curvularia* spp (primarily *C. lunata*) under Texas conditions. This is in contrast to experiments at ICRISAT where *C. lunata* was found to be more damaging than *F. moniliforme*. *F. semitectum* was relatively unimportant in Texas or at ICRISAT.

Infection

Field experiments in Texas and at ICRISAT Center have shown that the greatest damage, based on reduction in germination, occurs when sorghum heads are inoculated at anthesis or within 2 to 3 days of anthesis (Castor 1977; Rao and Williams 1977). This indicates that floral tissues (glumes, stigmas, styles, etc.) are most susceptible at flowering and become less susceptible thereafter. Additional support for early infection comes from information about the types of damage caused by grain mold fungi. Reduction in kernel weight or size would suggest an early and prolonged exposure to the pathogen's influence.

A comparison of seeds from heads inoculated with *F. moniliforme*, *F. semitectum*, and *Curvularia* has shown that *F. semitectum* produces minimal discoloration of kernels and no degradation of endosperm and germ tissues. *Curvularia* damage, a partial degradation of the endosperm, appeared to progress slowly inward from the pericarp. In contrast, *F. moniliforme* appeared to initially colonize the region where the hilum, scutellum, and endosperm join. Progressive colonization of the scutellum and endosperm followed. The embryo appeared to be the last tissue to be damaged. This observation is supported by data from experiments where embryos were excised. Seeds from inoculated susceptible lines exhibited very low germination (27%), even though embryo viability was high (68%) (Castor 1977). Glueck states that the germ appears to be the primary and initial area of preharvest grain deterioration, although no distinction is made between weather- or fungal-caused deterioration (Glueck 1979). Additionally, fungal isolations from glumes, seeds without embryos, and embryos have shown that embryos contain less fungi than any other tissue (Castor 1979; Mathur et al. 1975). The location of mycelial (mold) growth on kernels would support these observations. *F. semitectum* produced little or no mold. *Curvularia* spp first produced mold on portions of the seed not covered by the glumes. *F. moniliforme* mold was found initially beneath the glumes, around the germ and hilum. Eventually, mold of *Curvularia* sp and *F. moniliforme* could cover the entire kernel surface. There is some evidence suggesting that *F. moniliforme* mold growth over the kernel surface occurs only after complete colonization (and degradation) of the endosperm tissues. How infection, kernel development, and damage are related, based on current evidence for *F. moniliforme*, is shown in Fig. 1.

The preceding discussion is based on general observations. The avenue of entrance of a

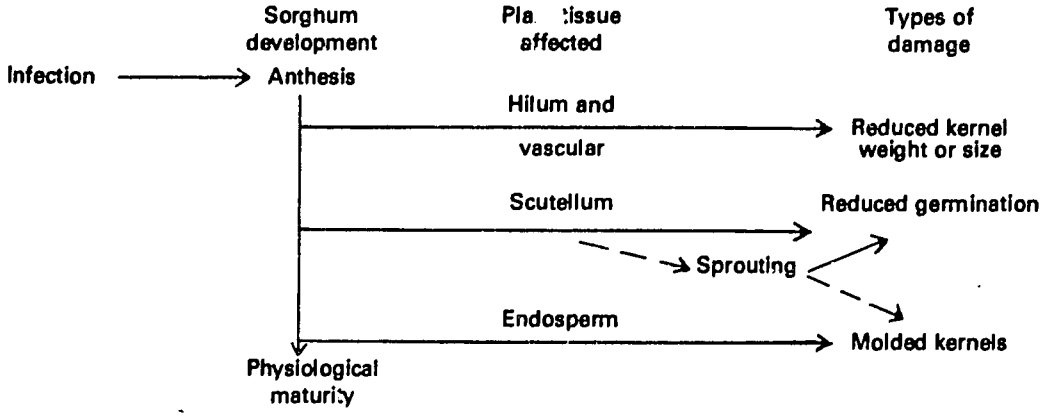

Figure 1. Schematic diagram showing the relation between infection and physiologic maturity of sorghum by common grain-molding fungi.

particular fungal species and the degree of damage could and probably does vary among sorghum lines. Specific studies are now in progress to show where pathogens (*F. moniliforme* and *C. lunata*) enter the kernels and when this occurs.

Screening Program

The screening of sorghum lines for resistance to grain mold fungi is complicated by several factors, including the large number of causal fungi, the different types of damage (probably indicating different modes of fungal attack), and the large number of lines. The following screening system is proposed as one method to identify lines with the best resistance to grain mold fungi:

Stage 1

Lines are grown at several locations known to be favorable for grain mold development. Susceptible lines are discarded, based on field ratings. (As a first step in a screening program, field ratings are acceptable, despite their shortcomings. Susceptible lines may escape detection due to differences in maturity among lines, or to variability in weather. Mold may be hidden by glumes. Lines with reduced kernel weight or size and sprouting may escape detection.)

Stage 2

Lines selected during Stage 1 are grown at one location and inoculated at flowering with a mixture of fungi. Noninoculated controls for each line are grown. Susceptible lines are discarded, based on field ratings. "Resistant" lines, including inoculated and noninoculated heads, are harvested. Threshed-seed measurements are taken and susceptible lines are discarded. Depending on the number of lines selected in Stage 1, this stage could be skipped entirely.

Stage 3

Lines selected in Stage 2 are grown at one location and inoculated with *individual species*. Noninoculated controls are grown with each line. Based on field and threshed-seed ratings, susceptible lines are discarded. Additional tests, such as 100-kernel weight, germination, sprouting, etc., are conducted to determine the levels of resistance of the remaining lines. At this point, selected lines could be crossed among themselves or to high-yielding agronomic varieties in an effort to increase the level of resistance.

(Artificial inoculation of early generation breeding progenies may be feasible as a way of identifying resistant sorghum lines. Data from Texas indicate that inoculation of

F_2 plants followed by selection of individual heads can be an effective means of rapidly developing grain mold resistant lines.)

Stage 4

Elite lines from Stage 3 are tested for other characters such as nutritional quality, protein content, and protein quality, as well as for reactions to other field fungi (*Phoma* and *Alternaria*) and to storage fungi (*Penicillium* and *Aspergillus*).

This program is comprehensive and theoretical; no other sorghum improvement program is as thorough, so far as is known. Most programs generally rely on Stage 1 or Stage 2 screening. Lines developed by such programs are relatively resistant to molding caused by the fungi, but may very well be susceptible to a reduction in kernel weight or size or to *F. moniliforme*-induced sprouting. As the more immediate and obvious problem of molding is overcome, an attempt to ensure that lines are resistant to the other types of damage must be made as well.

Future Research

Future research should include several important areas. *Phoma* grain mold may become an increasing problem as sorghum lines with greater levels of resistance to *Fusarium* and *Curvularia* spp are developed. Observations suggest that the presence of other fungi inhibits colonization of kernels by *Phoma*, which appears to colonize kernels superficially without doing much damage to the endosperm. However, the grain is discolored and appears to be of poor quality. In addition, *Phoma sorghina* has been reported to produce a mycotoxin (Boerema et al. 1977).

Generally, storage fungi such as *Penicillium* and *Aspergillus* spp occur in very low frequency in sorghum kernels. The presence of field fungi may also inhibit the development of storage fungi (Pettit and Taber 1978; Seitz et al. 1975). Unfortunately, susceptibility of lines to storage fungi or *Phoma* may be detected only after resistance to *Curvularia* and *Fusarium* spp has been developed.

Alternaria spp were considered unimportant in Texas, because they did not mold kernels or reduce germination. What about reduction in kernel weight or size? Grain mold resistant lines should be tested for their reaction to some of the less common grain fungi (*Alternaria* spp, *Helminthosporium* spp, etc.).

Attention should be given to nutritional quality. Is grain that is less "digestible" by fungi also less nutritious when used as a feed or food? This is perhaps not as improbable as it may seem. Sorghum lines having a testa are more resistant to grain mold fungi (and to birds). However, the testa contains relatively large amounts of polyphenolic compounds (tannins), which can complex with grain proteins, making them less available for digestion. Such grain is less nutritious.

The relationship between physical characteristics of grain (conductivity of seed leachate, rate of water absorption, etc.) and resistance to specific grain mold fungi needs to be worked out. Is resistance due to grain structure, to grain physiology, or to a combination of both? Grain physical characteristics may be more important during the postmaturity period, when grain deterioration due to weather and fungi, both saprophytic and pathogenic, occur.

Research on *Fusarium*-produced mycotoxins might seem less important today, since mold-resistant lines are being identified. However, many of these relatively resistant lines do show limited mold development, especially under environmental conditions favorable to the pathogen. Just how this limited fungal growth relates to the presence of mycotoxins and their concentration is not known. In this regard, the determination of the relationship among fungal growth (colonization), visible mold, and mycotoxin concentration would be of major value to researchers and consumers.

Much progress in the understanding of *Curvularia* and *Fusarium* grain molds and in the identification of mold-resistant sorghum lines has been made during the past few years. Mold-resistant agronomically acceptable varieties are currently being developed in many areas of the world. Much additional research needs to be carried out to determine how good these grain mold resistant varieties actually are, and how acceptable they will be to farmers.

Acknowledgment

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Screening for Sorghum Grain Mold Resistance at ICRISAT

K. N. Rao and R. J. Williams*

Native landrace sorghums are generally of long duration and are relatively low yielders. High-yielding sorghums are being developed using exotic germplasm and daylength-neutral sorghum-conversion lines. Improved varieties flower and mature early when soil moisture levels are adequate for grain filling. When rains persist beyond flowering and maturity, these improved varieties may develop molds on the grain, reducing its quality and quantity. The problem of grain molds is widespread and is considered one of the top-priority problems in the ICRISAT Sorghum Improvement Program. Systematic work on grain molds at ICRISAT Center began in 1974. Our priorities were sharpened with the information provided in the 1977 International Sorghum Workshop.

Objectives

The objectives of the ICRISAT sorghum grain mold projects are:

- to elucidate the epidemiology and biology of the disease complex;
- to develop effective large-scale field and laboratory screening techniques;
- to screen large numbers of germplasm and breeding materials for identification of sources of resistance; and
- to utilize resistance sources in developing mold-resistant cultivars with good agronomic traits, in a cooperative program with sorghum breeders.

Research activities aimed at the first three objectives are briefly discussed in this paper. Research activities on the fourth objective will be presented later in this session by our colleague, plant breeder Dr. D. S. Murty.

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Symptoms

Initial mold symptoms appear as white or grey mycelial growth on rachis, glumes, and anthers. The grains become discolored and at physiological maturity, discolorations are observed — black for *Curvularia* sp; pink for *Fusarium* sp; snow white for *Olpitrichum* sp; and grey for *Alternaria* or *Drechslera* sp. The fruiting bodies of *Phoma* sp and *Colletotrichum* sp appear as small raised black dots. Severely molded grains are generally lighter than clean grains and disintegrate when pressed between thumb and forefinger. Alternate wetting and drying coupled with infection by mold fungi cause grain deterioration. It is difficult to differentiate physical and physiological grain deterioration from deterioration caused directly by fungi.

Associated Microflora

Seventeen fungal species in 11 genera were isolated from field-collected molded sorghum grain (with assistance in identification from the Commonwealth Mycological Institute, England). The fungi isolated were: *Alternaria triticina*, *Cladosporium tenniussimum*, *Cochliobolus spicifer*, *Colletotrichum* sp, *Curvularia lunata*, *Curvularia verruculosa*, *Drechslera halodes*, *Drechslera* sp, *Fusarium semitectum*, *F. fusarioides*, *F. moniliforme*, *F. acuminatum*, *F. lateritium*, *Olpitrichum* sp, *Penicillium oxalicum*, *Phoma sorghina*, *Trichothecium roseum*.

The most frequently isolated genera were *Fusarium*, *Curvularia*, *Phoma*, and *Trichothecium*.

Effect of Mold on Seed Viability

Molded grain and apparently clean grain of the same cultivar were incubated on moist blotters, and observations on viability were recorded.

Molded grain of CSV-3 was subdivided on the basis of color and each color was again subdivided into two lots — surface-sterilized with 0.1% mercuric chloride solution for 1.5 min, and nonsurface-sterilized. Germination values after 7 days in moist chambers were 0.8% for pink nonsterilized, 0.8% for pink sterilized, 4.1% for black nonsterilized, and 9.2% for black sterilized. Apparently clean grain of the same variety from postrainy season harvest gave up to 100% germination. In similar studies with other cultivars, using 400 seeds in each case, loss in viability of molded grain up to 99% was observed in molded grain.

Screening Techniques

Host-plant resistance is the only control method for sorghum grain molds that will be viable economically and technically on peasant farms.

Grain mold resistance screening has been carried out in various programs utilizing natural mold development, when the materials were exposed to rains during flowering and maturity. There is a possibility that lines selected under such natural screening are not truly resistant; they could be escapes. To minimize escapes, there was a need to develop an effective screening method. The method developed at ICRISAT Center for field and laboratory screenings is briefly described here.

Field Screening

Sorghum earheads are covered with brown paper bags at emergence from the boot. Pathogenic isolates of *Fusarium moniliforme*, *F. semitectum*, and *Curvularia lunata* are grown on autoclaved sorghum grain for one week. Aqueous mycelial/conidial suspensions (2×10^3 conidia/ml) are prepared and sprayed onto the sorghum heads 7 days after bagging, and bags are replaced for another 14 days, after which they are removed. Scoring for mold development is carried out on inoculated and on open panicles 45 days after inoculation. On rain-free days, sprinkler irrigation is provided for half an hour in the evenings to maintain high humidity.

Laboratory Screening

Field screening allows only one screening in a year, and its success depends on the weather during the critical flowering period. In an attempt to make screening independent of season and local weather conditions, we experimented with laboratory screening procedures. At the present stage of development, the laboratory screening procedure is divided into two phases with progressively more severe pressure on the test materials.

Preliminary Screening

Forty grams grain per entry is taken from postrainy-season harvested (7 to 10 days after physiological maturity) bulk samples. Grains are soaked for 2 hr in tap water in 2-liter plastic buckets. Disposable plastic petri plate moist chambers are prepared containing blotting paper and cotton pads moistened with 10 ml of 0.2% 2,4-D solution. The lids of the petri plate are free from blotting paper and cotton pads to allow light to penetrate to the grain. The soaked washed grain is spread over the blotting paper in a single layer, and the petri plates are incubated at 25°C with 12-hr alternate light and dark regimes. Each entry is replicated four times and randomized to minimize position effects within the incubators. Three infection parameters are measured:

Percent Grain Infected

Simple calculations of percent grain infected are made on 400 randomly selected seeds (100 seeds from each of four petri plates).

Actual Severity

Individual grains are rated on a 1 to 5 severity scale (1 = no mold; 5 = severe mold) and the average of 400 grains is calculated.

Visual Scoring

Visual rating on a 1 to 5 scale, based on overall moldiness of the grain, is performed for individual entries.

Advanced Screening
(For reactions to *Curvularia* only)

Selected entries from the preliminary screening are given a more severe test by inoculating their

grain with an aqueous suspension of *Curvularia lunata* conidia (10×10^4 conidia/ml). A 40-g sample of seed is dip-inoculated after soaking and washing in tap water. Petri plates are incubated for 4 days and observations recorded as described in preliminary screening.

Screening at ICRISAT

Field Screening

A summary of the field-screening activities at ICRISAT Center from 1974 to 1978 is presented in Table 1. Initially, breeding materials were screened for mold resistance under natural mold infections. Most of the materials designated less susceptible were either brown or dark brown pericarp types. Single-head harvests were taken for further head-to-row progeny tests. In 1974, the field-screening technique was developed and it was used on a large scale in 1975 and 1976. Various groups of materials screened are briefly discussed below.

Germplasm

A total of 4036 germplasm lines with white, yellow, dull yellow, and light red grains were screened during rainy season 1975. Rainfall was frequent and heavy during grain filling and

Table 1. Summary of the grain mold resistance-screening activities at ICRISAT Center, 1974 to 1978.

Season, and material screened	Entries screened (no)	Entries selected (no)	Entries selected (%)
Rainy season 1974			
1. Sorghum breeding material	2617	27	1.0
2. Early maturing lines	280	26	9.3
3. Mold resistant nursery & zera zera lines	62	14	22.6
Rainy season 1975			
1. Sorghum germplasm	4036	93	2.3
2. Selected lines from 1974 tests	67	10	14.9
Rainy season 1976			
1. Sorghum germplasm	1421	12	0.8
2. Elite lines from 1975 tests	103	31	30.1
3. Advanced populations	2612	10	0.4
4. Grain grass material	1446	5	0.4
5. Population x variety crosses	234	4	1.7
6. JP-26 material	65	15	23.1
Rainy season 1977			
1. Mold resistance breeding material			
a) First sowing	3004		
b) Second sowing	4740	564	7.3
2. ISGMN 1977	35	7	20.0
3. Advanced populations	55	–	–
4. Grain grass material	636	–	–
Rainy season 1978			
1. ISGMN 1978	30	14	46.7
2. SEPON 1978	48	6	12.5
3. Less-susceptible lines from laboratory screening	82	14	17.1
4. International nurseries other than ISGMN-1978	109	5	4.6
5. Elite selections from mold resistance-breeding project	111	9	8.1
6. Single-head selections from rainy season 1977	564	112	19.7
7. Mold resistance-breeding progenies (F_4 and F_3)	2096	223	10.6

maturation, resulting in heavy mold development, both on inoculated and noninoculated panicles. Entries were scored on a 1 to 9 scale (1 = no mold; 9 = completely molded) and 103 entries were selected with a rating of ≤3. These entries were increased in the postrainy season 1975–1976 for further testing. During rainy season 1976, an additional 1441 sorghum germplasm lines were screened in the field. Of 1544 entries, only 43 were rated as low susceptible, and these were further screened during the 1977 and 1978 rainy seasons. Relatively less-susceptible entries were tested on a wide scale in a cooperative multilocation testing program — the International Sorghum Grain Mold Nursery (ISGMN). Results obtained in the period 1976 to 1978 are presented in a paper later in the session. Entries consistently low in susceptibility in all seasons were E 35-1, IS-9225, IS-2328, IS-2327, IS-2261, IS-2435, and IS-14332. These low-susceptible sources were supplied to the ICRISAT sorghum breeding group for utilization in the grain mold resistance breeding program.

Breeding Progenies

Several groups of materials from advanced populations, grain-grass materials, and population × variety crosses were screened in the 1976 rainy season. Most of the material planted was highly susceptible to grain molds with less than 2% of the entries rated as low susceptible.

Field screening of selected material from the mold resistance breeding project, involving crosses with adapted parents and resistant sources identified in the germplasm, was first done in the 1977 rainy season. A significant feature of that year's screening was the use of an early season planting (middle of April) so that flowering and grain filling would coincide with the time (1 to 15 July) when rain was most probable at ICRISAT Center. The normal main-season screening was also made. Severe molds developed in both early- and main-season plantings. In a collaborative effort with the sorghum breeding staff, selections were made for mold resistance and desirable agronomic traits. No line was uniformly free from molds, so 564 single-heads were selected, and the seed from these increased in postrainy season 1977.

Selected entries of breeding progenies from rainy season 1977, and new breeding lines (F_3 and F_4) were screened in rainy season 1978 under high inoculum pressure in the field. Many of the retested selections were relatively free from *Fusarium* and *Curvularia*. However, infection with *Phoma* sp was more on this clean background. Future efforts must include the identification and utilization of *Phoma*-resistant sources. From among the new material screened, only about 10% of the lines were low in susceptibility to molds. This year again, 254 single heads — representing different families involved in crossing — were harvested. Breeding lines M-35052, M-36284, M-36091, M-36008, M-36023, M-36040, and M-36088 showed high levels of grain mold resistance in addition to a good agronomic background. Screening large numbers of materials under high inoculum pressure and practicing intensive selection on an individual plant basis appear to be the key factors for success in dealing with the complex problem of sorghum grain molds.

JP-26 Mold-Resistant Lines

Lines selected under natural mold development at Samaru, Nigeria, in the JP-26 project were tested at ICRISAT Center in 1976. Of the 65 lines tested, two with dark seeds (A-2616 and A-2626) were mold-free, and 13 lines had only slight mold. During screening of the less susceptible entries in subsequent years, only one entry (JP-2579) was selected for wide-scale testing.

Laboratory Screening

Head-to-row progenies of selections made in the 1977 rainy season were increased in the following postrainy season and the increased seed was screened in the laboratory during summer 1978. In the laboratory test not one of the 659 entries tested with 4 days incubation was immune to grain mold infection, whereas 77 entries were rated as 2 by visual scoring. After 3 more days incubation — i.e., 7 days total incubation — 14 lines were rated as 2 (Table 2).

Correlations Between Three Grain Mold Infection Parameters Measured in the Laboratory

Rank correlations were made for the entries based on the three grain mold infection parameters (Table 3). Maximum correlation

Table 2. Grain mold-reaction of selected entries in laboratory screening.

Days incubated	Temp.	No. of entries in reaction category 1	2	3	4	5	Total
4	25°C	0	70	0	0	0	70
7	25°C	0	14	55	1	0	70

Table 3. Rank correlations between three grain mold-infection parameters in the laboratory screening for grain mold resistance.

Percent grain infected	1	
Average severity	0.72	1
Visual scoring	0.62	0.67

was obtained between percent grain infected and average severity. Visual scoring was correlated 67 and 62% with average severity and percent grain infected, respectively. Visual scoring can be used for large-scale screening, and all three parameters can be recorded for intensive screening of a small number of lines.

Relationship Between Field and Laboratory Ratings of Grain Mold Incidence

Entries with a visual rating of 2 in the laboratory were planted in a replicated trial in rainy season 1978 to study the relationship between laboratory and field testing. Eighty-three percent of the lines selected as promising in the laboratory gave a rating of ≤ 3 in the field under high inoculum pressure (Table 4). Known high susceptibles in field screening gave consistently high grain mold ratings in the laboratory (Table 5). Some entries which were low susceptible, even after inoculation in the laboratory, have also performed well in the field screening.

The laboratory screening techniques may be used to eliminate large numbers of high susceptibles from the breeding progenies, to screen photosensitive sorghum germplasm material, and to provide additional information on other less-susceptible entries. The laboratory screening is complementary to the field screening and will allow more rapid progress in the selection of grain mold resistant progenies.

Table 4. Relationship between grain mold ratings in field and laboratory screenings.

Screening location	No. of entries in reaction category 1	2	3	4	5
Laboratory	–	82	–	–	–
Field (rainy season 1978)	–	16	52	14	–

Table 5. Field grain mold ratings of selected entries from laboratory screening.

Entry	Laboratory ratings: Without inoculation	With inoculation	Field ratings, rainy season 1978
M-36091	1.25	2.2	2.3
M-36294	1.5	2.2	2.2
M-36088	1.5	2.5	2.2
M-36040	2.0	3.0	2.4
M-36023	2.0	3.0	2.4
PP_2B	3.5	5.0	5.0
ms bulk	5.0	5.0	5.0

Summary

The sorghum grain mold problem, its importance, and symptom development are discussed. Seventeen fungal species belonging to 11 genera were isolated from molded sorghum grain. In some cases complete loss in viability occurred in molded sorghum grain. An effective field-screening technique was developed and used to screen several thousand sorghum germplasm and breeding progenies from 1975 to 1978. Consistently low-susceptible source material identified from germplasm lines was utilized in a cooperative program with sorghum breeders to develop mold-resistant cultivars. Careful selection of individual heads from the susceptible populations under high inoculum pressure, and subsequent genetic manipulation, have yielded promising lines which com-

bine resistance with desirable agronomic qualities. Considerable progress has been made in developing a laboratory-screening technique to differentiate cultivar susceptibility to grain molds.

The International Sorghum Grain Mold Nursery

R. J. Williams and K. N. Rao*

The International Sorghum Grain Mold Nursery (ISGMN) program was initiated by ICRISAT in 1976 with the following objectives:

- to identify sources of stable grain mold resistance;
- to obtain information on the variability of the grain mold causal agents;
- to distribute grain mold resistant genotypes to scientists in national programs; and
- to promote the development of a communicating cooperating international network of scientists working on sorghum grain molds.

Cooperators in 13 countries in Asia and Africa (Table 1) have participated in the ISGMN program. The basic requirement of cooperators is that they should be able to expose the ISGMN test entries to sufficient grain mold pressure to adequately test reactions of entries to grain molds.

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Table 1. Cooperators and locations in the International Sorghum Grain Mold Nursery Program from 1976 to 1978.

Cooperator	Location		Year(s)
J. C. Girard	Nioro, Senegal Sefa, Senegal		1976, 1977
S. A. Clarke and J. A. Frowd	Sotuba, Mali		1976, 1977, 1978
J. A. Frowd	Farako-Bâ, Upper Volta		1976, 1977, 1978
O. Sidibe	Tarna, Niger		1977, 1978
S. O. Okiror and J. C. Selvaraj	Kano, Nigeria		1976
N. V. Sundaram	Samaru, Nigeria		1977, 1978
Bhrane Gebrekidan, Yilma Kebede and Mengistu Hulluka	Alemaya and Arsi Negele	Ethiopia	1977, 1978
V. Van Arkel and M. F. Vis	Lanet and Busia	Kenya	1977, 1978
S. Z. Mukuru	Ilonga, Tanzania		1978
B. D. A. Beck	Shire Valley, Malawi		1978
K. N. Rao	ICRISAT, India		1976, 1977, 1978
K. V. L. N. Rao and G. K. Rao	Adilabad, India		1977, 1978
Aftab Ahmed and G. K. Rao	Warangal, India		1977, 1978
H. L. Chauhan	Navsari, India		1977, 1978
C. S. Sangit Rao	Akola, India		1977, 1978
T. B. Garud	Parbhani, India		1978
K. N. Rao and D. S. Murthy	Bhavanisagar, India		1978
S. M. Naik	Udaipur, India		1977, 1978
M. N. Prasad	Coimbatore, India		1978
S. F. Hassan	Yousafwalla, Pakistan		1977, 1978
C. Pannabokke	Sri Lanka		1978
S. Patanothai	Thailand		1977, 1978

Selection of ISGMN Test Entries

The entries included in the ISGMN trials of 1976 and 1977 were those identified as relatively less susceptible (RLS) to grain molds in the ICRISAT screening program. In 1978 promising progeny of crosses among the RLS lines, and between them and elite grain quality lines, were included as test entires. Each year two known highly susceptible lines are included to serve as indicators of grain mold pressure.

Operation of the ISGMN Program

Seed of test entries is assembled and multiplied at ICRISAT Center. All cooperators receive seed from the same seed lot for each entry. This is important in order to avoid erroneous information on pathogen variability.

A set of entries is sent to each cooperator with a book of data sheets that includes information on the objectives of the trial, suggestions for planting, fertilization, inoculum provision, time and method of scoring, and data record sheets for climatic and plant-reaction data.

Cooperators are requested to return one copy of the data sheets to ICRISAT as soon as possible after completion of the trial. Data from all cooperators and a discussion of the important aspects of the results are published as a report and distributed to all cooperators.

The 1976, 1977, and 1978 Results

1976

The rains were particularly heavy in West Africa during October and early November, and thus the entries in the West African locations were subjected to a severe test. In many parts of India, unfortunately, September and early October were extremely dry. This is the time that most of the sorghum was maturing, so tests at Indian locations in 1976 were not exposed to conditions conducive to mold development.

Entry E 35-1, a zera zera with bold white grain from Ethiopia, was the best across locations, and was selected for uniform trials in Mali. Robledo and Frowd (personal communication) list the following as most promising in Upper Volta, IS-2327, IS-2261, IS-2328, IS-3443, and E 35-1 based on head mold ratings, and IS-9225 and IS-2388 were the best, based on threshed-grain mold ratings.

The high degree of mold development in West African tests indicates the importance of high rainfall during grain maturation for identification of mold-resistant lines. The 1976 ISGMN provided a modest but useful start to the ISGMN program, and enabled us to identify some lines thought promising by West African cooperators.

1977

The 1977 ISGMN saw a major expansion in cooperators and locations, and three groups of entries were tested under the ISGMN program. For those locations where main-season planting is carried out during January through March, an Early-Season ISGMN (ES-ISGMN) consisting of 22 entries was provided. For the majority of the cooperative locations in India and West Africa, planting takes place during the June-July period and these locations were provided with a 30-entry Main-Season ISGMN (MS-ISGMN); 15 entries were common with the ES-ISGMN. The third group of materials included entries (not multiplied and distributed by ICRISAT) supplied directly to selected cooperators by other sorghum scientists.

Results of trials with these three groups are reported separately in order to minimize confusions in comparisons.

Early-Season ISGMN

Flowering and mold data for the 22 entries at five locations are presented in Tables 2 and 3. Only field ratings were taken in the trials at Alemaya and Arsi Negele, while at Busia, Lanet, and Khon Kaen, Thailand, three grain mold infection parameters were recorded (Table 3). At Busia, which provided the greatest pressure (location mean of 4.4), entries with the least field grain mold ratings were IS-9504 and IS-5246 (Table 2). Five entries (IS-2327, IS-2328, IS-9225, IS-9504, and IS-9331) averaged a field grain mold rating of 3 or less. In Table 4 the relative rank of entries is compared for each parameter. Six entries appeared in the best ten

Table 2. Field grain mold ratings[a] of 22 entries in the 1977 Early-Season ISGMN at five locations.

Entry	DTF[b]	Khon Kaen	Alemaya	Arsi Negele	Lanet	Busia	Mean
IS-2327	87	1	2	2.5	2	5	2.5
IS-2328	86	1.5	2	3	2.5	4	2.6
IS-9225	91	1.5	2	3	2.5	4	2.6
IS-9504	86	3	2	–	3	2.5	2.6
IS-9331	96	3	2	–	4	–	3.0
E-35-1	90	1.5	3	2.5	3.5	5	3.1
IS-3443	86	1	3	3	3.5	5	3.1
IS-5246	103	4.5	2	3	3.5	2.5	3.1
IS-1545	99	2.5	3	2	5	3	3.1
IS-9533	91	3	2.5	2.5	4	5	3.4
IS-2495	93	2	4	4	3	4.5	3.5
IS-9521	100	3.5	1	–	5	5	3.6
IS-9327	95	3	3	–	4.5	4	3.6
IS-1087	87	2.5	3	–	5	4	3.6
IS-2261	84	1.5	4	4	4	5	3.7
IS-9468	99	4	2	–	5	–	3.7
IS-9544	94	4	2	–	5	–	3.7
CS-3541	90	3.5	3	3	4.5	5	3.8
IS-179	89	3	4	4	3.5	5	3.9
IS-2583	88	2	4	4	5	5	4.0
SY × IS 511	78	3.5	5	5	5	5	4.7
PP2B × 11167	91	4.5	5	5	5	5	4.8
Location Mean		2.7	2.9	3.4	4.0	4.4	3.4

a. 1 to 5 scale, where 1 = absence of mold; 5 = severe mold
b. Days to flowering (mean across locations)

for all infection parameters. The correlation matrix for the ranks of these three parameters is:

Field rating rank	1	
Lab rating rank	0.605	1
Lab ranking rank	0.776	0.623

Main-Season ISGMN

Data for 30 entries at 12 locations are presented in Tables 5, 6 and 7. Field grain mold rating averages varied from 1.6 to 4.4, and eight entries (Large glume 7, IS-2261, IS-9225, IS-3443, IS-2328, IS-2327, E 35-1, and IS-2583) had ratings of 3 or less at all locations (Table 5). Nine entries had a mean field grain mold rating of 2 or less, but it should be pointed out that eight of these entries took more than 70 days to flower whereas of the nine poorest entries (average more than 3), eight flowered in 66 days or less (the correlation coefficient of mean days to flowering and mean field rating is 0.772, significant at 0.1%). Thus there seems to be an element of escape in the lower mold values of the later flowering entries. However, entry IS-14332 combined early flowering (68 days) with good performance (overall fifth, Table 5) and thus is probably the most valuable entry in the trial. Rank values for three infection parameters of the 30 entries are listed in Table 8; seven entries appear in the best ten for all three parameters (IS-2327, IS-2328, IS-2261, IS-14332, E 35-1, Large glume-7, and IS-2435). The correlation matrix for the rank values for the three parameters is:

Field mold rating	1		
Lab mold rating	0.846	1	
Lab ranking	0.815	0.960	1

Table 3. Threshed grain mold rating[a] and ranking[b] of 22 entries in the Early Season ISGMN.

Entry	Lab rating				Lab ranking			
	Khon Kaen	Busia	Lanet	Mean	Khon Kaen	Busia	Lanet	Mean
IS-9225	1	4	3	2.7	1	7	4	4
IS-2327	1	5	3.5	3.2	1	12	1	4.7
IS-2328	1	3	5	2.3	1	5	9	5
IS-5246	3.5	3	2	2.8	6	4	–	5
IS-9504	4.5	1.5	4	3.3	11	1	1	4.3
IS-1545	3.5	2	–	2.8	8	3	–	5.5
IS-9521	4.5	–	4.5	4.5	9	1	–	5
CS-3541	4.5	2	4.5	3.7	9	2	5	5.3
IS-2435	2.5	5	5	4.2	3	9	8	6.7
E-35-1	3	4	5	4	7	6	9	7.3
IS-2261	1.5	5	5	3.8	2	11	11	8
IS-3443	2.5	5	5	4.2	4	13	10	9
IS-2583	2.5	5	5	4.2	5	15	9	9.7
IS-9331	5	–	4	4.5	13	–	3	5.3
IS-179	5	4.5	5	4.8	14	8	6	9.3
IS-1087	4.5	4.5	2.5	3.8	10	10	–	6.7
IS-9533	5	–	3.5	4.3	15	–	2	5.7
PP2B × 11167	5	5	4.5	4.8	14	14	7	11.7
IS-9327	5	–	3	4	10	–	–	10
IS-9544	5	–	2	3.5	11	–	–	11
BY × IS 511	5	5	5	5	14	16	12	14
IS-9468	5	–	4	4.5	12	–	–	12
Location Mean	3.6	4.0	4.0	–	–	–	–	–

a. 1 to 5 scale, mean of two replications at each location
b. based on the mean of two replications at each location

Local Program Entries

The entries IS-452, IS-455, IS-457, IS-472, IS-473, IS-474, and IS-855 were tested at Samaru, Sefa, Sotuba, Farako-Bâ, and at ICRISAT Center. IS-472 was the only entry with a mean field rating of less than 3 (mean of 2.6, maximum 4.5 at Sefa).

Entries from Texas A & M and from the ISRA (Senegal) program were tested at Sefa in Senegal and at two locations in Ethiopia. They arrived and were planted late at the Ethiopian locations, and consequently were not exposed to severe mold pressures there. At Sefa, they had considerable exposure to molds and the field grain mold ratings there are given for ten entries in Table 9. Again the negative relationship between flowering time and grain mold severity is clear.

1978

At the time of preparation of this paper, results have been returned from seven locations. As there were not sufficient time to prepare a full analysis (this will appear in the report of the 1978 ISGMN, to be circulated to cooperators in early 1979), reactions are given of the best entries carried forward from the 1976 and 1977 ISGMN trials, together with the two best breeding progenies in the trial and three lines from national programs, plus reactions of a susceptible check. The field mold ratings (on a 1 to 5 scale) are presented in Table 10, and the laboratory rankings are given in Table 11.

Table 4. Rank values of 22 entries for three grain mold parameters[a] based on averages of several locations.

Entry	Field rating	Lab rating	Lab ranking
IS 9225	3	2	1
IS 2328	2	1	4
IS 2327	1	5	3
IS 9504	4	6	2
IS 5246	8	3	4
IS 1545	9	4	6
E 35-1	6	11	9
IS 9331	5	19	5
IS 3443	7	13	11
CS 3541	18	8	5
IS 1087	14	10	8
IS 9533	10	16	7
IS 9521	12	17	4
IS 2435	11	15	8
IS 2261	15	9	10
IS 9327	13	12	14
IS 9544	17	7	15
IS 2583	20	14	13
IS 9468	16	18	16
IS 179	19	20	12
BY × IS 511	21	21	17
PP2B × 11167	22	22	16

a. Field rating ranks based on data from five locations. Lab rating and lab ranking ranks based on data from three locations

The severest mold ratings were recorded at Khon Kaen, where most of the entries were given a field rating of 5. Notable exceptions there were IS-9225 (2 and 1), IS-2261 (4 and 3), IS-2327 (3 and 4), and E 35-1, which was given a 1 in the first replication but strangely received a 5 in the second replication. Over all locations, five entries (IS-9225, IS-14332, E 35-1, IS-2327, and IS-2261) averaged a field mold rating of 2 or less, and an additional four (IS-2328, IS-2435, M-36285, and M-36284) averaged a field mold rating of less than 2.5. The known high-susceptible line averaged a field mold rating of 4.8.

The laboratory ranking data (Table 11) are interesting. Although the field mold ratings given at Khon Kaen were high, the seven entries there (IS-9225, E 35-1, IS-2327, IS-2261, IS-2328, M-36285, and M-36284) were regarded as being in the best ten entries in both replications, with IS-9225 and IS-2327 with an average rank of 1.5 and 2, respectively. The rankings at Warangal are a little difficult to understand, for entries with very low field mold scores (e.g., E 35-1, IS-2261, and IS-2327) have relatively high rank values. Over all locations six entries (IS-9225, IS-14332, E 35-1, IS-2327, M-36285, and M-36284) were in the best ten rank in at least 80% of the possible occurrences, and eight entries had a mean rank of ten or less.

Overall Performance

No entry has been scored highly resistant at all locations for the 3 years of testing. However, there are seven lines (E 35-1, IS-9225, IS-2327, IS-2328, IS-14332, IS-2261, and IS-2435) that are consistently better than others at most locations. The earliest flowering is IS-14332 (average of 68 days in 1977), and the remainder flower up to 10 days later. It is encouraging that the breeding lines M-36284 and M-36285 have performed as well as the best source lines, and that in the rankings they were in the best ten entries in 90% and 100%, respectively, of the possible occurrences. These are sister lines from crosses between SC 108-3 and E 35-1, these have good visible grain quality and flower in about 75 days.

Discussion

In its 3 years of operation to date, the ISGMN program has come a long way towards meeting its objectives.

Entries with consistently lower relative grain mold scores have been identified, and have been distributed to interested scientists in many programs in Africa and Asia.

There does not seem to be strong evidence for pathogenic variability among locations. Differences in general mold severity in any one year can probably be attributed to variation in climate during the critical flowering and grain-filling periods.

An extensive network of cooperators is now in existence in the ISGMN program, and there is a good coverage of the Indian and West African sorghum-growing regions.

In the future, entries are likely to consist

Table 5. Field grain mold ratings[a] of 30 1977 ISGMN entries at 12 locations.

Entry	DTF[b]	Locations[c] 1	2	3	4	5	6	7	8	9	10	11	12	Mean
Large glume 7	76	3	2	1	–	2	–	–	1	1.5	–	1	–	1.6
IS 2261	72	1.5	2	1.5	2	–	–	–	2.5	1.5	1.5	1	2	1.7
IS 9225	75	1	2	2.5	3	–	–	–	1.5	2	1	1	2	1.8
IS 3443	79	2	2	1	2.5	–	–	2	1.5	2.5	1	1.5	–	1.8
IS 2328	76	1	2	2	2	3	–	–	1.5	2.5	1	1	2	1.9
IS 2327	77	1	2	2	2	3	–	–	1.5	2	1	1.5	2	1.8
E 35-1	80	1.5	2	2	2	2.5	–	–	1.5	2	–	1.5	2	1.9
IS 14332	68	1	2	1	2	3	3.5	1	1.5	1	2	3	2.5	1.9
IS 2583	72	1.5	2	2.5	2.5	–	–	1	2	2.5	1	1	3	2.0
IS 2435	80	2	2	2	2	–	–	–	2	1	–	3.5	3	2.1
27006-10	64	1	2	1	2.5	3	4.5	1	3.5	2	3	1	–	2.2
IS 15788	74	2	2	2	2.5	5	–	–	3	2	1	1.5	3	2.3
IS 5246	79	1.5	2	3.5	–	4	4.5	–	1	1.5	–	1.5	–	2.3
LG Sel-1	65	1	1	–	3	3.5	4.5	1.5	2	3	1	1.5	5	2.5
Large glume 3	67	1	2	2	2.5	3	4.5	1	1	–	3	3	5	2.6
27006.43	60	4	2	–	–	–	5	2.5	2.5	2	1	1	5	2.6
CS 3541	73	1.5	1	2	2.5	3	4	–	2	2	2	4.5	4	2.6
IS 1545	74	3.5	2	2	2	3	4	1	2.5	2	2	2.5	4.5	2.6
IS 1087	70	1	2	1	2	4.5	4	2	2.5	3	3	3	3.5	2.6
CSH-6	65	2	3	2.5	2	3.5	4.5	1.5	2	2.5	3	2	4.5	2.7
IS 9521	65	3.5	2	1	2	4.5	3	1	2	3	2.5	5	4.5	2.9
IS 2404	62	1	2	4.5	2.5	3	4.5	1.5	2.5	2	4	4.5	5	3.1
G 69-1-3	58	1.5	2	1.5	2	5	4	4	3	2	4	4	5	3.1
CSH-5	73	1	3	3.5	2	4	5	–	3	2.5	2	5	4	3.2
IS 179	66	4	2	3.5	3.5	3.5	5	1	3	4	2.5	5	5	3.6
A 4186	65	3.5	2	3.5	3	4	5	2	3	3	4.5	4	5	3.6
G 81-111	60	2	2	4.5	3	5	5	4.5	2	2.5	5	3.5	5	3.7
NP × EL-18-2	62	1	2	4	3	4	5	3.5	3	4.5	5	4	5	3.7
BY × IS 5111	63	2	4	5	3	4.5	5	3	3	4	5	4	5	4.0
PP2B × 11167	60	4	4	5	3.5	5	5	4	3	4.5	5	5	5	4.4
Location Mean		1.9	2.1	2.4	2.5	3.7	4.5	2.1	2.2	2.4	2.6	2.7	3.9	2.6

a. 1 to 5 scale where 1 = absence of mold; 5 = severe mold

b. Days to flowering (mean across locations)

c. 1–6 India: (1. Pantnagar; 2. Akola; 3. Navsari; 4. Adilabad; 5. ICRISAT Center-2; 6. ICRISAT Center-1)
7. Pakistan (Yousafwala)
8–12. West Africa: (8. Samaru, Nigeria; 9. Tarna, Niger; 10. Sotuba, Mali; 11. Faroka-Bâ, Upper Volta; 12. Sefa, Senegal)

Table 6. Threshed grain mold rating[a] of 30 1977 ISGMN entries at 10 locations.

Entry	Location[b]										
	1	2	3	4	5	6	7	8	9	10	Mean
IS-7357	2	2	2	1	2	–	2	2	2	3.5	2.1
IS-14932	3	2	1.5	1	2	3	2	2	2.5	4	2.3
E-35-1	3	2	2	1	2	–	1.5	2	3	4	2.3
IS-2328	4	2	2	1	2.5	–	2	1	1.5	4	2.2
IS-2261	2	2	2	2	–	–	2.5	2	2	4	2.3
IS-1087	2.5	2	2	1	3	3	3	3	5	5	2.9
Large glume 7	4	2	1	–	2	–	1.5	2.5	2.5	4	2.4
CSH-6	3.5	3	2	1	3	2.5	3.5	2	2.5	5	2.8
CS-3541	3	2	2	2.5	2	3	2	2.5	4.5	4	2.7
IS-2435	4	2	2	2	–	–	1.5	2	3	4.5	2.6
IS-9225	3.5	2	2.5	2	–	–	3	2	2	4	2.6
IS-3443	4	2	2	3	–	–	2	3.5	2	4	2.8
Large glume 3	2.5	2	2.5	3	4	3.5	3	2.5	4	4	3.1
IS-1645	5	2	2	1	3.5	4	4	2.5	3	4.5	3.1
27008-43	5	2	–	–	–	4	1.5	4.5	2.5	5	3.5
IS-2583	2.5	2	2	3	–	–	2.5	3	4	5	3.0
IS-5246	3.5	2	3.5	–	4	4	1	2	2.5	4.5	3.0
LG Selection-1	2.5	2	2	3.5	4.5	3	3	3.5	3.5	5	3.2
IS-15788	3	2	2	2.5	5	–	2	–	3.5	5	3.1
G-69-1-3	4	2	2.5	2.5	4	4	4.5	2	3	5	3.3
27006-10	3	2	1	2.5	5	5	4.5	3.5	2.5	5	3.4
IS 9521	3.5	2	1	1	4	2.5	4	3	5	4.5	3.1
CSH-5	2	3	2	2.5	4	5	4	3	4	5	3.5
IS-179	3	2	3	3.5	4.5	4	4	3.5	3	5	3.6
A-4186	5	2	3.5	3	5	4.5	4	4.5	5	5	4.2
NP × EL-18-2	2	2	3	3.5	5	5	4	5	4	5	3.9
G-81-111	3	2	4	3	4	4.5	5	5	4	5	4.0
IS-2404	2	2	4	2.5	4.5	4	4.5	4.5	5	5	3.8
BY × IS 511	3.5	5	3.5	4	5	5	5	5	5	5	4.6
PP2B × 11167	5	5	5	4.5	5	5	2.5	5	5	5	4.7
Location Mean	3.3	2.3	2.4	2.3	3.7	3.9	3.0	3.0	3.4	4.6	

a. 1 to 5 scale where 1 = absence of mold; 5 = severe mold

b. 1–6 India: (1. Pantnagar; 2. Akola; 3. Navsari; 4. Adilabad; 5. ICRISAT Center-2; 6. ICRISAT Center-1).
7–10 West Africa: (7. Samaru, Nigeria; 8. Sotuba, Mali; 9. Faroka-Bâ, Upper Volta, 10. Sefa, Senegal)

Table 7. Laboratory rank values (based on the mean of two replications) of 50 1977 ISGMN entries at 8 locations.

Entry	Location[a]								
	1	2	3	4	5	6	7	8	Mean
IS-2327	1	16	4	2	9	3	3	1	4.9
IS-14032	10	2	4	11	6	1	4	4	5.3
IS-2261	4	7	10	–	14	8	1	5	7.0
IS-2329	23	17	1	4	6	5	2	2	7.5
S-35-1	11	16	6	3	2	16	10	3	8.4
Large glume 7	26	3	–	6	2	19	5	7	9.7
CSH-6	12	13	2	7	19	4	7	16	10.0
IS-2435	22	7	9	–	2	2	21	12	10.7
IS-2583	5	9	20	–	9	12	13	14	11.7
CS-3541	15	11	13	1	13	11	22	8	11.8
IS-9225	19	20	12	–	15	6	6	5	11.9
IS-3443	25	4	17	–	9	8	15	9	12.1
IS-5246	20	23	–	5	1	10	16	13	12.6
IS-15788	12	18	17	7	8	–	8	23	13.3
IS-1087	8	4	8	–	18	13	28	15	13.4
IS-1545	27	15	2	12	25	13	9	11	14.3
CSH-5	6	12	11	9	20	13	25	18	14.3
G 69-1-3	21	13	16	10	27	7	11	21	15.8
LG Sel-1	9	14	26	14	17	20	11	19	16.3
IS-9521	23	1	7	21	20	17	29	16	16.8
Large glume 3	7	19	22	–	16	18	27	10	17.0
27006-10	14	6	15	–	27	25	18	20	17.9
IS 179	15	10	20	13	23	21	17	25	18.0
27006-43	30	–	–	–	2	26	14	22	18.8
IS-2404	1	23	14	16	26	22	26	24	19.0
NF × EL-18-2	3	22	24	17	23	29	24	29	21.4
G 81-111	18	25	19	14	30	23	19	28	22.0
A-4186	28	21	22	18	20	28	23	26	23.3
PP2B × 11167	29	27	27	20	9	24	20	30	23.3
BY × IS 5111	17	26	25	19	27	27	30	27	24.8

a. 1–4 India: (1. Pantnagar; 2. Navsari; 3. Adilabad; 4. ICRISAT Center-2)
5–8 West Africa: (5. Samaru, Nigeria; 6. Sotuba, Mali; 7. Farako-Bâ, Upper Volta; 8. Séfa, Senegal).

Table 8. Rank values of 30 entries for three grain mold parameters based on averages of 12 locations.

Entry	Field rating	Lab rating	Lab ranking[a]
IS-2327	6	1	1
IS-2761	2	5	3
IS-2328	5	2	4
Large glume 7	1	6	5
IS-14332	8	4	2
E 35-1	7	3	6
IS-9225	3	8	11
IS-2435	10	7	8
IS-3443	4	11	12
IS-2583	9	13	9
CS-3541	17	9	10
CSH-6	20	10	7
IS-5246	13	14	13
IS-15788	12	18	14
IS-1087	19	12	15
IS-1545	18	17	16
Large glume 3	15	16	21
LG Selections-1	14	19	19
27006-10	11	21	22
IS-9521	21	15	20
G 69-1-3	23	20	18
CSH-5	24	22	16
IS-179	25	24	23
27006-43	16	23	24
IS-2404	22	25	25
NP × EL-18-2	28	26	26
G 81-111	27	27	27
A 4186	26	28	28
BY × IS 511	29	29	30
PP2B × IS 11167	30	30	28

a. Based on eight locations

Table 9. Flowering data and field grain mold ratings of ten sorghum entries at Bambey, Senegal.

Entry	Origin[a]	DTF[b]	GM rating
74-1055-051	ISRA	86	2
74-10-KL	ISRA	75	3
IS-3552-DER(Sc-745-5)	TAM	73	3
CE-90	ISRA	70	4
74-31-V 15	ISRA	70	4
Sc-630-11	TAM	70	4
95-40-63T × 3927-4	ISRA	68	4
IS-7254C (Sc-566)	TAM	58	5
IS-1260C (SC-97)	TAM	56	5
IS-7419C (SC-279)	TAM	48	5

a. ISRA — Bambey, Senegal (Program of J. Danis); TAM — Texas A & M (Program of R. A. Frederiksen)
b. DTF = Days to flowering

mainly of breeding progenies, in which the emphasis will be on increasing the mold resistance and combining it with earliness and good grain quality. The details of the program for the production of these progenies will be presented in the paper by Murty et al. during this session.

Some Questions

The ISGMN program should be contributing to national programs. In order that we can improve the program to better contribute, the participants' answers to the following questions will be valuable.

1. Is the ISGMN program considered to be worthwhile and should it continue?
2. What changes can be made in its structure and operation to make it more effective — e.g., should there be more entries, are the rating methods adequate for entry differentiation?
3. In how many days does a cultivar have to flower to be classified as "early?" Is a flowering time of between 70 and 80 days early enough for the majority of SAT situations?

Acknowledgment

We are indebted to all the cooperators in the ISGMN program who have given their time, energy, and resources in order to produce the results reported in this paper.

Table 10. Field grain mold ratings[a] of 13 sorghum lines in two replications at each of 7 locations in the 1978 ISGMN.

Entry	Sotuba		Farako-Bâ		ICRISAT Center		Adilabad		Warangal		Bhavanisagar		Khon Kaen		Mean
	Rep 1	Rep 2	Rep 1	Rep 2	Rep 1	Rep 2	Rep 1	Rep 2	Rep 1	Rep 2	Rep 1	Rep 2	Rep 1	Rep 2	
IS-9225	1	1	1	1	1	1	1	1	2	2	2	2.5	2	1	1.4
IS-14332	1	1	1	2	1.5	1	1	1	1	1	1.5	1.5	5	5	1.8
E-35-1	2	2	2	2	2	1.5	1	1	1	1	2	2.5	1	5(?)	1.8
IS-2327	1	2	2	2	1.5	1	1	1	2	2	2	2	3	4	1.9
IS-2261	1	1	1	2	2	1	1	1	1	2	4	3.5	4	3	2.0
IS-2328	1	2	1	2	1.5	1.5	1	1	3	3	3	2	5	5	2.3
IS-2435	2	2	2	2	2	1.5	1.5	1.5	1	1	3	3.5	5	5	2.4
M-36285	2	2	2	2	2	1.5	1.5	1.5	2	1	2	2.5	5	5	2.3
M-36284	2	2	2	2	2.5	1.5	1.5	1.5	1	2	2	2.5	5	5	2.3
JP-2579	2	4	4	5	3	2	1.5	1.5	1	1	2.5	2	5	5	2.8
CS-3541	3	3	3	4	3	2.5	2	2	2	2	2.5	2.5	5	5	3.0
IS-472	5	5	4	5	3	2.5	1.5	1.5	1	1	1.5	2	5	5	3.1
9991-Bulk	5	5	5	5	5	5	5	5	5	5	3.5	4	5	5	4.8

a. 1 to 5, where 1 = mold-free; 5 = severely molded

Table 11. Rank values[a] of 13 sorghum entries based on the evaluation of 30 entries in the 1978 ISGMN at 5 locations[b]

Entry	Farako-Bâ		ICRISAT Center		Adilabad		Warangal		Khon Kaen		Mean	% in 1st 10[c]
	Rep 1	Rep 2	Rep 1	Rep 2	Rep 1	Rep 2	Rep 1	Rep 2	Rep 1	Rep 2		
IS-9225	1	6	3	1	3	3	19	19	1	2	6.0	80
IS-14332	4	3	6	8	1	1	1	1	13	10	4.8	80
E-35-1	–	24(?)	5	7	5	5	8	9	5	6	8.2	89
IS-2327	3	8	2	2	2	2	16	15	3	1	5.4	80
IS-2261	17	23	9	5	6	6	14	14	4	4	10.2	60
IS-2328	24	1	1	3	4	4	24	25	2	5	9.3	70
IS-2435	7	2	4	4	8	8	6	6	13	10	6.8	70
M-36285	2	5	8	6	3	3	5	5	9	7	5.3	100
M-36284	21	9	10	9	10	10	7	7	8	9	10.0	90
JP-2579	22	7	17	12	9	9	3	3	13	10	10.5	50
CS-3541	11	17	23	24	22	22	9	8	13	10	15.9	20
IS-472	23	19	19	13	7	7	2	2	13	10	11.5	40
9991-Bulk	27	29	29	29	29	29	30	30	13	10	19.7	0

a. ranked from 1 = least molded to 30 = most molded
b. ranking was not done at Sotuba and Bhavanisagar
c. A 10 in the second rep at Khon Kaen is not counted because the 20 most severely molded were all ranked 10

Chemistry and Structure of Grain in Relation to Mold Resistance

J. A. Glueck and L. W. Rooney*

The purpose of this paper is to review some current findings regarding changes that occur in the kernel when sorghum deteriorates in the field as a result of attack by molds or other associated reactions. The role of kernel structure in water uptake will be discussed. Finally, the physical, structural, and chemical factors of significance in explaining why certain sorghums are more resistant than others to mold deterioration (weathering) will be discussed.

The term "molding" will be used in this paper interchangeably with the term "weathering," which is commonly used in the United States. Microorganisms play the dominant role in causing grain to deteriorate; however, enzymes from the grain itself also cause significant deterioration. Changes in the physical properties of the grain occur because of alternate wetting and drying. Insect damage during grain maturation can stimulate deterioration, and provide a means for entry of microorganisms into the sorghum kernel. Prolonged rainfall, high humidity, high temperature, and alternate periods of wetting and drying, before and after physiological maturity of the grain, all favor deterioration. The kind of microorganisms and their growth rate, as well as the initiation of kernel germination, are affected by environmental conditions. Significant losses from grain mold that occurred on the High Plains of Texas in 1974 were caused primarily by invasion of the kernels by microorganisms, while those losses in South Texas in 1976 were due to the combined effects of sprouting and microorganisms. High temperature during an extended period of rainfall accelerated the deterioration process in South Texas.

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Kernel Structure

The kernel or caryopsis is composed of three main parts, the outer covering (pericarp), the storage tissue (endosperm), and the embryo (germ). The structure has been discussed in detail (Rooney and Clark 1968; Rooney and Sullins 1977; Sanders 1955; Sullins and Rooney 1974, 1975).

Pericarp

The pericarp can be subdivided into the epicarp, mesocarp, and endocarp as illustrated in Figure 1a. The first or outermost portion is the epicarp, which usually consists of two to three cell layers. These cells are long and rectangular in shape, and contain wax and sometimes pigments.

The middle layer is the mesocarp, which may contain small starch granules that are visible under polarized light (Fig. 1b). This layer may vary in thickness from a thick starchy chalky appearance to a thin, translucent mesocarp. The mesocarp is usually thickest opposite the embryo. Mesocarp thickness is controlled by the Z-gene, where thin is dominant to thick. Thin mesocarp sorghums appear to withstand weathering better than those with thick mesocarps. Milling yields from sorghums with thin mesocarp are superior to those from sorghum from thick mesocarps. The thin mesocarp contains few, if any, starch granules.

The innermost layer of the pericarp is the endocarp, consisting of cross and tube cells. The cross cells are long and narrow, and the long axis is at right angles to the long axis of the kernel. The tube cells are 5μ wide and up to 200μ in length, and the long axis of each cell parallels the long axis of the kernel. One of the main functions of cross and tube cells is the transport of moisture. These cells are also the point of breakage when the pericarp ("bran," in

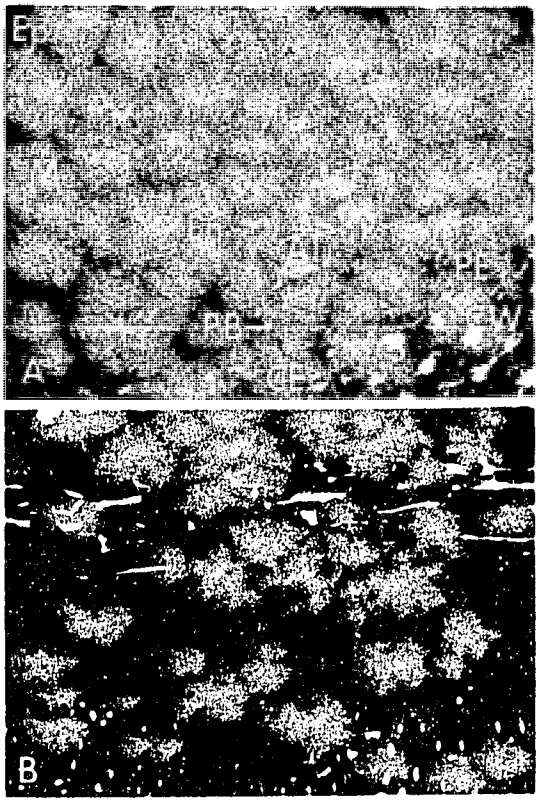

Figure 1. Photomicrographs of a thin section of a mature sorghum kernel viewed with ordinary light (A) and with plane-polarized light (B). 400X. Ep – epicarp, M – mesocarp, En – endocarp, Al – aleurone layer, PE – peripheral endosperm, CE – corneous endosperm, S – starch granule, CW – cell wall, PB – protein body. in photo B, the starch granules appear as Maltese crosses and can be observed in the endosperm and mesocarp.

milling terminology) is removed during milling of the grain. It is probable that fungi enter into the kernel with water.

The presence or absence of pigments in the pericarp is controlled by R-Y-I genes. Grain color (appearance) varies greatly, and is affected by the actual color of the pigments, by the thickness of the mesocarp, presence of a testa, color of the endosperm, and plant and glume color. In a white-kerneled sorghum, pigments from the glume may leach into the grain. During milling, the pigments can cause an off color in the grits, flour, or other products.

Testa

Just beneath the pericarp, some sorghum kernels have a highly pigmented layer called the testa or subcoat (Fig. 2; compare Fig. 2a with 2b). The presence or absence of the testa is controlled by the B_1 and B_2 genes. Testa is present when genes are B_1-B_2-, while testa is absent with all other gene combinations. Some sorghum lines contain a pigmented partial testa that is found at certain places around the kernel. The testa also varies in thickness from one line to another and from one area of the kernel to another. It is usually thickest at the crown of the kernel and thinnest over the embryo. The color of the testa varies among sorghum lines.

The testa originally, in the immature seed, is the inner integument which has a definite structure. However, as the kernel matures and the endosperm expands, the cellular configuration of the testa gives way to a continuous layer. Pigmentation is associated with a high concentration of polyphenols or tannins. High tannin content appears to improve weatherability by retarding preharvest seed germination and reducing seed molding.

The tannins also serve as a deterrent to birds. Tannins in the bird-resistant sorghums reduce dry-matter digestibility by binding proteins and possibly complexing with digestive enzymes. Compared with low-tannin sorghums, the high-tannin grains have poor feed efficiency when fed to livestock. Some color precursors are located in the endosperm. When some white sorghum flours are treated with dilute alkali, these precursors produce greenish yellow to off-white food products. There is considerable variability in intensity of color amongst different white sorghum varieties.

Endosperm Structure

The endosperm of sorghum consists of the aleurone layer and the peripheral, corneous, and floury portions. The aleurone cell layer, located beneath the pericarp (Fig. 1, 2a), or testa (Fig. 2b) if it is present, is a single layer of block-like rectangular cells. When viewed under greater magnification, the aleurone cells contain spherical bodies that vary in size and

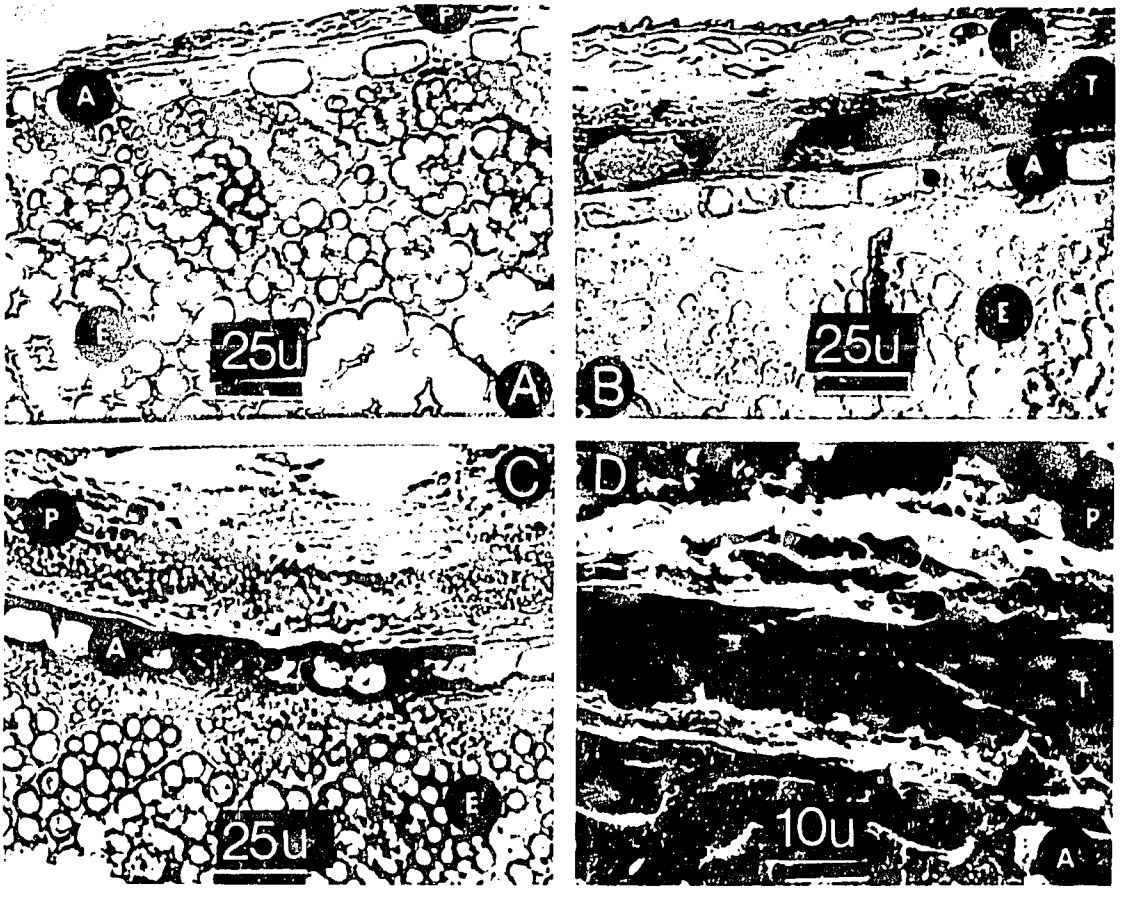

Figure 2. Cross sections of the sorghum kernel. (A) White pericarp without testa. P – pericarp; A – aleurone; E – endosperm. (B) White pericarp with testa. T – testa. (C) Red pericarp without testa. In this section, heavily pigmented dark spots were observed in the aleurone layer. There was no visible damage to the kernel by insect or disease that penetrated the pericarp. (D) Scanning-electron photomicrograph of the testa (1000X). Light photographs are 400X.

contain protein and perhaps phytin. The aleurone cells contain a high level of oil, large amounts of minerals, water-soluble vitamins, and autolytic enzymes. They do not contain starch granules. The aleurone proteins are of good nutritive quality. The aleurone cells play an essential role in autolysis and mobilization of kernel components during germination.

The peripheral endosperm is beneath the aleurone layer, and is an ill-defined area consisting of the first two to six endosperm cells. These cells are small and blocky, and contain small starch granules (Fig. 3) embedded in a dense proteinaceous matrix. The matrix protein is comprised mainly of glutelins or alkali-soluble proteins and prolamins. The prolamins, alcohol-soluble protein, exist in small spheres called protein bodies. The amorphous matrix protein can be preferentially removed by pronase enzyme to expose the protein bodies. However, alpha-amylase will remove the starch, which also exposes some of the protein bodies. The endosperm contains both free protein bodies and those located in the matrix protein. When the active cell cytoplasm, which is in greatest concentration around the periphery of the endosperm, dries during maturation of the kernel, it encases the protein bodies and becomes the cementing protein matrix. The protein bodies of the peripheral and

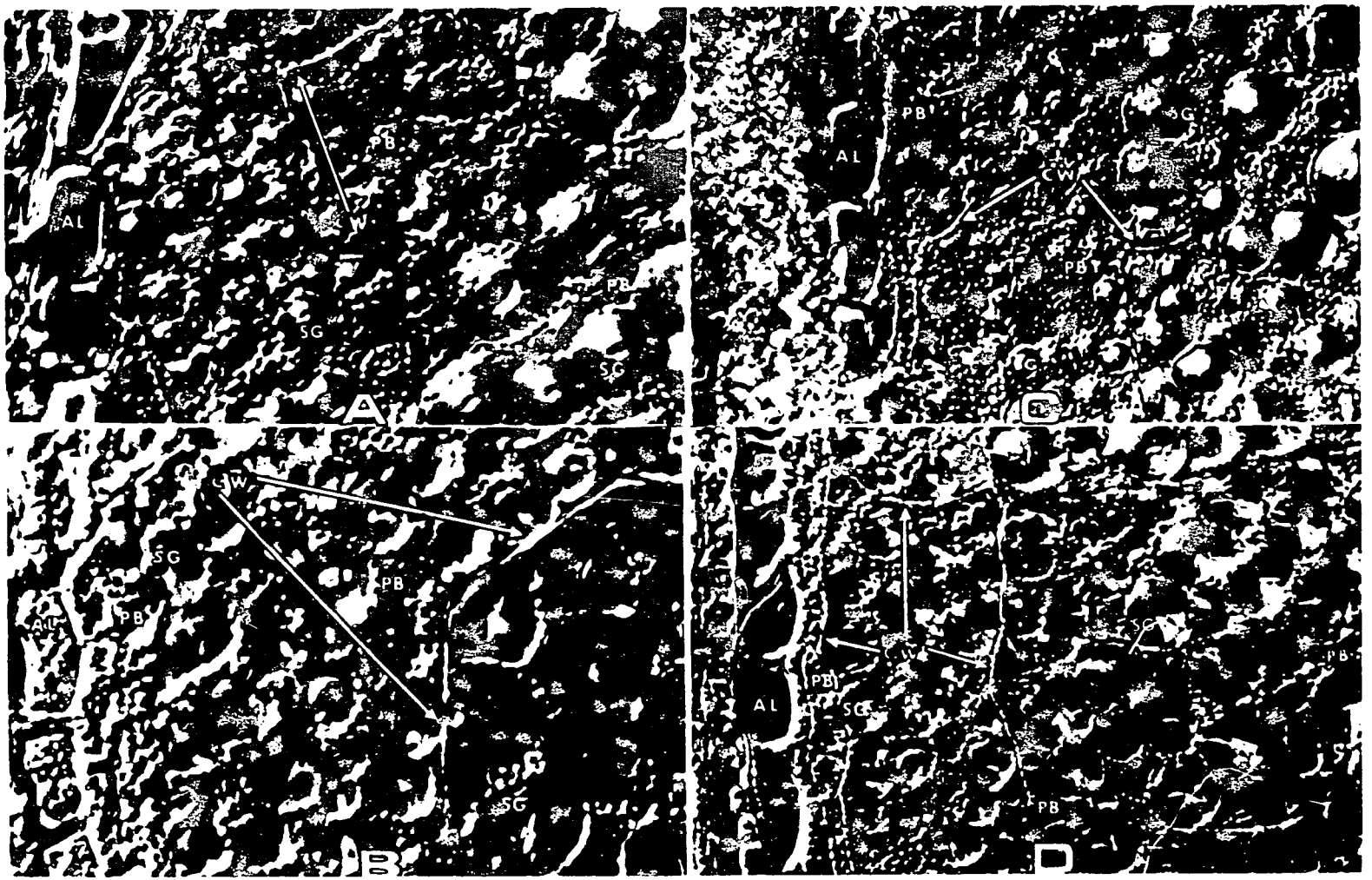

Figure 3. Sections of untreated nonwaxy and waxy sorghum endosperm viewed with the light microscope at magnification of 320X. AC - aleurone cells; CW - cell walls; PB - protein bodies; SG - starch granules. Photo A is a nonwaxy Kafir, B is a waxy Kafir, C and D are waxy and nonwaxy Redlan, respectively.

corneous endosperm range from 0.3 to 3.0μ in diameter and decrease in size and number toward the center of the kernel. The protein bodies in the floury endosperm range from 0.3 to 1.5μ in diameter.

Figures 3 and 4b illustrate how the starch granules are embedded in a nest of protein bodies and matrix. Enzyme treatment of thousands of kernels has consistently indicated that hydrolysis of the starch in the peripheral area is slowed by the high concentration of protein bodies and matrix. The enzymes or digestive fluids have a hard time contacting the starch granules to facilitate digestion. This renders much of the starch in the peripheral area unavailable for utilization. Newer processing

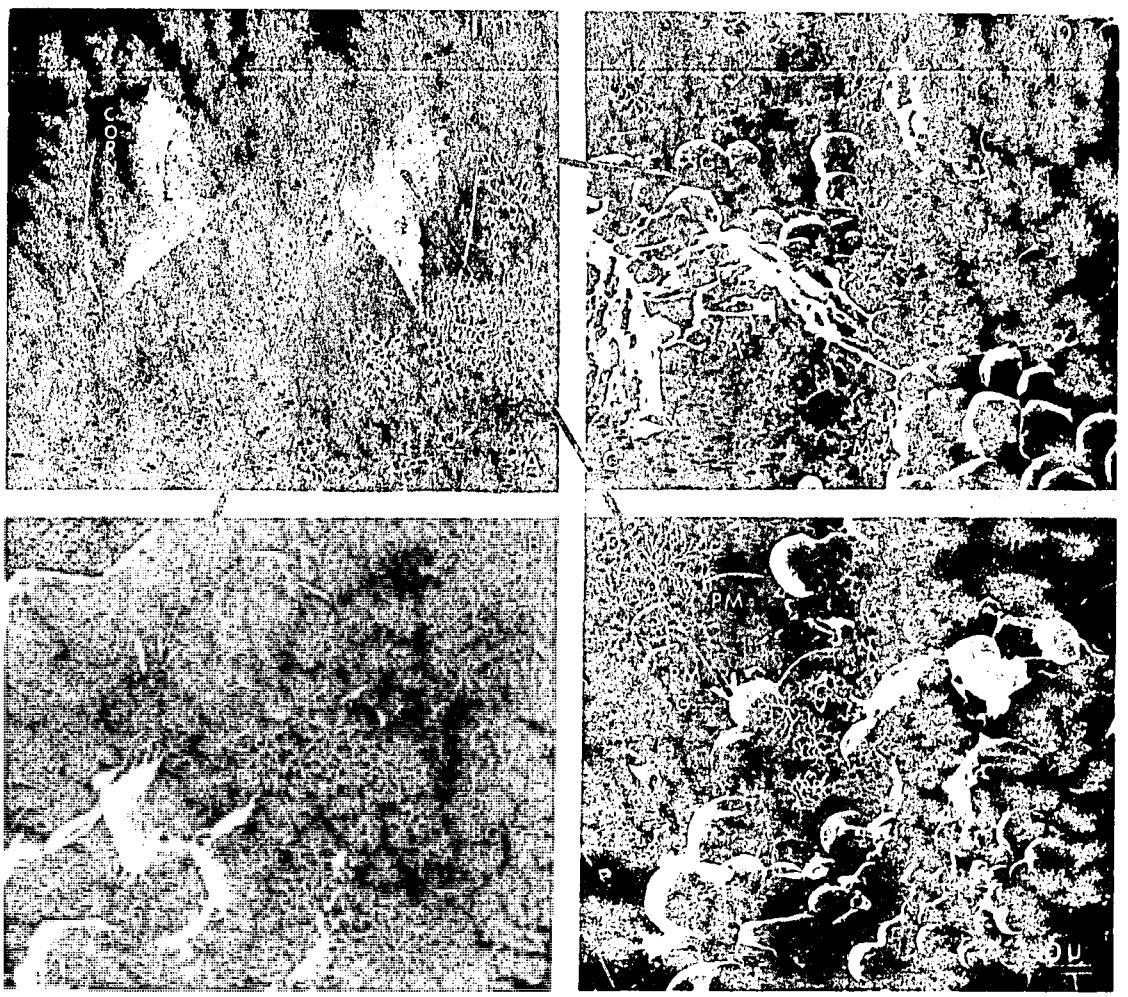

Figure 4. Scanning-electron photomicrographs of the corneous and floury endosperm areas within a sorghum kernel. Kernels were cut longitudinally and examined at areas labelled in Photo A. Photo B illustrates the typical structure of the corneous endosperm where there is a continuous interface between the starch and protein, resulting in no air space between the starch granules. The corneous area, therefore, appears extremely dense and vitreous. In contrast, the floury endosperm (Photo D) contains air spaces of areas between the starch granules not filled with protein, which gives a loosely packed or chalky appearance to the center portion of the endosperm. Photo C is a transition area where some endosperm cells are corneous and some are floury. SG – starch granule; PM – protein matrix; PB – protein bodies; BG – broken granule.

methods mechanically disrupt the organization of this area, which greatly increases the accessibility of the starch and protein to the digestive enzymes. The organization of the peripheral endosperm area is affected by genetic and environment factors.

The corneous endosperm (hard, flinty, horny), located beneath the peripheral endosperm (Fig. 4), has a continuous interface between the starch and protein (Fig. 4b). The starch granules are very angular or polyhedral in shape, with depressions where protein bodies were trapped between expanding starch granules. The starch/protein bond is strong enough to allow some starch granules to break rather than pull from the matrix. The hardness and appearance of the corneous endosperm is explained by this structure.

The floury endosperm area (Fig. 4d) has loosely packed endosperm cells. The starch granules are spherical, and they are not held together by protein matrix. In addition, small voids occur between starch granules, and it is difficult to see any protein matrix. The air spaces alternating with cell constituents diffuse light as it passes through the endosperm, which explains the chalky or opaque appearance of the floury endosperm. Protein bodies and matrix are present in the floury endosperm (Fig. 4d), but the matrix protein is not continuous and consists of relatively thin sheets spread over the surface of the starch granules. Within the sorghum endosperm, the protein content is lowest in the floury area. The floury endosperm is soft and extremely susceptible to enzyme attack.

At some place in the endosperm, there is a transition from corneous to floury endosperm; Figure 4c shows this transition zone. Some cells in the floury portion contain starch granules that appear well-meshed together, while other cells are loosely packed and contain large voids between the starch granules.

Embryo

The embryo, or germ, of sorghum contains approximately 10% of the kernel's total dry weight. The scutellum and the embryonic axis are the major parts of the embryo. The scutellum surrounds the embryonic axis, and is thought to facilitate movement of nutrients into the developing roots and leaf tissues of the embryonic axis during germination. The scutellum consists primarily of large vasculated parenchyma cells with a surface layer of epithelial cells on the posterior surface (Paulson 1969). Apparently, a well-developed vascular system in the scutellum facilitates movement of materials to the embryonic axis. The vascular system in the scutellum may be connected to the transfer cells in the endosperm adjacent to the scutellum. The structure of the embryo has not been characterized fully.

Other Structural Features of Importance

The stylar and hylar areas of the sorghum kernel may be of great importance in grain deterioration, since these are natural openings in the kernel. The hylar region has been referred to as closing tissue, or "dark layer" and "placento-chalazal" regions (Eastin et al. 1973; Giles et al. 1975; Glueck 1979; Kiesselbach and Walker 1952; Wolf et al. 1952). In maize, the enlargement of the scutellum crushes a layer of cells external to the placento-chalazal pad, which upon maturity becomes dark in color and is called a "black layer." Harrington and Crocker (1923) described the closing tissue in *Sorghum halepense* as an intensely pigmented layer of thick-walled cells forming a protective cover to the large hilar opening through the inner integument. When the closing tissue was completely bleached, the cells of this dark central region and those above and below it were almost identical in appearance. In sorghum the placento-chalazal pad lies between the band of phloem parenchyma tissue on the abgerminal side of the pericarp and the "transfer cells", which are modified basal endosperm cells (Giles et al. 1975). The "transfer cells" may function in solute transfer from the phloem vascular system of the pedicel to the transport systems of the developing sorghum caryopsis.

In addition to the hilar and stylar regions, Kiesselbach and Walker (1952) describe a thin noncellular membrane located between the nucleus and inner integument; the membrane completely surrounds the maize seed, except over the placento-chalazal region. Such a membrane has not been reported in sorghum. Since the genetic variability among sorghums is great, grain containing a membrane may exist, and it would be expected to affect solute and solvent transfer.

Changes in Physical and Structural Properties of Grain

Grain-weathering tests have been conducted on a statewide basis in Texas since 1975. Data in Table 1, taken from some of these tests in 1975, show typical changes that occur as grain deteriorates in the field (Glueck 1979; Glueck et al. 1978).

Deterioration results from physical, physiological and chemical changes in the kernel, causing breakdown of kernel structure and eventual loss of viability. Deteriorated grain is usually dark and discolored in external appearance, has a dark discolored germ, and the inside of the kernel is chalky in appearance due to partial hydrolysis of starch and protein (Fig. 5). When environmental conditions are favorable, sprouting may occur, as in southern Texas in 1976 (Fig. 5f). Among 15 selections grown in the Corpus Christi test nursery and harvested after a 12-day rainy period, sprout damage was extensive in all heads, ranging from 38 to 100%. In general, grains with highest sprout damage also had more mold damage and greater discoloration. Bushel weight of the line that showed the greatest degree of deterioration was 42 lbs. Grain of the same line grown at a different location and harvested without deterioration weighed 58 lb/bu. Although the percent of trash is regulated by combine conditions, deteriorated grain tends to be more "trashy," perhaps as a result of attempts to maximize the amount of grain harvested. The percentage of fines and broken kernels was also higher in the deteriorated grain. The processing properties of this grain were altered, because it readily fell apart when handled.

Physical, physiological, and chemical changes result in loss of dry matter and decreased test weight, 1000-kernel weight, density, and hardness of the grain (Table 1). Changes in physical properties can be explained by comparing scanning-electron micrographs of undamaged and deteriorated sorghum kernels (Fig. 6, 7). Undamaged starch granules are spherical and have a smooth surface. In contrast, starch granules of deteriorated grain show considerable pitting on the surface, resulting from enzyme attack (Fig. 7d), and the protein matrix is weakened and partially hydrolyzed (compare Fig. 6b, c with Fig 7b, c). The fungal mycelium, which appears as a threadlike network, penetrated through the endosperm and partially hydrolyzed the protein and starch (Fig. 7b, c). Other enzymes are produced by the kernel itself during the initial stages of germination. The individual or combined action of the fungal and/or grain enzymes produces a softer kernel which may break when handled and produce fine particles when processed into food by milling processes.

Chemical composition of weathered grain was not very different from that of nonweathered grain, as indicated by average values for 12 lines grown at Corpus Christi and harvested before and after the rain in 1976 (Table 2). Weathering decreased the proportion of crude protein and fat, and increased crude

Table 1. Mean and range of measurements of the effect of field deterioration on physical properties of grain from 25 sorghum lines grown at three Texas locations in 1978.

	Test weight[a]			1000-Kernel weight			Density			Hardness[b]			Germination		
	Lub	CS_1	CS_2	Lub	CS_1	CS_2	Lub	CS_1	CS_2	Lub	CS_1	CS_2	Lub	CS_1	CS_2
	(lb/bu)			(g)			(g/cc)						(%)		
Mean	63	59	56	28	26	25	1.36	1.36	1.35	22.9	22.3	20.0	95	78	32
Minimum	56	52	47	20	19	18	1.27	1.29	1.29	0.5	1.8	2.7	87	33	2
Maximum	65	62	59	38	33	34	1.39	1.39	1.37	33.7	34.9	30.6	99	93	75

a. All lines were grown at Lubbock (Lub) and College Station (CS_1, CS_2). Lubbock grain was essentially free of deterioration, while grain from CS_1 and CS_2 had moderate to severe deterioration, respectively. Sprouting was not observed.

b. Hardness was determined by a standardized pearling procedure. The highest values have the greatest resistance to pearling, which is an index of hardness

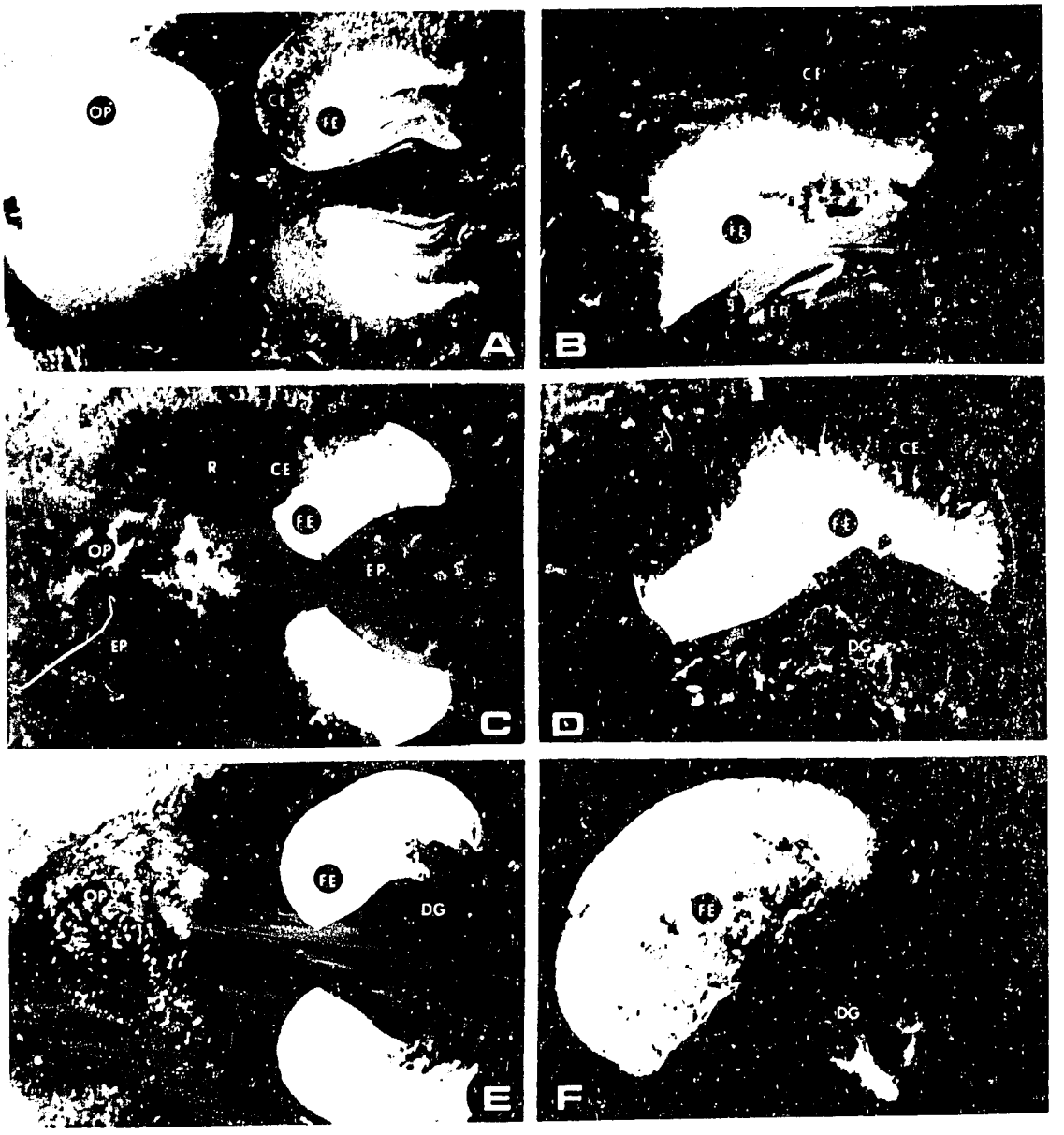

Figure 5. Light photomicrographs of undamaged and deteriorated sorghum kernels. Photo A) Undamaged whole and half kernels with yellow endosperm, intermediate texture, magnification about 4.0X. CE – corneous endosperm; FE – floury endosperm; OP – surface of pericarp. Photo B) Undamaged half kernel with yellow endosperm, intermediate texture, ca 6.4X. S – scutellum; EP – epicotyl, radicle, and coleorhiza of germ. Photo C) Whole and half kernels with normal intermediate texture and sprout damage, ca 4.0X. EP – epicotyl that has emerged from seed; R – elongated radicle. Photo D) Half kernel of grain with sprout and microbial damage, ca 6.4X. DG – damaged germ. Photo E) Whole and half kernels with yellow endosperm, intermediate texture, and severe damage, ca 4.0X. Photo F) Half kernel with yellow endosperm, intermediate texture, and severe damage, ca 6.4X.

fiber and ash; however, only the difference in ash was significant, and the variability among lines was quite great, as indicated by the standard deviations. Nitrogen-free extract (NFE) and starch content were not affected. Other data have shown a slight decrease in NFE and slight increases in protein, fat, ash, and fiber as compared with nonweathered grain. For weathered grain, a portion of the carbohydrates is utilized to provide energy for growth and development of the fungi and respiration of the grain. Therefore, dry weight is lost as carbon dioxide and water. The grain protein may be partially hydrolyzed and used to synthesize

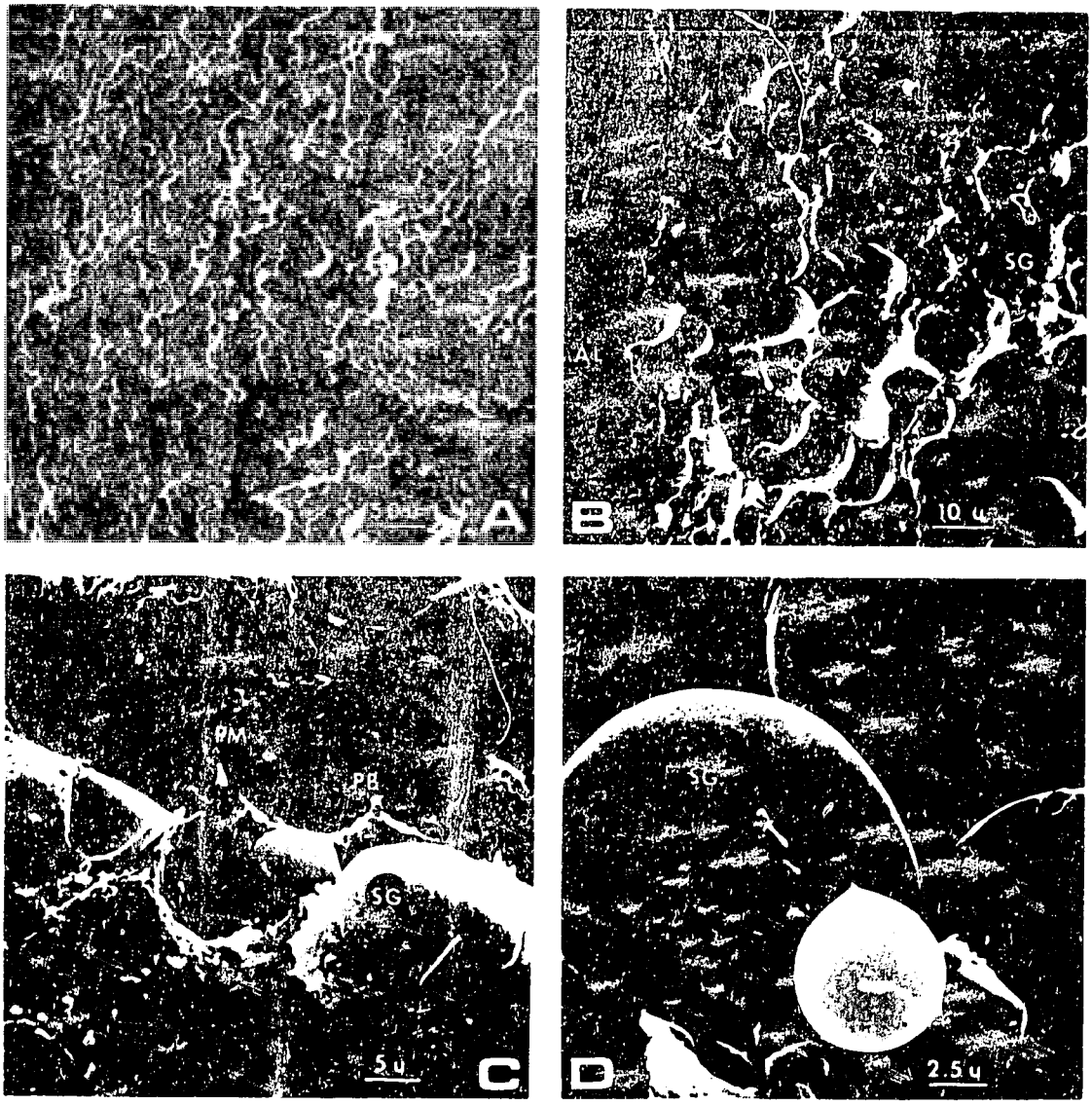

Figure 6. Scanning-electron photomicrographs of undamaged yellow-endosperm intermediate-texture sorghum kernels. Photo A) Endosperm cross section. P – pericarp; AL – aleurone cell layer; PE – peripheral endosperm; CE – corneous endosperm, ca 200X. Photo B) Corneous-endosperm area; SV – starch void; SG – starch granule, ca 1000X. Photo C) Protein and starch of corneous endosperm; PM – protein matrix; PB – protein bodies; SG – starch granule, ca 2000X. Photo D) Starch of floury endosperm, ca 4000X.

fungal protein which remains in the grain. Seed protein used in production of protein in the sprout may or may not remain with the grain, depending upon whether or not it dries, breaks off, and is lost during harvest and cleaning operations. Slight losses of NFE relative to other grain components could explain the increased percentages of protein, ash, fat, and fiber. The degree of weathering, ranging from surface discoloration caused by microorganisms to extensive growth of mold and the penetration of the seed by mycelia accompanied by sprouting,

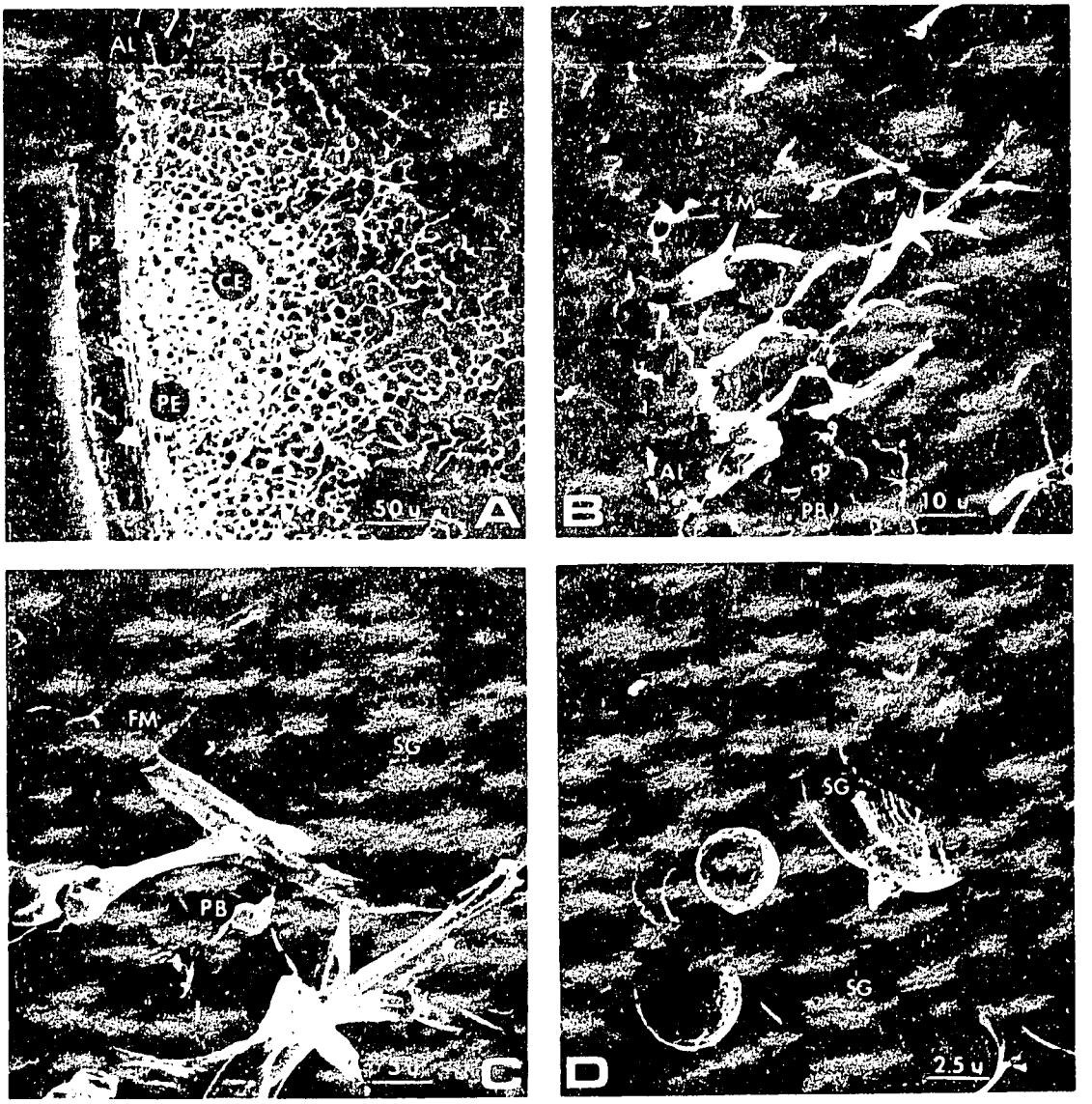

Figure 7. Scanning-electron photomicrographs of deteriorated yellow-endosperm, intermediate-texture sorghum kernels. Photo A) Endosperm cross section; P – pericarp; AL – aleurone cell layer; PE – peripheral endosperm; CE – corneous endosperm; FE – floury endosperm, ca 200X. Photo B) Corneous-endosperm area; PB – protein body; FM – fungus mycelium; SG – starch granule, ca 1000X. Photo C) Fungus, protein, and starch or corneous endosperm, ca 2000X. Photo D) Starch of floury endosperm, ca 4000X.

may have quite variable effects on composition.

The severely weathered grain from southern Texas in 1976 was fed to livestock with no reduction in animal health or performance (Glueck et al. 1978). Aflatoxin was not found in the weathered grain samples. In general, sorghum grain, for unknown reasons, appears to have a low incidence of aflatoxins, at least under our conditions in the USA. Certainly the quality of these sorghums for food preparation for human consumption was destroyed.

Characterization of Sorghum Grains with Resistance to Weathering

Several sorghum lines that consistently ranked among the most resistant to weathering at all locations and over several years of testing have been identified. These lines were included in studies designed to determine why they were resistant in comparison to susceptible checks. The approach was to combine observations in the field with detailed studies of grain structure and physical and chemical properties. The suspected genotypes of the resistant and susceptible lines with respect to pericarp characteristics are described in Table 3. Since genetic studies were not designed, many of the genotypes are postulations based on F_3 populations of limited crosses.

Kernel Characteristics and Physical Properties

All resistant as well as susceptible lines, except SC-0103 and SC-0719 had intermediate endosperm texture. The proportion of corneous to floury endosperm was approximately equal; however, most resistant selections had slightly more corneous endosperm relative to floury. SC-0103 and SC-0719 had a more floury endosperm texture; the grain of each contained a testa layer. The corneous endosperm characteristics are not necessary for resistance to weathering; however, if all other things were equal, a line with more corneous grain would resist deterioration more than would a floury endosperm line, because of the more dense structure and organization (Clark et al. 1973; Ellis 1972, 1975). Apparently water and fungi are not able to proceed as readily through a more organized structure. Some F_5 progeny of a TX-09 (floury endosperm texture) by SC-0170 (intermediate texture) cross had some resistance, even though the grain had floury endosperm and did not contain a testa. The grain did contain one or two dense peripheral cell layers surrounding the endosperm, which probably imparted the resistance to the floury endosperm sorghum.

In general, sorghum kernel size did not appear to be a major factor related to weathering resistance. Obviously, extremely large kernels

Table 2. Effect of weathering on chemical composition of grain of 12 sorghum lines grown in the coastal belt of Texas in 1976.

Grain[a]	Constituent (%)					
	Moisture	Protein	Fat	Fiber	Ash	NFE[b]
Nonweathered	13.9	10.7	3.1	2.3	1.7	82.3
Weathered	12.5	10.5	3.0	2.4	1.9	82.4
Weathered as per cent of nonweathered						
Percent	89.9	98.1	96.8	104.4	111.8	100.1
Std. deviation	9.2	6.8	11.0	13.4	9.6	1.2

a. Average of 12 lines. Nonweathered grain was harvested prior to the rains, while the weathered grain was harvested from the same plots after the rains.
b. Nitrogen-free extract.

Table 3. Genotypes and grain appearance of mold resistant and susceptible lines.

Line	Genotype	Mesocarp thickness	Endosperm texture[a]	Reaction to grain deterioration[b]	Appearance
SC0279-14	$B_1B_1b_2b_2ss$	thin, pearly	I	R	bright red translucent
SC0566-14		thin, pearly	I	R	bright red translucent
SC0748-5	$b_1b_1B_2B_2$'SS' rrYYIIzz	thin	I	R	lemon yellow
74PR759		intermediate	I	R	red
SC0103-12	$B_1B_1B_2B_2$SSRR	thick chalky	Fl	R	brown
TAM428		thin	I	M	white
BTX398	$b_1b_1B_2B_2$SSRRYY IIzz	intermediate	I	R	red
TX2536	$b_1b_1B_2B_2$SSRR yyiiZZ	thin pearly	I	S	yellow
SC0630-IIE-4	$b_1b_1B_2B_2$SSRR YYIiZZ	thin	I	R	bright red – red[c]
SC0719-IIE	$B_1B_1B_2B_2$SSRR YYIIZZ	thick chalky	Fl/I	R	dark red
SC0599-6		thin	I	R	red

a. I-Intermediate, Fl-floury.
b. R-resistant, M-moderate resistant, S-susceptible.
c. SC0630 was segregating for intensity of pericarp color, which is controlled by the i loci.

are usually more susceptible than medium or small kernels. Kernel hardness and density was in general positively related to weathering resistance. The soft floury grain of SC-0103 has resistance because of the high tannin content.

Glume Size and Shape

A grain type enclosed in long enveloping glumes is not guaranteed to resist deterioration. The conspicuum type has long glumes, and although SC-0279 is resistant (especially to microorganism degradation), many of the conspicuum types have little grain mold resistance. The open-glume type probably promotes sprouting in the panicle, since the open cupped glumes hold water that is taken up by the grain. In addition, some microorganisms, especially the *Fusarium* spp, probably enter the grain prior to physiological maturity through the connective tissue between the developing grain and pedicel.

Grain shape probably has an indirect effect on resistance or susceptibility to deterioration. Field observations and studies involving the uptake of water by dry grain suggest that the durra-type grain is prone to fissure. Breaks in the pericarp due to fissures or insect damage are entry sites for microorganisms and moisture into the grain (Fig. 8). We have observed this happening in the field; but we do not know how frequently it happens.

Grain and Glume Color

Pigmentation in the pericarp and glume may impart a slight degree of resistance to grain deterioration. The scope of this study did not allow conclusions to be made on this parameter. All of the identified resistant lines contained some coloration; however, there were some colorless or white pearly lines included in our field trials at most locations (SC-0283 and several zera zera types) that had good resistance to grain mold at some locations. Many of the more resistant lines have tan plant color; however, the characteristic does not seem essential for the expression of resistance. Grain of SC-283 has a very high proportion of corneous endosperm. A sorghum kernel with a testa, thin

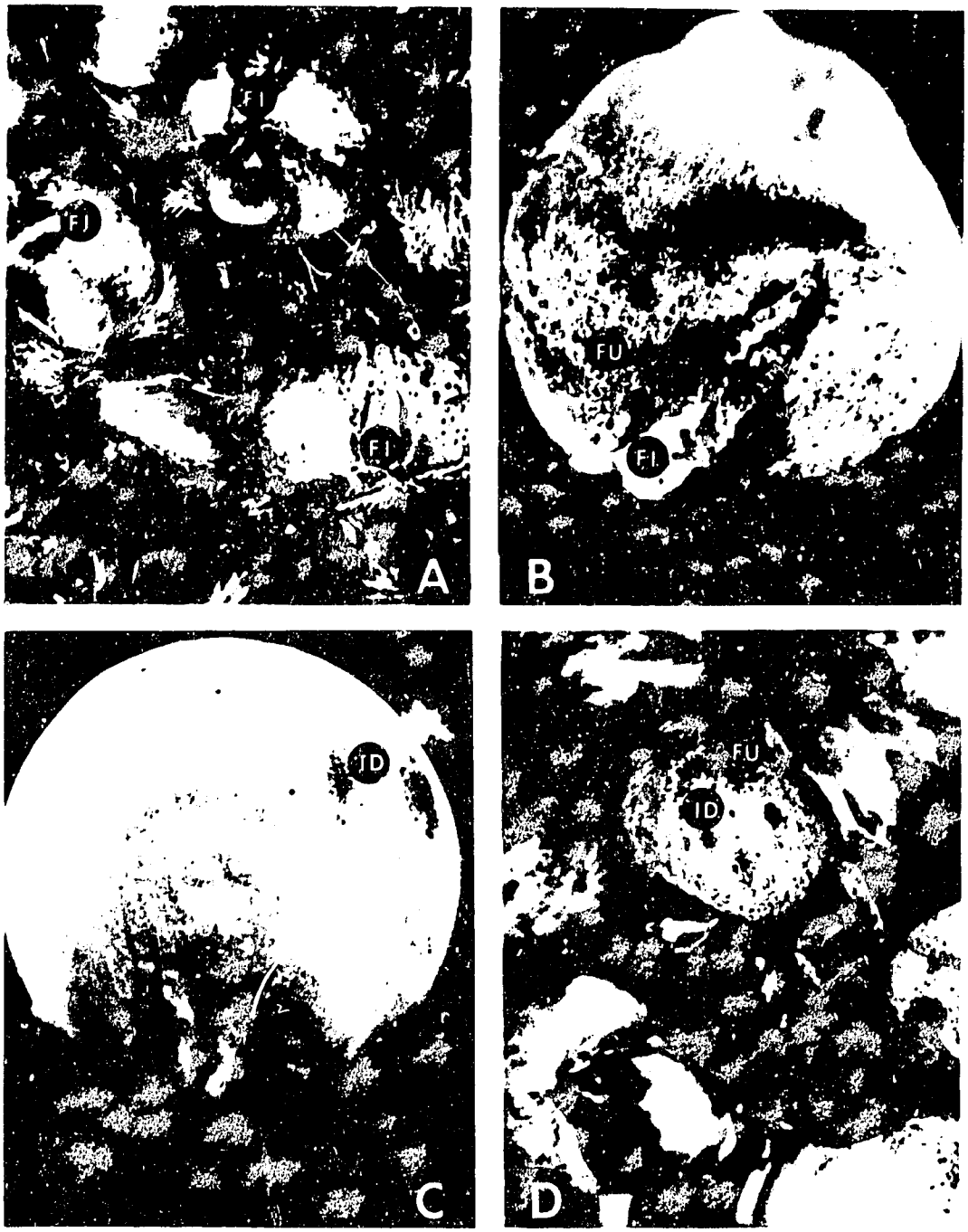

Figure 8. Fissures and insect damage of sorghum grain. Photo A) Fissured grain in the panicle which occurred after exposure to high moisture while in the field. FI – fissures, ca 2.5X. Photo B) Close-up of a sorghum kernel with a large fissure or break across the crown area as well as surface fungal growth. FU – fungal growth on the kernel surface, ca 5X. Photo C) Sorghum kernel damaged by an insect with piercing mouth parts. Darkened area surrounding and spreading from the puncture is due to fungal growth. ID – insect damage, ca 5X. Photo D) Grain with insect damage fungal growth in the panicle, ca 2.5X.

pericarp, and a highly corneous texture should give excellent mold resistance. Unfortunately, its food quality would not be good.

Chemical Characteristics

The grain of sorghum lines with resistance to deterioration does not differ significantly from susceptible grains in protein, fat, ash, fiber, and starch content. The tannin content of the brown grains (with a pigmented testa) was high, and probably accounted for their resistance. There were no real differences in tannin content that could be detected among the nonbrown grains. The nonbrown grains have polyphenolic compounds, which are possibly important. For instance, water extracts of all the nonbrown grains in the study retarded the growth of sorghum seedlings placed on filter paper containing the concentrated extract. Normal seedling growth was resumed when seedlings exposed to the water extract for 60 hours were switched to distilled water. The extracts did not inhibit germination, but they did retard the growth rate.

Paper chromatographic separation of the grain extracts resulted in great variability among lines in number and quantity of separated compounds (Fig. 9). Two high tannin lines, SC-0719 and GA-615, had fewer separated bands than other lines. Preliminary assays of the recovered separated bands indicated inhibitory as well as stimulatory responses of seedling growth to various bands. These data indicate the complex nature of these extracts. Further detailed analyses are needed.

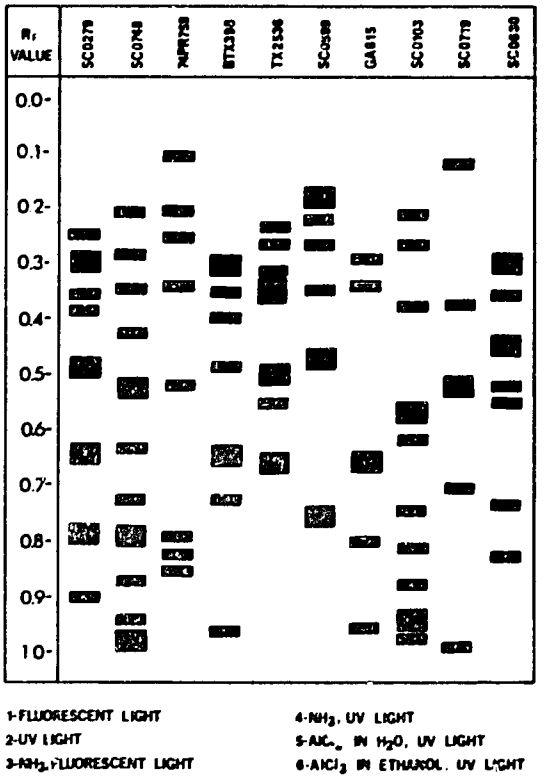

Figure 9. Chromatographic separations of water-soluble heat-stable extracts of sorghum grain. Bands from the extract of each grain type were visible under the conditions indicated by the number in the shaded band.

Epicarp Cell Appearance and Surface Wax

Initial observations of resistant and susceptible lines suggested that the surface wax quantity or coverage on the grain affected water uptake and grain mold. Measurements of surface wax on selected lines indicated that the quantity of wax was probably not a factor, since the more susceptible lines had equal or greater quantities of surface wax when compared to resistant lines (Table 4).

Examination of the surface of normal and dewaxed grains of susceptible and resistant lines revealed differences in the appearance of surface wax and the dewaxed epidermal cell surface. Alterations in the distribution of the wax on the surface of the pericarp (Fig. 10) may affect water uptake by the grain. The fungus found on the grain surface causes discoloration. However, penetration of the fungus through the epicarp cell surface was not observed. This supports the hypothesis that primary microbial entry is through the hylar and stylar areas, and not through direct penetration of the epicarp.

Water Uptake and Movement in the Grain

Water uptake and movement in wheat and sorghum has been studied (Grosh and Miller 1959; Jowett 1965). We have tried to determine the pathways by which water enters the kernel.

Table 4. Surface wax content of grain of sorghum lines susceptible and resistant to grain mold.

Lines	Quantity of surface wax	
	College Station[a] (%)	Dallas[b] (%)
SC-0279-14	0.267	0.290 d[c]
SC-0748	0.210	0.248 e
PR-759	0.327	0.356 c
SC-0103	0.220	0.218 f
BTX-398	0.233	0.260 de
TX-2536	0.387	0.453 b
SC-0630-IIE	–	0.520 a
SC-0719-IIE	–	0.233 ef
SC-0599	–	0.322 c
GA-615	–	0.288 d

a. Values from Dr. Larry Seitz, U.S. Grain Marketing Research Center, Manhattan, KS. Represents an average of early and late harvested grain from College Station in 1975 and grain from Dallas. Determinations were made with a benzene extraction methodology.

b. Average of two replications of 1977 grain samples from Dallas extracted for 10 minutes with hot chloroform and reported as percent wax of a dry grain weight basis.

c. Means followed by the same letter in each column are not different at the 5% level of significance, by Duncan's Multiple Range Test.

For a floury-endosperm sorghum, TX-09, the primary entry pathway for water is the disrupted connective tissue between the pericarp and rachis branch (Fig. 11). Water enters the cross and tube cells of the pericarp, and rapidly moves around the seed. Concurrently, water movement appears to be through the hilum (black layer) into the germ. After 30 min, water moved into the endosperm in the void area where endosperm, germ, and pericarp meet. Some water enters the kernel through the style, and moves around the kernel in the cross and tube cells.

After 60 min soaking time, water movement into the endosperm was greatest near the upper area of the scutellum. It is possible that water moves via the scutellum vascular system into the endosperm. This observation was more pronounced after 90 min.

Once the water was in the endosperm, it moved readily through the less-organized central floury endosperm. After 3 hr the water began to move into the corneous endosperm. At 7, water had moved through all areas of the kernel. The movement of water was followed by inbibing the kernels for a given time, cutting them in half, and observing them after exposure to iodine vapor. The starch grains turn blue when the moisture content reaches a certain level.

Breaks in the kernel altered the rate and pattern of water uptake. Field observations and laboratory water-uptake studies indicate that the rapid increases in moisture of sorghum grain may cause stresses which disrupt the cellular compactness of the caryopsis (Fig. 8). Kernels of some lines are more prone than others to breakage when subjected to moisture. Cracks in the kernel would allow more rapid water uptake as well as entry ways for fungi. In the corneous endosperm of wheat, uptake of water causes fissuring, which allows a more rapid entry of water into the endosperm (Grosh and Miller 1959). A similar situation may exist with sorghum; however, it was difficult to assess the amount of damage of advance of moisture in the corneous endosperm with the iodine-vapor methodology.

Coating the germ area with paraffin impaired water uptake by the kernel (Fig. 12). Covering the stylar half of the kernel with paraffin caused a reduction in water uptake compared with the control; complete control of water uptake occured when the hilar area was covered. This simple exercise supports the microscopic observations which indicate that the germ area is the primary pathway for water to enter the dry kernel. In the field, the stylar area may be of greater importance because the kernel is still attached to the glumes.

Evaluation of sorghum lines for rate of water uptake indicates that lines more susceptible to grain mold absorb moisture more readily than do resistant lines with similar grain characteristics (Fig. 13). Both TX-2536 and SC-0279 have thin translucent pericarps, average kernel size, and intermediate endosperm texture. The susceptible line, TX-2536, had a high rate of water uptake, whereas SC-0279, a resistant line, had a low rate of water uptake. Other lines with varying levels of resistance had water uptake rates that primarily reflect differences in mesocarp thickness and endosperm texture. Water uptake by grain of lines used to study a grain-leachate test was greater for susceptible lines than for resistant lines. Grain characteristics (endo-

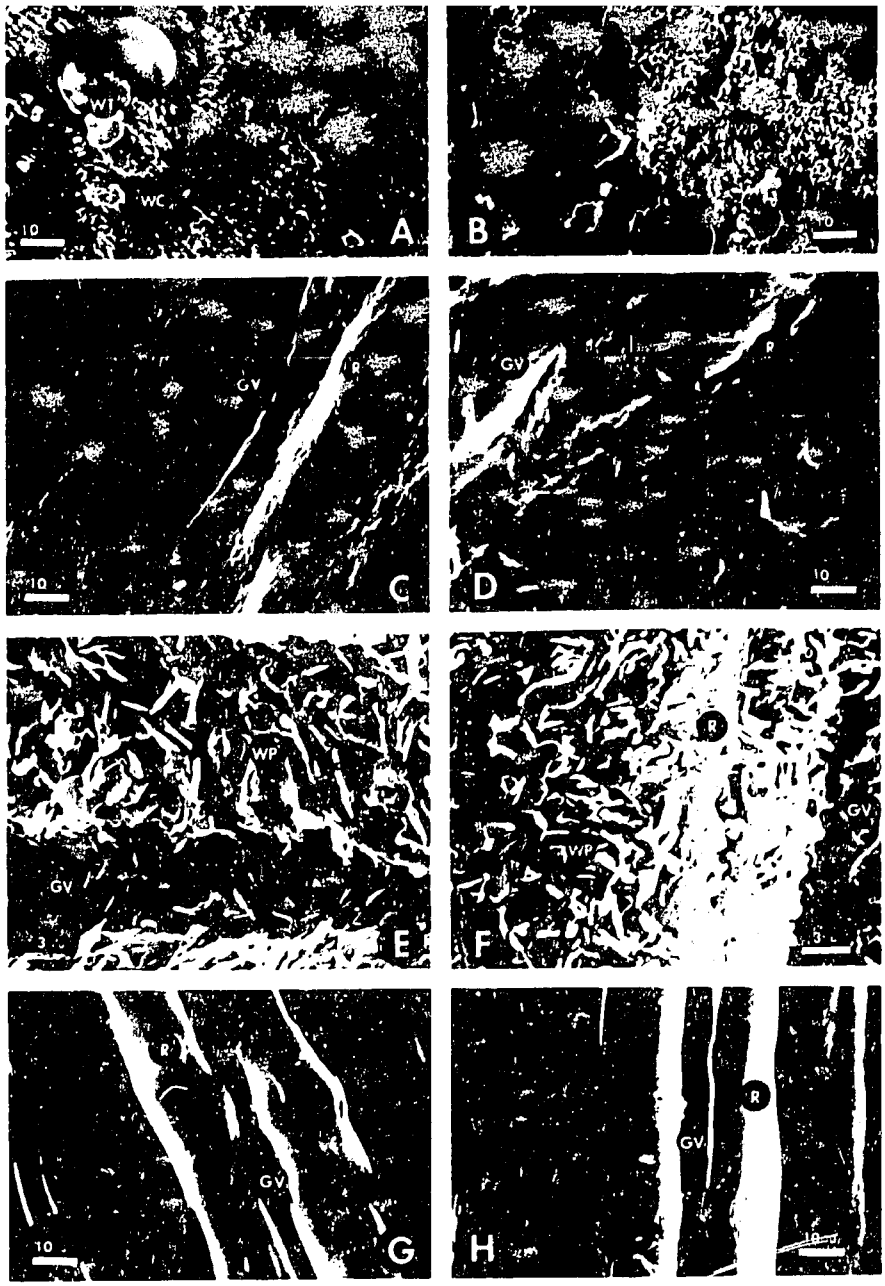

Figure 10. Surface wax and dewaxed epidermal cell-surface appearance of sorghum grain. Photo A) Surface wax of TX 2536 over the dorsal area of the grain, illustrating various wax formations. WT – wax tuft; WC – continuous wax layer; WP – flattened wax projections. Photo B) Surface wax of SC0279 over the dorsal area of the grain. Photo C) Dewaxed epidermal cell surface of TX 2536 over the dorsal area of the grain illustrating slight surface pitting and elongated appearance of cells. GV – grooves or valleys formed by epidermal cells; R – ridges. Photo D) Dewaxed epidermal cell surface of SC0279 over the dorsal area of the grain, illustrating a more-netted surface appearance than TX 2536. Photo E) Surface wax of TX2536 over the grain germ. Photo F) Surface wax of SC0279 over the germ. Photo G) Dewaxed cell surface over the germ of TX 2536, illustrating elongated tubular cell appearance. Photo H) Dewaxed cell surface over the germ of SC0279

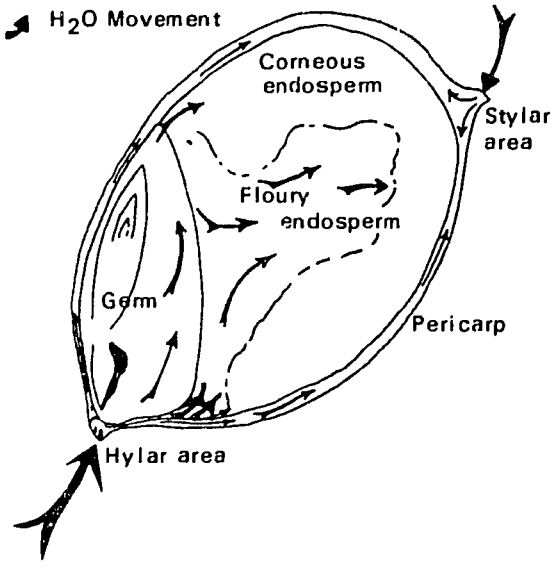

Figure 11. Water uptake and movement in a sorghum kernel. Arrows indicate primary water-entry areas and subsequent movement in the kernel.

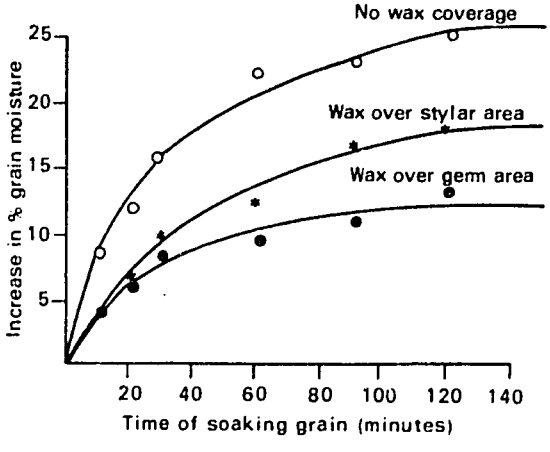

Figure 12. Effect of wax coverage of the germ or stylar areas of sorghum grain on the rate of increase in grain moisture.

sperm texture and pericarp thickness) and extent of deterioration both affect the rate of water uptake. Observations suggest that glumes slightly retard water uptake and loss. The relative differences among lines, however, were small.

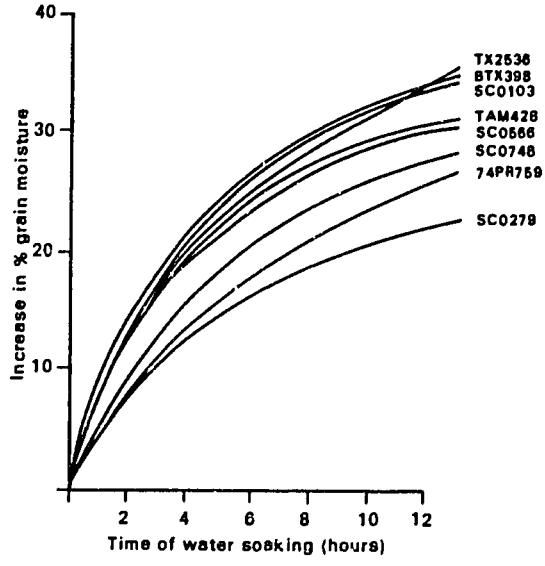

Figure 13. Effect of sorghum type on the rate of increase in grain moisture.

Stylar Structure at Maturity

Portions of the style branches remain attached to the mature caryopsis on the rounded apex opposite the point of caryopsis attachment (Fig. 14). Variability exists among lines in respect to length, position, or curvature of the style remnants, organization of the internal style tissue, and possible closing or plugging of the style openings with pectin or mucilage compounds.

The stylar branches of harvested threshed grain of TX-2536 are usually broken off, and the mesocarp tissue of the pericarp is exposed and vulnerable to entry by microorganisms or water (Fig. 14c). Harvested threshed grain of SC-0279 (Fig. 14b) and many of the more resistant lines tend to retain their stylar branches or greater portions of the branches when compared with TX-2536. This difference in breakage of the style indicates a structural difference among lines. Longitudinal sections of SC-0279, as well as scanning-electron microscopy (SEM) observations, suggest that the stylar tissue may be closed with pectin- or mucilage-type materials. The deposit of materials in the stylar tissue was also evident in other resistant lines — SC-0748 especially, as indicated by staining with safranin. In addition, TX-2536 was the only line examined

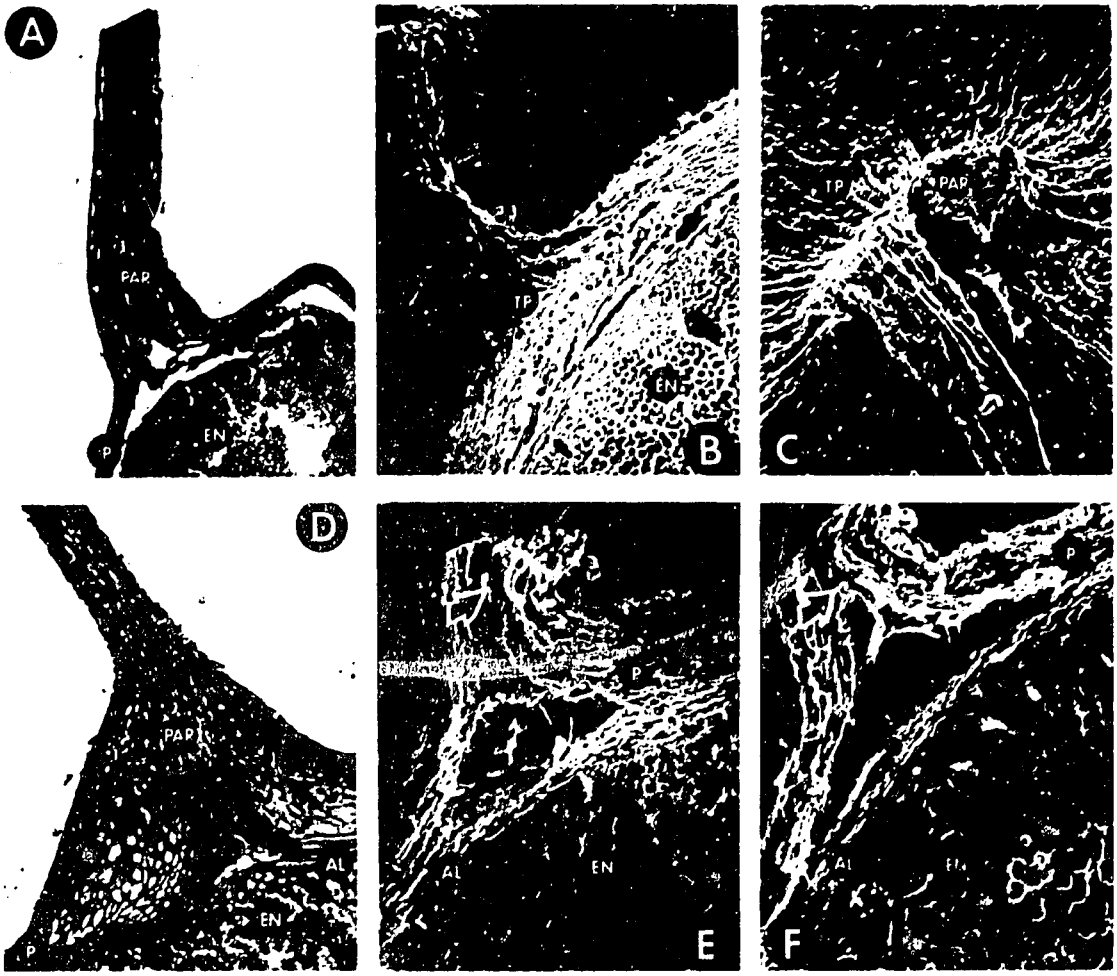

Figure 14. Stylar area of sorghum lines resistant and susceptible to preharvest grain deterioration. Photo A) Longitudinal section of style of SC0279 (resistant), 18 days postanthesis. En – endosperm; P – pericarp; PAR – parenchyma tissue of inner style; ca 40X. Photo B) Styles of SC0279 kernel. TP – tubular shaped outer pericarp cells; ca 100X. Photo C) Surface view of stylar area of TX2536 (susceptible) with one style broken off near the base, exposing the inner parenchyma tissue, and the other style lying perpendicular with the pericarp surface; ca 100X. Photo D) Longitudinal section of TX2536, 18 days postanthesis; ca 40X. Photo E) Longitudinal section of SC0279, 48 days postanthesis, illustrating constricted stylar opening (arrow). AL – aleurone cell layer; ca 200X. Photo F) Longitudinal section of susceptible line 48 days postanthesis, illustrating more-open stylar area; ca 200X.

in which surface discoloration proceeded from the stylar area. Appearance of the grain of TX-2536 suggested that the microorganisms had entered the pericarp through the style, and as they advanced through the pericarp the translucent pearly appearance was lost due to cellular disruption.

Hilar Development and Structure

Comparison of the hilar region of mature grain of TX-2536 and SC-0279 (Fig. 15) suggests a more organized complete closure of the translocation tissue in SC-0279, compared to TX-

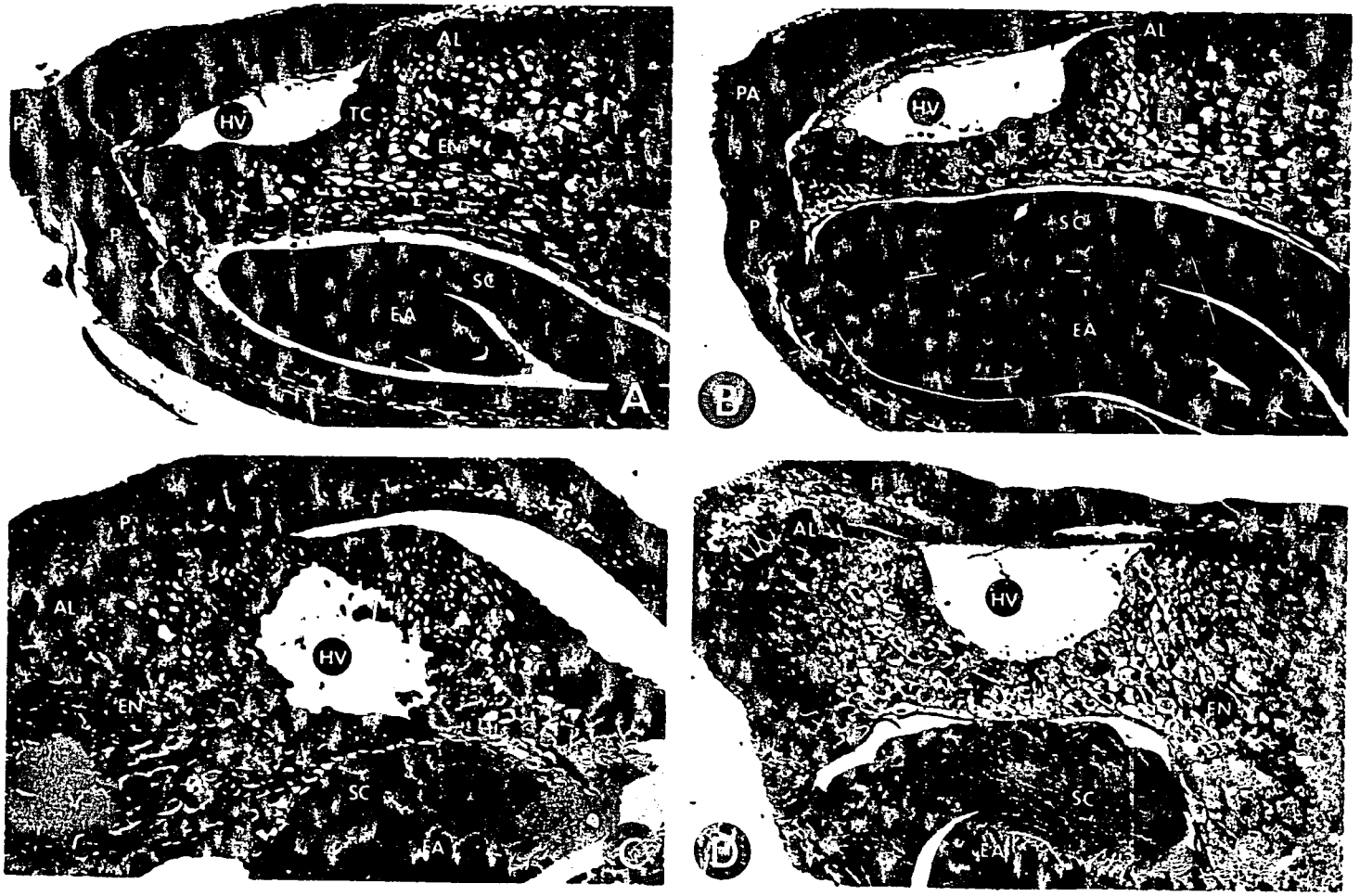

Figure 15. Hilar area of sorghum lines, both resistant and susceptible to preharvest grain deterioration. Photo A) Longitudinal thin section of the susceptible line, TX2536, 10 days postanthesis. EN – endosperm; EA – embryonic axis; HV – hilar void; TC – transfer cells or modified endosperm cells; AL – aleurone cell layer; SC – scutellum; P – pericarp; PA – point of grain attachment; ca 25X. Photo B) Longitudinal thin section of the resistant line SCO279, 18 days postanthesis; ca 25X. Photo C) Cross section of TX2536, 38 days postanthesis; ca 25X. Photo D) Cross section of SCO279, 38 days postanthesis; ca 25X.

2536. Cross sections of TX-2536 depict disruption of the closure tissue along with voids in the endosperm, which would provide reduced resistance to water and microorganism entry. In contrast, cross sections of the hilar area of SC-0279 suggest an organized complete closure of the hilar region. Although much of the pigmented black layer can be removed by removal of the pericarp, pigmented tissue remains, and it appears continuous with the aleurone layer. This continuous tissue layer is especially evident in grain of SC-0279 and other more resistant lines. It is not so apparent in TX-2536, a susceptible line. Wolf et al. (1952) reported the hilar layer splitting into an upper and lower layer in corn; however, Giles et al. (1975) failed to mention hilar layer separation in the sorghum grain they examined.

During caryopsis development, the hilar area serves as the site of translocation of nutrients from the vegetative plant portions into the ovule. If microorganisms enter into the vascular system of the plant, they could enter the caryopsis and possibly gain entry into the embryo and endosperm through the hilar area. Translocation of nutrients into the developing endosperm appear to be via specialized basal endosperm cells or transfer cells (Giles et al. 1975; Gunning and Pate 1969) (Fig. 15). Sections of grain from different lines were 15μ thick, and it was not possible to discern differences among lines in transfer-cell structure. Since one of the primary differences between susceptible and resistant lines was the rapid degradation of endosperm of susceptible lines, some type of barrier must be restricting microorganism movement into the endosperm of resistant lines. This barrier is probably due to differences in transfer-cell structure and/or the structure and continuity of the aleurone cell layer, as well as the overall organization of the endosperm in this critical area of pericarp, endosperm, and germ association.

Grain shape, especially in the abgerminal area near the black layer, appears to affect the progression of grain deterioration. As grain of TX-2536 reaches physiological maturity and begins to lose moisture, extreme indentation of the black layer in relation to the yoke of surrounding endosperm is observed. This mature grain conformation may relate to the cellular disruption observed in the area where the hilum, endosperm, and germ come together.

Summary

A multitude of factors affect the development of expression of mold and weather deterioration of sorghum grain. Nine lines with high levels of resistance — SC-0279, SC-0748, SC-0599, SC-0566, SC-0630, PR-759, BTx-398, SC-0103, and SC-0719 — were identified. The first seven lines are of primary importance because they do not contain the testa layer and have low tannin levels, whereas SC-0103 and SC-0719 possess a pigmented testa layer. The identification of these lines resulted primarily from visual field ratings of the grain at six to eight locations throughout Texas for 2 to 4 years of evaluation. Other lines exhibited high levels of resistance at limited locations or during some years, but they did not possess those qualities imparting general resistance.

The seven nonbrown resistant lines originated from varied backgrounds. Amount and appearance of surface wax, as well as dewaxed epicarp surface appearance, varied among grain types. In SC-0279 and BTx398 sorghum kernels, the pericarp surface near the black layer is more completely covered with wax than are kernels of TX-2536. However, there were no consistent differences that would explain the improved resistance to deterioration. Separation of the water-soluble heat-stable grain extracts indicated variability in number, type, and quantity of compounds in the different grains. The lemon-yellow line, SC-0748, had several unique bands of compounds which restricted seedling growth; however, other lines also restricted seedling growth. The extract of SC-0630 had only a few bands, which was also true of the brown sorghum lines SC-0103, SC-0719, and GA-615. SC-0630 had less sprouting in the panicle during the rains in southern Texas in 1976. Since the brown sorghums usually possess some level of grain dormancy, similarities in grain extracts suggest a chemical inhibition to germination in SC-0630.

The more prominent factors associated with resistance to preharvest grain deterioration relate to moisture loss, movement in the grain, and uptake. The longer the period of time the moisture content of the grain is at 18 to 20% or more, the more prolific will be microorganism growth. TX-2536 tends to dry down slowly in comparison with more resistant nonbrown lines; thus a favorable atmosphere for micro-

organism growth is maintained. Grain that has undergone some deterioration and cellular disruption takes up water readily; thus the progression of deterioration is enhanced. Undamaged excellent quality grain of TX-2536 has increased rates of water uptake when compared with thin-pericarp resistant types (SC0279). This may relate to grain structure in the hilar area. The more disorganized appearance of the hilar area and associated endosperm and germ tissue of TX-2536 suggests greater accessibility to microorganism establishment and growth. Grain shape, continuity of the aleurone layer in the hilar area, and the organized solid appearance of grain of most resistant nonbrown lines would affect water uptake and movement in the grain, and microorganism entry and ultimate expression of preharvest grain deterioration. In addition, the initial discoloration of the pericarp of TX-2536 occurs at the stylar area, which suggests the entry of microorganisms and moisture through the style. Since this type of pericarp discoloration was not observed in the resistant lines, there may be some obstruction in the style tissue that prevents entry. This obstruction appears to be a pectin- or mucilage-type material.

The studies discussed here have shown that certain structural features of the sorghum kernel may play an important role in limiting the movement of water and entry of microorganisms into the sorghum kernel. Additional studies are needed to compare sorghum kernels from lines with closely related plant and kernel characteristics that have good and poor resistance to deterioration. Close interaction and collaboration among scientists will be necessary to conduct the critical tests required to clarify which of the multitude of factors have the greatest influence.

Acknowledgment

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Mycotoxins in Sorghum: Toxigenic Fungi During Storage and Natural Occurrence of T2 Toxin

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The importance of mycotoxins in human foods has been recognized in recent years. Mycotoxins may be potentially harmful to various human organs such as the liver, kidney, and gastrointestinal tract. From the standpoint of health, harmful effects could be appreciable when staple foods are contaminated, since the amount ingested over time will be considerable. Under normal conditions of harvest and storage, the risk of aflatoxins in a staple like rice is much less than when it is stored under abnormal conditions — such as cyclone, floods, and unseasonal rains (Tulpule and Bhat 1978). Two major disease outbreaks — aflatoxic hepatitis and enteroergotism — attributable to mycotoxins have been reported from India (Bhat and Krishnamachari 1978).

In India, mycotoxins in sorghum have not been studied extensively. A screening investigation on head molds of sorghum grains in Pantnagar, UP, showed contamination with *Aspergillus, Colletotrichum, Curvularia, Fusarium, Penicillium*, and *Phoma*. The species of *Aspergillus* isolated resembled *A. flavus*. Aflatoxins B^1, B^2, G^1, and G^2 were detected in samples of sorghum infected with head molds (Tripathy 1973).

One of the promising methods of preventing aflatoxin accumulation in food grains is to select varieties that will support the least amount of aflatoxins. Varieties of corn, peanut, sunflower, and soybean promising in this regard have been identified (Tulpule et al. 1978). However, the stability of the resistance character under different climatic conditions must be studied. Research in the Department of Plant Pathology, Andhra Pradesh Agricultural University at Hyderabad, is under way to select varieties of sorghum that resist aflatoxin production.

Besides aflatoxins, ergot alkaloids may occur in sorghum. Most, if not all, varieties of sorghum grown in Karnataka and Maharashtra in India are susceptible to *Claviceps* spp. Ergoty grains of sorghum generally ramain soft and rarely harden.

As part of the program of mycotoxin surveillance of staples, studies have been undertaken at the National Institute of Nutrition on screening various commodities such as rice, maize, pearl millet, and sorghum for mycotoxins (Tulpule and Bhat 1978). This paper deals with our studies on sorghum.

Mycoflora Pattern in Stored Sorghum Samples

Sixty-four samples of sorghum collected from the villages of Krishna and Kurnool in Andhra Pradesh were examined. The samples were collected mostly from "Patra," the most common storage structure in either district. Patra is an underground pit lined with paddy straw or straw rope. In Kurnool district, samples were also collected from other storage structures such as "jade," "garise," and gunny bags. Jade is a big earthern jar. Garise is a stone or cement bin constructed inside the house, sometimes provided with a stone slab bed that may be plastered with cement/mud/dung. The moisture content of samples obtained from Krishna district ranged from 10.3 to 14.4%, while that of the samples obtained from Kurnool district ranged from 10.0 to 15.5%. *Aspergillus flavus* was isolated from 50% of the samples collected from Kurnool district and from 77.5% of the samples obtained from Krishna district. The percentage of grain infected in each sample ranged from 4 to 28%. In about 40% of samples with *A. flavus*, the percentage was 8 or less. In 57% of the samples, other species of *Aspergillus* were also isolated. In 42% of the samples, other fungi — such as *Alternaria, Curvularia, Chaetomium, Fusarium*, and a few nonsporulating fungi — were present. Aflatoxin was not found in any of the samples.

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These studies have indicated that toxigenic fungi can be present in the sorghum samples stored under usual conditions. However, unless certain environmental conditions (such as excess moisture) exist, toxin production will not take place.

Natural Occurrence of T2 Toxin in Ingested Moldy Sorghum

Fungi such as *Fusarium* and *Myrothecium*, which belong to the group "fungi imperfect," produce a class of mycotoxins collectively known as Trichothecenes. Among these, T2 toxin is the most important (Saito and Ohtsubo 1974). Circumstantial evidence has implicated T2 toxin in disease outbreaks in man and in animals. "Alimentary toxic aleukia," prevalent during World War II in the USSR, may have been due to T2 toxins.

Species of *Fusarium* are often found in sorghum infected with head molds. Other fungi that invade sorghum grains during rainy harvest periods are *Drechslera*, *Alternaria*, and *Curvularia.* Extracts from moldy sorghum and from nonmoldy sorghum were injected intraperitoneally into rats; toxic symptoms in rats receiving extracts from the moldy sorghum included reduction in weight gain, general weakness, sluggishness, and untidy fur. Acute inflammatory lesions were revealed on pathological examination of the gastrointestinal tract.

When the extracts were applied externally to the shaved skin of guinea pigs, rats, and rabbits, the skin showed marked redness, inflammation, and crust formation. Other signs included pinpoint hemorrhage, scab formation, and subdermal hemorrhage.

Some of the fusarial toxins — such as zearalenone — exhibit uterotrophic activity. To determine if moldy sorghum extracts show such toxicity, they were given orally to weanling female rats. After 24 hours, the animals were sacrificed, and uterine weights were taken. Uterine weights of the treated animals were not significantly different from those of control rats.

The *Fusarium* species isolated from moldy sorghum was identified as *Fusarium incarnatum*, belonging to the arthrosporiella group. It may be appropriate to mention that the system of speciation in *Fusarium* is far from satisfactory. However, since the isolate of *Fusaria* obtained is a tropical one, the classification proposed by Subramanian (1971) was followed in species identification.

The fungus *F. incarnatum* was inoculated on autoclaved rice. After 7 days of incubation, the artificially contaminated rice was dried and extracted with solvents such as petroleum ether, ether, and ethyl acetate. Similar extracts were obtained from noninfected rice for use as a control.

Toxicological investigations with extracts of *F. incarnatum*-infected rice included intraperitoneal injection of rats, dermal application to guinea pigs, and oral feeding to weanling rats. Effects were similar to those with moldy sorghum extracts, clearly indicating that the toxicity of moldy sorghum was primarily due to mycotoxins elaborated by *F. incarnatum.*

Extracts from moldy sorghums as well as from rice infected with *F. incarnatum* were partially purified by passing over a column of silica gel after partitioning with petroleum ether. The solvent mixture of n-hexane-ethyl acetate in the ratio of 1:3, 1:5, and 1:7 was successively passed through the column. The effluents were collected separately, and spotted over silica gel-coated thin layer chromatography plates. The plates were developed separately in various solvents systems, such as chloroform-methanol (97:3), ethyl acetate-n-hexane (3:1), and toluene-ethyl-acetate-ethyl ether (90:5:5). After development, the plates were air dried at 110°C for 10 min. In the second fraction, i.e., n-hexane-ethyl acetate (1:5), a sky-blue fluorescent spot was visible under ultraviolet light. This compound was identified as T2 toxin. Further chemical confirmation of T2 was done after crystallizing the toxin. Melting point, IR spectrum, NMR, and GLC pattern of the T2 toxin from rice infected with *F. incarnatum* were identical with authentic T2 toxin.

Although natural occurrence of T2 toxin in sorghum is known, disease outbreaks attributable to this mycotoxin have so far not been identified in sorghum-eating populations. This may be mainly because of the lack of correlation between vague symptoms of poisoning — such as diarrhoea and vomiting — and identification of the *Fusarial* toxin in moldy sorghum This is precisely the area that needs an in depth collaborative study by epidemiologists, clinicians, mycologists, and toxicologists.

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Factors Affecting the Development of Sorghum Grain Molds in Senegal

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Sorghum is the second most important cereal in Senegal — after pearl millet — and there are plans to increase its production through intensification. This is not possible with traditional varieties because of their height, photoperiod sensitivity, lateness, and little or no response to mineral fertilization. Thus there are increased efforts to obtain improved sorghum varieties.

Two sorghum improvement programs are under way, one covering the northern and central northern zones of Senegal and the other covering the more humid regions to the central south and southeast of the country. One of the important objectives of the second program is the development of varieties of relatively short duration (90 to 105 days) that are grain mold resistant. These varieties mature early, before the end of the rainy season, and should resist molds to ensure that the grain is fit for human consumption.

Methods

In an earlier article (Denis and Girard 1977), we described in detail the different methods that could then be used to identify mold resistant varieties. In this paper, we present only those that are most commonly used in Senegal.

Field Trials

Since the climate is unpredictable, mold development is not always adequate for proper screening of varieties. In Senegal there are two methods for ensuring high infestation pressure while selecting for mold resistance:

- Planting in Bambey in June under irrigation, before the start of the rainy season, so that generally photoinsensitive varieties mature during a very wet period (Aug to Sep).
- Planting in Sefa in mid-Casamance, in a region where conditions are more humid than those required for the varieties.

At a later stage the selected lines are tested over several years and in locations in the area where they will be ultimately cultivated.

Methods for Analysis and Evaluation of Molds

Several methods are used for evaluating the resistance capacity of varieties:

- A rapid technique where threshed grains are compared, using a hand lens, to grain ranging from nonmolded (1 rating) to very molded (10 rating), (a technique developed by Dr. Natale Zummo and thus called the Zummo scale).
- A laboratory technique for qualitative and quantitative evaluation of molds, through estimation of the grain surface covered by mold and the proportion of grains carrying the fungus.
- Seed germination tests.

As the results are expressed in percentage values, the observations are subjected to angular transformation before statistical analysis.

Presentation and Discussion of Results

Grain Infection Stage and Relative Importance of the Fungi

Ten days after flowering, grains were taken at 5-day intervals from panicles of two sorghum varieties (CE 90 and 68–25) that had flowered on the same day. These samples were analyzed in the laboratory to determine the percent molded area and the composition of the mycoflora by

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incubating the grains on filter paper for 24 and 48 hr at 27 to 28° C.

Damage due to molds is first apparent on grain about 30 days after flowering (Table 1). However, certain types of molds (*Fusarium, Curvularia*) can be identified from the first sampling 10 days after flowering; this signifies that they are established relatively early in the grains, but develop only when the grains are close to maturity (Table 2). It was possible to verify that these fungi can be present on the panicles as early as anthesis. *Curvularia* predominates at the start of grain development, while *Fusarium* predominates at maturity (Table 2). *Curvularia* and *Fusarium* tend to infect grain in inverse proportion during grain development. These two fungi were the most important and *Fusarium* is apparently more closely associated with percent molded area.

Yield, Height, Duration Relationships with Moldiness

A four-replicate trial with 54 F_5 progeny derived from 67–17 × CE 90, and the parents, was planted at Bambey, Darou, and Nioro in

Table 1. Index[a] of molded area of sorghum grain taken at different dates after flowering (17 Sept 1974).

Varieties	Number of days after flowering						
	10	15	20	25	30	35	40
CE 90	0	0.1	0.2	2.8	50.9	54.1	58.7
68-25	0	0.6	1.1	15.0	79.6	79.1	81.5

a. $\text{Index} = \dfrac{n1 \times 0 \times n2 \times 10 + n3 \times 50 + n4 \times 100}{n}$

Where n1, n2, n3, n4 are the number of grains belonging to respective categories 1, 2, 3, 4 given below:

1 = visible molded grain area < 1%
2 = visible molded grain area ≥ 1% and ≤ 19%
3 = visible molded grain area ≥ 20% and ≤ 79%
4 = visible molded grain area ≥ 80%
and n is the total number of grains observed

Table 2. Percentage of sorghum grains infected with various fungi harvested at different dates after flowering.

Varieties/Fungi	Number of days after flowering						
	10	15	20	25	30	35	40
CE-90							
Fusarium	26	22	35	46	51	76	61
Curvularia	71	89	75	46	41	10	27
Helminthosporium					6	4	5
Pycnidial fungus	6			5	6	1	4
Others				5	3	10	8
68-25							
Fusarium	21	30	49	48	52	59	67
Curvularia	41	76	78	45	44	39	30
Helminthosporium			1	1	6		2
Pycnidial fungus	37				6	3	2
Others				1	7	2	2

Senegal. Moldiness (Zummo scale) was determined twice by three observers on grain samples taken at threshing. The mean of observations per variety and location were used to calculate the coefficients of correlation between yield, height, plant maturity, and moldiness of grains (Table 3).

Table 3. Coefficients of correlation with the Zummo scale.

	Yield	Height	Maturity
Bambey	–.468**[a]	–.569**	–.240
Darou	–.278*	–.070	–
Nioro	–.216	–.120	–.244

a. Significance thresholds of the coefficient of correlation with 54 degrees of freedom; 5%(*).264, 1%(**).342.

The results for Bambey indicate a negative relationship between moldiness and yield as well as plant height. Apparently, the least molded plants are the tallest.

At Darou, where plant height was low and mold infection was high in comparison with Bambey (Table 4), the relationship between moldiness and yield was negative, but it is less negative than in Bambey. Moreover, there was no relationship with plant height.

At Nioro, where plant height was much lower but mold infection was similar to that in Darou, there was no relationship between these different criteria and moldiness. This is undoubtedly due to the presence of more important limiting factors. We believe that when there are not many factors limiting plant development, grain molds have a more definite relationship with yield and plant height. In all cases, plant maturity seems to be less related to moldiness in these varieties.

Table 4. Average yield, height, time to flowering, and mold rating of the 56 varieties tested.

Locations	Yield (kg/ha)	Height (cm)	Flowering (days)	Zummo (1–10)
Bambey	6157	245	71	5.04
Darou	5112	229	–	5.85
Nioro	2846	179	64	5.90

Relationship between Locations and Grain Molds

In an eight-replicate trial, four varieties were tested at four ecologically different locations. An analysis of combined variance of the results (Table 5) has shown that differences between locations are highly significant for all the characters considered — percent molded area and percentage of grains carrying *Fusarium*, *Curvularia*, and a pycnidial fungus.

The varieties differ only for percent molded area, while the variety × location interaction, like the locations, is always significant. Performance of varieties in relation to fungi was not significantly different over all locations but differences occur between them at each individual location (Table 6). Varieties should therefore be recommended according to locations. These results require confirmation by long-term experiments. If these results are consistent, they should be explained by interaction between fungi in terms of their qualitative and quantitative presence and environmental conditions.

Relationship between Locations, Years, and Grain Molds

Eight varieties were compared in a trial with three replicates conducted in 1975 and 1976 at Bambey and Nioro.

Combined analysis of variance (Table 7) indicates that locations, and years, as well as their interaction, differ significantly for percent molded area, nongermination rates, and percentage of grain carrying the pycnidia. These sources of variation do not differ significantly for the percentage of grain carrying *Fusarium* and *Curvularia*, except the locations × years interaction for *Fusarium*, which is highly significant. We interpreted this by saying that these fungi are always present in large quantities, whatever the year or location.

The results also show that the performance of varieties, in relation to all the fungi considered, changed with location and year. This verifies the earlier hypothesis, knowing that the fungi interact among themselves.

Table 5. Analysis of combined variance and averages of sorghum varieties grown at Boulel, Darou, Nioro, and Sefa for molded area and genera of fungi present.

Sources of variation	D.F.	Molded area	Fusarium	Curvularia	Pycnidial fungus
A. Variances					
Location	3	1432.01**[a]	2272.67**	2401.94**	4455.09**
Error (a)	28	1.62	15.17	49.35	56.28
Replicates between trials	31	140.04	233.64	277.02	481.97
Varieties	3	95.38*	88.6	93:49	389.49
Varieties × Locations	9	21.46**	261.55**	74.46*	311.68**
Error (b)	84	4.90	34.56	30.02	67.37
Total	127				
B. Averages of varieties					
1. 7531-V33=7410-122-5		18.31	33.24	35.15	23.12
2. 7531-V1=7410-140-1-1		19.54	36.91	32.56	26.71
3. 7531-V36=7410-122-3		20.65	34.82	36.57	28.45
4. 7531-V35=7410-186-1		22.37	33.56	35.58	31.48
LSD 5%		1.10	2.92	2.73	4.08
C. Averages over locations					
Boulel		10.37	26.26	25.76	15.69
Darou		22.26	29.99	42.94	29.87
Nioro		22.95	36.85	41.76	21.37
Séfa		25.28	45.43	29.41	42.83
LSD 5%		0.65	2.00	3.60	3.84

a. Differences are significant at 5% (*) and 1% (**) levels, respectively.

Relationships between Certain Sorghum Grain Molds

During the 1977 rainy season, 44 varieties were studied in a trial with four replicates for their performance in relation to grain molds. The following fungi were studied: *Fusarium, Curvularia, Helminthosporium, Alternaria, Cladosporium, Colletotrichum*, the pycnidial fungus and other unidentified fungi referred to as "other fungi." The different coefficients of correlation between the fungi were calculated using the averages over four replicates (Table 8).

Fusarium and *Curvularia* are the most important fungi among those studied. These fungi interact with all the other fungi. *Fusarium* is negatively related with *Helminthosporium*, the pycnidial fungus, and *Colletotrichum*, and positively with the "other fungi." *Curvularia*, on the other hand, is negatively related with "other fungi" and positively with *Helminthosporium, Alternaria*, and *Cladosporium*. There is probably an antagonistic relationship between *Fusarium* and *Curvularia*.

Relationship between *Fusarium* and *Curvularia* on Sorghum Grains

A trial was conducted at Bambey during the 1974 rainy season with staggered planting of seven varieties showing different performance in relation to grain molds. Observations were carried out on six flowering dates at 3-day intervals for percent molded area and percentage of grains carrying *Fusarium* and *Curvularia* (Table 9).

Table 6. Development of grain mold and frequency of isolation of *Fusarium Curvularia* and the pycnidial fungus at Boulel, Darou, Nioro, and Séfa.

Varieties	Boules	Darou	Nioro	Séfa
		Molded area		
V1. 7531V33 (7410-122-5)	8.17	18.73	21.34	25.00
V2. 7531VI (7410-140-1-1)	8.10	24.54	21.80	23.71
V3. 7531V36=7410-122-3	11.46	21.31	23.42	26.42
V4. 7531V35 = 7410-186-1	13.76	24.47	25.25	26.00
		Frequency of *Fusarium* isolation		
V1. 7531V33 (7410-122-5)	19.63	32.64	32.21	48.49
V2. 7531V1 (7410-140-1-1)	24.40	37.30	40.93	45.00
V3. 7531V36 = 7410-122-3	36.49	24.49	35.02	43.27
V4. 7531V35 = 7410-186-1	24.53	25.52	39.24	44.96
		Frequency of *Curvularia* isolation		
V1. 7531V33 (7410-122-5)	26.02	40.00	45.79	28.79
V2. 7531V1 (7410-140-1-1)	19.15	42.98	37.90	30.22
V3. 7531V36 = 7410-122-3	30.33	43.61	41.51	30.85
V4. 7531V35 = 7410-186-1	27.56	45.15	41.82	27.79
		Frequency of Pycnidial fungus isolation		
V1. 7531V33 (7410-122-5)	9.91	28.31	13.05	4.22
V2. 7531V1 (7410-140-1-1)	7.38	23.19	30.54	45.72
V3. 7531V36 = 7410-122-3	18.92	32.92	22.32	39.64
V4. 7531V35 = 7410-186-1	26.56	35.06	19.56	44.73

Effect of Molds on Sorghum Grain

The effects of molds on sorghum grain observed to date are given in Tables 10 and 11. The molded area of grains increases with the concentration of *Fusarium* and other fungi (Table 10). It is also seen that the unidentified fungi contribute to increased moldiness of grains (Zummo scale), and the nongermination of seed as well as the number of abnormal seedlings. Therefore it would be useful to identify them. Finally, we can say that *Fusarium* impedes seed germination.

The interpretation of the positive relationship presented is not arbitrary and is therefore acceptable. This is not the case for interpretation of negative relationships. With present knowledge, it is difficult to understand that grains are less molded and germination is better as the pycnidial fungus increases, or that grain appearance and germination are better and there are fewer abnormal seedlings as *Alternaria* increases.

It also appears that molds have a depressive effect on the chemical composition of grain (Table 11). Results show a significant decrease in protein rates in the highly molded sample compared to other samples. Content of amino acids isoleucine and valine is also lower in highly molded samples. Lysine content is a limiting factor in both healthy and molded sorghum.

Fusarium, Curvularia, Helminthosporium, and the pycnidial fungus could all be involved in the deterioration of grain food value (Table 11). However, further details are required to verify if the observed differencs are statistically significant and biologically important, and to determine the fungi causing most damage.

Conclusions

These few results enable us to appreciate the complex nature of the problem. Several fungi have been identified, and those that are yet to be identified are relatively important. A certain hierarchy could be established however, with

Table 7. Combined analysis of variance for microflora of sorghum grains at Bambey and Nioro, 1975–1976.

Sources of variation	D.F.	Molded Area	Non-germinated	Fusarium	Curvularia	Pycnidial fungus
A-Variance						
Between trials	(3)					
Locations	1	60.87***[a]	5398.03**	0.69	258.33	2582.75**
Years	1	3.05**	1902.73*	33.58	54.90	3192.43**
Locations × Years	1	853.71**	1383.43*	420.01**	182.99	252.27*
Error (a)	8	0.19	239.69	15.88	76.08	41.71
Replicates	(11)					
Varieties	7	656.27**	956.43**	107.44**	139.51**	598.05**
Varieties × Locations	7	39.05**	266.13**	80.82**	39.33**	270.76**
Varieties × Years	7	39.53**	157.06**	38.25**	90.98**	631.28**
Var × Loc × Years	7	36.52**	164.92**	23.84	84.76**	324.07**
Error (b)	56	0.48	35.26	12.03	12.85	24.97
Total	96					
B-Average of varieties						
1 NKX-3183		20.22	48.15	38.27	27.23	35.43
2 B-3197		25.31	46.98	44.70	27.15	28.87
3 TAM-680		21.23	48.26	41.32	28.96	29.05
4 TAM-420		22.63	56.99	48.38	20.74	15.99
5 CE-90		21.46 56.58	43.95	28.58	31.36	
6 7420062-2		25.96	48.78	44.32	33.07	22.74
7 7403048-1		28.43	52.64	42.92	28.76	36.23
8 M 35-1		42.98 73.53	45.59	28.29	21.53	
LSD 5%		0.57	4.85	2.83	2.93	4.08

a. Differences are significant at 5%(*) and 1%(**) levels, respectively

Table 8. Correlation coefficients among the fungi observed on molded sorghum grains on 44 varieties at Séfa 1977.

	Fusarium	*Curvularia*	*Helminthosporium*	*Alternaria*	*Cladosporium*	*Pycnides*	*Colletotrichum*	*Others*
Non germinated	.481***[a]	–.445	–.360**	–.444**	–.277	–.442**	.082	.481**
NG and Abnormal	.167	–.421**	–.141	–.528**	.012	–.264	.196	.457**
Fusarium	–	–.206	–.492**	–.202	.225	–.516**	–.304	.377*
Curvularia	–.206	–	.356*	.383*	.343*	–.080	.039	–.453**
Helminthosporium	–.492	.356	–	.082	.161	.178	.155	.182
Alternaria	–.202	.383	.082	–	.190	.004	–.257	–.162
Cladosporium	–.225	.343*	.161	.190	–	–.155	–.195	–.190
Pycnides	–.516**	–.080	.178	.004	–.155	–	.259	–.207
Colletotrichum	–.304*	.039	.155	–.257	–.195	.259	–	–.105
Others	.377*	–.453**	.182	–.162	–.190	–.207	–.105	–

a. Significantly different coefficients of correlation from 0 to probability thresholds of 5%(*) and 1%(**) respectively

Table 9. Relations between grain molds and flowering dates (Bambey 1974).

Varieties	Ranking by mortality		Flowering dates 14/9	17/9	20/9	23/9	26/9	29/9	Averages
		Molded area	70.2	23.3	–	14.8	20.8	25.6	30.9
68–19	1	*Fusarium*	69.0	57.0	–	74.0	58.0	72.0	66.0
		Curvularia	22.0	23.0	–	15.0	27.0	13.0	20.0
		Molded area	41.1	23.3	–	16.0	28.5	13.1	24.4
Meloland	2	*Fusarium*	48.0	68.0	–	67.0	50.0	74.0	61.4
		Curvularia	41.0	25.0	–	8.0	35.0	19.0	25.6
		Molded area	36.4	47.2	–	34.8	50.5	37.4	41.3
68–17	3	*Fusarium*	55.0	53.0	–	68.0	42.0	81.0	59.8
		Curvularia	29.0	30.0	–	25.0	45.0	14.0	28.6
		Molded area	51.3	33.4	69.1	8.2	30.7	13.0	34.3
67–17	4	*Fusarium*	54.0	66.0	84.0	77.0	49.0	72.0	67.0
		Curvularia	41.0	19.0	18.0	14.0	28.0	12.0	22.0
		Molded area	32.1	31.3	31.7	16.6	38.1	16.3	27.7
69–4	5	*Fusarium*	43.0	50.0	44.0	44.0	45.0	67.0	48.8
		Curvularia	42.0	38.0	39.0	27.0	19.0	15.0	30.0
		Molded area	19.8	19.4	48.5	15.8	18.1	8.9	21.8
CE-90	6	*Fusarium*	47.0	24.0	45.0	57.0	32.0	35.0	40.0
		Curvularia	26.0	39.0	49.0	22.0	19.0	17.0	28.7
		Molded area	–	43.4	41.8	20.3	19.9	6.9	26.5
CE-99	7	*Fusarium*	–	38.0	68.0	60.0	59.0	70.0	59.0
		Curvularia	–	46.0	29.0	16.0	22.0	13.0	25.0
	–	Molded area	41.8	31.6	47.8	18.1	29.5	17.3	29.6
Averages	–	*Fusarium*	52.7	50.9	60.3	63.9	47.9	67.3	57.4
	–	*Curvularia*	33.5	31.4	33.8	18.1	27.9	14.7	25.7

Table 10. Relationship between moldiness, germination, and grain molds (Séfa 1977).

Character	Molded area	Zummo Scale	Nongerminated	Nongerminated and abnormal
Nongerminated	.7122**	.474**	–	.620**
Nongerminated & abnormal	.507**	.618**	.620**	–
Fusarium	.708**	.152	.481**	.167
Curvularia	– .275	– .658**	– .445**	– .421**
Helminthosporium	– .303*	– .233	– .360*	– .141
Alternaria	– .279	– .361	– .444**	– .528**
Cladosporium	– .170	.157	– .277	.012
Pycnidial fungus	– .701**	– .022	– .442**	– .264
Colletotrichum	– .084	– .007	.082	.196
Others	.505**	.379*	.481**	.457**

a. Significantly different coefficients of correlation from 0 to probability thresholds of 5(*) and 1(**)% respectively.

Table 11. Relations between molds and chemical composition of grains.

Samples	Molds[a]					Chemical composition[b]				
	Percent molded area	*Fusarium*	*Curvularia*	*Helminthosporium*	Pycnidial fungus	Protein g/100 g	Coefficient of protein efficiency	Isoleucine g/100 g proteins	Valine g/100 g protein	Limiting factor[c]
CE-90 Healthy	0	–	–	–	–	13.43	0.73	4.3	5.3	Lysine-56
CE-90 Slightly molded	5.1	46.5	26.5	5.5	14.5	14.67	0.69	–	–	
CE-90 Very molded	28.7	43.0	33.0	15.0	19.5	11.68	0.66	3.4	4.2	Lysine-54

a. According to analyses done at CNRA, Bambey, Senegal 1978. b. According to M. CANONNE, ORANA, DAKAR, Senegal, 1978. c. FAO type combination percentage.

Table 12. Microfloral evaluation of grain-mold "tolerant" sorghum varieties grown at Séfa 1977.

Varieties	Rank	Moldy surface	Nongerminated	Nongerminated & abnormal	Scale	*Fusarium*	*Curvul.*	Pycnidial fungus	Others
1. IS-2327	1	13.0b[a]	10.5a	31.0a	4.50	28.0bcd	38.5c	55.0b	11.5a
2. IS-14332	2	10.0a	39.5cd	59.0bc	6.25	17.0a	14.0ab	76.0e	14.5a
3. IS-2328	2	16.6d	26.0b	55.0b	6.25	23.0abc	14.0ab	65.5cde	21.5ab
4. E-35-1	4	12.1b	36.5bcd	72.5cde	6.50	27.0bcd	14.5ab	73.0de	19.5ab
5. 74-10SS.051[b]	5	21.6e	33.0bc	89.0fg	6.0	26.5bcd	16.0ab	57.5bcd	18.5ab
6. Large glume-7	6	31.3f	82.0e	93.0g	6.75	45.0e	12.5ab	30.0a	23.5b
7. 74-10-KH[b]	6	22.7e	46.5d	82.5ef	6.0	29.5cd	15.0ab	57.0bcd	21.5ab
8. IS-7225	8	15.5c	26.0b	72.5cde	6.75	22.0abc	27.0b	47.5b	14.0a
9. IS-2261	9	11.5b	31.0bc	79.5def	6.75	21.0ab	14.5ab	69.5cde	17.5ab
10. CS-3541	9	22.0e	37.0bcd	64.0bcd	6.75	45.0e	7.5a	59.0bcd	23.0ab
11. CE 90[b]	10	17.5d	37.0bcd	80.0def	7.0	32.5d	8.0a	61.0bcde	24.0ab

a. Only values with different letters, within columns, differ significantly from each other at 5% level
b. Varieties developed at CNRA, Bambey, Senegal

Table 13. Evaluation of the International Sorghum Grain Mold Nursery at Séfa 1977.

Varieties	Rank	Characters evaluated[a]		
		Zummo scale	Field scoring	Days to flowering
IS-2327	1	4.63	2	74
IS-2328	4	5.88	2	79
E-35.1	4	6.00	4	81
IS-14332	5	5.88	3	81
7410-SS051[b]	6	6.13	2	84
2KH1-E20	7	6.50	–	94
7410-KH[b]	8	6.25	3	72
IS-2261	8	6.50	2	78
IS-7225	8	6.38	6	85
Large glume-7	9	6.25	–	81
CS-3541	10	6.50	4	67
IS-3443	11	6.63	–	91
Large glume-3	15	6.75	5	57
95-40-63	18	6.88	4	65
CE-90[b]	18	6.88	4	67
7531-V15[b]	19	6.88	4	67
IS-1067	21	7.00	4	65
IS-452	22	7.63	5	72
IS-3552 DER (SC-7488-5)	22	7.00	3	73
IS-7254 C(SC-566)	23	7.13	5	56

a. Average of 2 replicates
b. Varieties developed at CNRA, Bambey, (Senegal).

Fusarium and *Curvularia* among the most important fungi followed by the pycnidial fungus and other unidentified fungi. The problem is made more complex by the fact that the fungi, being living organisms, develop according to their own properties and ecological conditions and interact. Thus, for Senegal, it would be useful to first study *Fusarium* and *Curvularia* individually and then study their interactions. Laboratory trials under controlled conditions would be suited for individual studies of these fungi and natural field conditions for studying their interactions. This will not affect accuracy of observations which will be taken on a hourly basis.

These results also reveal the urgent need for sources of genetic resistance to grain molds (Tables 12, 13). In two trials involving accessions from several countries, we observed that there is only one variety (IS-2327) that can be called resistant; a few others, generally accepted for want of anything better, can be classified only as slightly tolerant to grain molds. Therefore, additional varieties should be identified and/or large-scale detailed screening should be carried out to identify genetic resources of resistance available for work on sorghum grain molds.

One aspect of sorghum grain molds not discussed in this paper concerns the relationship between dormancy and sorghum grain molds. We suspect that dormancy could prevent development of fungi on grain. This should be investigated.

Finally, where sorghum is used for human consumption, attention should be paid to toxicity incidence sometimes caused by certain fungi.

Summary

In Senegal, local varieties of sorghum usually mature at the end of the rainy season, thus escaping grain mold infection. The decision to introduce early varieties in one area, where in general only late and/or photosensitive sor-

ghums are grown, stimulates the need for grain mold resistant varieties. Grain molding is a complex problem — several species of fungi are involved, and they interact with one another in a dynamic fashion, depending on the conditions of the grain and the environment.

Presumably this is one of the reasons why sources of multiple (for many fungi) genetic resistance are scarce. This situation calls for a fragmentation of the problem, working first with the most important fungi as far as grain appearance and deterioration are concerned. In Senegal, besides some unidentified fungi, *Fusarium, Curvularia*, and *Phoma* would be the first to be studied. Sources of resistance to these fungi are urgently needed. In Senegal, IS-2327 appears to be grain mold resistant followed by IS-14332 and E 35-1, which should be called grain mold tolerant. Some varieties developed in our program are classified as "acceptable" when harvest is not delayed.

Reference

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Breeding for Grain Mold Resistant Sorghums at ICRISAT

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Sorghum is grown in the semi-arid tropics (SAT) mostly as a food crop in the rainy season. Traditional cultivars in this region generally flower at the end of the rains and their grains mature essentially during dry weather. Therefore, grains retain the original characteristics which farmers prefer. On the other hand, the recently improved genotypes have a higher harvest index and give stable and high yields across environments, but they flower early and their grains fill and often ripen in wet weather. These genotypes pose the following problems:

1. Susceptibility to parasitic and saprophytic fungi that destroy the grain;
2. Pigmentation and grain weathering;
3. Loss of seed viability and/or (in extreme cases) sprouting on the panicle; and
4. Poor food quality.

These interrelated problems are usually referred to by the all-inclusive term "grain deterioration." In recent years, grain deterioration has been receiving increased attention in many sorghum-growing areas, and significant developments have been reported by Glueck et al. (1976), Murty (1977), and Rana et al. (1978).

Since farmers' preferences depend upon the consumption value of the grain and its market price, grain-deterioration problems become crucial for the extension of high-yielding genotypes. The ICRISAT Sorghum Improvement Program has given high priority to this area of research, and has specific collaborative projects on each of the problems listed. This paper reviews the progress of the ICRISAT sorghum breeding work on resistance to the two most important grain molds, *Curvularia* and *Fusarium*, during the last 4 years.

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Objectives

The project on improvement for grain mold resistance has the following objectives:

1. Incorporation of mold resistance into elite material (varieties and hybrids) of good grain quality and yield;
2. Identification of additional sources of resistance to various grain molds and their further utilization; and
3. Improvement of the available degrees of mold resistance by appropriate selection and recombination techniques.

Progress

The Breeding Scheme

Grain molds appear to be a universal phenomenon in regions where breeders are attempting to breed for sorghums that flower earlier than the local types. Therefore mold resistance needs to be incorporated into appropriate maturity groups adapted to broad ecological zones in the SAT. To achieve this objective, ICRISAT breeders have attempted an elaborate program (Fig. 1) to be carried forward with intensive selection in the segregating generations for mold resistance and good grain quality. Parents are selected from various sources and appropriate crosses made in large numbers. Selected F_1 hybrids are carried forward to F_2. Selection in F_2 populations is carried out under natural conditions, but from the F_3/F_4 generation onwards artificial screening is done. The F_2 populations are less protected, and are grown under moderate stress conditions so as to eliminate genotypes highly susceptible to shoot fly and borer. Protection against midge and head bug damage is provided, as such damage might interfere with grain mold ratings. Grain yield assessments are made from the F_4 generation onward. Evaluations at Dharwar (15°27'N),

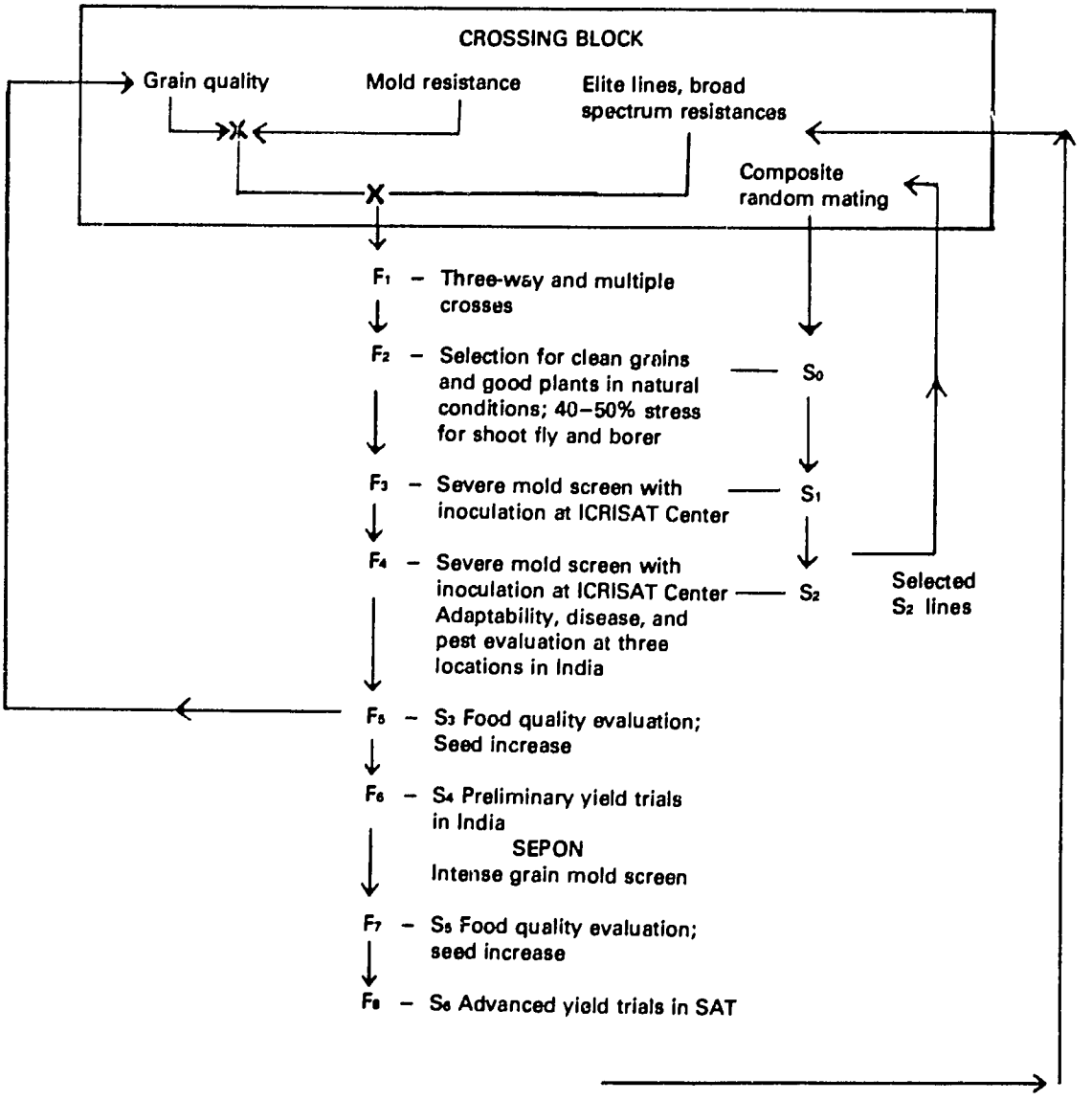

Figure 1. Improvement for grain mold resistance and quality breeding scheme.

Bhavanisagar (11°27'N), and ICRISAT Center (17°27'N) provide an opportunity to select for better adapted types less susceptible to diseases and pests. Food-quality evaluations are done generally from the F_5 generation onward, either at ICRISAT Center or at the ICRISAT regional stations. Selected lines are distributed to cooperators as a nursery called "Sorghum Elite Progeny Observation Nursery" (SEPON). The scores we receive from cooperators help us identify widely adapted mold-resistant progenies. Selections from this observation nursery will enter advanced yield trials in the SAT. There is also a parallel recurrent selection program in a broadbased composite for improving mold resistance. Artificial inoculation is done in the S_2 generation and the selected families recombined for the next cycle. They can also be advanced by pedigree breeding and the S_4 and S_5 lines could go into SEPON. Selections from preliminary yield trials are recycled into the crossing block.

Breeding for semiphotosensitive and photosensitive groups of material cannot be carried out at ICRISAT Center because of flowering problems. In the near future we expect to do part of this work at Bhavanisagar, using the scheme essentially as outlined, but with minor modifications.

Crossing Block

Parents were selected from the world collection, international nurseries, and advanced yield trials of national programs for their superiority in grain mold resistance, good grain quality, adaptation, earliness, and yield. New sources of resistances to *Curvularia* and *Fusarium* were identified at ICRISAT (e.g., IS-9327, E 35–1, IS-14332, IS-2328, IS-9225) by inoculation and intensive screening of the world collection. These, along with CS-3541, a released variety from the All India Coordinated Project, have been used extensively. Several of the zera zeras are known to be free of pigmentation and weathering problems, and hence have been used frequently. Parents possessing broad-spectrum resistances to various other diseases and pests were also used. Single, double, and three-way crosses were made between adapted, good grain, and mold-resistant varieties based on height and maturity. The range of parents included in the crossing program in the last 3 years is presented in Table 1. To date about 12 700 crosses have been made and the F_1 generations evaluated. Of these, only 1967 F_1 hybrids (17%) were advanced. The philosophy behind making a large number of crosses is to (a) generate enormous variation and broaden the genetic diversity, (b) sample many crosses and select more among rather than within crosses, and (c) identify useful combiners for various traits in the world collection and breeding stocks. A summary of the number of crosses and segregating families evaluated for grain molds in the last 3 years is presented in Table 2. It may be noted that

Table 1. Crossing block – parental array in grain mold resistance and grain quality improvement trials at ICRISAT Center.

Group	No. of parents
Zera zeras and other less mold susceptible lines	52
High-yielding and adapted lines from India, Upper Volta, Nigeria, Senegal, Sudan, Egypt, Uganda, Ethiopia, Kenya, USA, and ICRISAT	116
Good rain quality entries from the world collection	45
Popular Indian farmer types	48
Yellow endosperm types from Karper's nursery	60
IS conversion lines from Puerto Rico	18
Waxy and highly corneous grain types	16
Disease- and pest-resistant lines	15
Total	370

Table 2. Breeding material evaluated by ICRISAT for grain molds during 1975–78.

Generation	1975–76	1977	1978
F_1	3574	3400	5735
F_2	427	592	948
F_3	2820	2119	612
F_4	–	1576	1364
F_5	–	770	–
F_6	–	–	663

approximately 3000 early generation progenies are generated for grain mold-resistance screening each year.

Selection for Grain Mold Resistance

Generally 800 plants were grown for each F_2. However, in some interesting crosses 1600 plants were grown. In 1976, a mixture of *Curvularia* and *Fusarium* inoculum was sprayed onto the heads a few days after flowering, and the heads were left open. Artificial inoculation of a large number of hectares is impractical, so selection for resistance was later limited to mold and weathering under natural conditions. Agronomically desirable plants with clean and good grains were selected. The weather was favorable for selection against molds in 1976 and 1978. Individual panicle selections were advanced in the offseason, and each of the rows was bulk harvested with minimal selection. Some of the F_2s were grown in the summer at Bhavanisagar, and the F_3s were advanced in the rainy season under artificial inoculation at ICRISAT Center.

The F_3/F_4 progenies were planted during the rainy season in separate rows, and several panicles were inoculated a few days after flowering. In 1978, however, panicles were bagged before anthesis to ensure self-pollination. The selfing bags were removed after anthesis and the panicles sprayed with a mixture of *Curvularia* and *Fusarium* (22 000 conidia/ml) and the bags replaced. Twenty days later the bags were removed. Head mold ratings were taken on inoculated as well as noninoculated heads 50 to 55 days after flowering. All rows were evaluated for their reaction to *Curvularia* and *Fusarium*, on a scale of 1 to 5, where 1 = no molds, 2 = less than 10% of the grains moldy, 3 = 11 to 25% of the grains moldy, 4 = 26 to 40% of the grains moldy, and 5 = more than 40% of the grains moldy. Relatively clean panicle grains (with a score of 3 or less) were selected from low susceptible progenies. During the postflowering period, humidity in the field was ensured by operating sprinklers for a few hours each day. The sprinklers were helpful in promoting mold development, and they created a severe stress for this trait, but posed extreme problems of sprouting on the head. Therefore, sprinkler use was later restricted to 30 minutes in the evening on each rain-free day.

Selection was successful, and the details of results are presented in Tables 3 and 4. It may be noted that in rainy season 1977 we made 487 less susceptible selections, out of which only 50 had a score of 2. Advanced generation lines from the 487 selections were retested in rainy season 1978, when the majority of them had a score of 2. We retained only 62, however, after discarding sister selections and lines of similar origin and genetic background. F_3 and F_4 progenies from a fresh lot of crosses contributed 192 additional selections.

Laboratory Evaluation

The seed increased in the postrainy season of 1977 (November to February) from the 487

Table 3. Mold-resistant selections obtained in rainy season 1977 by screening 4465 F_3, F_4 and F_5 progenies after inoculation with *Curvularia* and *Fusarium*.

Origin	Score		Total
	2	3	
Single crosses	34	330	364
Double crosses	–	49	49
Three-way crosses	16	58	74
Total	50	437	487

Table 4. Mold-resistant selections obtained in rainy season 1978 by screening 2639 F_3, F_4, F_5, and F_6 progenies after inoculation with *Curvularia* and *Fusarium*.

Origin	Score				Total
	1.5	2.0	2.5	3.0	
1977 selections					
Single crosses	11	44	2	-	57
Three-way crosses	–	4	–	–	4
Segregating generations:					
Single crosses	10	29	4	2	45
Double crosses	5	2	1	–	8
Three-way crosses	42	86	8	4	140
Total	68	165	15	6	254

selections made during rainy season 1977 was evaluated in the laboratory for relative fungal infection. Grain samples were inoculated and incubated for 4 days (12 hr dark and 12 hr light) at 25°C. The severity of infection was visually rated on a scale of 1 to 5 (increasing order of infection) in replicated sets of petri dishes. Fifteen percent of the 487 selections had an infection severity score of 2. Some of the selections which showed the least infection in the laboratory as well as in field testing in rainy seasons 1977 and 1978 were (SC-108-4-8 × CS-3541)-29-1, (SC-423 × CS-3541)-57-1, (2219B × CS-3541)-12-1, (SC-108-3×E 35-1)-1-1, (SC-108-3×E 35-1)-29-2, (10680×CS-3541)-6-1, and (SC-108-3×Uchv$_2$)-15-1. Single, double, and three-way crosses, involving parents like CS-3541, E 35-1, IS-9530, IS-9327, IS-2328, 2219-B, SC-108, SC-423, CS-3687, 2KX-2, 8272, Uchv$_2$, and IS-3443, contributed mostly to the less mold susceptible selections.

Resistance to other important fungi, such as *Phoma* and *Alternaria*, is yet to be identified and incorporated into the breeding program. We propose to begin to do this soon.

Selective Intermating

An examination of the available degree of mold resistance in the world collection and various breeding stocks at ICRISAT indicates that there is no line possessing absolute resistance to either one or all the molds, and that the term "resistance" to grain molds is relative. Resistance to different fungi is present in various degrees in various lines, and its genetic basis is probably polygenic. An improvement in natural levels of resistance should be possible through selective intermating of resistant progenies in specific crosses or recurrent selection in random mating populations. We have attempted both these approaches, using our mold less-susceptible selections.

In the offseason of 1977, we divided the 487 mold less-susceptible selections into 12 broad groups, based on origin, pedigree, plant morphology, maturity, and grain quality parameters. We intercrossed members between these 12 different groups. A total of 2045 such intercrosses were advanced in the summer, and a selected set of 182 crosses were grown under natural conditions favorable for grain mold screening. The general level of resistance to *Curvularia* and *Fusarium* was better in these than in other groups of material. We selected 1009 plants scoring 1.5 or 2.0. Advanced-generation progenies of these selections will be tested under artificial inoculated conditions in the next rainy season (1979).

Mold Resistance Composite

As a long-term approach, a broad-based composite is being built, wherein recurrent selection for grain quality and mold resistance in a good agronomic background will be practiced. In 1976, bulk pollen from many of the F_1 hybrids was dusted on male steriles segregating in an early ms$_7$ sterile bulk. Later, the first backcross to the steriles segregating in the F_2 population was done, using confirmed mold-resistant sources and other elite F_4 selections. The second backcross was made in the 1977 postrainy season, using the mold less-susceptible selections from artificial screening. Another backcross will be made in the postrainy season of 1978 with the latest mold-resistant selections and elite material. In addition to grain mold resistance, we want to incorporate good food-quality characters into this composite, and it will then be handled in three subpopulations, based on maturity.

Genetics of Grain Mold Resistance

Information on the genetics of grain mold resistance is scanty. Rana et al. (1978) and Dabholkar and Baghel (1978) found that general combining ability effects seem to be important.

In 1976 at ICRISAT, three parents less susceptible to *Curvularia* and *Fusarium* infection, three moderately susceptible parents, and three highly susceptible parents were crossed in all possible combinations. Six of the hybrids were backcrossed to both parents, while 14 were advanced to F_2. The parental, F_1, F_2, and backcross generation material was evaluated for grain molds under inoculated conditions. Unfortunately, severe damage by head bugs interfered with assessments of the panicles for grain molds. However, the wide range of maturities and grain colors in the segregating generations seemed to have affected the mold scores. Nevertheless, a cursory examination of the generation mean data showed some evidence for partial dominance.

In 1978, we initiated a new set of crosses, where segregation for days-to-flower, plant-color, and seed-color factors is expected to be minimal. Nine parents exhibiting various degrees of susceptibility to *Curvularia* and *Fusarium*, 12 F_1 hybrids involving these parents, and 12 F_2 populations were planted under comparable conditions on the same date in the rainy season. A few days after flowering, panicles were sprayed separately either with *Curvularia lunata* or *Fusarium moniliforme* inoculum, and then bagged. The bags were removed 20 days after flowering and grain mold scores given on a scale of 1 to 10 (increasing order of infection) 50 days after flowering. The number of plants inoculated in the nonsegregating parental and F_1 generations varied between 3 to 20, while in F_2 it ranged from 85 to 288. Generation means for all the 12 crosses are presented in Tables 5 and 6 for *Curvularia* and *Fusarium*, respectively. The frequency distribution of mold scores showed that the variation for resistance to the fungi studied is continuous, and is probably controlled by polygenes. Keeping in mind that sampling errors exist, we may observe from the generation means in Tables 5 and 6 that, in general, the F_1 means are close to the midparent, indicating mostly additive type of gene action. Occasionally, however, the F_1 mean is slightly higher than the average of parents, probably because of partial dominance of susceptibility in that particular cross. The average scores of F_2 progenies of several crosses also indicate the same situation. Overall, these preliminary observations indicate the predominance of additive gene action and the presence of partial dominance in some crosses. This is in conformity with the conclusion drawn from 1977 data. Some of the interesting crosses are being followed up in a more detailed manner for a generation mean and variance component study.

Table 5. Generation means of scores on reaction to *Curvularia lunata* inoculation.

Cross ($P_1 \times P_2$)	P_1	P_2	Mid-parent	F_1	F_2
IS-9327 × H 112	2.3	6.4	4.35	4.5	6.1
IS-9327 × IS 9530	2.3	2.6	2.45	2.5	4.1
SPV-104 × CS 3541	8.3	2.1	5.20	6.2	5.7
SPV-104 × E 35-1	8.3	2.5	5.40	7.0	5.5
370 × IS 9327	6.5	2.3	4.40	4.7	5.5
CS-3541 × 370	2.1	6.5	4.30	5.0	6.2
IS-9530 × IS 10939	2.6	6.5	4.55	5.8	6.1
SC-120 × IS 9327	5.0	2.3	3.65	3.3	5.2
SC-120 × CS 3541	5.0	2.1	3.55	5.1	5.6
SC-120 × SPV 104	5.0	8.3	6.65	7.1	6.0

Table 6. Generation means of scores on reaction to *Fusarium moniliforme* artificially inoculated at ICRISAT Center.

Cross ($P_1 \times P_2$)	P_1	P_2	Mid-parent	F_1	F_2
IS-9327 × H 112	2.2	6.4	4.30	4.5	4.5
IS-9327 × IS 9530	2.2	2.8	2.50	2.4	3.8
SPV-104 × CS 3541	6.3	2.6	4.45	4.9	5.6
SPV-104 × E 35-1	6.3	2.7	4.50	–	5.8
E 35-1 × CS 3541	2.7	2.6	2.65	3.1	3.9
370 × IS 9327	7.6	2.2	4.90	5.3	5.1
SC-120 × 9327	4.4	2.2	3.30	2.7	4.8
SC-120 × CS 3541	4.4	2.6	3.50	3.5	4.9
SC-120 × 10939	4.4	7.0	5.70	–	4.5
SC-120 × SPV 104	4.4	6.3	5.35	5.4	5.8

Fertility Restoration Studies

Development of A-lines possessing good grain quality and mold resistance could be a significant contribution to hybrid breeding. About 600 selections in F_4, F_5, and F_6 generations were tested as pollinators on two cytosteriles from the All India Coordinated Sorghum Improvement Project (AICSIP), namely 2219A and 2077A, and fertility-restoration studies on these hybrids are under way. Preliminary results indicate parents (10680 × 3541)-7 and (Bulk Y × GPR 165)-4-3 to be maintainers.

Yield Tests

Grain yield was measured on 210 F_5 bulks grown in 7.5 m^2 plots planted in a randomized block design, with three replications at ICRISAT Center in rainy season 1977. Several of the entries yielded 4000 to 5000 kg/ha grain, compared to 4800 kg/ha of the hybrid check, CSH-6. Selections from rainy season 1977 tests were advanced under inbreeding, and again yield

tested in replicated trials in rainy season 1978 at ICRISAT Center, Dharwar, and Bhavanisagar. Grain yield data on some of the promising entries is presented in Table 7, along with adjunct notes on days to flowering, plant height, and grain-mold scores under artificial inoculation. It may be noted that the best entries gave average yields of 5300 to 5600 kg/ha.

Sorghum Elite Progeny Observation Nursery (SEPON)

Seed of 72 elite selections from Adapted × Mold Resistant crosses in the F_5 generation was supplied to 12 cooperators in various regions of the SAT in the form of a nursery called 1977 Sorghum Elite Progeny Observation Nursery (SEPON 1977). In addition to the local check (contributed by the cooperator), a varietal check (SC-108-3) and a hybrid check (CSH-6), selected for wide adaptation in earlier nurseries, were also included. Overall scores and comments given by the cooperators for grain molds, yield, and adaptation revealed that a majority of the lines exhibited satisfactory to good performance in comparison with local checks, and were likely to contribute to national programs. The entries selected in each location (with an overall performance of 2 on a scale of 1 = very good to 5 = poor) are listed in Table 8. Notable among the entries that performed well across several locations are Nos. 10, 29, 30, 36, 37, 38, 42, 53, and 74, CSH-6. In the Ethiopian high altitudes, however, the nursery did not show good adaptation, although resistance to leaf diseases was pronounced. In Thailand, some of the lines were reported to be free from gray leaf spot, and yielded well compared with the checks. In Mali, some of the lines yielded 4500 to 4900 kg/ha in replicated trials, far surpassing the local checks, while some of the others were susceptible to lodging and possibly charcoal rot. In Senegal, some of the lines yielded 3700 to 4200 kg/ha, and a few were selected in the field as well as in the laboratory for less susceptibility to grain molds. In Upper Volta, also, selections with good yield and less susceptibility to grain molds were made. Many lines exhibited grain-mold resistance in the low altitudes of Mexico (Poza Rica), while some appeared to be good in Tanzania. SEPON 1978 comprising 46 selections in F_5 to F_7 generations, has been despatched to 15 cooperators throughout the

Table 7. Performance of some elite selections in rainy season 1978 yield trials at three locations in India.

Pedigree	Days to[a] flower	Plant[a] height (cm)	Grain Yield (kg/ha)[b]				Grain mold score
			ICRISAT Center	Dharwar	Bhavanisagar	Mean	
(SC 108-3 × CS 3541)-3-1	68	152	5348	5817	4557	5240	3.0
" -3-3	68	156	5630	5680	4668	5326	2.5
" -19-1	71	172	5496	5849	5661	5668	2.5
" -14-1	74	163	4731	4835	5096	4886	2.5
(SC 108-4-8 × CS 3541)-40-1	74	183	4825	5527	5650	5334	2.5
(SC 108-3 × E 35-1)-29-2	74	181	5405	5877	5455	5575	2.0
(CS 3541 × IN 15-2)-26-1	69	174	5463	5333	5026	5274	2.5
CSV-3 (370)	70	176	4748	2601	4056	3801	5.0
CSV-4 (CS 3541)	73	141	4131	4460	3125	3905	3.0
CSH-6	64	167	6231	6576	4017	5608	3.0

a. Average over locations b. Plot size was 18 m² at ICRISAT, 7.2 m² at Dharwar, and 4 m² at Bhavanisagar

Table 8. Sorghum Elite Progeny Observation Nursery 1977 (SEPON) – Overall performance of lines across location.

Entry No.	Pedigree	Location[a] 1	2	3	4	5	6	7	8	9	10	11	12
1.	(IS 12622C × CS 3541)-1								x[b]	x			
2.	(SC 423 × CS 3541)-16						x						
3.	(SC 423 × CS 3541)-47								x				
4.	(SC 423 × CS 3541)-23								x				
5.	(SC 423 × CS 3541)-24						x		x				
6.	(SC 423 × CS 3541-27	x							x				
7.	(SC 423 × CS 3541)-52						x		x				
8.	(SC 423 × CS 3541)-61												
9.	(SC 423 × CS 3541)-85						x		x				
10.	(SC 423 × CS 3541)-113	x		x			x		x	x			
11.	(0222 × CS 3541)-10	x					x					x	
12.	(0222 × CS 3541)-13												
13.	(10596 × CS 3541)-6												
14.	(10262 × CS 3541)-1		x						x				
15.	(10262 × CS 3541)-2	x	x	x									
16.	(10262 × CS 3541)-13	x	x										
17.	(10262 × CS 3541)-20											x	x
18.	(10262 × CS 3541)-21					x			x	x			
19.	(10262 × CS 3541)-22												
20.	(10680 × CS 3541)-2					x			x	x			
21.	(10680 × CS 3541)-4					x			x	x			
22.	(10680 × CS 3541)-6	x								x			
23.	(2219 B × CS 3541)-12	x	x										
24.	(2219 B × CS 3541)-22	x	x										
25.	(SC-108-3 × CS 3541)-1	x										x	
26.	(SC-108-3 × CS 3541)-3	x										x	x
27.	(SC-108-3 × CS 3541)-7	x	x				x			x			
28.	(SC-108-3 × CS 3541)-14	x					x	x		x			x
29.	(SC-108-3 × CS 3541)-28	x					x	x	x	x			
30.	(SC-108-3 × CS 3541)-30	x	x		x	x		x	x			x	
31.	(SC-108-3 × CS 3541)-39		x			x			x	x	x		
32.	(SC-108-3 × CS 3541)-51	x								x	x		
33.	(SC-108-3 × CS 3541)-53		x							x	x		
34.	(SC-108-3 × CS 3541)-57							x				x	
35.	(SC-108-3 × CS 3541)-3						x		x				
36.	(SC-108-4-8 × CS 3541)-11	x	x	x		x	x		x				x
37.	(SC-108-4-8 × CS 3541)-13			x		x	x		x	x	x		
38.	(SC-108-4-8 × CS 3541)-14	x				x	x	x	x	x			
39.	(SC-108-4-8 × CS 3541)-35			x									
40.	(SC-108-4-8 × CS 3541)-37	x	x				x						
41.	(SC-108-4-8 × CS 3541)-38	x				x			x		x		x
42.	(SC-108-4-8 × CS 3541)-43	x					x	x	x	x			
43.	(SC-108-4-8 × CS 3541)-46	x					x			x			x
44.	(SC-108-4-8 × CS 3541)-55	x								x			
45.	(SC-108-4-8 × CS 3541)-60						x						
46.	(SC-108-4-8 x CS 3541)-64						x		x		x		
47.	(SC-108-4-8 × CS 3541)-70												
48.	(SC-108-4-8 X CS 3541)-73					x			x				
49.	(SC-108-4-8 × CS 3541)-77						x		x	x	x		x

Continued.

Table 8. *Continued*

Entry No.	Pedigree	Location[a] 1	2	3	4	5	6	7	8	9	10	11	12
50.	(SC-108-4-8 × CS 3541)-81	x					x		x		x		
51.	(SC-108-4-8 × CS 3541)-92	x					x		x				
52.	(IS 12645C × CS 3541)-40	x											
53.	(IS 7994C × CS 3541)-8	x	x				x			x	x		
54.	(IS 146 × CS 3541)-28	x											
55.	(CS 3541 × GPR 148)-2												
56.	(CS 3541 × GPR 148)-4												
57.	(CS 3541 × GPR 148)-5												
58.	(CS 3541 × GPR 148)-18												
59.	(CS 3541 × ET 2039)-4								x				
60.	(CS 3541 × ET 2039)-11								x				
61.	(2219 B × GPR 148)-8			x					x				
62.	(SC-108-3 × GPR 148)-6								x				
63.	(SC-108-3 × GPR 148)-12	x				x							
64.	(SC-108-3 × GPR 148)-26					x							
65.	(SC-108-3 × GPR 148)-27					x							
66.	(2219 B × Swarna)-1								x	x			
67.	(CS 3541 × Swarna)-4			x		x			x	x			
68.	(SC-108-3 × Swarna)-12			x		x			x	x			
69.	(SC-108-3 × Swarna)-18					x							
70.	(WABC × Entomology)		x										
71.	(Bulk Y × GPR 165)-4-3					x							
72.	Bulk 'Y'												
73.	SC 108-3		x		x	x	x						
74.	CSH-6	x	x		x	x				x			
75.	Local check		x		x	x	x		x				

a. 1. ICRISAT Center
2. Kovilpatti, India
3. Khon Kaen, Thailand
4. Alemaya, Ethiopia
5. Busia, Kenya
6. Kamboinse, U. Volta
7. Farako-Bâ, U. Volta
8. Maradi, Niger
9. Sefa, Senegal
10. Massantola, Mali
11. Ilonga, Tanzania
12. Poza Rica, Mexico

b. Overall performance of the line was better and was selected for one or more attributes, including less susceptibility to molds.

SAT. Yield data (Table 7) pertains to some of the entries included in SEPON 1978. Preliminary reports indicate that a number of these progenies are performing well, and are useful to the cooperators.

Summary

A large number of crosses between mold-resistant, good grain, and adapted types, followed by intensive inoculated screening in early segregating generations, revealed lines less susceptible to *Curvularia* and *Fusarium*. Some of those in advanced generations appear to be valuable to national programs, their yield potential being moderately high. Gene action governing grainmold resistance appears to be largely additive, though there is some evidence for partial dominance. The progress of selective intermating in pedigree crosses and the possibilities for recurrent selection in random-mating populations are discussed.

Acknowledgment

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Grain Mold Discussion Session

Causal Agents and Time of Infection

Frederiksen:

What is the relative importance of early infection compared with infection on the mature grains?

Castor:

Early infection is probably very important in causing severe damage. Although infection can occur at any time during grain development, less damage results from late infections.

K. N. Rao:

We carried out daily inoculations with *Fusarium* and *Curvularia* during a period following inflorescence emergence. Inoculations from 3 to 8 days after head emergence resulted in maximum mold incidence. Inoculation at later stages of grain development did not produce so high a degree of mold. We also took infected florets out of the panicle following inoculations and found that the *Fusarium* had penetrated the ovary.

Denis:

In our studies with several planting dates (whereby different flowering stages could be examined at the same time), we found that *Curvularia* generally infects very early and decreases in importance as the crop matures. Conversely *Fusarium* increases in importance as the crop matures and is most important at harvest. However, indications of the infection by any fungus remains visible up to maturity, regardless of the time of infection.

Rosenow:

In Texas severe mold infestation on mature grain can develop in as short a period as 6 days of rain. I think the late-season infections are more important, and suggest that the bags should be removed earlier than 14 days after inoculation, to allow the heads to be exposed for the maximum time to natural weathering. Has anyone observed a case in which grain mold developed when the environment was wet and humid during flowering and early grain development, but dry at physiological maturity? The majority of mold problems occur when the weather is wet at and from physiological maturity.

Denis:

Mold will develop on grain after a single rain when fungi are already present and the grain is at or near the hard-dough stage.

Williams:

Infection occurs early in the grain-development process. The symptoms manifest themselves near or at maturity if the environment is conducive. The appearance of symptoms at or near maturity does not mean that the infection took place only at that time.

Zummo:

In Nigeria, short Kaura and Fara-Fara mature at about the same time, after the end of the rainy season. In storage, short Kaura deteriorates under high humidity, whereas Fara-Fara does not. We attribute this difference to differences in susceptibility to the mold fungi during the wet period before maturity.

N. G. P. Rao:

While there has been a great deal of work done on the fungi that invade the florets at an early stage under wet conditions, there is a need for more information about fungi that infect the grain at a later stage. Which fungi infect the grain after it starts to lose moisture and during postmaturity? Does bagging interfere with the development of these later-infecting fungi? In the effort to reduce deterioration problems, both the early and the late infecting fungi have to be tackled.

Williams:

Curvularia and *Fusarium* have been so overwhelmingly important that it was not until we had material that could resist their infections that we could begin to work with the later-infecting fungi. A *Phoma sp* can be severe on lines which have low susceptibility to *Fusarium* and *Curvularia*. I do not think bagging prevents later-infecting fungi from getting to the grain, for we remove the bags 14 days after inoculation.

Murthy:

IS-14332 has performed well not only against *Fusarium* and *Curvularia*, but also against *Phoma*.

N. G. P. Rao:

SPV-102 is also resistant to these three fungi.

Screening for Resistance

Inoculation Methods

Frederiksen:

What are the best inoculation procedures for resistance screening?

Denis:

In Senegal, factors important for successful screening are:

1. Provision of inoculum and humidity at flowering time. If natural inoculum is relied upon, then it is very important to promote high humidity during flowering;
2. A good distribution of susceptible lines throughout the nursery to provide additional inoculum;
3. Use of several replications and locations to avoid local variations due to several factors — including rainfall duration and distribution; and
4. Several readings in the same trial.

Bidinger:

When you use cultures of the grain mold pathogens to inoculate dry mature grain in laboratory tests, are the fungi acting as parasites or saprophytes? Does the answer to this have any bearing on the correlations between the laboratory and field screening?

Williams:

This is a question I have raised within our group. If grain molds are primarily a problem of fungi infecting florets at early stage of grain development, how can we have a reliable method of laboratory screening using dry mature grain? We are not sure we know the answer, but we are able to differentiate between cultivars in our laboratory tests. When we incubate the grain from the dry-season crop we find out what fungi entered the seed during its development, and the degree of colonization that occurred. There is probably enough dew to initiate infection of the grain of highly susceptible lines in the dry-season crop, but the environment is not conducive to symptom development. By incubation of the mature grain, we can evaluate the degree of such infection.

Sundaram:

While bagging of heads may help promote molds in dry regions, will it not reduce mold development in wet areas by preventing the rain getting to the head?

K. N. Rao:

Bagging of heads does not reduce mold development in wet weather. Results from India, from Senegal, and from the USA show that bagging helps promote mold development. The bags are not water proof; they become wet and the heads inside become wet. The heads dry out more slowly after rain if they are bagged, and this promotes mold infection and development.

N. G. P. Rao:

We need to combine field screening, using inoculations with *Fusarium* and *Curvularia* at anthesis, with laboratory screening of mature grain for resistance to *Phoma*.

Murthy:

It is important to use "checks" of varying maturities. We now use three checks — early, medium, and late maturing — after every 30 to 40 test rows, so that meaningful comparisons can be made. In ICRISAT

screening, not all heads in a row are bagged, and the bags are removed from the bagged heads 14 to 20 days after inoculation. The *Phoma* infection is estimated on noninoculated heads to avoid any possible *Phoma-Curvularia-Fusarium* antagonism. I think this gives ample chance to measure adequately reactions to the major mold fungi.

Balasubramanian:
Can one use the rate of imbibition of water when screening for grain-mold resistance?

Rooney:
Yes, it is possible. But you have to keep comparisons within the same grain types to avoid confounding factors.

N. G. P. Rao:
We have developed a laboratory selection technique based on the capacity for water absorption (the uptake of water in 2 hr), grain hardness, and the presence of a tan background. The selection is based on appropriate weightage of these three parameters.

Rating Scales

Frederiksen:
How good are rating systems such as the Zummo scale? How well do field ratings relate to laboratory ratings?

Denis:
The Zummo scale is very useful. Checks rated at 4 or 5 (on a 1–9 scale) can be used throughout the field, and used for comparisons with experimental lines planted in a replicated manner. One makes a rating in comparison with the checks. Only the check lines need to be common from one location to the other.

K. N. Rao:
There appears to be good correlation between field and laboratory ratings. Field rating alone will eliminate a vast number of susceptible materials, but both field and laboratory ratings are needed for more critical comparisons. A third stage could involve laboratory inoculation of threshed grain to further differentiate lines.

Castor:
But we must remember that field ratings and laboratory ratings are only fairly highly correlated under favorable environmental conditions. In less favorable conditions the correlation may not exist, since mold can develop beneath the glumes, and will not be seen until the grain is threshed.

Denis:
The two sets of observations should be complementary. It is important to look at the frequency of a "good" rating in the field (one line might appear "good" in 10% of the tests and another might appear "good" in 90% of the tests). Postharvest handling may influence the appearance of molds on seed prepared for laboratory examination.

Sundaram:
The presence of an easily visible fungus on the seed exterior, like *Phoma*, may confound the evaluation of grain quality.

Rosenow:
I have observed that some lines develop extensive mold on the grain exterior, but the internal quality is good, while others may be quite the reverse. We need a rating system which takes this into account.

Williams:
If the environmental conditions in the field have not been conducive to severe mold development, then it may be necessary not only to examine threshed grains, but also to plate them out and see which fungi emerge. Lines apparently similar can be differentiated in this way.

Mechanisms and Genetics of Resistance

Riccelli:
We have heard this morning that there is no relationship between individual seed characters and grain-mold resistance. From my observations in Venezuela I think brown-seeded sorghums are much more resistant to grain molds than are other types. SC-103, which has a floury endosperm, is resistant to grain molds, because of the presence of a testa layer.

Rooney:

I agree that the presence of a testa layer is associated with resistance.

Williams:

In our experience at ICRISAT, even dark-seeded lines, particularly dark red-seeded lines, can become severely molded under prolonged hot and humid conditions. We have concluded that there is no necessary correlation between any one particular character and the ability to resist molds. We think resistance is the result of a complex of additive effects of several different characters, and we know that we can develop acceptable levels of resistance in white-seeded types.

Denis:

A new version of NK-300 has no tannin, and is less resistant to grain molds than the version with tannin.

Doggett:

I would like to draw attention again to the potential value of sorghums with large membranaceous glumes. While they may not be useful against *Curvularia* and *Fusarium*, they may offer protection from *Phoma* and other late-infecting fungi, for they wrap the grain fairly tightly. They may, however be difficult to work with, and probably will not be useable in hybrids.

K. N. Rao:

Large Glume-7 has done well in the ISGMN program, but some other large-glume lines were not good.

Denis:

Large glumes may allow more fungal spores and moisture to remain in close contact with the grain, and thus they can also have detrimental effects. Good head exsertion and a moderately loose panicle to allow aeration may also contribute to resistance. Seed dormancy may also relate to grain-mold resistance. I have observed varieties which take 120 to 125 days to reach maturity with both a high level of resistance and a high level of dormancy. In summary, I think the characteristics of grain mold resistant lines are as follows: (a) a barrier exists to prevent fungal penetration; (b) a barrier exists to prevent fungal colonization if penetration occurs; (c) large glumes protect the grain, (d) pigments or other chemicals in the glumes inhibit fungal development.

Crosses between lines with intermediate levels of resistance might be more beneficial than crossing resistant lines with a high-yielding susceptible line.

Rosenow:

Dormancy needs additional research in relation to grain mold.

Murthy:

We need more information on preharvest sprouting induced by *Fusarium.*

Dodd:

Is there a relationship between host-plant senescence and grain-mold development? Plants that die prematurely develop more severe mold than do others. Other factors — such as the association of grain molds with early maturity, reduced grain weight, maturity differences within the panicle, nearly saprophytic fungi, and less corneous endosperm — suggest an association between grain mold and senescence. Can we select for grain-mold resistance by selecting nonsenescent types?

Williams:

How necessary and useful are detailed studies on (a) the genetics of resistance, and (b) the mechanisms of resistance?

Craig:

It is important to have some idea of the numbers of genes involved in grain-mold resistance, because the selection for mold resistance will occur at the same time as selection for several other characters.

Rosenow:

There is a need for identification of resistance factors, because a breeder has to determine which lines to use as parental lines. Also, a general knowledge of inheritance is necessary to be successful in breeding efforts. A knowledge of breeding

behavior might be even more valuable than inheritance studies.

Rooney:
We definitely need fundamental studies on factors associated with resistance, so that we can tell the breeders what specific characters to breed for — e.g., we know that seeds with very hard corneous endosperm, brown pericarp, and persistent testa should be low-susceptible. We need to see what other factors are associated with resistance. Selections for all these parameters might not be desirable without a precise knowledge of how the grain is to be used.

Doggett:
Such detailed basic work should be done at cooperating universities. A program such as that at ICRISAT should concentrate on the identification of the resistant lines.

Resistance Sources

Bhrane:
We need to expand our sources of resistance, for at present the number of lines with resistance is small. The area in Ethiopia where E35-1 originated is a high-rainfall, long-season, high-temperature area. Other sorghums collected from this area are likely to have mold resistance. There must be other locations where mold resistance occurs. Can ICRISAT conduct specific collections for grain-mold resistance?

House:
Yes, we have plans for this.

Doggett:
It would be helpful to know the origins of resistant lines, so that we can collect more material from these areas. We should, however, try to broaden the genetic base of resistance by collections in several geographical locations. If we have a broad genetic base for grain-mold resistance, the material can be utilized in population breeding.

Williams:
Are changes necessary in the operation of ICRISAT's International Sorghum Grain Mold Nursery?

Rosenow:
To aid in evaluation of sorghums of differing maturities, I suggest you add susceptible check entries which span the maturity range in the ISGMN. The experimental entries could then be compared with the susceptible check of the corresponding maturity.

Sundaram:
The ISGMN should be continued, but the number of entries should be restricted and only the very best retained from the previous season. The cooperators should be allowed flexibility in managing the nursery to guarantee maximum infection pressure.

Denis:
The entries in the ISGMN should include some good agronomic types. Checks should be distributed throughout the field to demonstrate variations in inoculum or in local environmental conditions which might influence mold development. Experimental entries should then be rated in comparison with these widely distributed checks.

Selvaraj:
The success of screening for grain molds at most ISGMN test locations depends on natural mold development, and thus environmental factors at a critical stage of plant development are important. As all test lines do not flower at the same time, some may get conditions more conducive to molds than others at the critical growth stage. Planting the test lines at different dates could allow flowering at the same time.

Williams:
This is an important problem which is encountered in many disease-resistance screening programs. It is not practicable to have different planting dates of different entries, for several reasons. The dates of flowering should be recorded and used in the analysis of the final data.

Grain Quality and Mycotoxins

Frederiksen:

What is the need for marketability and yield-loss studies? Is the only good product completely mold-free?

Denis:

One needs to see the appearance of the grain before making a decision. For example, if it is for human consumption, is anthocyanin present? We must also know the consumer's circumstances — does he have the opportunity to choose between a molded product and a nonmolded product? If there is food shortage, relatively moldy grain has to be, and is, consumed. Even very susceptible lines may be useful for other purposes, such as beer making.

Frederiksen:

Although there are reports of mycotoxins in sorghum they seem to occur rarely. Is this so, and why?

Bhat:

Maize, groundnuts, rice, and wheat are more important for mycotoxins than sorghum. We do not know why.

Frederiksen:

Apart from mycotoxins, do other potentially injurious products occur in sorghum grain? As we develop higher resistance to grain molds, is there a chance that we will build up levels of plant-produced carcinogens or other toxic products?

Bhat:

I do not think so. I know of no naturally occurring plant-produced carcinogens detected in sorghum grains.

Chemical Control

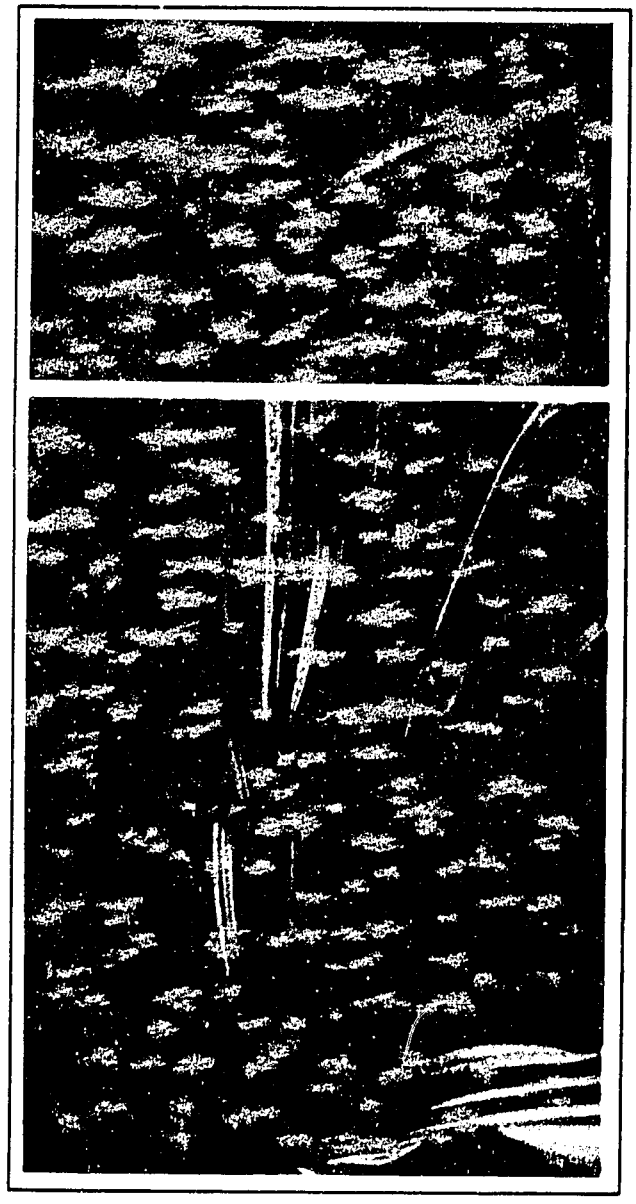
Anahosur:

Is chemical control of grain molds feasible for seed-production crops?

Williams:

It probably is feasible to think of controlling grain molds with fungicides in the case of a few valuable breeding stocks. I do not think it is feasible on a crop basis for several reasons. For seed production, the best control method is avoidance, by planting the crop at a location in a season when grain molds are not a problem.

Sorghum
Downy Mildew



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Sorghum Downy Mildew in Asia: Assessment of Present Knowledge and Future Research Needs

K. M. Safeeulla and H. Shekar Shetty*

Sorghum downy mildew (SDM), caused by *Peronosclerospora sorghi* (Weston and Uppal) Shaw, was first reported in India by Butler (1907). Subsequently, it has been reported from China, the Phillippines (Anon. 1966), and Thailand (Exconde 1970). Downy mildew on sorghum is of economic importance in the South Indian states of Karnataka, Tamil Nadu, and in Maharashtra. Considerable work on many aspects of the disease and its causal organism has been documented by Safeeulla (1976a), including information on distribution, host range, taxonomy, morphology of the asexual and sexual spores, disease symptoms, cytology, spore production, liberation, dispersal and germination, inoculation techniques, cross inoculation studies, tissue culture studies, host variety reactions, and an extensive bibliography.

In the present paper, we attempt to bring into focus recent work with special emphasis on the work which is being done at the Downy Mildew Research Laboratory at Mysore. Critical gaps in our knowledge and areas that require further investigation or confirmation will be highlighted.

Host Range

Sorghum downy mildew is reported to infect the following species in Asia:

- *Euchlaena mexicana* Schrad.
- *Heteropogon contortus* (L.) Beauv.
- *Panicum typheron* Schultz.
- *Sorghum halepense* (L.) Pers.
- *Sorghum bicolor* (L.) Moench
- *Zea mays* Linn.

H. contortus is reported as a host of *P. sorghi* in Rajasthan, India (Dange et al. 1974). Oospores have not been reported on maize in Rajasthan, but occur there on *H. contortus*, which is an important collateral host in perpetuating the disease. In other parts of India where SDM is common on sorghum and maize, and abundantly produces oospores, the role of collateral hosts is limited. In Venezuela, collateral hosts play a major role in the perpetuation and spread of SDM (Malaguti 1976). Considering the extent to which SDM is spreading, there is a possibility of finding more collateral hosts. In Thailand, *Sclerospora* spp have been reported on *Dichanthium caricosum, Panicum cambogiense* and sorghum (Pupipat 1976; Renfro and Pupipat 1976). These DM pathogens need to be studied in detail, and assigned to the proper species.

Taxonomy

The causal agent of SDM was first reported as *Sclerospora graminicola* (Sacc.) Schroet. on the basis of its oogonial phase. Kulkarni (1913) observed the asexual phase of the fungus and described it as a variety of *S. graminicola.* Weston and Uppal (1932) assigned Kulkarni's variety to a new species as *Sclerospora sorghi.* Shaw (1978) has separated *S. sorghi* from *S. graminicola*, and included it in the genus *Peronosclerospora*, along with other species of conidia-producing *Sclerosporas.* The causal organism is now known as *Peronosclerospora sorghi* (Weston and Uppal) Shaw. The synonyms are:

- *S. sorghi* Weston and Uppal (1932);
- *S. graminicola* var. *andropogonis-sorghi* Kulkarni (1913);
- *S. graminicola* auct. non Schroeter (1879);
- *Protomyces graminicola* auct. non Schroeter (1876).

The fungus that causes SDM in Karnataka, Tamil Nadu, Maharashtra, and Andhra Pradesh

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belongs to the same species; however, the same cannot be said about the SDM fungus that occurs in Rajasthan (India), Thailand, and the Philippines. There is very little information available on the SDM fungus reported from China. It is difficult to comment on the similarity or differences between the pathogens reported from China and other Asian countries until comparative studies have been made. However, there is a clear distinction between the SDM pathogens present in south India on sorghum and on maize, and in Rajasthan on maize which has been identified as *P. sorghi*. There are differences in the morphology of the asexual phase in degree of oospore formation and in host range. The SDM fungus in Rajasthan does not infect sorghum, infects maize and *Teosinte*, producing only the conidial stage in these two hosts, but producing abundant oospores in *Heteropogon contortus*. The SDM fungus common in some parts of South India infects sorghum and maize forming oospores, but does not infect *H. contortus*. The malformation of tassels on SDM infected maize commonly observed in Karnataka is not noticed in Rajasthan. These differences have been documented by various workers (Safeeulla et al. 1975a; Safeeulla 1976a; Payak 1975). Another important difference between the SDM fungi from South India and Thailand is in the number of nuclei present in the conidia. The number of nuclei per conidium at Mysore varies from 16 to 34 (average 22) whereas the number observed in the conidia from Thailand varies from 8 to 15 (average 11.6) (Bhat, personal communication). Counts of nuclei in the conidia from Rajasthan will be helpful in identifying the downy mildew on maize there.

Kenneth (1976) expressed the opinion that *S. maydis* (Racib.) Butler and the SDM fungus belong to the same species, existing as two different physiological races, one infecting sorghum and maize and the other exclusively related to maize or maize-teosinte hybrids. Information now available does not support this view. Ample evidence exists to prove that the fungus from Rajasthan is distinct from *P. sorghi*, and is so considered in this paper. Recent investigations at the Mysore Downy Mildew Research Laboratory indicate that the morphology of the oospores of the SDM fungus from Rajasthan is different from the morphology of *P. sorghi* oospores from South India. Differences in oospore morphology are of taxonomic significance as reported by Safeeulla and Shaw (1963).

Conidia

In *P. sorghi* conidia are borne on erect conidiophores with a basal cell and a main axis which is usually dichotomously branched. The basal cell is knobbed and bulbosed at the bottom and is 100 to 150μ long. The main axis extends in length from 80 to 150μ from the septum of the basal cell to the beginning of the branched system. The diameter of the main axis varies from 10 to 25μ. The branched system consists of a succession of short, dichotomies usually involving primary, secondary and tertiary branches terminating in tapering sterigmata usually about 13μ long. Conidia vary in size from 15 to 28.9 x 15 to 26.9μ. Most frequently they measure 21 to 24.2 x 19 to 22.9μ under natural conditions. Conidia are hyaline with a thin wall, continuous at the apex, unmodified and without any papilla of dehiscence, and germinate invariably by hyphae. The morphology and measurements of the conidiophores and conidia are important in differentiating *P. sorghi* from other downy mildew (DM) fungi. Under excessive humidity and temperatures exceeding 30°C the morphology of the conidiophores changes (Safeeulla 1976). When defining the fungi involved in the DM complex, measurements of the basal cell, main axis up to the level of branching, and branches with conidia, should be given separately.

Conidia can produce local and systemic infection (Safeeulla and Thirumalachar 1956; Safeeulla 1976). Inoculations of healthy leaves with conidia confirmed that secondary infection of neighboring plants occurs by the conidia liberated from systemically infected plants and from local lesions. A big leaf canopy and high humidity favor secondary infection by conidia. Conidia not disseminated after production germinate on the surface of the leaf, producing a downy mycelial mat.

Wind plays an important part in the dissemination of conidia from infected plants. *P. sorghi* conidia were detected in the air-spora from a sorghum field at Mysore (Shenoy and Ramalingam 1976a) during 137 days of the year. The yearly mean concentration of *P. sorghi* conidia was 10.6/m^3. The period of occurrence

of conidia was between May and December. The peak period was during July and August. The highest number of conidia 8109/m³ were trapped in the month of June. The percentage in total air-spora being 0.23.

More than 12 000 conidia/cm² of infected leaf surface have been recorded (Safeeulla 1976a; Shenoy and Ramalingam 1976b). More conidia are liberated from the lower surface of the leaf than from the upper surface. Maximum sporulation takes place at 100% relative humidity (RH). At 80% RH and below, no conidia are produced. Optimum temperature for sporulation is 21 to 23°C. The optimum temperature for conidial germination is 23°C. Conidial germination does not take place below 10°C or above 32°C. Like RH and temperature, light also plays an important part in sporulation. Darkness undoubtedly promotes sporulation, but exposure of the infected plant to light is a prerequisite for sporulation. Our experiments have shown that exposure of the infected plant to light for at least 1 hr prior to incubation is necessary for sporulation.

Chemical analysis of the asexual phase of *P. sorghi* may give a clue to the nutritional requirements and chemical composition of the fungus. Rasheed et al. (1978) gave corroborative evidence for recognizing physiological differences within *S. graminicola* on the basis of examination of sporangia for soluble proteins using polyacrylamide gel electrophoresis. Similar studies with conidia of *P. sorghi* may also be rewarding.

Systemic infection results if conidia come in contact with seedlings. Sorghum plants are susceptible to conidial inoculum up to 25 days after sowing. Plants inoculated just after emergence produced systemic infection. Plants inoculated at the 1- to 2-leaf stage showed systemic symptoms at the 4- to 5-leaf stage. In plants inoculated at the 3- to 4-leaf stage symptoms took 12 days to appear (at the 7 to 8 leaf stage). As the leaf stage at inoculation advances, the incubation period for systemic symptoms becomes longer. When plants are inoculated after the 6th leaf stage (20 days after seeding) systemic symptoms are not observed, and only local lesions appear (Table 1). Local lesions appear even if the plant is inoculated at the boot-leaf stage. With increase of conidial inoculum load, the percentage of infected plants also increases. At conidial concentration of 100 to 400 conidia/ml it takes 10 to 14 days for expression of the disease. More than 20 days are required if the conidial concentration is lower. Seedlings inoculated with higher concentration of conidia (more than 1000 conidia/ml) show symptoms earlier, and the symptoms appear on the first leaf.

Shenoy and Ramalingam (1976b) in their aerobiological and epidemiological studies on SDM, have developed scales for estimating the severity of SDM in the field from data on the

Table 1. Downy mildew incidence in sorghum plants inoculated at nine growth stages.

Inoculation at*		Rating at		Systemically infected plants %	Plants showing local spots %
Leaf stage	Days after seedling	Leaf stage	Days after inoculation		
0 (coleoptile)	4	1-2	5	100	0
1-2	5	4-5	8	100	0
2-3	8	5-6	10	100	0
3-4	10	7-8	12	100	0
4-5	14	8-9	12	90	0
5-6	18	8-9	12	90	10
5-6	20	9-10	12	5	95
6-7	25	9-10	12	0	100
7-8	30	11-12	12	0	100

*Inoculum concentration: 25 000–40 000 conidia/ml

occurrence of conidia. They describe the following stages in the development of local lesions:

1. Water-soaked spots of 0.2 to 0.6 mm in length;
2. Leaf decolorized in the infected area, with spots increasing in yellow areas;
3. Spots appearing yellow on the upper surface of the leaf with downy growth on the lower surface;
4. Spots turning brown;
5. Dark greyish or brown necrotic spots.

Symptoms appear on the lower leaf surface in the form of water-soaked, chlorotic or brown patches. Sporulation takes place in the latter two stages. The spots are more abundant on the tips than at the base, because the conidia first come in contact with the tips of the unfolding leaves. Local lesions are common in susceptible sorghum cultivars.

Oospores

In South India *P. sorghi* produces abundant oospores in sorghum and it occasionally produces oospores in some varieties of maize, but does not in others. The occurrence of oospores in 11 entries of maize, when inoculated with conidia and held in the glasshouse, suggests that the production of oospores is not mainly dependent upon host variety but is governed by temperature, humidity, and other environmental factors (Safeeulla et al. 1975a).

As many as 155 800 oospores have been obtained from 1g dry weight of systemically infected sorghum leaf (Shenoy and Ramalingam 1976b). This is equivalent to 856 oospores per 1 cm^2 of the leaf.

There is ample evidence to prove that *P. sorghi* is perpetuated from season to season and transmitted to new areas through oospores. Transmission of the pathogen in the form of oospore-infected soil has been reported by McRae (1924). Oospores have also been located on seed, and in glumes and pericarp of systemically infected sorghum plants (Safeeulla 1976a).

In aerobiological studies of a downy mildew infected sorghum field oospores were detected in spore catches on 21 days in a year during the months of May, July, and December (Shenoy and Ramalingam 1976b). The same authors determined the yearly mean concentration of oospores to be 0.12/m^3. The percentage of oospores among the total air-spora was 0.01. Viable oospores have been found in droppings of cattle fed with infected leaves (Safeeulla 1976a).

Oospores of *P. sorghi* remain viable in soil and in the laboratory for several years. For the oospores stored in the laboratory in the form of debris, the percentage of infected plants obtained was 22.5, 10, and 3.6 respectively at the end of the first, second, and third year of storage. The percentage of infected plants from oospores mixed with soil and exposed to weathering was higher than that from oospores stored in the laboratory in all the 3 years. However, there was a gradual reduction in the percentage of infected plants after 1 year. Up to 60% of the plants were infected by fresh oospores.

Oospore Germination

Many workers have failed to observe oospore germination in the laboratory although they have succeeded in inoculating sorghum plants with oospores in soil. Weston and Uppal (1932) reported direct germination of oospores by following the technique of Hiura (1930). Safeeulla (1970) reported up to 10% oospore germination of 1-year-old oospores in contact with the germinating seeds of susceptible sorghum varieties. This was confirmed by Safeeulla et al. (1975a), who observed 5 to 20% germination of oospores in contact with susceptible sorghum and maize varieties. Germination of oospores has been observed in the presence of roots of maize, wheat, oats, cotton, and sorghum seedlings (Pratt 1978). No germination was observed in oospores incubated with resistant cultivars. Safeeulla et al. (1975a) described the germination process. The germ tubes emerge from any point in the spore wall. They were branched in some cases. The contents of the oospores migrated into the germ tubes, leaving the oospores empty. Septa were formed at the bases of the germ tubes. Twelve to 14 nuclei migrated into the germ tubes immediately after their formation. Weathered oospores had higher percentage germination than fresh oospores (Table 2). SDM infection was obtained in a series of crops grown in soil to which oospores were added only once. This indicates that all oospores do not germinate at the same time. Active substances from host

Table 2. Germination capacity of oospores of different ages when inoculated with seedlings of SDM – susceptible DMS 652 sorghum.

Date of collection	Date of incubation	Dates oospores germinated	Oospore germination %
5.12.1972	5.12.1972	9.12.1972	2.5
5.12.1972	5.3.1973	8.3.1973	4.2
5.12.1972	6.7.1973	9.7.1973	6.6
5.12.1972	5.11.1973	8.11.1973	12.8
5.12.1972	5.2.1974	8.2.1974	3.0
5.12.1972	5.6.1974	8.6.1974	4.5
5.12.1972	5.10.1974	8.10.1974	7.2
5.12.1972	5.11.1974	8.11.1974	6.0
16.8.1969	10.12.1972	15.12.1972	4.4
16.8.1969	12.11.1973	16.11.1973	2.0
16.8.1969	11.11.1974	15.11.1974	1.5

roots may trigger the process of germination, and only those oospores which come within the sphere of host root exudates germinate.

The ability of oospores to remain viable for several years is important for the spread of SDM. As all oospores do not germinate at the same time, the fungus is able to perpetuate for several seasons. Considering the genetic differences among the varieties and physiological changes due to aging, one would expect the root exudates to have definite effects on the behavior of the pathogen in the rhizosphere. This has been emphasized by several workers. Since oospores of *P. sorghi* germinate only under the influence of living susceptible host plant roots, investigations have been carried out (Safeeulla et al. 1975b) on biochemical differences in susceptible and resistant varieties (Tables 3, 4). Fifteen free amino acids were detected in the susceptible and resistant varieties. Of these, there was more cysteic acid, cysteine, valine, and proline in the susceptible variety DMS-652 than in the resistant variety. Aspartic acid, glutamic acid, serine, lysine, arginine, hydroxyproline, methionine, and phenylalanine were present in medium concentrations in both varieties.

No qualitative differences in the free amino acids and carbohydrate content of root exudates of susceptible and resistant varieties of sorghum were observed. However, quantitative differences are evident, and concentration may be a factor in stimulating oospore germination.

Table 3. Comparison of relative quantities of free amino acids in 3-day-old seedlings of susceptible and resistant sorghum varieties.

Amino acids	Susceptible variety (DMS 652)	Resistant variety (IS-184)
Cysteine	***	**
Cysteic acid	***	**
Aspartic acid	**	**
Glutamic acid	**	**
Glycine	*	*
Lysine	**	**
Serine	**	**
Arginine	**	**
Hydroxyproline	**	**
Valine	***	**
Methionine	**	**
Phenyl alanine	**	**
Leucine and isoleucine	*	*
Proline	***	**

* Low ** Medium *** High

Germination of *P. sorghi* oospores was not stimulated by pure samples of any of the amino acids or carbohydrates detected in the root exudate, individually or in combination. Other substances, such as organic acids and phenolic

Table 4. Free amino acid content in root-exudates from 4-day-old seedlings of susceptible and resistant sorghum varieties.

Amino acids	Susceptible variety (DMS-652)	Resistant variety (IS-184)
Cysteic acid	**	*
Glutamic acid	**	*
Glycine	*	*
Lysine	**	*
Arginine	**	*
Serine	*	*
Alanine	**	**
Hydroxyproline	**	*
Valine	*	*
Phenyl alanine	*	*

* Low ** Medium *** High

compounds, which may affect germination, have not been studied. It is possible that root exudates may harbor organisms which synthesize growth factors, which in turn may stimulate oospore germination. Exudate from a highly susceptible variety shows a higher concentration of some amino acids than exudates from the resistant variety (Table 3). The presence of some of these amino acids in higher concentration may be conducive to the growth of microorganisms, thus creating a complex of rhizosphere microflora.

Infection Process

Infection process of the oosporic germ tube through host root has been followed (Safeeulla et al. 1975a). Germ tubes towards the root and the length of the germ tubes depended upon the distance between the germinating oospore and host root surface. Soon after the germ tube came in contact with the surface of the root, an appressorium was formed which developed a narrow finger-shaped infection peg which penetrated the epidermal cell. Most of the germinated oospores brought in contact with nonhost seedlings of pearl millet and finger millet (*ragi*) developed appressoria, but there was no entry. Although infection pegs were noticed on ragi seedlings, there was no subsequent spread of the fungus into the tissue. Sorghum seedlings, killed and brought in contact with germinated oospores, did not induce appressorial formation.

Infection by germinating oospores in soil results in systemic symptoms, which appear at different stages of plant growth from the 3-leaf stage to maturity. First symptoms of the disease in the field occur on the second leaf onwards, never on the first leaf. If the symptoms do not appear before 35 days of sowing, third and fourth leaves escape infection. After 55 days, symptoms appear only at the 5-to 10-leaf stage. In 75-day-old plants, symptoms appear on 5-to 12-leaf stage (Table 5). However, downy mildew symptoms can appear on basal and nodal tillers in the later stages. In resistant sorghum cultivars, symptom expression is normally on basal and nodal tillers. More investigations are necessary to determine the relative importance of the various sources of inoculum, the different types of symptoms expressed, and the extent of damage caused at different stages of plant growth.

Reaction of Sorghums to *Peronosclerospora sorghi*

Safeeulla and Thirumalachar (1955) screened a few lines of sorghum and maize, using both conidial and oosporic inoculum. Sundaram (1972) listed sources of resistance to SDM as a result of field evaluation in India. Safeeulla (1976a) screened more than 500 sorghum entries against *P. sorghi* in sick plots at Mysore, and found 18 lines free from infection, 87 moderately resistant, and the remaining entries highly susceptible. None of them were resistant to conidial inoculation in the laboratory. Subsequently, a large number of inbreds, hybrids, and varieties have been screened in sick plots at Mysore, including the materials of the International Sorghum Downy Mildew Nursery and All India Coordinated Sorghum Improvement Project. There is good SDM resistance in different groups of sorghums from different countries. Some of this resistance has been incorporated in the hybrids and varieties released for cultivation in India. However, scientists should be vigilant about the performance of the released cultivars, and replace them with suitable varieties when the resistance breaks down. Safeeulla (1977) indicated that SDM resistance

Table 6. Sorghum reaction to downy mildew in plots at Mysore infected by *Peronosclerospora sorghi* oospores.

Entry	Total plants	Plant age: 35 days	55 days	75days	Total plants with systemic infection
		No. of plants with systemic infection			
Ag 1002	50	0	0	0	8.33
Ag 1010	36	2 (4,5)[a]		1 (7,8,9)	8.33
Ag 1012	74	1 (5,6)			1.35
Ag 1003	43	1 (5,6)	1 (6,7,8)	1 (7,8,9)	6.97
Ag 77007	33	–	–	5 (6,7,8)	15.15
Ag 77012–2	54	–	–	1 (6,7)	1.85
Ag 77012–3	42	1 (6,7)	–	–	2.38
Ag 77018–1	41	–	–	1 (7,8)	2.43
Ag 77018–2	54	3 (5,6,7)	1 (6,7)	8 (8,9,10)	22.22
Ag 77018–3	49	1 (4,5,6)	–	2 (5,6,7)	6.12
Ag 77559	75	3 (4,5)	2 (6,7)	14 (5,6,8)	25.33
Ag 77567	41	1 (4,5)	–	–	2.43
Ag 77603	47	–	–	–	0.00
Ag 77711	49	–	–	–	0.00
Ag 77735	70	–	1 (5,6)	4 (7,8,9)	7.14
Ag 771002	71	3 (5,6)	4 (6,7,8)	–	9.85
Ag 771006	54	–	–	2 (6,7)	3.70
Ag 771009	68	2 (5,6)		6 (7,8,9)	11.76
Ag 771014	62	1 (5,6)	–	2 (10,11,12)	4.83
Ag 771041	50	1 (4,5)	–	4 (6,7,8)	10.00
Ag 77045	110	–	–	–	0.00
Ag 771049	81	2 (3,4,5)		6, (7,8,10)	9.87
Ag Sart	79	1 (4,5)	1 (9,10)		2.53
Ag Brandes	100	4 (5,6)	3 (6,7,8)	28 (6,7,8,9,12)	35.00
Control DMS 652	148		41	60	68.24

a. Number of leaves on which chlorotic symptoms first appeared is indicated in parentheses.

is breaking down in some cultivars. Subsequent investigations and observations have confirmed this (Table 6). There can be no disagreement that some of the resistant cultivars become vulnerable to either a new pathogen, a new race of the old pathogen, or both. Pathologists have to strive constantly to locate and incorporate genes that are resistant to new pathogens subsequent to the release of new hybrids and varieties of sorghum.

At the Downy Mildew Research Laboratory at Mysore, resistance breaks down under artificial epiphytotic conditions earlier than it would in the farmers' fields. Such information could be used in providing information to farmers and breeders.

Biochemical Status of the Susceptible and Resistant Sorghum Plants

Aspartic acid, B-alanine, cysteine, glutamic acid, serine, glycine, lysine, arginine were in higher concentration in healthy leaves than in diseased leaves (Table 7). Cysteine and arginine were absent from diseased leaves. These results indicate that the physiology of the sorghum plant with SDM is greatly altered. Other groups of substances such as carbohydrates, organic acids, phenols, etc., may also change with SDM infection.

There is no qualitative or detectable quantitative difference between protein-bound amino acid of healthy and mildewed leaves of sor-

Table 6. Sorghum reactions to sorghum downy mildew in sick plots at Mysore[a] (1972–1978).

Entry No.	IS-8850	IS-8865	IS-8867	IS-163	IS-2382	IS-3364	IS-3926	IS-4324	IS-9283	IS-148	CS-3541	CSH-5	CSH-6	IS-184	IS-2918
	Percentage of systemically infected plants														
Trial															
1.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
3.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
4.	6	4	4	0	0	0	0	0	0	0	0	0	0	0	0
5.				2	1	5	16	1	12	0	0	0	0	0	0
6.										2	4	2	2	0	0
7.														0	0
8.														0	0
9.														0	0
10.														4	6

a. Susceptible check, DMS-652 showed infection ranging from 70 to 100%

ghum (R-147). This indicates that the fungus uses up only the free amino acids which are in the cytoplasm, and does not degrade or break down the protein which contains the amino acids in bound form.

Crude protein content of sorghums resistant to SDM (IS-184, 148, CSV-5) and susceptible (DMS-652 and Swarna) is given in Table 8. The total crude protein content was significantly more in the leaves of resistant varieties than in the susceptible ones. After infection, leaves of susceptible varieties had more protein than their healthy counterparts.

Table 7. Amino acid content of healthy and SDM infected sorghum leaves (R-147).

Amino acids	Healthy	SDM infected
B-Alanine	+++	++
Leucine	++	++
Phenyl alanine	++	++
Aspartic acid	+++	++
Cysteine	+++	0
Glutamic acid	+++	++
Serine	+++	++
Glycine	+++	+
Lysine	+++	++
Arginine	+++	0
Threonine	+	+
Valine	++	+
Proline	+	+

The intensity of the spots was categorized into low (+), medium (++), and high (+++)

Table 8. Crude protein content of leaves of sorghum varieties susceptible and resistant to sorghum downy mildew.

Class	Variety	State	Protein content (g/100g)
Susceptible	DMS-652	Healthy	18.60
		Diseased	21.20
	Swarna	Healthy	17.25
		Diseased	22.42
Resistant	IS-184	–	24.65
	148 (CSV-5)	–	23.15

Anthocyanin content of seeds of resistant varieties (IS-5314, IS-184, IS-2042) is more than in the susceptible varieties (DMS-652, IS-302 and IS-555, Table 9).

Susceptible sorghum (DMS-652, Swarna) infected with *P. sorghi* contain more tannic acid than the healthy plants. In the resistant varieties CSV-5 and IS-184, a lesser amount of tannic acid is detected, compared to the susceptible ones. The total amount of phenolic compounds in sorghums in the healthy regions adjacent to the areas colonized by the fungus is higher (Table 10).

Healthy leaves of susceptible varieties show a lesser amount of phenolics than the infected ones. The tannic acid and total phenols are more in seedlings of susceptible than in the resistant varieties. However, their concentration increased in 2-week-old plants and the same trend was recorded up to 6 weeks. This increase in tannic acid and total phenolic compounds can be correlated with the increase in resistance to SDM infection. Sorghum plants are normally SDM susceptible up to 3 weeks and become resistant after this period (Shetty et al. 1975).

Table 9. Anthocyanin content of sorghum seeds, susceptible and resistant to sorghum downy mildew.

Class	Variety	Optical density*
Susceptible	DMS-652	0.925
	555	0.248
	302	0.523
Resistant	IS-184	1.91
	IS-2042	1.96
	IS-5314	1.928

* Solvent: Solute ratio = 4:1

Seed Transmission

Butler (1918) postulated the presence of *P. sorghi* mycelium in the seeds of systemically infected sorghum plants. Jones et al. (1972) reported the occurrence of hyphae of *P. sorghi* in the style, ovary wall, nucellus of carpellate flowers, pedicels, and pericarp of mature seeds of maize. Safeeulla and Shetty (1974) observed mycelium not only in the pericarp and endosperm of maize, but also in the coleoptile and coleorhiza regions. Reduction in the moisture content of the infected seeds as a factor affecting transmissibility of the pathogen has been reported for *P. sorghi* in maize (Jones et al. 1972). Sorghum seeds without glumes developed systemic infection when they were sown in sterilized soil without drying or storage. Seeds with persistent glumes containing oospores developed downy mildew symptoms irrespective of drying on storage (Safeeulla and Shetty 1974).

Table 10. Total phenolic contents of sorghum leaves susceptible and resistant to sorghum downy mildew.

Class	Variety	State	Age of the plants in weeks mg/g		
			1	2	6
Susceptible	DMS-652	Healthy	0.665	1.85	2.1
		Diseased	1.25	2.32	2.27
		Adjacent to diseased area	1.92	2.92	3.36
	Swarna	Healthy	0.72	1.92	2.42
		Diseased	1.65	2.92	3.15
		Adjacent to diseased area	1.95	2.98	3.85
Resistant	148 (CSV-5)		0.625	1.32	1.85
	IS-184	–	0.613	1.12	1.54

Ahmad (personal communication) found oospores in 13 out of 20 seed samples obtained from Argentina, Venezuela, and India. All the seed samples were collected from systemically infected plants, except the seeds of four samples from India, which were collected from artificially inoculated earheads. Six of the 20 seed samples showed the presence of internally seed-borne inoculum. The infection in the glumes varied from 33 to 76%, whereas in the pericarp, the infection was 38 to 59%. In many of the seeds the glumes and peduncles were completely occupied by oospores.

More investigations are essential to determine the factors affecting viability of *P. sorghi* in and on sorghum and maize seeds in order to know what risks there are in seed shipment, and to provide guidance to technical personnel.

Chemical Control

The chemical seed treatment long sought for to control SDM has been found. CGA-48988, a new systemic fungicide (developed by CIBA-GEIGY), is effective against *P. sorghi* on sorghum and maize. Applied as seed dressing, CGA-48988 gave excellent control under heavy inoculum pressure from oospores present in the sick plots, oospores coating on the seed, addition of oospore material in the form of leaf debris, and conidial inoculum from infector rows planted 3 weeks earlier than tested plants. The control of SDM under these circumstances is encouraging. The chemical seed treatment can be used to prevent introduction of seed-borne inoculum, check the spread of mildew to disease-free areas, and reduce soilborne oospore inoculum. We already have preliminary data on the efficacy of the new chemical (Venugopal and Safeeulla 1978; Safeeulla and Venugopal 1978).

Detailed investigations, in collaboration with scientists from CIBA-GEIGY, on the efficacy of the different formulations, method of application, optimum dosage, phytotoxicity, effect on seed germination, field and laboratory evaluation of different chemical formulations, artificial inoculation of chemically treated plants, and the effect of the new chemical along with other fungicides recommended for controlling other important diseases and pests, have been undertaken, and will be published elsewhere.

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Sorghum Downy Mildew in the Americas

Gino Malaguti*

Sorghum (*Sorghum bicolor* [L.] Moench) was long considered a "stranger" in the Americas, where its cultivation was limited to experimental plots, frequently as a curiosity.

The first variety of sorghum to be extensively planted in almost all the countries of the Americas was forage sorghum, "forraje criollo." It is a tall, easy tillering plant, and we now know that it is very susceptible to sorghum downy mildew (SDM). The area of these forage sorghums has gradually decreased, being replaced by grain sorghums, which are widely grown today.

In Venezuela in 1947 to 1948, only small experimental grain sorghum plots were cultivated, in order to observe their adaptation and yield. From these tests the first Venezuelan studies on smut (*Sphacelotheca sorghi*), leaf spots (*Cercospora sorghi* and *Gloeocercospora sorghi*), and midge (*Contarinia sorghicola*) were initiated.

There is no doubt that the first experimental sorghum seeds, and with them probably many diseases, were introduced into Latin America from the United States. The USA in turn received sorghum seeds, and probably with them many sorghum pathogens, from some African and Asian countries.

In the case of *Peronosclerospora sorghi* (Weston and Uppal) Shaw it is well known that oospores of the fungus contaminate seed surfaces. They may be contained in the glumes of the seeds, and in this way be easily disseminated.

We think it is irrelevant to discuss or to speculate where the SDM agent came from. It is an inevitable fact that when the cultivated area was expanded year after year, diseases started to appear. Often genetic improvement for better quality, higher yield, and uniformity leads to a greater vulnerability to pathogens, with more possibilities of epiphytotics, e.g., the recent epidemics caused by *Helminthosporium maydis* among maize hybrids with "T" cytoplasm.

Thirty years ago *P. sorghi* was limited to four African and two Asian countries, and Italy. According to Harlan (in Frederiksen and Renfro 1977) the first sorghums might have been cultivated in some savannas of tropical Africa, and from there taken to India and Pakistan. Later, improved sorghums might have gone back to Africa from India, probably carrying some new diseases, among them SDM, whose pathogenic agent might have existed in a local wild host such as *Heteropogon contortus*. We can also assume that the first sorghums were brought to America in the seventeenth century along with the slave traffic from Africa. In many Central and South American countries, there are some "indigenous" or "criollo" big, white-grained sorghums that natives use mostly for preparing beverages or toasted foods. It is evident that the present commercially cultivated sorghums or their ancestors, were reimported from Asia and Africa into the American continent, especially into the USA, and from there to the other American countries.

It seems that the hypothesis suggested by Dickson (1956), that the SDM pathogen might have been previously present in the Americas on teosinte (*Euchlaena mexicana*) or maize, both hosts of American origin, is unlikely, but not impossible.

P. sorghi was unknown in the Americas before 1960, but two other graminicolous DM pathogens were known: *Sclerospora graminicola* and *Sclerophthora macrospora.* The former was reported in 1909 on maize in Argentina and on *Setaria viridis* and maize in the USA between 1925 to 1928 (Melhus et al. 1928; Frederiksen and Renfro 1977); the latter — *S. macrospora* — was reported on various graminicolous plants (Whitehead 1958) around 1920 in the USA. Later it was observed on wheat in Mexico and the USA; on sorghum

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and maize in the USA and on sugarcane in Peru (Revilla 1955). Recently, it was observed by the author in Venezuela on *Eleusine indica*.

The fact the *S. graminicola* has been found only sporadically, and *S. macrospora* only occasionally and under special flooding conditions on maize and sorghum in the Americas, indicates that *P. sorghi* is the only serious downy mildew (DM) pathogen of sorghum and corn, and it is a real threat to the cultivation of these cereals.

The grain sorghum cultivated areas have increased enormously in the Americas in the last decade (Maunder 1975). Sorghum has increased in Venezuela from 1000 ha in 1965, to 6000 ha in 1973, to 300 000 ha in 1978. According to FAO data for 1974, Argentina increased its sorghum cultivated area from 865 000 ha in 1965 to 2.5 million ha in 1974; Mexico from 200 000 ha to 1.2 million ha; and South America in general from 800 000 ha to more than 3.2 million ha. This increase is due to the great adaptability of sorghum to marginal soils and different climatic conditions of the semi-arid regions, and, to a certain extent, to its resistance to drought and diseases. It also lends itself well to combine harvesting. In many regions, sorghums have been replacing other crops such as maize, cotton, rice, and sugarcane, which require better soil quality, larger quantities of water, and more labor and management. It is probable that in the near future the diet of our people will have to change because of the greater availability of sorghum.

Peronosclerospora sorghi was first observed on the American continent in Texas between 1958 and 1961 (Reyes et al. 1964). Not long afterwards, it was reported in other states of the USA and different countries of Central and South America. The disease has now been reported in five countries of South America; four countries of Central America, and 13 states of the USA. It is probable that the disease will extend to other regions of the Americas, where sorghum and maize are planted, due to the likelihood of the fungus spreading through oospores on infested sorghum seeds.

Sorghum Downy Mildew in the Americas

In their excellent review, Frederiksen and Renfro (1977) offered a panoramic view of the global status of the DM diseases of maize, including SDM. Historical aspects, geographic distribution, incidence, and information about etiology, epiphytology, and control are partly summarized in this review.

P. sorghi became established in the Americas some time in the decade 1965 to 1975, a period in which sorghum cultivation greatly increased. In Venezuela, Brazil, Bolivia, and Mexico sorghum and maize are commonly both hosts of the disease. Sometimes the disease is prevalent in sorghum and maize, as in Argentina, parts of Mexico, and some states of the USA. Sometimes SDM may be more prevalent in maize. SDM symptoms display great variability in the two main hosts, e.g., in maize, phyllody and tassel malformations may or may not be prominent. It is difficult to assert whether different biotypes of the fungus are present, or if the above-mentioned variations are due to the influence of environmental conditions, or if they are simply related to host reactions.

In almost all countries SDM was first observed on forage sorghum or on sudangrass (*Sorghum sudanense*) and sorghum × sudangrass hybrids, which are undoubtedly the most susceptible hosts. At the same time it has been reported on johnsongrass (*S. halepense*) or "false" johnsongrass (*S. verticilliflorum* and *S. arundinaceum*), and on many natural hybrids between them. *P. sorghi* frequently produces local lesions with abundant conidia in old leaves of these species, *Cercospora* infection is generally found in the same lesions.

In this paper I present up-to-date information on SDM obtained from different countries, based on the available literature and personal communications received from the plant pathologists, plant breeders, and agronomists of South and Central America who answered my requests for information.

North America

United States of America

The USA is the largest sorghum producer on the American continent, where more than 6 million ha are grown. This is about twice the area planted in all Latin America. It is therefore not surprising that, in the Americas, SDM first appeared in the USA. Sorghum genetic material

was introduced into the USA a century ago, and research has been conducted for more than 60 years (Edmunds 1975). As might be expected, the disease first appeared in three areas of northern Texas (Futrell and Frederiksen 1970; Reyes et al. 1964), the region where 40% of the national sorghum area is cultivated. Later it appeared in the nearby state of Kansas, which produces 24%.

Between 1961 and 1978 the disease spread to Alabama, Oklahoma, Arkansas, Georgia, Louisiana, Mississippi, New Mexico, Tennessee. In 1972 it appeared in Kentucky (Williams and Herron 1974) and Indiana (Warren et al. 1974), and in 1974 in Illinois (White et al. 1978). SDM has not been reported in Nebraska, a state accounting for 17% of the national production, or in Florida; however, *Sclerospora macrospora* has been observed in this state on Augustine grass (Jones and Amador 1969).

The presence of SDM in the states of Kentucky, Indiana, and Illinois is alarming since they form part of the famous "corn belt" region. In Illinois the disease was reported in both sorghum and maize, with the highest incidence in maize, where it reached 80% in certain areas far from the original infection focus.

The symptoms of the mildewed maize plants in Indiana are brown discoloration of the internal pith, and the formation of abundant brace roots along the stem, up to the sixth node (Warren et al. 1974).

In Texas, incidence of more than 25 to 30% has been reported in sorghum, with up to 80% in certain areas of some fields. Symptoms induced by *P. sorghi* have been compared with those incited by *S. macrospora* (Frederiksen and Bockholt 1977; Futrell and Frederiksen 1970). In most of the states johnsongrass and shattercane are common wild hosts that act as natural and perennial inoculum reservoirs of the SDM fungus.

Undoubtedly, the most complete and most extensive research on DM in the American continent has been conducted in the USA. Many aspects of the disease have been studied, and valuable information on etiology, inoculation techniques, epiphytotic characteristics, cultivar reactions to the disease, sources of resistance, and possibilities of control have been made available.

Many of these results have been usefully applied in many other countries of the Americas where similar tests and research have been or are being carried out.

Mexico

SDM was first reported in Mexico in 1964 in the north-eastern region, north of Tamaulipas State. In 1966 there was an SDM epidemic, suggesting that the pathogen had been present there some years before. Later, it was observed in the higher regions of Guerrero State, at an elevation of 1200 m. At present, the disease is found in the states of Michoacan, Guanaguato, Veracruz, Tabasco, and Nuevo Leon.

Research deals more with breeding for genetic resistance in both maize and sorghum and is mainly conducted in the Centro de Investigaciones Agricolas de Tamaulipas (CIAT), where sources of stable resistance are available. The common hosts are: maize, teosinte, sorghum, and johnsongrass. Besides planting at different dates (anticipating or retarding), researchers at CIAT use oversowing to destory the diseased plants when young, and probably use seed protectant. Tests with Ridomil (CIBA-Geigy 48988) are being conducted at the present time. Among entries in the ICRISAT SDM Nursery, QL-3, CSV-4 and IS-5273 were found to be resistant.

Central America

Among Central American countries, SDM has been reported in El Salvador, Guatemala, and Honduras, but not in Nicaragua, Costa Rica, and Panama.

El Salvador

Among Central American countries El Salvador is the largest sorghum producer, cultivating about 150 000 ha. The disease was detected in 1973 on sorghum, maize, johnsongrass, sudangrass, and broomcorn.

In general the disease has not developed in epiphytotic proportions, but has remained localized in several limited areas. A sorghum improvement project is under way, testing genetic materials by conidial inoculations, and evaluating disease resistance of crystalline endosperm selections of CENTA S-1 sorghum (Wall et al. 1977).

Guatemala

SDM was recorded in 1976 in the Jutiapa Department, in the southeast of the country, in separate areas of five counties (Cordova and Poey 1979). Projects designed to incorporate resistance into the local maize varieties by using Philippine and Thailand DM resistance sources are in progress at the Instituto de Ciencias y Tecnologia Agricola de Guatemala (ICTA). This improvement program is carried on in collaboration with CIMMYT, Mexico.

Honduras

SDM is present in certain areas with a variable incidence.

Costa Rica

The disease is not present in this country.

Panama

It seems that the disease is not present, in spite of previous doubtful reports (Frederiksen and Renfro 1977).

Caribbean Countries

There is no information about the presence of SDM in Puerto Rico, Cuba, Santo Domingo, Haiti, Jamaica, Barbados, Trinidad, etc. It is assumed that sorghum cultivation is very limited in these regions.

South America

Among South American countries, SDM has been reported in Argentina, Brazil, Bolivia, Uruguay, and Venezuela.

Argentina

SDM was first observed on sudangrass and sorgo × sudan hybrids about 1967 to 1968 (Frezzi 1970; Nider et al. 1969). The following year the disease became severe with high incidence (up to 90%) in the northern provinces of Chaco, Santa Fe, northern Cordoba, Buenos Aires, and Tucuman.

The cultivar FS-26 showed good resistance in both Argentina and Texas. This could be an indication that the pathogen strain present in both places is similar. The disease in maize is considered sporadic and unimportant.

Screening of genetic material is conducted in an open field, where a naturally highly infected soil is available. Conidial inoculation in a moist chamber is also carried out.

The outstanding paper by Frezzi (1970) is the most complete phytopathological work from Argentina. He describes the symptomatology of SDM diseased sorghum plants, and makes interesting observations about fungus characteristics, especially regarding conidial formation in relation to humidity content and low night temperatures. As a result of research carried out for 1 year, this author found that conidial production is highest at 18 to 20°C; and that it is drastically reduced when the temperature falls (11 to 15°C in Mar to Apr).

The simple method of placing oospores on humid filter paper in petri dishes, kept at a temperature of 23 to 25°C, (used by Hiura in 1939 for *S. graminicola*) was used to study oospore germination.

Pathogenicity tests were done by "dusting" the seeds with oospores before planting. More than 50% of the infected plants were observed in susceptible cultivars such as Supek-INTA.

In Argentina too the forage sorghums are the most susceptible. Maunder (1973) reported differences of more than 15 tonne/ha in yields of five SDM-resistant and five SDM-susceptible sorghum × sudan hybrids. A reduction of 15 to 20% was found in diseased grain sorghum plots. Resistant forage hybrids like sudan Sx-121 and Sx-131 gave good results where disease is endemic. The resistance was polygenic and partially dominant, and results were obtained when both parents were resistant.

Brazil

Although SDM was first officially reported by Frederiksen in 1974, in the state of São Paulo, the disease might have been present some years earlier (1968 to 1971) in the state of Rio Grande do Sul. In this state the disease has been reported in 25 counties, and among them, in an epiphytotic form, in the county of Santo Antonio de Patrulha, in 1974 (Almeida 1975). Because of the potential vulnerability of maize which is widely cultivated in this area when cultivation in the area is greatly increased, some

quarantine measures — even if for purely experimental purposes — were adopted in order to control the seed trade, especially seed importation. At the same time the reaction of maize and sorghum cultivars to *P. sorghi* was also evaluated. These tests were first carried out in Texas and later in Brazil (Rio Grande do Sul and Sao Paulo) under the coordination of the "Centro Nacional de Pesquisa." At present, the disease is spread over the states of Sao Paulo, Santa Caterina, and Rio Grande do Sul, in an area where 87% of the maize and 80% sorghum of the country are produced.

Fernandes and Nakamura (1977) presented details of SDM in the state of Sao Paulo for 1975 to 1977, describing symptoms observed in maize and sorghum. The occurrence of local and systemic infections in sorghum, and the many symptoms in maize — half leaf, leaf spot, tassel malformation, phyllody, smut association, and brace roots emission along the stem-nodes — were pointed out. The symptoms described are very similar to the ones reported by Malaguti (1977) in Venezuela, and in the USA by Williams and Herron (1974) and Warren et al. (1974).

According to information given by F. T. Fernandes, screening for the selection of commercial or experimental materials is done in the field. High levels of soilborne inoculum are maintained by planting a highly susceptible broomcorn variety, and later plowing it into the soil. Tests are made by planting each cultivar twice, at 1-month intervals, and evaluating reactions at flowering time.

Sorghum and maize materials with good resistance do exist, and they exhibit some resistance stability. In 1977 to 1978 tests, no commercial maize cultivar was fully resistant (up to 10 to 25% infection). About 40% sorghum cultivars were resistant, while others were about 10% infected. Reactions of sorghum material to *P. sorghi* in the USA and Brazil were similar.

The common hosts of the fungus are maize, grain sorghum, forage sorghum, broomcorn, *S. halepense*, and *S. verticilliflorum*.

Bolivia

Although not mentioned in official reports on sorghum problems (Rodriguez et al. 1978), the disease is apparently frequent and severe in sorghum and maize in the important agricultural region of Santa Cruz de la Sierra (Drs. G. R. Granados and J. B. Barnett personal communication).

Uruguay

SDM has been reported in experimental plots of forage sorghum hybrids at "La Estanzuela" Experiment station (Chiara and Artola 1974). The occurrence of the disease in this country is not surprising, considering that it is widespread in nearby Argentina.

Venezuela

The first official occurrence of SDM in Venezuela, on both sorghum and corn, was recorded in limited areas of Yaracuy state in 1973 (Fernandez et al. 1975). However, the disease had been observed some years earlier (1968 to 1969) in a grain sorghum experimental plot at a location of the same state of Yaracuy, on NK-222 hybrid by Ing. Agr. D. Tovar (personal communication). The following year the disease was observed in three other states (Portuguesa, Barinas, and Aragua), and has actually been reported in 14 out of the 20 states of the country, that is, almost every region where corn and sorghum are cultivated. The disease has usually occurred 1 or 2 years after sorghum cultivation has been introduced into a new area.

Studies on SDM in Venezuela were handicapped by the initial mistaken assumption that three distinct diseases were supposed to be present: one on sorghum, exhibiting all the symptoms described in the literature for SDM caused by *Peronosclerospora sorghi*; another on maize, with the conspicuous symptoms of the disease (phyllody, tassel malformation and proliferation — witches' broom condition) described in the literature as "crazy top" (punta loca) and caused by *Sclerophthora macrospora*; the third on maize, showing erect, narrow, yellowish leaves, and ascribed to sugarcane mosaic virus (SCMV) or maize dwarf mosaic virus (MDMV) (Malaguti 1977, Malaguti et al. 1975).

Later, the exact etiology of the disease was established through the abundant downy sporulation observed in all cases on leaf surfaces, and after the positive results obtained in cross inoculations on different hosts. It was

ascertained that the distinct manifestations and reactions of maize, sorghum johnsongrass, false johnsongrass, and sudangrass, artificially cross inoculated with conidia or oospore from each host, were due to the same SDM pathogen (Malaguti 1976; Malaguti and Tellechea 1977; Malaguti et al. 1977).

In spite of these findings, the disease continued to be called "punta loca" (crazy top) or "falsa punta loca" (false crazy top), even though no tassel proliferation, but only erect, narrower leaves were observed in maize, and discolored, barren panicles were seen in sorghum (Malaguti 1977).

Inoculation methods using conidia — in a moist chamber, resembling a simplified version of that used by Craig (1975) — or oospores (pelleting or dusting the seeds, or mixing oospores to the soil) have been studied and employed. In field tests susceptible plants are used as conidia donors, as is done in many countries (Renfro 1977), and are planted in advance around or at the border of the experimental plots. In most tests, seeds were also inoculated with oospores.

Plants of the hosts listed above showed systemic infection when they were properly inoculated, regardless of the inoculum type (conidia or oospore) and regardless of the inoculum source (different hosts of the fungus). The diseased young plants transplanted in the field have produced conidia and oospore, and exhibited symptoms as in the case of naturally infected ones. Oospore production was frequent, and sometimes abundant in susceptible diseased corn cultivars (Antigua G-2; H. Arichuna) (Malaguti 1978).

It is difficult to appraise the incidence of SDM and the losses of sorghum due to it in Venezuela. It can severely attack up to 80% of the plants in some limited areas cultivated with susceptible cultivars, but this high incidence is not commonly found in extensive commercial fields or in the whole region.

During the last 3 or 4 years many studies have been carried out in different locations of the country, testing the reaction to *P. sorghi* or sorghum or maize materials (inbred lines, varieties and commercial hybrids). The results of these studies and observations are partly published (Agudelo 1976; Bejarano 1977, Cabrera and Gatica 1976; Gatica 1977; Malaguti and Tellechea 1977; Nass et al. 1976; Riccelli and Barboza 1976), or they are found in mimeographed reports in different institutions. Mimeographed reports of Ings. Agrs.: Hector Mena and Arnoldo Bejarano, (Programa Cereales, CENIAP, Maracay), Douglas Tovar and Hernan Nass (Estacion experimental Araure, Portuguesa); Carlos Gonzalez, Henry Gatica, and Samuel Cabrera (Foremaiz, Araure, Portuguesa), Venezuela.

Among sorghum hybrids; TE-Hondo, Topaz, Pioneer B-815, Dekalb D-42, and NK-266, were the most resistant to SDM. The most susceptible were: Dorado M, Dorado A, Pioneer 8417, Pioneer 8454, NK-222, and V. Red Swazy. However TE-Hondo and Dekalb D-42 have been found to be very susceptible to sugarcane mosaic virus (SCMV) or to maize dwarf mosaic virus (MDMV).

Among the new national hybrids (Semillas Proseca C.A.) some were SDM susceptible (H. Barinas 2, H. Araure 1, V. Guarico) and others were resistant (Chaguaramas 3, Aguasay 2). The resistance in sorghum seems to be polygenic and dominant, and therefore transmissible to the hybrids. (Riccelli 1977; Riccelli and Barboza 1976).

Among entries of the ICRISAT SDM nursery for 1978, recently tested at Maracay, the following were SDM free: QL-3, UCV-1, UCV-2, SC-120–4, IS-2223, IS-3799 while the following were susceptible: DMS-652, CSV-2, IS-2550, IS-2042, IS-5272, 3660-B, 2077-B.

In maize, sources of resistance have been found in Philippine DM resistance sources, Thailand composites, Swan 1, 3, and Texas 601.

The importance of johnsongrass and "false" johnsongrass, not only as a reservoir of the SDM pathogen from one season to the next, but also as conidia inoculum spreader all year round, has been pointed out and evaluated (Nass et al. 1977). Tests on the influence of some agronomic practices (soil preparation, field fertilization, date and method of planting) gave inconsistent results (Cabrera 1977). Seed treatment and spraying of young plants in the field with the fungicides Benlate, Dithane M-45, Orthocide 50, and Manol (four times at 4-day intervals from emergence) gave poor or no response (Malaguti and Tellechea 1977).

Satisfactory results have been obtained by treating seeds with other chemicals, such as Ridomil (Ciba-Geigy 48988), Melprex (Dodine), and Bayleton. The first gave almost 100% pro-

tection in field and laboratory tests; the second and the third gave 30 and 12% protection, respectively (Tables 1, 2). Further experiments are being undertaken to confirm these results on susceptible sorghum and maize, and to determine the influence of treatments on germination and plant growth.

Other Countries of South America

SDM has not been reported in Colombia, Ecuador, Peru, Paraguay, or Chile to date. It is interesting that SDM has not been observed in Colombia, a neighboring country of Venezuela and Brazil, where sorghum cultivation has notably increased (from 2000 ha in 1962 to about 200 000 ha today), and where about 10% of the cultivated area is planted with cultivars Dorado M and NK-222 that have shown to be susceptible to SDM in other countries. The remaining area is planted with national cultivars, notably the improved variety ICA-Nataima (Torregroza 1978).

According to information received, SDM has not been observed in Ecuador, Peru, or Paraguay. In these countries sorghum cultivation is very limited at the present time.

Table 1. The numbers of SDM infected plants from 60 maize seeds treated with fungicides and planted in pots containing oospore inoculated soil.

Treatment	Seedlings emerged (No.)	Diseased plants (No.)	Infection (%)
Seeds not treated (check)	48	28	58
Seeds treated With Ridomil*	16	0	0
Seeds treated with Melprex*	40	22	55
Seeds treated with Bayleton*	50	22	44

* Slurry treatment, 0.8%

Table 2. The numbers of SDM infected plants out of 300 maize seeds, treated with fungicides and inoculated with oospores, in the hole, at planting in the field (average of 3 replications).

Treatment	Seedlings emerged (No.)	Diseased plants (No.)	Infection (%)
Seeds not treated (check)	223	60	27
Seeds treated with Ridomil*	53	1	2
Seeds treated with Melprex*	50	8	16
Seeds treated with Bayleton*	213	37	17

* Slurry treatment, 0.8%

Summary and Discussion

Literature review and available information received from Central and South American countries suggest the following conclusions:

1. Downy mildews are 'Old-World' diseases. When maize was introduced into Asia and Africa it was found that there were some DM pathogens capable of infecting it: when sorghum, on the other hand, was introduced into the Americas there were probably no local DM agents. Why was SDM, unlike the other DMs, introduced along with seeds, into the Americas? According to Shaw (1976) it could have been due to the better adaptability of *P. sorghi* to tropical and subtropical regions.
2. The introduction of the SDM pathogen into another continent, or from one country, state, or region into another, seems to be associated with the movement of oospore-infested seeds (grain, forage, sudan, or sorghum × sudan hybrids) or the importation of grain sorghum for the feed industry.
3. There is a lack of information about SDM in South and Central America; countries that have no problems with this disease usually do not care about it until the disease becomes a threat. Personal communications on this from Bolivia, Peru, Colombia, Costa Rica, etc., have been received. When SDM appeared in Venezuela and Brazil in 1974, the disease was well known and had been studied in

Texas (Frederiksen and Bockholt 1977; Frederiksen et al. 1973), and Argentina (Frezzi 1970) and, of course, other continents (Safeeulla 1976).

4. In most American countries, forage sorghum, sudangrass, or sorghum × sudan hybrids were the first SDM hosts. "Johnson" and "false johnson" grasses, and shattercane are common wild hosts of the pathogen, and represent a permanent inoculum reservoir, making control measures very difficult. Teosinte is mentioned as a common host in Mexico.
5. SDM has spread mostly in the warm regions of the Americas, between the Equator and the 35° latitude. Commonly it starts suddenly and virulently, decreasing in severity later in the original areas and appearing again in new ones with greater incidence, until it becomes endemic. It is possible that a gradual biological control of the pathogen takes place, as has been pointed out for *Sclerotium rolfsii*.
6. The similarity of reactions of sorghum and maize cultivars to SDM in different American countries suggests that probably only one strain of the pathogen exists in this region.
7. Conidia and oospores are produced in sorghum hosts. Oospores are found embedded in the sorghum leaf tissue between vascular bundles, never in the bundle sheath cells (as it occurs in *S. macrospora*). Conidia shape and size are similar in the American countries (averaging 19 to 25μ). Table 3 gives the measurements of oospores of two pathogens on some hosts. Oospores (averaging 30 to 36μ in diameter) are frequently found in very susceptible maize cultivars in special environments (Malaguti 1978). Leaf shredding, which is a very common symptom in sorghums, is rare in maize. Conidia are formed in young leaf tissue, oospores in old and necrotic tissue. Both structures may coexist in the same tissue in sorghum, but not in maize.
8. In some American countries or states, SDM is more prevalent on sorghums, and in others more on maize. The description of symptoms of SDM diseased sorghum plants is very similar in all regions. On maize these symptoms often differ, showing sometimes only erect, yellowish striped leaves, or a severe phyllody and tassel and ear proliferations. The latter

Table 3. Comparative measurements (in μ) of oospores of *Sclerophthora macrospora* and *Peronosclerospora sorghi* in some hosts.

Host	*S. macrospora*	*P. sorghi*	Author and Year
Alopecurus spp	60-65		Saccardo 1890
Eleusine coracana	35-64		Thirumalachar et al.
Eragrostis major	56, 1-72, 7		Noble 1934
Triticum vulgare	52, 9-75, 6		Saccardo 1891
Triticum vulgare	45-64, 2		Peglion 1910
Triticum vulgare	52, 2-77, 4		Goidanich 1932
Zea mays	36-69, 4		Peglion 1910
Zea mays	42-75		Ullstrup 1952
Saccharum officinarum	41-45		Revilla 1955
Sorghum halepense	–	28, 4-39, 5	Kenneth 1966
Sorghum vulgare	–	31-36, 9	Weston and Uppal 1932
Sorghum bicolor	–	34-35, 5	Fernandez et al. 1976
Sorghum bicolor	–	24-40	Malaguti 1978
Eleusine indica	34-72		Malaguti 1978
Zea mays		24-36	Malaguti 1978
Averages	45, 42-68, 03	28, 28-37, 58	

can cause confusion between this disease and the "crazy top" (Fernandes and Nakamura 1977; Malaguti 1977; Malaguti et al. 1975). Stem pith discoloration and formation of brace roots symptoms can also be seen.

There are no extensive studies on disease-induced histological and physiological transformations.

9. Inoculation techniques are similar in most countries. Field tests are made by inoculating soil or seed with oospores, which are sometimes associated with the use of susceptible conidia donor plants. Local infections, where abundant conidia are formed, are frequently found in the old leaves of sorghums. No leaf shredding or oospore production is observed in these leaf spots, commonly associated with *Cercospora sorghi* invasion. It seems that besides inoculation date and leaf age, host hypersensitivity may be involved in the induction of localized rather than systemic infections.

   Systemic infection can follow oospore as well as conidia inoculation.

10. The need for resistant cultivars is pointed out by most SDM workers, whose first line of investigation is screening for resistant material and breeding for resistance. Resistant cultivars secure production in SDM infected areas, and reduce fungus propagation and dissemination.
11. The effect of planting date, grazing (in forage sorghums), land and culture management, fertilizing and population density are among the topics under study. Fungus perpetuation in plant debris and soil, inoculum dispersal and survival, pathogenesis and disease development, evaluation of all available genetic materials that react to SDM and other DMs, and seed dressing with fungicides are the main areas that will be explored.
12. Seed treatment in order to protect plants during the first 16 to 20 days after germination (the period when plants are susceptible) is the most promising control method.

    The effectiveness of certain fungicides — Metalaxyl (Ridomil, Ciba-Geigy) in particular and possibly Dodine — against SDM agents and their effects on germination, plant growth, and yield have to be investigated.
13. The possibility of disease reduction by soil amendment with chemicals or green manuring should be investigated bearing in mind the known effect of this treatment on *Phytophthora cinnamomi* in avocado.
14. The following are the main control measures suggested:
    a. Discard susceptible (especially forage) cultivars, and use only resistant ones within the infected areas.
    b. Choose an appropriate planting date, depending upon local climatic conditions.
    c. Rogue out diseased plants when young, specially in the seed production fields.
    d. Burn infected plant debris after harvesting.
    e. Do not use fields for seed production where diseased plants are observed.
    f. Practice crop rotation, avoiding maize or sorghums for some 3 to 5 years.
    g. Increase seed rate in order to assure a good plant population despite the disease.
    h. Seed dress with an effective systemic fungicide in order to protect seedlings and young plants.

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Sorghum Downy Mildew Research at Texas A & M University

Jeweus Craig*

The research program on sorghum downy mildew (SDM) at Texas A & M started in 1961 when the first reported observation of this disease in the United States occurred in Texas (Reyes et al. 1964). SDM, caused by *Peronosclerospora sorghi* (Weston and Uppal) Shaw (1978), has spread to 13 other states (Frederiksen et al. 1973b, 1977; Warren et al. 1974; White et al. 1978; Williams and Herron 1974). Texas, however, is the only state in which SDM is a major threat to crop production. Consequently, Texas continues to be the center of research on SDM in the United States.

Our SDM research program is a cooperative effort of federal and state personnel, conducting research in the following areas:

1. etiology of SDM;
2. testing for SDM resistance;
3. inheritance of reaction; and
4. chemical and cultural control of SDM.

Etiology of Sorghum Downy Mildew

Current research is concerned with factors affecting oospore germination, histopathology of hosts infected with *P. sorghi*, and variations in virulence of the pathogen.

Oospore Germination

Inability to induce consistent germination of the oospores of *P. sorghi* has hampered research on SDM. Recent studies of oospore germination by Pratt (1977) show that germination is induced by incubating oospores on nucleopore membranes in soil near the roots of sorghum seedlings. Oospore germination was by germ tube, and the percentage of germination was low (less than 1%). He found that seedlings of maize, wheat, oats, cotton, and soybeans, as well as sorghum, induced oospore germination. Pratt's results suggest that oospore inoculum in the field can be reduced by the planting of nonhost crops. These observations on the ability of nonhost crops to induce oospore germination support Vudhevonich's (1975) contention that nonhost crops reduce the incidence of SDM more than does fallowing.

I have observed occasional germination of oospores incubated on water agar. Most of the incubated spores either gave rise to hyphae of mycoparasites, or burst to release masses of biflagellate zoospores. I did not identify these zoospores, but assumed they belonged to a species parasitic on *P. sorghi*. The *Phylcrochytrium* sp, reported by Kenneth et al. (1975) as a parasite of oospores in Israel, was not found in these oospore collections. Heavy parasitization of oospores was characteristic of all the oospore collections I examined, and may account for the low germination percentages.

Histological Studies

Histological studies were used to determine differences in host-parasite relationships between SDM-resistant and SDM-susceptible cultivars inoculated with conidia of *P. sorghi*. Yeh (1977), in studies of inoculated sorghum lines, found no differences between resistant and susceptible cultivars in their reaction to stomatal penetration and initial infection by the pathogen. However, in resistant cultivars, the pathogen was unable to progress from the area of initial infection. In the susceptible host, *P. sorghi* moved rapidly through leaf and leaf-sheath tissue. Shabani (1978) conducted histological studies of resistant and susceptible maize inbreds. He found that the pathogen invaded resistant and susceptible maize cultivars with equal ease. As in sorghum, after the

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initial infection phase, the progress of *P. sorghi* was restricted in resistant lines and rapid in susceptible lines.

Histological studies were made of the rate at which the pathogen progressed from localized conidial inoculation sites in maize inbreds representing three types of SDM reaction. The reaction types were highly resistant, moderately resistant, and highly susceptible. The rates of pathogen growth through the leaf and leaf sheath in these inbreds were closely correlated to their SDM susceptibility. Progress was fastest in the highly susceptible, slowest in the highly resistant, and intermediate in the moderately resistant. Lateral progress of the pathogen from leaf sheath to leaf sheath was noted in the highly susceptible cultivars, but not in the other reaction types. The lateral movement was accomplished by mycelium which emerged from stomata on the adaxial surfaces of the pathogen-colonized leaf sheaths. The mycelial strands invaded the stomata of the adjacent leaf sheath. Safeeulla (1976) reported the development of similar mycelioid structures from the stomata of infected leaves incubated at high humidity.

Histological studies now under way are concerned with the development of the pathogen in resistant and susceptible cultivars inoculated with oospores of *P. sorghi*. Observations made to date indicate that resistance is expressed by inhibition of pathogen growth after penetration.

Biotype Studies

Surveys for virulent variants of *P. sorghi* in Texas are conducted by planting SDM-resistant sorghum cultivars at many sites. If an appreciable incidence of SDM is found in any of the test cultivars, inoculum is secured from the infected plants, increased, and compared to the standard population *P. sorghi* for virulence. In addition to these field nurseries, commercial plantings of SDM-resistant sorghum hybrids are monitored for increases in susceptibility.

Increased susceptibility of SDM was recently noted in some presumably resistant commercial hybrids. We found that the increased susceptibility was not caused by a new race of *P. sorghi*. In two cases, the reduction in resistance was caused by genetic modifications of the hybrids by breeders. In another, the cause has not yet been determined, but may have been climatic.

Testing for Resistance

Sorghum and maize cultivars are tested for resistance to SDM by exposure to natural infection in the field disease nurseries, and by artificial inoculation with conidia or oospores in the greenhouse.

Field Nurseries

In the past, most of our tests for resistance to SDM were conducted in field nurseries. The results of these tests were of great value (Craig et al. 1977; Frederiksen et al. 1973a, 1973b; Horne et al. 1978; Miller et al. 1968). Field testing identifies resistant genotypes, but a reliable method of greenhouse testing is preferable because of reduced costs, fewer seasonal limitations, and reduced probability of escapes among susceptible entries.

Conidial Inoculation

In recent years, we have made extensive use of conidial inoculation (Craig 1976) for greenhouse testing. The usefulness of greenhouse tests is determined by how well these tests foretell the reactions of the cultivars to natural infection in the field. We found that the reactions of sorghum cultivars to conidial inoculation at the one- to two-leaf stage were very similar to their reactions to natural infection under conditions that favour high disease incidence in susceptible cultivars in the field.

In maize, the relationship between reactions to conidial inoculation and reactions to natural infection is complex. Schmitt et al. (1977) reported positive correlations of reaction to field infection and reaction to conidial inoculation for the maize cultivars in their study. Others (Frederiksen et al. 1975; Kenneth and Shahor 1973; Reyes et al. 1964) noted that some maize cultivars were resistant to SDM in the field and very susceptible to conidial inoculation in the greenhouse. It is assumed that the observed differences were caused by differences between the reaction to oospores (field inoculum) and reaction to conidia.

Four maize inbreds, representing a range of

field-reaction types, were compared for reaction to conidial inoculation at different stages of growth. The degree of correlation between their field reactions and their reactions to conidial inoculation was dependent on the stage of growth at inoculation. In this group of inbreds, frequency of infection from conidial inoculation at the two-leaf stage of growth was similar to the frequency of field infection (Table 1).

Conidial inoculation is an efficient method of screening for resistance to SDM, but it has some detriments. It requires precise control of temperature and humidity during inoculation, and maintenance of populations of infected plants to provide inoculum. In addition, some forms of field resistance in maize may not be identified by conidial inoculation. For these reasons, an effective method of testing SDM resistance with oospores would be a valuable alternative.

Oospore Inoculation

Greenhouse tests of host resistance to oospores have been characterized by failure to achieve consistently high levels of disease in susceptible cultivars. I studied various aspects of oospore inoculation, and concluded that most of this variability was caused by differences in the infection potentials of different inoculum lots (Craig 1977).

Three lots of oospores were tested for infectivity at successively higher rates of soil infestation. The inoculum lots differed in the amount of inoculum per unit of soil required to produce 90% infection in a highly susceptible maize inbred. After the levels of soil infestation required for infection were determined, each inoculum lot was retested three times with 3- to 4-week intervals between tests. Each lot of oospores continued to induce high levels of infection in these tests.

Resistant and susceptible cultivars of sorghum and maize were tested to determine the relationship between their reactions to this method of oospore inoculation and their reactions to field infection. The results indicated a high degree of similarity between infection frequencies in the greenhouse and the field (Table 2).

Genetic Studies

Genetic studies to determine the mode of inheritance of reaction to *P. sorghi* in selected maize inbreds are under way. These studies will utilize crosses of resistant and susceptible inbreds and the F_2, F_3, and backcross progenies to determine the number of genetic factors conditioning reaction to SDM. Information acquired to date indicates intermediate dominance for resistance, with variations among pedigrees in the level of this dominance (Table 3).

Table 1. Reactions of maize inbreds inoculated with conidia of *Peronosclerospora sorghi* at different stages of growth.

Inbred	Field reaction[a]	Growth stage[b] 1 SDM[c] (%)	2 SDM (%)	3 SDM (%)
B-68	100	100	96	95
Oh-43	19	65	22	0
Va-26	24	65	22	0
Tx-601	0	20	0	0

a. Maximum percentage of SDM infection observed in four or more field tests.
b. 1 = One-leaf stage; 2 = Two-leaf stage; 3 = Three-leaf stage.
c. %SDM = percent of inoculated plants showing SDM symptoms.

Table 2. Reaction of sorghum and maize cultivars to field infection by *Peronosclerospora sorghi* and inoculation with oospores of *P. sorghi*.

Entry	Field reaction	Oospore infection[a] (%)
Maize		
Tx-601	Resistant	0
N-28	Susceptible	100
Sorghum		
Tx-2519	Resistant	7
Tx-412	Susceptible	91

a. Percent of inoculated plants showing downy mildew symptoms

Table 3. Incidence of downy mildew (SDM) in maize lines and F_1 crosses inoculated with oospores of *Peronosclerospora sorghi*.

Entry	Percent SDM
Tx-601	0
Tx-303	100
Tx-508	100
T-232	95
Tx-601 × Tx-303	40
Tx-601 × Tx-508	7
Tx-601 × T-232	10

Chemical and Cultural Control

Chemical Control

Current studies are concerned with the evaluation of Ridomil, a fungicide, for control of SDM in maize and sorghum. Exconde and Molina (1978) found this fungicide to be extremely effective in the control of Philippine downy mildew.

Cultural Control

Shabani (1978) conducted a study of the effects of depth of planting on SDM incidence in maize. He found that maize planted at 15 cm had significantly less disease than maize planted at shallower depths.

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Current Sorghum Downy Mildew Research in the All India Sorghum Project

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Sorghum is an important grain and fodder crop in India, and about 90% of the country's sorghum is grown in the states of Maharashtra, Karnataka, Andhra Pradesh, Tamil Nadu, Madhya Pradesh, Rajasthan, and Gujarat. Sorghum downy mildew (SDM), caused by *Peronosclerospora sorghi* (Weston and Uppal). Shaw, is one of the potentially destructive diseases of sorghum. It can reach epiphytotic conditions on susceptible entries — such as CSV-2, CSV-3, CSV-6, 296, 269 and others — under favorable environmental conditions. The disease is most destructive during the rainy season, and is normally rather mild or sporadic during the postrainy and summer seasons. However, a planting at the Dharwar farm was completely destroyed during postrainy season in 1978, thus indicating that it may also become destructive during this season.

SDM can cause 100% loss on a susceptible variety. In farmers' fields, SDM is generally sporadic in nature and the infection may range up to 5%, because the cultivars and hybrids grown — CSH-1, CSH-5, CSH-6, CS-3541, and 148/168 — possess high degrees of resistance. Local varieties such as GM-2-3-1, Nandyal, and others have shown infection up to 10%.

SDM has been reported to be common in Karnataka, Tamil Nadu, Maharashtra, Gujarat, Madhya Pradesh, Andhra Pradesh, and Uttar Pradesh (Ramakrishnan 1963). It is particularly destructive in rainfed areas of Karnataka, Tamil Nadu, Andhra Pradesh, and in southern and eastern parts of Maharashtra. The endemic areas are Satara, Sangli, and Ahmednagar districts of Maharashtra; Dharwar, Hubli, and Bangalore of Karnataka; and Salem, Dharmapuri, Coimbatore, and Tirunelveli in Tamil Nadu (Sundaram 1977).

Systematic studies on SDM were initiated with the inception of the All India Coordinated Sorghum Improvement Project (AICSIP) and are continuing, mainly concentrating at Dharwar and Coimbatore. Weather conditions at Dharwar and Coimbatore are highly favorable for the development of SDM. The Agricultural College research station, Dharwar, is situated at 15°26'N lat 75°7'E long, 678 m elevation. Average rainfall, temperature, and relative humidity during June to September are 86 cm, 23.9°C, and 86%, respectively. Average maximum and minimum temperatures are 27.6 and 20.2°C, respectively. During the period from June to September, rainy days are well distributed, thus providing favorable conditions for infection. Similar conditions prevail at Coimbatore. Hence, under these conditions SDM is endemic in nature and is seen at these stations every year in epiphytotic form on susceptible entries.

The current SDM research work in India pertains to:

a. standardization of screening procedures,
b. screening of the sorghum entries,
c. genetics of resistance,
d. chemical control, and
e. oospore-germination studies.

Standardization of Screening Procedures

The main objective is to find an effective screening technique to evaluate the reaction of different sorghum entries within a limited number of days. Although several methods have been used (such as oospore incorporation into the soil, seed treatment with oospores, alternate-row method, infector-row technique, seed treatment with oospores plus oospore incorporation in soil), the author has found the "infector-row" method to be successful in promoting 100% systemic infection on main shoots in a highly susceptible check (DMS-652) (Table 1, 2). With this method, finely powdered

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oospore material is incorporated into the seed furrows and the susceptible entry (DMS-652) is planted 2 to 3 weeks prior to the planting of test lines in 1-meter bands at the end of each line, 45 cm apart. The test lines are planted in seed furrows previously treated with oospore material. Two test lines are sandwiched between two rows of indicator DMS-652 lines. In every tenth plot, seeds of susceptible check (DMS-652) are planted in two rows, sandwiched between two test rows as indicator plots.

The reaction of the test entries is recorded by counting systemically infected plants, and calculating percent infection based on the total number of plants. Local lesions are not considered. A uniform rating scale to evaluate resistance to the disease should be adopted in order to obtain uniform results.

Screening of Sorghum Entries

The objectives of this program are: (a) to determine the level of resistance/susceptibility of entries being considered for release, and (b) to identify new sources of resistance useful in the breeding program.

Several entries of sorghum received from AICSIP have been screened at Dharwar and

Table 1. Percent systemic SDM infection on main shoots of four sorghum cultivars exposed to oospores and/or conidia by various inoculation methods.

Entry	Inoculation methods			
	Conidia supplied by infector rows (%)	Oospore incorporated in soil (%)	Seed treated with oospores (%)	No oospore; No infector rows (%)
DMS 652	96.06	67.01	23.08	2.39
302	93.33	55.71	17.56	2.89
296	88.55	57.78	18.83	1.63
555	94.33	57.33	17.91	2.63

LSD (0.05) for inoculation methods: 14.41

LSD (0.05) for entries: 2.34

Table 2. Percent systemic SDM infection in sorghum cultivars DMS 652 and CSV-2 exposed to oospore and/or conidial inoculum by various inoculation methods.

Inoculation method	Time of evaluation			
	DMS 652		CSV-2	
	30 DAP[a]	Dough stage	30 DAP	Dough stage
Infector row[b]	87.5	100.0	84.3	96.4
Alternate row[b]	29.3	84.3	29.1	77.1
Soil treatment[c]	23.4	72.3	19.2	68.2
Seed treatment[c]	2.2	27.7	2.6	21.3
Seed treatment + Soil treatment[c]	29.4	75.5	21.3	73.1
No oospore	1.3	4.3	0.3	6.3

a. Days after planting
b. Conidial inoculum and natural oospores in soil
c. Artificial infestation of soil with oospores

Coimbatore by "oospore incorporation into the soil prior to sowing," and the results of the entries are presented in Table 3, 4 and 5. Generally, the hybrids have shown less than 10% systemic infection. Hybrids SPH-24, SPH-107, MSH-40, CSH-6, CSH-5, and others have shown low levels of infection. Amongst the several varieties screened, SPV-104 was found completely SDM free in 3 years of testing; SPV-126, SPV-166, SPV-81, CS 3541, and others have shown low levels of infection. Amongst the several parental lines screened, 2219-B, 3660-B, CS-3541, and 2077-B have consistently shown less than 5% infection, whereas 3677-B, 1258-B, 3691-B, and 285 have shown less than 10% infection.

Since 1976, entries have been tested in the cooperative International Sorghum Downy Mildew Nursery (ISDMN) by the infector-row method. In 3 years of testing (Table 6), QL-3 was consistently immune, and IS-173, CSV-4, Uchv-2, Uchv-1, and IS-3164 had less than 10% infection.

Genetics of Resistance

Puttarudrappa et al. (1972), using IS-84 and

Table 3. Percent systemic SDM infection in 30 sorghum hybrids in up to 5 years of testing in the AICSIP program.

Entry	1974	1975	1976	1977	1978	Mean
SPH-24	–[a]	0	0	–	–	0
SPH-107	–	–	–	–	0	0
MSH-40	–	–	–	–	0	0
CSH-6	0.9	0	0	0	0	0.2
SPH-39	–	–	0	1.2	–	0.6
CSH-2	1.4	0	1.2	1.2	1.4	1.0
SPH-68	–	–	0	0.9	2.4	1.1
CSH-4	2.0	0	1.6	–	–	1.2
SPH-57	–	–	0	1.3	2.4	1.2
CSH-5	1.1	0	0	2.6	2.5	1.2
SPH-59	–	–	0	1.2	2.8	1.2
SPH-112	–	–	–	–	1.4	1.4
SPH-10	1.6	1.6	0	1.8	2.3	1.4
SPH-11	0.6	2.1	1.6	–	–	1.4
SPH-12	2.7	1.8	0	–	–	1.5
SPH-7	2.2	1.3	1.2	–	–	1.6
CSH-3	1.0	2.2	–	–	–	1.6
SPH-58	–	–	0	2.9	4.7	1.7
SPH-95	–	–	–	–	1.7	1.7
SPH-108	–	–	–	–	1.7	1.7
SPH-4	2.0	0	1.8	1.9	4.2	2.0
SPH-80	–	–	0	0.7	6.5	2.4
SPH-30	–	0	1.3	3.3	8.3	3.2
SPH-110	–	–	–	–	3.3	3.3
SPH-99	–	–	–	–	3.5	3.5
SPH-98	–	–	–	–	3.8	3.8
SPH-1	..	–	2.9	6.0	–	4.4
CSH-1	2.8	0	0	7.0	22.9	6.5
SPH-93	–	–	–	–	7.5	7.5
SPH-61	–	–	3.1	4.4	20.3	9.3

a. Entry not tested

IS-2941 as resistant parents and DMS-652 as the susceptible parent, studied the inheritance of resistance to SDM. Susceptibility was dominant to resistance and was controlled by two complementary factors. The presence of either or both factors in a recessive condition leads to the resistance. Rana et al. (1978) studied SDM resistance, using CS-3541 and 148 as resistant parents and 296 and 302/303 as susceptible parents, and found that SDM resistance is quantitatively inherited. Hybrids between susceptible and resistant parents show heterosis for resistance. Additional doses of resistant parents in backcrosses increase the level of

Table 4. Percent systemic SDM infection in 37 sorghum varieties in up to 5 years of testing in the AICSIP program.

Entry	1974	1975	1976	1977	1978	Mean
SPV-104	–[a]	–	0	0	0	0
SPV-238	–	–	–	–	0	0
SPV-166	–	–	0.4	0	0	0.1
SPV-126	–	–	1.1	0	0	0.4
SPV-81	–	–	0	0	1.2	0.4
SPV-33	–	–	0	1.2	0.4	0.5
CS 3541	0.8	0	1.3	1.0	1.1	0.8
SPV-129	–	–	0	1.2	1.9	1.0
SPV-130	–	–	0	–	2.5	1.2
DJ 6514	–	–	1.2	1.5	1.2	1.3
SPV-228	–	–	–	–	1.4	1.4
SPV-250	–	–	–	–	1.5	1.5
SPV-109	–	–	0	2.1	2.5	1.5
SPV-225	–	–	–	–	1.6	1.6
SPV-189	–	–	–	–	1.7	1.7
SPV-105	–	–	2.3	1.7	1.5	1.8
SPV-35	1.4	0	1.3	0.9	5.7	1.9
CSV-5	0.3	0	1.3	3.9	5.3	2.2
SPV-121	–	–	0	5.7	–	2.9
SPV-101	–	–	1.4	2.0	8.2	3.9
SPV-261	–	–	–	–	4.6	4.6
SPV-13	2.9	0	3.2	7.6	10.6	4.9
SPV-117	–	–	0	4.6	9.2	4.9
SPV-262	–	–	–	–	5.0	5.0
SPV-138	–	–	3.1	6.8	5.4	5.1
SPV-34	2.6	0	2.5	7.9	13.2	5.3
SPV-220	–	–	–	–	5.3	5.3
SPV-80	–	–	3.5	4.6	8.6	5.6
SPV-100	–	–	2.8	6.7	8.6	6.0
SPV-110	–	–	0	2.9	17.7	6.9
SPV-102	–	–	1.2	7.0	13.9	7.4
SPV-115	–	–	0	8.9	14.3	7.7
CSV-6	9.6	0	10.8	11.5	32.4	12.9
CSV-1	3.1	0	13.8	13.5	22.2	13.2
SPV-9	21.9	2.5	3.7	20.8	23.2	14.4
CSV-3	11.2	0	17.1	26.3	32.9	17.5
CSV-2	18.4	0.3	22.9	46.7	73.5	32.4

a. Entry not tested

Table 5. Percent systemic SDM infection in 20 sorghum lines in up to 5 years of testing in the AICSIP program.

Entry	1974	1975	1976	1977	1978	Mean
2077-BE	–[a]	–	–	0	–	0
CS-3541	0	1.2	0	1.6	1.1	0.8
2219-B	0	0.9	4.2	0	2.0	1.4
3660-B	0	1.0	2.3	1.2	3.5	1.6
3677-B	0	0	3.9	2.9	8.7	3.1
CK-60-B	0	0.7	3.6	0.8	10.9	3.2
1258-B	–	–	–	0	7.3	3.7
3691-B	0	3.1	2.0	6.2	7.2	3.7
148/168	0.3	1.1	0.9	2.7	16.2	4.2
2077-B	4.6	3.3	3.1	2.6	2.9	4.7
285	10.0	–	–	0.9	4.9	5.3
36-B	5.2	1.6	4.1	13.3	15.4	7.9
SPV-106	–	–	2.7	1.8	24.5	9.3
IS-84	6.8	7.2	5.2	9.7	20.6	9.5
1202-B	–	–	–	1.5	20.3	10.9
323-B	–	–	–	0	22.0	11.0
2947-B	0	4.3	4.0	16.2	37.9	12.5
Swarna	6.2	10.6	7.2	10.2	34.7	13.8
PD 3-1-11	–	–	–	23.0	73.5	48.2
296-B	20.0	26.2	48.9	54.5	79.8	48.8

a. Entry not tested

resistance proportionately. High degrees of determination of F_2 by F_1 (77%) and F_3 by F_2 (47%) indicate the highly heritable nature of SDM resistance (Rana et al. 1978).

Chemical Control

Attempts have been made and are continuing to find an effective chemical to control SDM. Balasubramanian (1976) at Dharwar obtained control of systemic infection up to 84% by four sprayings of Dithane M-45 (0.4%) at 7-day intervals, starting from the 7th day after planting. At Coimbatore during rainy season 1977, three chemicals were tested, Dithane M-45 (0.3%), Bavistin (0.1%), and Bafin (0.04%). The incidence of the disease was reduced up to 46.4, 52.6, and 53.6% with Dithane M-45, Bavistin, and Bafin, respectively (Bavistin has no effect on phycomycetes, so these are surprising results). During rainy season 1978, my experiments demonstrated that Ridomil is the most effective chemical; 100% control of systemic infection was obtained by seed treatment at 4g/kg and spraying with 2 g/l of water 40 days after planting (Table 7).

Oospore-Germination Studies

Oospores were detected on seed surfaces, on and in the glumes, and also in the rachis of the panicle. Microscopic examination of the rachis shows the presence of oospores distributed between the vascular bundles. Repeated efforts to trace the presence of oospores in the seed met with no success. Germination of oospores, both freshly collected and 12 months old, was made with potassium permanganate treatment. No definite germination was obtained and no germ tube was seen coming from oospore. However some oil-like globules or bodies were seen around the oospores; the importance of these needs to be investigated. Oil-like globules have also been seen associated with *S. graminicola* oospores (Bhat 1973).

Future Work

Areas of study to be pursued include:

1. Studies on physiologic specialization in *Sclerospora sorghi.*
2. Exploring further the use of conidial inoculum in the screening program.
3. Determining the susceptibility period of sorghum during its growth period, both for systemic and local lesions.
4. Identification of more stable resistant genotypes with good agronomic characters.
5. Genetics of resistance.

Table 6. Percent systemic SDM infection on the ISDMN entries in 3 years of testing at Dharwar, India.

Entry	1976	1977	1978	Mean
QL-3	0	0	0	0
IS-173	0	2.1	7.9	3.3
CSV-4	0	4.2	6.2	3.4
Uchv-2	0	6.5	4.0	3.5
Uchv-1	2.8	1.7	8.4	4.3
IS-3164	1.7	8.2	–	4.9
IS-2042	0	2.3	15.4	5.9
SC-120-14	0	4.9	17.5	7.5
SC-599-6-3	7.6	–[a]	–	7.6
IS-7254	–	–	7.9	7.9
SC-120-6-8-8	0.9	15.4	–	8.1
SC-239-14	0	16.8	–	8.8
IS-3799	0	9.5	17.6	9.0
SC-170-6-17	5.0	13.7	–	9.4
CSV-5	1.3	4.9	22.4	9.5
IS-6380	10.0	–	–	10.0
IS-5273	0	6.5	26.3	10.9
SPV-35	–	–	11.5	11.5
CK-60-B	–	–	11.5	11.5
SC-175-14	7.2	17.8	–	12.5
TAM-428	1.6	24.0	–	12.8
SC-173-12	0	28.3	–	14.2
IS-2918	0	29.2	–	14.6
E-35-1	–	–	15.2	15.2
SC-110-14	1.0	9.3	42.0	17.4
3660-B	–	–	19.8	19.8
IS-2223	–	–	19.9	19.9
NSA-440-12	0	44.1	–	22.1
2077-B	–	–	22.9	22.9
SC-414-12	2.2	17.1	70.6	29.9
1258-B	–	–	30.1	30.1
1202-B	–	–	32.0	32.0
GPR-269	38.1	–	–	38.1
IS-2327	–	–	39.5	39.5
IS-2550	–	–	53.8	53.8
CSV-2	23.6	66.5	91.3	60.5
DMS-652	21.5	84.4	100.0	68.6

a. Entry not tested

Table 7. Chemical control: percent systemic infection on main shoot.

Treatment	Replications		
	RI	RII	RIII
Ridomil (0.4%) (Seed treatment)	1.1	3.4	1.2
K.W.G. 0519 (0.2%) (Seed treatment)	35.7	40.2	37.5
KT 19827 (0.5%) (Seed treatment + three sprays)	19.7	32.4	26.7
Dithane M-45 (0.4%) (Four sprays)	31.6	17.7	18.0
Ridomil seed treatment + one spray	0	0	0
Nontreated (control)	80.4	89.5	82.2

L.S.D. (0.05) = 0.687

6. Evolving an economic schedule of chemical control.
7. Obtaining a reliable *in vitro* oospore-germination technique.

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Factors Affecting Sorghum Downy Mildew Development

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Sorghum downy mildew (SDM), caused by *Peronosclerospora sorghi*, is endemic in the Indian states of Tamil Nadu, Karnataka, and Maharashtra. In this paper, data are presented on the influence of fertilizers, trace elements, and seeding dates on SDM incidence in experiments conducted at Dharwar in Karnataka.

Effect of Nitrogen and Phosphorus Fertilizers

Nitrogen, in the form of ammonium sulfate, was applied at 0, 44.8, 89.7, and 134.5 kg N/ha and phosphorus, in the form of superphosphate, was applied at the rate of 0, 33.6, 67.3, and 100.9 kg P/ha. The experiment was laid out in a split-plot design, with nitrogen treatments as main plot and the phosphorus treatments as subplots. Plot size was 6.8 by 5.8 m. Spacing between subplots was 1.5 m. Spacing between rows and plants was 45 and 15 cm, respectively. The soil was artificially infested with *P. sorghi* oospores.

Increase of phosphorus fertilization from 0 to 67.3 kg/ha significantly increased SDM incidence in susceptible variety DMS-652 (Table 1). In the resistant hybrid CSH-1, increased susceptibility was observed only when phosphorus application was increased from 0 to 33.6 kg P/ha (Table 2). Additional phosphorus applications did not influence the disease significantly. Nitrogen did not significantly affect the incidence of the disease in DMS-652 or CSH-1. The initial status of nitrogen and phosphorus in the soil was not estimated. The pH of the soil was 5.8.

Table 1. Effect of nitrogen and phosphorus on the incidence of downy mildew in sorghum cultivar DMS-652 in trials conducted at Dharwar, India.

Nitrogen level (kg N/ha)	Phosphorus level (kg/P/ha)			
	0	33.6	67.3	100.9
	% incidence			
0	25.5	41.3	51.6	54.5
44.8	29.9	62.1	57.6	50.2
89.7	27.8	45.6	55.1	60.0
134.5	28.7	45.8	64.3	54.3

Nitrogen standard error = 2.25 LSD (P = 0.01) 8.32
Phosphorus standard error = 2.98 LSD (P = 0.05) 6.14

Effect of Zinc and Manganese

Zinc in the form of zinc sulfate ($ZnSO_4.7H_2O$) and manganese in the form of manganese sulfate ($MnSO_4.H_2O$) were applied to the seed furrows with ammonium sulfate and superphosphate. The rate of zinc and manganese sulfates applied were 0, 5, 10, 15, 20, 25 kg/ha. The rates of ammonium sulfate and superphosphate applied were 44.8 N_2 and 47.3 P_2O_5 kg/ha. The plot size and spacing were as in the previous experiment. Zinc and manganese were in main plots and their concentrations were varied in subplots. Susceptible cultivar DMS-652 was used as the test variety. Zinc and manganese application did not influence SDM incidence significantly (Table 3).

Effect of Planting Date

In an experiment with several planting dates of DMS-652 and CSH-1, the SDM incidence was the lowest in the earliest planting (last week in June) and was highest when planted during the last week in July (Table 4).

Estimation of available soil moisture by gravimetric method showed a high soil moisture (76 to 79%) during the first 16 days following the June planting, and 44 to 47% available soil moisture during the same period in July.

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Table 2. Effect of nitrogen and phosphorus on the incidence of downy mildew in sorghum cultivar CSH-1, Dharwar, India.

Nitrogen application (kg/ha)	Phosphorus application (kg/ha)			
	0	33.6	67.3	100.9
	(% incidence)			
0	5.1	4.3	6.3	7.3
44.8	7.1	14.0	14.5	12.2
89.7	4.6	12.4	15.9	14.1
134.5	7.8	15.1	15.0	10.9

Nitrogen standard error = 2.03
Phosphorus standard error = 2.35
LSD (P = 0.05) = 4.84

Table 3. Effect of zinc and manganese on incidence of sorghum downy mildew.

Trace element	Application of trace element (kg/ha)[a]					
	0	5	10	15	20	25
	(% incidence)					
Zinc	57.7	71.2	70.2	58.2	69.0	67.8
Manganese	73.8	59.2	64.6	59.6	64.8	56.5

a. Trace element standard error = 3.47;
Concentration standard error = 4.42
Differences among means were not significant.

Table 4. Effect of planting date on the incidence of sorghum downy mildew in experiments at Dharwar, India.

Cultivar	Date of planting[a]				
	1	2	3	4	5
	(% incidence)				
DMS 652	9.4[b]	29.9	61.9	74.5	51.7
CSH 1	0.5	1.1	4.2	12.3	10.5

a. 1 = Jun (last week); 2 = July (first week); 3 = July (second week); 4 = July (last week); 5 = Aug (first week)
b. Standard error for cultivars = 3.04; Standard error for dates = 4.61
LSD (P = 0.05) = 9.67 LSD (P = 0.05) = 9.50
LSD (P = 0.01) = 17.75 LSD (P = 0.01) = 12.87

It is evident that higher phosphorus and lower moisture levels can encourage incidence of SDM, but applications of trace elements like zinc and manganese do not influence the incidence of downy mildew.

The ICRISAT Sorghum Downy Mildew Program

S. R. S. Dange and R. J. Williams*

Sorghum downy mildew (SDM), caused by *Peronosclerospora sorghi* (Weston and Uppal) Shaw, has great destructive potential and appears to be increasing in importance in many sorghum-growing regions in Asia, Africa, and the Americas, on maize as well as on sorghum (Frederiksen et al. 1973; Frederiksen and Renfro 1977).

In the ICRISAT sorghum-improvement program, we have recognized from the outset the need to identify and utilize SDM resistance, but we have not been able to mount a major research effort on this disease until recently, because our limited staff have mainly concentrated their efforts on the widespread major problem of sorghum grain molds. In late 1977, we were fortunate to have Naron Singburaudom from Kasetsart University, Thailand, spend 3 months at ICRISAT Center examining factors affecting conidial production and successful field inoculations (Singburaudom and Williams 1977). An intensified program on SDM was initiated at ICRISAT in June 1978.

In this paper, we attempt to summarize the SDM research conducted at and from ICRISAT Center, to project our plans for the next few years, and to pose some questions which we feel should be considered by pathologists participating in this Workshop.

Symptoms

On sorghum we have observed systemic and local lesion infections by *P. sorghi*.

The first indication of a systemic infection is the emergence of partially infected leaves. Subsequently fully infected leaves emerge. The first two to four infected leaves produce conidia. Subsequent leaves produce oospores, become necrotic, and shred as the plants mature.

During cool humid weather, local rectangular chlorotic lesions develop; these produce conidia but apparently do not produce oospores. The local lesions can be densely grouped and may coalesce to give the leaf a blighted appearance, mostly toward the tips of the lower leaf blades. Local lesions generally become necrotic and can support the growth of other fungi.

Host Range

At ICRISAT Center, we have inoculated several graminaceous species with conidial suspensions of *P. sorghi*, and have obtained systemic infection in maize, teosinte, *Sorghum caffrorum, S. sudanense, S. halepense, S. verticilliflorum,* and *S. almum*, in addition to grain sorghum.

Although several maize and sweet corn cultivars readily become infected with *P. sorghi* at ICRISAT Center, the symptoms observed are generally quite different to those obtained on maize at Mysore and Udaipur in India, and at Farm Suwan in Thailand. For example, maize cultivar VL-54 first produces one or two relatively normal-appearing partially infected leaves and then remains stunted, with the unfolding leaves rolling and wilting in the whorl, almost as if damaged by stem borer. Nodes of the infected plants are discolored. An occasional plant in VL-54 will develop more normal symptoms and some cultivars (e.g., CM 500) develop predominantly normal symptoms, except that conidial production is scarce and no deformity of tassel or ear is observed. We have observed a few oospores in infected sweet corn leaves, but they are sparse.

The host range of *P. sorghi* appears to be highly variable, depending upon location. In Rajasthan, India, *P. sorghi* infects maize and *Heteropogon contortus* and does not infect sorghum (Dange et al. 1974). In Thailand, *P. sorghi* readily infects maize, rarely infects sorghum, but does not infect *H. contortus* (Renfro

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personal communication). A thorough investigation of the host range of *P. sorghi* is needed, and we have proposed an international cooperative project on this subject; the project will include researchers in Asia, Africa, and the Americas.

Identification of SDM Resistance

At ICRISAT Center

Conidial Production

The first requirement of a resistance-identification program is a reliable and meaningful screening technique. For SDM, reliance on oospores in a "sick plot" has many drawbacks and we have examined several methods of inoculating with conidia. To do this, we needed to produce large quantities of conidia and have them available at a more convenient time than that during which they naturally mature (0200 to 0400 hr). We found that leaves that have received 6 to 8 hr daylight (they need more time in overcast cloudy weather than in sunny weather) and are then maintained in dark moist chambers at 20°C, produce abundant conidia that are mature after 7 to 8 hr of incubation. The normal practice in our program now is to harvest infected leaves between noon and 1300 hr and to inoculate between 2000 and 2100 hrs. We can, however, produce conidia whenever needed by manipulating light and temperature in the incubators; for example, leaves collected at 1700 hr are placed in the moist chambers at 20°C in an incubator programed to remain at 20°C for 7 hr and then to cool to 5°C, which allows conidia to be harvested at any time on the subsequent morning. Leaves harvested at 1700 hr and maintained under fluorescent lights overnight can be moist-chambered at 20°C at 0900 hr the following morning and the conidia can be harvested for inoculation after 1700 hr.

Inoculation Method and Age of Plants

We have examined several ways of exposing sorghum seedlings of various ages to *P. sorghi* conidia, including injection deep into the whorl, direct spray, exposure to older conidia-producing infected plants, and injection of conidia into the collar region.

Germinating cultivar DMS-652 seedlings (20 and 36 hr after placement of seed in moist chambers at 30°C) were sprayed with conidial suspension and were planted in pots after 3, 6, and 9 hr of postinoculation incubation. The amount of systemic infection was less with increased preinoculation and postinoculation incubation (Table 1).

In a staggered-planting pot experiment, plants of cultivar DMS-652 sorghum of six ages were inoculated by injection of a conidial suspension into the whorls. The greatest infection developed in the plants youngest at the time of inoculation (Table 2). These data add to the existing evidence that the younger the plant when exposed to conidia, the greater the prob-

Table 1. Percent systemically SDM-infected DMS-652 sorghum plants from germinating seeds sprayed with conidia of *Peronosclerospora sorghi* after incubation at 30°C followed by postinoculation incubation.

Postinoculation incubation (hr)	Preinoculation incubation 20 hr	Preinoculation incubation 36 hr
3	65	35
6	49	33
9	32	24

Table 2. Percent plants systemically infected with SDM, following inoculation with conidial suspensions at six plant ages.[a]

Plant age at inoculation (days)	Systemic infection 28 days after inoculation (%)
5	37
10	24
15	13
20	10
25	11
30	11

a. Plantings were made sequentially so that the inoculation of plants of all ages was conducted at the same time.

ability of systemic infection. In subsequent studies, we produced high levels of infection by deep injection of conidial suspensions into the collar regions of 15-to 25-day-old plants.

In order to test the possibility of using infector rows for large-scale field screening, we planted seed of CSV-2, DMS-652, and IS-2550 sorghum, and CM-500 and sweet corn maize in sterilized soil in trays and placed the trays between rows of systemically infected conidia-producing sorghum plants.

High levels of infection were obtained in all test materials (Table 3). These data suggest that the infector-row system can be effectively used for SDM screening. However, the relatively short conidial-production phase of infected plants and the relatively short susceptible stage of seedlings, make critical the correct time of planting test entries, and the provision of the right environmental conditions for conidial production and infection.

Inoculation of the ISDMN Entries

In late 1977, the 25 entries in the 1977 International Sorghum Downy Mildew Nursery were inoculated by injecting of conidial suspensions deep into the whorls of seedlings 5 days after emergence. Very high levels of infection were obtained on several cultivars which consistently are much more resistant when exposed to natural inoculum pressure at SDM "hot-spots," such as Dharwar and Mysore (Singburaudom and Williams 1977; ICRISAT 1978). Cultivars varied greatly in the degree of difference between SDM-infection levels from the artificial and natural inoculations, indicating the possibility of different types of resistance.

Table 3. Percent systemic SDM infection obtained with infector rows in sorghum and maize 33 days after planting.

Crop	Cultivar	Total plants	Infected plants	% systemic infection
Sorghum	DMS-652	160	134	84
	IS-2550	138	97	70
	CSV-2	72	48	67
Maize	CM-500	86	76	88
	Sweet corn	75	61	81

These results highlight the importance of finding realistic inoculum concentrations and inoculation methods in order to avoid failure to recognize useful field resistance.

Evaluation of Maize for Infector Rows

There are two major drawbacks to using sorghum as the infector-row conidial source for SDM field screening. The first is shoot fly, which can be devastating on seedlings emerging later than the surrounding crop; the second is the relatively short time period during which systemically infected sorghum plants produce conidia before switching over to oospore production. The use of maize as the conidial producer would avoid these drawbacks. However, as indicated above, we have not been able to obtain in a good level of conidial production of SDM-infected maize at ICRISAT Center.

Plans for Field Screening

At present we plan to screen for SDM resistance in the field during the rainy season and the immediate postrainy cool season of each year. We will use sorghum cultivar DMS-652 as the conidial donor and will infect the infector row plants at the seedling stage by syringe inoculation. Mist irrigation will be used in the evening hours to promote conidial production, and the test entries will be planted when the infector row plants show a high level of conidial production. We need to determine if the natural conidial inoculation will need augmentation by direct spraying of conidia onto test-entry seedlings.

Multilocational Cooperative Screening

The International Sorghum Downy Mildew Nursery, initiated in 1976, has been in operation at several locations each year, through the cooperation of many scientists. The results are reviewed in a separate paper in this session.

Discussion and Questions

The most important question to be answered is what is the appropriate inoculation procedure? Should oospores or conidia, or both, be used? How should the test plants be inoculated with conidia — by syringe injection, by spray, or by

exposure to older infected plants? The logical answer is that the method adopted should be epidemiologically meaningful for the particular location. If there is an effective natural conidial source at the time sorghum is planted, then the inoculation procedure should involve conidial showers; conversely, if oospores are the main infective propagules at a particular location, there is a strong argument for using oospore-inoculation methods. To properly answer the question, we need to know the relationship between reaction to oospore and reactions to conidia at various inoculum concentrations, using different methods of inoculation.

A second, related, question concerns the relationship between reactions to oospores and reactions to conidia. Can reactions to one be accurately used to predict reactions to the other? Another related question is the relationship between local lesion reaction and systemic reaction. If a cultivar develops severe local-lesion infection when unfolded leaves are exposed to conidial showers, is this an indication that the cultivar is also highly susceptible to systemic infection?

The variability in *P. sorghi* from region to region needs further investigation. There is a great deal of evidence for pathotypes varying in reaction to host species, but there is no definite evidence for the existence of races within the sorghum-infecting pathotype. However, if the pathogen species is capable of variability at the host interspecific level, it will probably be capable of producing biotypes in response to selection pressures of variability within a single host species. We believe that the ISDMN program and the proposed international project on host range will provide answers to these questions.

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The International Sorghum Downy Mildew Nursery

R. J. Williams, K. N. Rao, and S. R. S. Dange*

The International Sorghum Downy Mildew Nursery (ISDMN) program was initiated in 1976 with the following objectives:

1. To identify sources of stable SDM resistance;
2. To obtain information on the variability of the SDM pathogen;
3. To distribute SDM resistant genotypes to scientists in national programs;
4. To promote the development of a communicating cooperating international network of scientists interested in SDM.

In its first year of operation there were three cooperators, all within India. Cooperators in Africa and the Americas were added in 1977and 1978, and the number of cooperators in India was expanded (Table 1).

The basic requirement of cooperators is that they should be able to expose the ISDMN test entries to local SDM pathogen populations with sufficient pressure to provide meaningful data on the SDM reactions of the test entries.

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Selection of ISDMN Test Entries

Initially, entries reported as SDM resistant in various national and regional programs were included. As the world sorghum collection is screened for SDM reactions at ICRISAT Center, entries found to be SDM resistant will be fed into the ISDMN. Entries from any SDM worker are welcomed, provided that the cultivar has clearly been SDM resistant at its home location. In 1978 the best entries from other international disease nurseries were also included in the ISDMN. Each year two known high-susceptible lines are included as "indicators."

Operation of the ISDMN Program

Seed of test entries is assembled and multiplied at ICRISAT Center. All cooperators receive seed from the same seed lot for each entry. This is important, if erroneous information on pathogen variability is to be avoided.

A set of entries is sent to each cooperator with a book that includes information on the objectives of the trial, suggestions on planting, fer-

Table 1. Cooperators and locations in the International Sorghum Downy Mildew Nursery Program in 1976, 1977, and 1978.

Cooperator	Location	Year(s)
K. H. Anahosur	Dharwar, India	1976, 1977, 1978
Kausalya Gangadharan	Coimbatore, India	1976, 1977, 1978
K. M. Safeeulla	Mysore, India	1976, 1977, 1978
B. B. More	Digraj, India	1977, 1978
K. N. Rao & S. R. S. Dange	ICRISAT, India	1977, 1978
K. A. Balasubramanian	Hyderabad, India	1978
Max Boling	Gaborone, Botswana	1977, 1978
Gino Malaguti	Maracay, Venezuela	1978
R. A. Frederiksen	Texas, USA	1978

tilization, inoculum provision, time and method of scoring, and duplicate sets of data-record sheets for climatic and plant-reaction information.

Cooperators are requested to return one copy of the data sheets to ICRISAT Center as soon as possible after completion of the trial. The data will be analyzed and published in a report that contains data from the cooperators, along with discussion of the important aspects of the results. It is distributed to all the cooperators.

The 1976, 1977, and 1978 Results

1976

Two of the 32 entries, QL-3 and Sc 120-14, were free from systemic infection at all three locations. Eleven entries had a mean incidence of less than 5% with a maximum incidence of less than 10% (Tables 2, 3).

1977

Data on the 25 entries, received from cooperators at five Indian locations and one African location, are summarized in Tables 4, 5, 6. Cultivar QL-3 was again immune at all locations and four entries had less than 10% incidence at all locations except ICRISAT Center.

The ICRISAT Center results are interesting and require some discussion. The high level of infection, compared with the other locations, is probably the result of the injection of a conidial suspension deep into the whorls of young plants 5 days after emergence. At the other locations, test entries were exposed to oospores in the soil (in sick plots) and in some cases to conidia provided by infector rows and adjacent infected crops. Under the severe artificial inoculation at ICRISAT Center some of the entries, which showed a high degree of resistance under "natural" inoculation developed

Table 2. Percent incidence of SDM in the 13 most resistant lines at three locations in the 1976 ISDMN, compared with percent incidence in the local susceptible lines and the location means for all test entries.

Entry	Location			Entry Mean
	Dharwar	Coimbatore	Mysore	
	(%)			
QL-3	0.00	0.00	0.00	0.00
SC 120-14	0.00	0.00	0.00	0.00
CSV-4	0.00	0.00	0.83	0.28
SC 173-12	0.00	2.55	1.87	1.47
IS 3799	0.00	1.10	3.45	1.52
IS 5273	0.00	2.55	2.35	1.93
UchV-2	0.00	0.00	5.69	1.89
IS 2042	0.00	3.80	1.90	1.90
SC 239-14	0.00	3.10	3.29	2.13
CSV-5	1.25	3.00	2.30	2.18
UchV-1	2.75	0.00	4.28	2.34
IS 173	0.00	3.80	5.02	2.94
TAM-428	1.55	3.45	7.55	4.18
Local susceptible	–	12.50	80.00	46.25
Location mean[a]	3.65	6.15	11.86	7.22

a. Derived from all test entries.

high levels of infection (exceptions to this were UchV-1 and CSV-4). However when relative performance of the entries is examined by comparing rank values (Table 5), it can be seen, that of the best 10 entries at locations where there was natural inoculation, nine were also in the best ten at the location where the severe infection inoculation occurred. It seems from these results that there are two types of resistance in the ISDMN test entries: one that breaks down with increased inoculum concentration (e.g., IS-173 and Uch V-2), and one that remains relatively stable when exposed to different inoculum concentrations (e.g., Uch V-1 and CSV-4). Cultivar QL-3 remained immune, even following syringe inoculation.

Table 3. Mean and maximum[a] percent incidence for 32 sorghum entries tested at three locations in the 1978 International Sorghum Downy Mildew Nursery.

Entry	Mean incidence (%)	Maximum incidence (%)
QL-3	0.00	0.00
SC 120-14	0.00	0.00
CSV-4	0.28	1.65
SC 173-12	1.47	2.16
IS 3799	1.52	5.81
IS 5273	1.63	3.33
UchV-2	1.90	9.25
IS 2042	1.90	3.35
SC 239-14	2.13	6.58
CSV-5	2.18	4.60
UchV-1	2.34	7.46
IS 173	2.94	8.63
SC 110-14	3.84	10.48
TAM-428	4.18	9.28
SC 108-14	4.28	11.60
SC 414-12	5.60	16.00
IS 3164	6.06	10.12
SC 120-6-8-8	6.67	13.13
SC 170-6-17	7.21	14.67
SC 175-14	7.92	16.25
NSA 440-12	0.32	17.70
TAM 2566	8.45	14.94
IS 2918	9.37	42.79
IS 2858	9.82	24.56
IS 3168	9.97	29.76
SC 599-6-3	10.14	15.61
IS 159	11.18	34.67
SC 423-14	11.92	22.20
IS 6380	15.07	25.96
CSV-2	29.75	57.00
DMS 652	31.32	50.00
GPR 269	33.66	69.77

a. Based on individual replication values.

1978

At the time of preparation of this paper, results

Table 4. Downy mildew incidence in 28 sorghum entries in the 1977 International Sorghum Downy Mildew Nursery at Gaborone, Botswana.

Entry	Incidence %	
	Rep. 1	Rep. 2
QL-3	0	0
UchV-1	0	0
SC-120-14	0	0
CSV-4	0	0
IS-5273	0	0
IS-173	0	6.9
IS-2042[a]	25	0
UchV-2	0	0
IS-3799	0	0
CSV-5	0	C
SC-414-12	0	0
SC-110-14	0	0
SC-239-14	0	0
SC-170-6-17	0	0
SC-120-6-88	0	0
TAM-2566	0	0
IS-3164[a]	4	52.3
SC-108-14	0	0
SC-173-12	0	0
TAM-428	0	0
SC-175-14	0	0
IS-2918[a]	34.6	0
NSA-440-12	31.3	29.2
CSV-2	86.7	100.0
DMS-652	81.3	90.0

a. The major discrepancies between replicates of these two entries indicates a possible error in recording or in the original seed packeting. As IS-2042 is included in the 1978 ISDMN, its reaction at Gaborone can be reexamined.

Table 5. Downy mildew incidence in 25 sorghum entries at five locations in the 1977 International Sorghum Downy Mildew Nursery.

Entry	ICRISAT Center		Dharwar		Mysore		Digraj	Coimbatore		Entry Mean[a]	
	Rep. 1	Rep. 2	Rep. 1	Rep. 2	Rep. 1	Rep. 2	Rep. 1	Rep. 1	Rep. 2	Incl. ICRISAT	Excl. ICRISAT
	(%)										
QL-3	0	0	0	0	0	0	0	0	0	0	0
UchV-1	0	12.5	0	3.4	3.2	4.8	1.9	2.9	3.2	3.6	2.9
SC-120-14	33.3	50.0	0	9.8	5.8	5.3	0	0	0	11.6	3.0
CSV-4	15.8	5.3	4.0	4.3	7.6	6.2	0	2.9	4.2	5.5	4.2
IS 5273	16.0	10.5	8.6	4.4	8.1	7.0	0	2.7	0	6.4	4.4
IS 173	22.2	25.0	0	4.2	8.8	10.1	0	3.3	6.9	8.9	4.8
IS 2042	20.0	21.1	0	4.5	11.9	6.8	0	6.7	3.6	8.3	4.8
UchV-2	36.8	26.3	10.8	2.1	9.5	4.7	0	6.9	2.9	11.1	5.3
IS 3799	28.6	63.2	11.1	7.9	9.9	9.8	0	3.3	2.9	15.2	6.4
CSV-5	26.3	33.3	12.0	11.4	4.3	3.6	2.3	5.9	6.3	11.7	6.5
SC-414-12	80.0	66.7	16.1	18.0	8.3	3.0	4.5	10.7	2.8	23.4	9.1
SC-110-14	89.5	53.8	12.0	14.0	5.7	10.7	7.9	11.1	6.7	23.5	9.7
SC-239-14	31.6	50.0	20.0	23.5	7.0	3.0	10.5	3.8	3.7	17.0	10.2
SC-170-6-17	81.2	82.4	21.4	18.4	4.4	6.2	3.8	8.0	10.7	26.3	10.4
SC-120-6-88	85.7	50.0	9.1	29.2	2.6	1.8	10.2	14.3	10.0	23.7	11.0
TAM 2566	61.9	52.9	15.6	6.1	30.4	9.4	0	9.4	15.2	22.3	12.3
IS 3164	50.0	57.9	6.1	12.2	13.9	16.1	14.0	6.5	17.9	21.7	12.4
SC-108-14	57.9	36.8	2.9	15.0	2.8	20.2	33.3	10.7	6.9	20.7	13.1
SC-173-12	86.7	75.0	35.5	24.2	17.2	7.9	5.1	0	3.6	28.3	13.4
TAM-428	50.0	43.8	22.9	25.0	35.4	11.5	10.5	8.8	4.3	23.6	16.9
SC-175-14	40.0	93.3	25.9	31.1	44.3	16.3	4.9	12.9	9.4	30.9	20.7
IS 2918	61.1	38.9	36.0	26.5	36.9	21.5	9.5	16.7	12.9	28.9	22.9
NSA 440-12	80.0	84.6	41.0	66.7	72.4	36.4	38.5	9.1	13.2	49.3	40.5
CSV-2	94.7	77.8	74.1	90.3	100	100	33.3	20.0	19.4	67.7	62.5
DMS 652	81.8	100	96.3	88.6	100	100	62.5	33.3	42.9	78.4	74.8
Location Mean	48.8		20.4		19.5		10.1	8.5			

a. As the inoculation method used at ICRISAT Center was very severe, entry means are given including (Incl. ICRISAT) and excluding (Excl. ICRISAT) ICRISAT Center data.

Table 6. **Comparison of entry ranking in the 1977 ISDMN based on incidence and infection index values at ICRISAT Center and the average rank from the four other locations.**[a]

Entry	Incidence Rank		Infection Index Rank	
	ICRISAT	Others	ICRISAT	Others
QL-3	1	1	1	1
UchV-1	2	2	2	2
CSV-4	3	4	3	4
IS-5273	4	5	4	6
IS-2042	5	7	5	5
SC-120-14	10	3	10	3
IS-173	6	6	6	7
UchV-2	8	8	8	8
CSV-5	7	10	7	9
IS-3799	18	9	11	10
SC-239-14	9	13	9	13
SC-120-6-08	16	15	18	15
SC-110-14	17	12	19	11
SC-414-12	19	11	20	12
IS-3164	12	17	15	16
SC-108-14	11	18	12	17
TAM-2566	20	16	16	18
SC-170-6-17	23	14	21	14
TAM-428	13	20	13	20
SC-173-12	21	19	23	19
IS-2918	14	22	14	22
SC-175-14	15	21	17	21
NSA-440-12	24	23	22	23
CSV-2	22	24	24	24
DMS-652	25	25	25	25
Correlation Coefficient	0.78		0.83	

a. Dharwar, Mysore, Digraj, Coimbatore.

have been received from the Indian locations, Mysore, Dharwar, Coimbatore, and Digraj, as well as from ICRISAT Center (Table 7). For the third successive year, QL-3 is immune to SDM at all Indian locations. Three other entries — Uch V-2, CSV-4, and IS-173 — have a mean incidence of less than 3% and a maximum incidence of less than 10% across replications and locations.

Dharwar provided the greatest SDM pressure (location mean of 28.9%) followed by Mysore (12.2%) and ICRISAT Center (8.1%). SDM incidence in most of the entries was low at Coimbatore and Digraj.

IS-7254, the best line in 2 years of the International Sorghum Leaf Disease Nursery, was resistant (<10% SDM) in nine of the ten replications, but, strangely, had 16% SDM in one replication at Digraj. The two elite grain mold-resistant lines, E 35-1 and IS 2327, had relatively low SDM incidence at all locations except Dharwar, where they averaged 19 and 33%, respectively.

Of the -B lines in the trial, CK 60-B was the most resistant, with mean and maximum SDM-incidence values of 4 and 14.9%, respectively.

Recent personal communication with our cooperators in the USA and Venezuela indicate

Table 7. Downy mildew incidence in 24 sorghum entries at five locations in the 1978 International Sorghum Downy Mildew Nursery.

Entry	Digraj		Coimbatore		ICRISAT		Mysore		Dharwar		Entry Mean
	Rep. 1	Rep. 2	Rep. 1	Rep. 2	Rep. 1	Rep. 2	Rep. 1	Rep. 2	Rep. 1	Rep. 2	
	(%)										
QL-3	0	0	0	0	0	0	0	0	0	0	0
UchV-2	0	0	0	0	2.9	0	9.2	2.4	3.6	4.0	2.2
CSV-4	0	0	0	0	4.1	4.0	1.5	2.7	3.4	9.0	2.5
IS 173	0	0	0	0	0	0	7.7	3.0	6.6	9.2	2.7
UchV-1	0	0	0	0	1.6	0	4.6	6.0	8.9	16.0	3.7
CK 60-B	0	0	0	0	5.0	0	4.6	7.5	14.8	8.2	4.0
SPV-35	0	0	0	2.9	5.9	1.4	6.4	8.2	7.0	15.9	4.8
IS 7254	16.1	5.3	2.4	1.9	5.0	0	2.4	4.0	6.8	9.6	5.3
SC 120-14	0	0	0	0	16.1	0	5.4	0	13.6	21.6	5.6
IS 2042	0	0	0	2.9	7.1	1.3	11.7	8.2	19.2	11.5	6.2
IS 3799	0	0	4.3	0	17.2	4.3	0	6.2	10.7	21.3	6.4
CSV-5	0	3.0	4.5	0	0	7.5	4.9	0	18.2	26.7	6.5
E 35-1	0	0	0	2.1	9.3	3.6	13.5	5.2	17.5	20.9	7.2
1258-B	0	0	3.3	0	2.3	3.7	4.3	0	32.5	27.7	7.4
3660-B	0	2.9	0	0	11.8	0	15.9	11.1	18.2	21.4	8.1
IS 5273	0	0	0	0	5.3	2.1	9.1	6.0	25.8	26.8	8.3
1202-B	0	0	3.1	2.8	12.8	0	0	7.9	27.0	37.0	9.1
IS-2327	9.1	6.5	1.9	0	10.0	1.5	5.5	3.1	46.3	20.4	10.4
SC 110-14	0	0	0	0	15.8	3.6	5.5	5.5	49.3	34.6	11.4
2077-B	0	0	21.4	2.9	0	14.0	26.4	7.4	25.4	20.5	11.8
SC 414-12	0	0	0	2.2	11.1	5.8	14.3	6.2	84.3	92.2	21.6
IS 2550	18.8	3.4	13.0	25.0	17.1	31.0	60.8	52.3	56.5	46.7	32.4
CSV-2	20.0	3.8	24.3	25.0	20.4	37.0	47.3	51.5	100	93.0	42.2
DMS-652	51.6	38.2	28.6	26.1	33.9	55.6	02.0	38.5	100	100	55.4
Location Mean	3.7		4.2		8.1		12.2		28.9		

that QL-3 is also immune to SDM in these countries.

Overall Performance

The entries QL-3, CSV-4, Uch V-2, IS-173, and Uch V-1 have consistently shown a high degree of resistance at all locations in all years (Table 8). QL-3 was free from infection in all the trials.

Discussion

From the results presented above, can we examine whether or not the ISDMN program is meeting the objectives listed on the first page of this paper?

Entries have been identified that are resistant across several locations in three seasons, i.e., QL-3, CSV-4, Uch V-2, IS-173, and Uch V-1. We cannot predict with certainty that these entries will not "break down" to SDM in the future, but they certainly are the most stable sources of SDM resistance available at the present.

There is no consistent evidence of races of the SDM pathogen from the ISDMN results. The greater SDM incidence at Dharwar in 1978 is probably due to a combination of higher inoculum pressure and favorable environment rather than to more virulent SDM races occurring there. There was a report that QL-3 developed up to 30% SDM in Texas trials (R. A. Frederiksen personal communication), but the

Table 8. Summary of SDM incidence in the best ISDMN entries in 3 years' trials.

Entry	Years	Locations[a]	SDM incidence (%) Mean	SDM incidence (%) Maximum
QL-3	1976	3	0	0
	1977	6	0	0
	1978	5	0	0
CSV-4	1976	3	0.3	1.7
	1977	6	4.2	7.6
	1978	5	2.5	9.0
UchV-2	1976	3	1.9	9.3
	1977	6	5.3	10.8
	1978	5	2.2	9.2
IS 173	1976	3	2.9	8.6
	1977	6	4.8	10.1
	1978	5	2.7	9.2
UchV-1	1976	3	2.3	7.5
	1977	6	2.9	4.8
	1978	5	3.7	16.0

a. ICRISAT Center 1977 data not used because of the severe artificial inoculation procedure employed.

seed source was different and subsequent results with ICRISAT QL-3 indicate that this was resistant in Texas.

The ISDMN cooperative network is relatively small at the present time. We need to develop links with more African and American SDM workers. We hope that as a result of the present workshop, we will have suggestions for additional cooperators in Asia, Africa, and the Americas, and that we will be provided with suggestions for additional ISDMN entries.

Acknowledgment

We are indebted to all cooperators (Table 1) for their vital role in the operation of the ISDMN program, and for the time, energy, resources, and effort they have provided, in order to produce the results reported in this paper.

Prospects for Chemical Control of the Cereal Downy Mildews

F. J. Schwinn*

Until recently, the control of diseases caused by the Peronosporales (downy mildews, damping-off, root rots etc.) was mainly based on dithiocarbamate fungicides, such as zineb, maneb, mancozeb, and on phthalimide derivatives, i.e., captan, folpet, captafol. Over the last 30 years these chemicals have been widely used throughout the world on a large variety of crops. They do a good job against the foliar downy mildews, as far as these cause local infections, and as long as they are used as protectants. Since they are purely residual fungicides without any major movement in the plant tissue, they are not active against systemic downy mildews, nor do they exhibit any curative activity.

Thus, there has been a clear-cut need for a more versatile fungicide against the Peronosporales. Whereas a great deal of progress has been made in the control of other fungal diseases during the last decade by the discovery of curative and systemic fungicides, the situation has remained unchanged until recently as far as the Peronosporales are concerned.

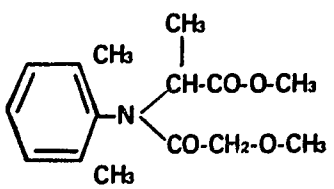
In 1973, Ciba-Geigy in Basel, Switzerland, so far unimportant in the fungicide market, discovered a new class of chemicals, the acylalanines, which showed promise with regard to curative activity and systemicity. A chemical with the code number CGA-48988 (US-code = 1-82-50 W) was finally selected for further development. We had two precursors of it, which you may have heard about, i.e., CGA-29212 (which was abandoned) and CGA-38140 (which has been introduced for use on ornamental plants). CGA-48988 is now in the phase of market introduction.

* Professor, Ciba Geigy Ltd., Basel, CH-4002, Switzerland.

General Information

Chemical name: Methyl D L-N-(2, 6 – dimethyl Phenyl) – N(2'methoxyacetyl)-alaninate.

Structural formula:



Solubility: 0.71% (=7100 ppm) in water at 20°C; readily soluble in most organic solvents.

Compatibility in tank mixes: compatible with most insecticides, acaricides, and fungicides in common use. In case of doubt, however, a compatibility test should be carried out.

Spray liquids must be used up within the day of mixing.

Toxicity: acute LD_{50} oral, rat 700 mg/kg
dermal, rat 3100 mg/kg
Irritation skin, eye (rabbit) minimal
Chronic effects in long-term studies (ongoing): so far no adverse findings.

Ecotoxicology: CGA 48988 is practically non-toxic to fish and bees; only slightly toxic to birds.
No bioaccumulation in aquatic organisms.
Favorable behavior in soil: slight leaching in nonabsorptive soils.

Residues: So far no residue problems in potatoes, grapevines, hops, lettuce, tobacco.

Tolerances: established for lettuce, potatoes, grapes.

Resistance: In greenhouse experiments no resistant strains of *Phytophthora infestans* and *Pythium* spp have been found so far; these studies are being continued.

Status of registration: (as of December 1978) registered in Australia, France, Greece, Ireland, Spain, South Africa, Switzerland, UK. Registration under way in many other countries.

Biological Properties

CGA-48988 is specific in its spectrum of activity (Table 1). It controls plant diseases caused by Oomycetes (*Phycomycetes*), especially those caused by Peronosporales. It controls diseases on leaves, fruits, stems, collars, and roots at

Table 1. Spectrum of activity of CGA-48988.

Crop	Pathogen	Route of application		
		leaf	soil	seed
Potatoes	*Phytophthora infestans*	xx[b]	x[a]	
Vines	*Plasmopara viticola*	xx		
Tobacco, transplanted	*Peronospora tabacina*	xx	xx	
	Phytophthora nicot. var. *nicot.*		x	
Seedbed	*Peronospora tabacina*	xx	xx	
	Pythium spp		xx	
Hop	*Pseudoperonospora humuli*	x	xx	
Maize, sorghum	*Sclerospora* spp		xx	xx
	Peronosclerospora spp		xx	xx
	Sclerophthora spp			
Millet	*Sclerospora graminicola*		xx	xx
Lettuce	*Bremia lactucae*	xx	x	
Brassicas	*Peronospora parasitica*	xx	xx	
Cucurbits	*Pseudoperonospora cubensis*	x		
Onions	*Peronospora destructor*	x		
Paprika	*Phytophthora capsici*		x	
Tomatoes	*Phytophthora infestans*	xx		
Vegetables	*Pythium* spp		x	x
Sugar beets	*Pythium* spp		x	x
Rapeseed	*Albugo candida*	x	x	x
Pineapples	*Phytophthora* spp		x	x*[c]
Avocados	*Phytophthora cinnamomi*		x	
Cocoa	*Phytophthora palmivora*	x		
Citrus, apples	*Phytophthora* spp		x	

a. x= positive result; *b*. xx = advanced trial stage
c. * = dips of material

markedly lower dosage rates per ha than the standard products. CGA-48988 is a systemic fungicide that penetrates rapidly and is translocated acropetally. It is absorbed through leaves, green stems, and roots. Due to its swift penetration it escapes adverse weather conditions. New growth is temporarily protected from inside (up to 21 days, depending on the local situation and growth pattern of the crop).

Spray intervals can be markedly prolonged compared with those of standard protective and residual fungicides in current use. This is especially true when the latter must be applied frequently.

In the field the action is protective as well as curative, i.e., new attacks will be prevented, and infections in the early stages of development will be stopped.

The formulations and trade names of CGA-48988 are given in Table 2. The APRON 35 SD formulation was specifically developed for a slurry seed treatment. It contains a higher amount of stickers in order to assure good adhesion to the seed. Only this formulation is recommended and will be sold for seed dressing application against cereal downy mildew. The methods used for control of SDM are given in Table 3.

At present both RIDOMIL and APRON, are under broad testing on maize, sorghum, and millet in a number of southeast Asian, South American and African countries, by CIBA-GEIGY field trial groups as well as by external cooperators and organizations.

Literature and Samples

Reprints of several CIBA-GEIGY publications on CGA-48988 are available from the author.

CIBA-GEIGY will gladly provide samples of RIDOMIL and APRON for field evaluation upon request. Please contact your local representative of the company, or Dr. P. A. Urech, c/o CIBA-GEIGY SA, AC 2.82, CH 4000 Basel, Switzerland.

Table 2. Formulations and trade names of CGA-48988.

Trade Name	Formulation	Active Ingredient Content	Use
RIDOMIL(R)	25 WP	25% CGA-48988 Wettable Powder	Foliar
RIDOMIL(R)	1 G 2 G 5 G	1/2/5 % CGA-48988 Granules	Soil
RIDOMIL(R) plus	50 WP	15% CGA-48988 + 35% Copper Wettable Powder	Foliar
APRON(R)	35 SD	35% CGA-48988 Seed dressing	Seed

(R) = registered trademark of CIBA-GEIGY Ltd., Basel, Switzerland,

Table 3. Methods used for SDM control with CGA-48988.

Crop	Application	Product	Rate
Maize	Foliar	RIDOMIL 25 WP	25g a i/100 liters of water, 300–500 liters/ha, 3–4 applications per season.
	Soil	RIDOMIL 2 G	25 kg/ha
	Seed	APRON 35 SD	500–750 g/100 kg seed 750–1000 ml water
Sorghum and Pearl millet	Seed	APRON 35 SD	750 g/100 kg seed 1000 ml water

Downy Mildew Discussion Session

Variability in *Peronosclerospora sorghi*

Williams:

One of the major points of interest with SDM is the apparent variability within the species *Peronosclerospora sorghi*. In Asia there is a Thai strain, a Rajasthan strain, and probably more than one southern Indian strain. An international project is needed to classify these different strains on the basis of their host range, symptoms, and capacity for conidial and oospore production.

Safeeulla:

I agree. The sorghum downy mildew in Thailand is morphologically different from that in southern India and the biotype in Rajasthan (northern India) is also different.

Williams:

Dr. Malaguti mentioned *Sclerospora graminicola* in the Americas. I believe that this species does not naturally infect pearl millet in the Americas. It's a *Setaria* pathogen there, whereas in Africa and India it is primarily a pearl millet pathogen.

Malaguti:

The similar pattern of reactions of sorghum and maize to SDM throughout the Americas suggests that only one strain is present there. *Sclerospora graminicola* also has been identified in the Americas, but is rarely observed. It could be that it was wrongly identified. *Sclerophthora macrospora* occurs more commonly.

Balasubramanian:

Can we exploit polyacrylamide gel electrophoresis for analysis of soluble proteins to help in identification of physiologic races within *Peronosclerospora sorghi?*

Safeeulla:

I believe we can.

Epidemiology

Brhane:

I would like to know, from Drs. Safeeulla and Malaguti, the history of the pattern of spread of SDM from the time it was first noticed in your respective countries (India and Venezuela, respectively) to the time when it became epidemic. In Ethiopia, where we grow mixed populations of sorghum, the disease occurs only on occasional plants and does not seem to be increasing, although it has been recognized for some years. I am particularly interested in the situation where farmers do not grow improved varieties.

Safeeulla:

Although the disease has been reported in India for 80 years, the pathogen has not spread as much as we might have expected. The pathogen is highly sensitive to temperature, and thus is localized in its importance. Where conditions are conducive for infection, higher disease incidence follows inoculum buildup and genetic uniformity of the sorghum crop. The genetic diversity in commercial sorghums could explain the lack of SDM epidemics in Ethiopia.

Frederiksen:

In Venezuela, the perpetuation of the downy mildew fungus in the off-season occurs in wild grasses as well as by oospores. When it encounters uniformly susceptible populations of hybrid sorghum, it develops in epidemic proportions. In the USA, it has spread only as far as there are collateral hosts for oospore production.

Wall:

In El Salvador, the sorghum crop is nonuniform — farmers grow mixed populations — and this is probably the reason why SDM epidemics do not develop there.

Resistance Screening

Craig:

With maize there are three reaction types to downy mildew. The highest level of resistance is effective for both conidia and oospores; the next is effective for either conidia or oospores; the lowest is susceptible to both. The rate of growth of the inoculum and the location of the inoculum with respect to the infection site is important.

Williams:

This fits well with the reactions we have obtained with different inoculation methods. Some entries remain resistant with high inoculum pressure, whereas others become more susceptible.

Safeeulla:

In my experience, we can use conidial inoculation to knock down all varieties.

Williams:

In our tests, QL-3 has been resistant following several inoculation methods.

Anahosur:

In our tests, using conidia, QL-3 showed no infection, not even a single local lesion, over 3 years of testing. QL-3 has a waxy leaf, and resistance could be related to this.

Balasubramanian:

I agree that it is possible to get resistance to both conidial and oospore inoculation.

Selvaraj:

In the absence of conclusive evidence of the existence of different types of resistance, perhaps we should use both oospore and conidial inoculum together. That is, in a "sick" plot infested with oospores, infectors should be planted so that the lines under test are subjected to both types of inoculum.

Williams:

As Dr. Dange and I have pointed out in our paper, the method of inoculation to be adopted should be epidemiologically meaningful for the particular location. This could involve conidia, oospores, or both. If, as we have heard in the case of Venezuela, conidial showers from collateral hosts are important epidemiologically, then you should screen for resistance using conidial showers.

At other locations, such as Mysore, where oospores seem to be far more important than conidia, oospores should be used. We probably need to determine the reactions of cultivars to conidia and to oospores, at different concentrations, and if we find distinct differences in reactions then we can blend the different resistances by breeding.

Chemical Control

Andrews:

Ridomil seems to have a specific effect on fungal growth. What is the possibility of the fungus developing resistance to the chemical? What is the implication for the strategy of use of the fungicide, and for the development of resistant varieties as a control measure?

Schwinn:

In vitro studies on the development of tolerance to Ridomil have been done using *Pythium* sp and *Phytophthora infestans.* We have not been able to identify resistant strains in *Pythium*, but from a large number of isolates of *P. infestans* some tolerant strains were selected. However, these were nonpathogenic. This is the type of behavior found with other new systemic fungicides such as the triaremoles, tripherine, and the triazole fungicides used for the control of other diseases. This is quite different from the tolerance which develops to the benzimidazole fungicides, where tolerant strains maintain their pathogenicity. Only long-term field testing will fully answer the question. This is under way in a number of countries.

We also try to use strategies to avoid or delay buildup of resistance to the fungicide, by mixing with or alternating with other fungicides. This works well with leaf spot-type foliar diseases, but is more difficult in diseases with systemic infection.

Tyagi:

Is there a possible residue problem in harvested grain following seed treatment with Ridomil?

Schwinn:

In our preliminary studies, residue has not been detected in grain following seed treatment, but we require more data.

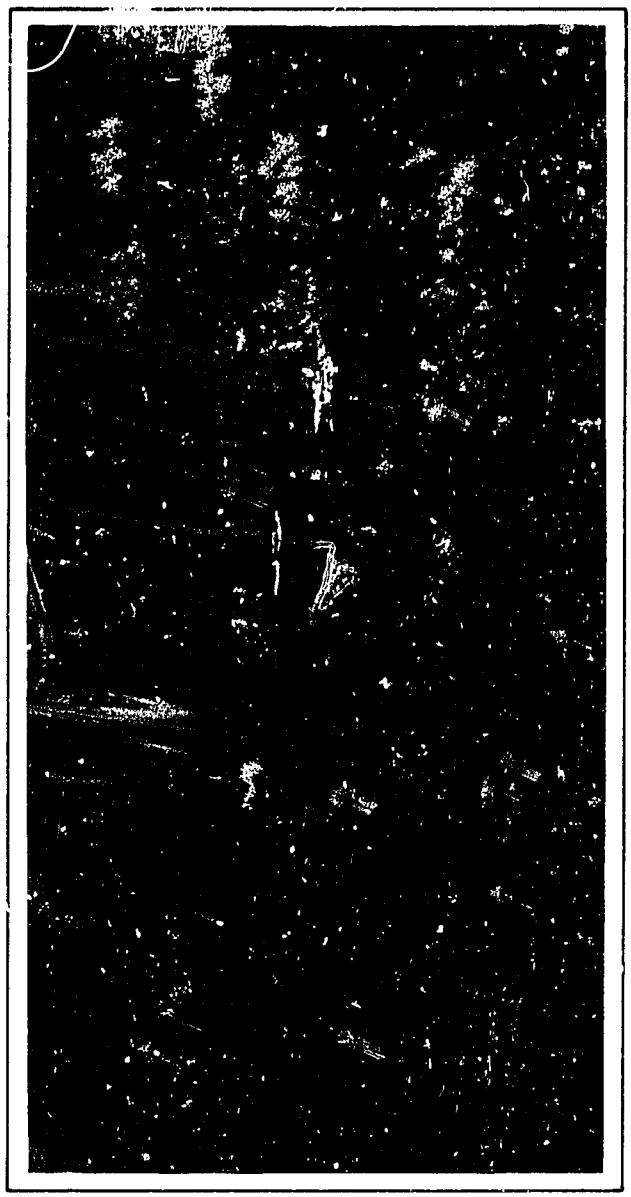
Andrews:

Now that effective fungicidal control of downy mildew is available, should we continue to develop genetic resistance? Should we perpetuate the use of highly susceptible varieties protected by Ridomil?

Zummo:

Ridomil should not be used to perpetuate a highly susceptible variety in commercial fields. Higher levels of genetic resistance combined with Ridomil in severe downy mildew areas will provide growers with added insurance for crop protection.

Sorghum
Leaf Diseases



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A Review of Sooty Stripe and Rough, Zonate, and Oval Leaf Spots

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Sooty Stripe

Sooty stripe, caused by *Ramulispora sorghi* (Ellis and Everhart) Olive and Lefebvre, is one of the most common leaf diseases of sorghum. It was discovered in 1903 in Alabama by Ellis and Everhart on johnsongrass, *Sorghum halepense* (L.) Pers., but was erroneously attributed to *Septorella sorghi*, of the order Sphaeropsidales. In 1920 in Manchuria, Miura described the disease on sorghum, and named the fungus *Ramulispora andropogonis* placing it in the Melanconiales. In 1932, Tai studied the same disease in China but named the pathogen *Titaeospora andropogonis* (Miura) Tai. In 1943, Bain and Edgerton in the USA showed that the fructification of the parasite was actually a sporodochium. In 1946 Olive, Lefebvre, and Sherwin described the fungus in some detail under the name *Ramulispora sorghi* (Ellis and Everhart) Olive and Lefebvre, transferring it to the Tuberculariaeae (Tarr 1962).

Elongate elliptical lesions develop on the leaves with straw-colored centers of dead tissue and purplish to tan lesion margins, depending on the host cultivar. The mature lesions can be several centimeters long and 1 to 2 cm wide. The lesions may coalesce to produce large areas of necrotic leaf tissue. As the lesions age, the centers darken and become greyish when conidia are produced, and then blackish or sooty as numerous small black sclerotia are produced. The sclerotia are superficial and are easily rubbed off. The sooty stripe lesions are somewhat similar to those of leaf blight, but the presence of the superficial sooty sclerotia is a clearly distinguishing feature of sooty stripe. In addition mature sooty stripe lesions are surrounded by distinct yellow haloes, which makes them readily distinguishable from lesions of leaf blight.

Description of the Pathogen (Olive et al. 1946)

The hyphae are intercellular in the parenchyma of the leaves but are intracellular in the vessels. The mycelium aggregates just beneath the stomata to form more or less compact stroma from which the conidiophores arise. These frequently mass into bundles that make up the sporodochia. The sporodochia emerge through the stomata and, under favorable conditions, produce abundant conidia which become aggregated into gelatinous masses. The conidia are slender, elongate (3.8–86.3 × 1.9–3μ), multiseptate (3–8 septa). Eventually the stroma and the conidiophores produce large black sclerotia (53–170μ in diameter). Most of the sclerotia that appear on the surface of the lesions seem to arise independently of the sporodochia. Each sclerotium is linked to a substomatal stroma by a column of hyphae that passes through the stomata.

Geographic Distribution

R. sorghi has been reported in many countries. In Africa: Botswana, Central African Republic, Nigeria, Rhodesia, Sudan, Tanzania, Chad (Tarr 1962), Mali (Chevaugeon 1952), Upper Volta (Delassus 1964), Senegal (Bouhot and Mallamaire 1965), Niger (Jouan and Delassus 1971). In Asia: China, India (Tarr 1962). In North America: in 12 states of southwestern USA (Odvody et al. 1973). In South America: Argentina (Tarr 1962), In Oceania: Australia (Ramakrishnan 1963). It is very probable that *R. sorghi* is present in other countries.

The ICRISAT International Sorghum Leaf Disease Nursery (ISLDN) was planted in the 1977 rainy season in Niger, Nigeria, Upper Volta, Senegal, and various regions in India. It was

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observed that among the different leaf diseases of sorghum that were studied (*Colletotrichum graminicola, Exserohilum turcicum, Gloeocercospora sorghi, Cercospora sorghi, Puccinia purpurea, Ascochyta sorghina, Ramulispora sorghi), Ramulispora sorghi* was the one for which selection pressure was adequate at most locations (6 out of 8).

Damage

R. sorghi can cause variable damage, ranging from a few lesions on senescent leaves at the base of the plant to complete destruction of the foliage. Ramakrishnan (1963) considers this disease to be very common in India in certain states — Uttar Pradesh, Andhra Pradesh, and Tamil Nadu — though not very serious. But Futrell and Webster (1966) report that in 1965 at Samaru, Nigeria, the leaf surface in most of the germplasm of a collection of 2693 sorghum lines from the world collection was entirely destroyed by *R. sorghi*. In Senegal, *R. sorghi* is the most important disease problem in sorghum after grain mold, especially in the central, south, and southeast of the country.

There is not much accurate information available on yield losses due to *R. sorghi* and other foliar fungi. Sharma and Jain (1975) studied the effect of seven leaf diseases, including *R. sorghi*, on five sorghum varieties and found a negative correlation between disease intensity and grain yields. But, in a study carried out in 1977 in Senegal on 288 sorghum lines there was no correlation between disease intensity and yield (Girard 1978).

Odvody et al. (1973) indicate that in Nebraska symptoms of *R. sorghi* can be observed at two stages of plant growth — seedling and late stages. Early attack can probably affect yields adversely. Late attacks — the most frequent — only affect yields when the upper leaves are attacked, as these leaves are involved in grain filling.

Biology and Epidemiology

Studies on the biology and epidemiology of *R. sorghi* are of two main types: in vitro culture of the fungus, and conservation of the parasite and spread of the disease.

The first type of study was carried out by Olive et al. (1946). They tried to grow *R. sorghi* on various culture media. On most of the agar media growth was slow and fructification of the fungus was abundant. The authors also indicate that carrot juice plus 1% dextrose is a favorable medium for fructification of *R. sorghi*.

In 1946 Olive et al. also initiated studies on the seasonal carryover of the parasite. After overwintering outside, the sclerotia were capable of producing conidia when incubated in moist chambers. In Nebraska, Odvody and Dunkle (1973) made a detailed study on the overwintering capacity of *R. sorghi*. They concluded that the sporodochia are the primary survival structures of *R. sorghi* and that, under certain field cultural practices, the maintenance of the connection between subepidermal sporodochia within the leaves and the sclerotia on the leaf surface could be important for survival of the parasite. Therefore, the disease could be started by conidia produced from sclerotia that have spent the unfavorable season in the soil or on sorghum leaf debris. Sorghum grains infected by *R. sorghi* could also ensure seasonal persistence of the disease (Tarr 1962). The perennial grass *Sorghum halepense* also ensures survival of the parasite (Rawla et al. 1975).

In the same cropping season, disease initiated by conidia produced by germination of sclerotia could be spread in the field by wind and rain (Tarr 1962). Development of the disease in the field has yet to be studied in detail.

Factors Promoting Disease Development

Disease development is influenced by several factors. Of these, climatic conditions are probably the most important, but not much is known about their role. Most of the authors indicate that the disease is promoted by high humidity (King 1972; Odvody et al. 1973; Sundaram 1977). In Senegal, it appears that the disease occurs mainly in areas between the 700 and 1000 mm isohyets. In the more humid southern regions it gives way to leaf blight (*Exserohilum turcicum*).

Soil fertility appears to be another important factor; Naik et al. (1977) have shown that incidence of five leaf diseases of sorghum (*Colletotrichum graminicola, Ramulispora sorghi, Gloeocercospora sorghi, Ascochyta sorghi*, and *Cercospora sorghi*) increased with nitrogen fertilization.

Other factors promoting disease development are location of the crop on low-lying clay soils (Jouan and Delassus 1971), and the proximity of high population of johnsongrass (*Sorghum halepense*) to sorghum plots (Odvody et al. 1973).

Screening Techniques for Resistance to *R. sorghi*

The use of genetic resistance is the main control method against sorghum diseases. Identification of genetic resistance and its rational use by breeders requires the development of appropriate screening techniques. This implies very sound knowledge of the biology and epidemiology of the disease. We have seen that, although the seasonal persistence of the disease has been studied in detail, little is known of disease development during the same cropping season. Therefore, screening methods used to date need to be improved.

Most scientists use natural infection. Futrell and Webster (1966) benefited from a serious natural outbreak of the disease in 1965 in Samaru, Nigeria, to test 2693 sorghum lines from the world collection. Tests were conducted under natural conditions in an area in south-central Senegal that is known to promote development of *R. sorghi*. Varieties highly susceptible to *R. sorghi* were planted in border rows to provide a source of inoculum. Similarly, ICRISAT recommends that for the International Sorghum Leaf Disease Nursery (ISLDN) trials sorghum susceptible to the principal leaf diseases should be planted at each end of the test rows.

Olive et al. (1946) carried out artificial inoculations on greenhouse-grown sorghum plants by spraying a suspension of conidia produced from carrot juice cultures. This technique enabled them to detect differences in susceptibility among test varieties.

Odvody et al. (1973) also carried out artificial inoculations in the greenhouse on 64 sorghum lines. They were able to conclude that sources of resistance to *R. sorghi* probably exist.

The major disadvantage of techniques based on infection under natural conditions is that these conditions depend on climatic factors which are unpredictable. As disease development cannot be controlled it could be inadequate or even absent. Thus, out of 7850 sorghum lines grown at Rajendranagar in 1968, only 215 showed symptoms of sooty stripe (Nagarajan et al. 1972). Obviously selection pressure was much too low for meaningful screening of varieties for resistance to *R. sorghi*.

Disease development can be controlled through artificial inoculation especially if it is carried out in a greenhouse and not in the field. The disadvantage is that these techniques cannot be used by all breeders (greenhouse facilities, preparation of inoculum, proper inoculation, etc are not available.)

Testing in areas known to favor disease development, with application of inoculum to compensate for lack of natural inoculum, should be a satisfactory compromise between controlled artificial inoculation and natural infection. But due to the lack of reliable data on disease development during the same cropping season it is not possible, at present, to rationally determine how inoculum can be applied (spreading of infected leaf debris, inoculation using syringes, use of infector rows, etc., are possibilities).

Scoring Scale for Disease Reactions

Different scoring scales have been used to evaluate susceptibility of sorghum varieties to *R. sorghi*.

Futrell and Webster (1966) considered as resistant those varieties with only small lesions and with less than 5% destroyed leaf area.

Rawla (1973) and Rawla and Chahal (1975) carried out detailed studies on the comparative needs of *R. sorghi* and *R. sacchari* cultures. The scale used by Nagarajan et al. (1972) is given in Table 1.

In Senegal, Galiba (personal communication) uses a very accurate scoring scale for his studies. He determines disease incidence (percentage of affected plants) and severity. For severity he uses a scale of 1 to 6, for ten leaves per plant and 20 plants per row:

1. Completely healthy leaf
2. Small lesions widely scattered; affected leaf area varying from 0 to 5%
3. Large lesions (1 to > 1 cm) widely scattered; affected leaf area varying from 6 to 20%
4. Numerous large lesions; affected leaf area varying from 21 to 35%

Table 1. Scoring scale for leaf disease reactions.

Grade	Percentage of affected leaf area	Remarks
1	Trace to 2	Moderately resistant
2	3-10	
3	11-20	Moderately susceptible
4	21-30	
5	31-40	
6	41-50	
7	51-60	
8	61-75	Susceptible
9	76-85	
10	86-100	

Source: Nagarajan et al. (1972).

5. Large lesions more or less connected; affected leaf area varying from 36 to 50%
6. Large coalescent lesions; affected leaf area more than 50%

An index is calculated on the basis of this scoring. There is also the scale recommended by plant pathologists at Texas A & M University, originally proposed by Zummo (1971) for scoring sorghum leaf diseases:

0 – evaluation not possible (no disease);
1 – resistant; disease is hardly apparent or present in certain plants only;
2 – heavy attack; more than 50% of the plants show low intensity symptoms; not of economic importance apparently;
3 – severe attack; 100% of the plants are affected; destroyed leaf area can be up to 25%; the disease seems to be of economic importance;
4 – same as 3 but destroyed leaf area is more than 25%
5 – leaves or plants are destroyed because of the disease.

Finally Mohammad and Mahmood (1973) have proposed a scoring system from which they calculated an infection value in an attempt to construct a quantitative system for intervarietal comparison.

Grade Leaf symptoms

0 – Leaves with no disease symptoms (Value 0)
1 – light brown stripe; severity trace to 20% (Mean 10)
2 – dark brown stripe; severity trace to 21–40% (Mean 30)
3 – dark brown stripe; severity trace to 41–60% (Mean 50)
4 – beginning to lose normal green color with rather long stripes; severity 61–90% (Mean 75)
5 – drying with stripes running through the leaf; severity 91–100% (Mean 95)

$$\text{Infection value} = \frac{\Sigma\ (\text{No. of diseased leaves in each grade} \times \text{mean value of each grade})}{\text{Total number of leaves in all grades}}$$

This system has been applied to several leaf diseases of sorghum (Mathur and Prakash 1975), although, to our knowledge, it has not been used for sooty stripe.

The problem of selecting a scoring scale is very important. In fact, each scale should be suited to the requirements of the users. No scale can be universally employed. The scoring technique used by Futrell and Webster has the advantage of being very simple but it does not enable us to distinguish between different degrees of susceptibility. The scale used by Nagarajan et al. (1972) is much more precise but quite difficult to use because without practice it is not easy to distinguish, for example, between 20 and 30% of affected leaf area. The technique used by Galiba in Senegal is more precise but more complicated, as the scoring has to be carried out leaf by leaf on individual plants. However, this scale is not meant for screening but for specific studies on *R. sorghi*. Similar considerations apply to the scale proposed by Mohammad and Mahmood (1973). Finally, the 1 to 5 scale proposed by the Texas plant pathologists has the disadvantage of including subjective criteria (economic importance). But experience proves that it is very easy to use and that ratings by different people on the same material generally agree.

Sources of Resistance

According to Leukel et al. (1951) resistant varieties should be used for disease control, but no resistant variety was identified.

Olive et al. (1946) indicate that in an experiment conducted in 1944, at the North Florida Experiment Station at Quincy, infection was

very low to moderate on the following varieties: Rex, Planter, Colman, Saccaline, Leoti, Denton, Rox, Orange, Sapling, Brown Durra, Norken, Atlas, Silver Top, and Gooseneck. In an artificial inoculation experiment only slight infection was observed on the following varieties: Honey, Early Folger, Atlas, Iceberg, Leoti, Plainsman milo, Spur feterita, and Early Hegari.

At Samaru in Nigeria Futrell and Webster (1966) tested 2693 entries from the world sorghum collection during a natural high-intensity disease outbreak. They found that only 5% of the varieties could be considered resistant to sooty stripe (small lesions and destroyed leaf area less than 5%) Forty seven percent of the lines from Upper Volta, 10% of those from Nigeria, and 6% of those from Mali, were resistant. Seventeen percent of the resistant lines belonged to the Conspicuum and 10% to the Caffrorum race. They concluded that West Africa was the area of origin for sorghums resistant to sooty stripe, and that most of the resistant lines had been found in the Conspicuum race, which is endemic in W. Africa.

In a test conducted at Nioro du Rip in Senegal in 1977, 25 of 288 lines were disease-free at the time of scoring. Of these 25 lines, 22 were derived from MN 1056×68–20, 2 from Ramada×68–18, and 1 from 63–[illegible] 18–20.

Results of the ICRISAT ISLDN-[illegible] revealed that, out of 30 lines tested in Senegal (Nioro), Upper Volta (Kamboinse), Nigeria (Samaru), Niger (Tarna), and India (Kovilpatti, Indore, Parbhani, Udaipur, and Delhi), six were given a 1-rating (no symptoms) or 2-rating (few hardly visible symptoms) at all locations where selection pressure was adequate for sooty stripe.

In the IDIN (International Disease and Insect Nursery) developed by the Texas A & M University, two lines were rated 1 at Bambey, Senegal, in 1975 and 1976. These were SC 326–6 and SC 599–610.

It is, therefore, not certain whether immune lines are available, but resistant parents probably exist.

Very little data is available on the stability of resistance to sooty stripe and its variability. Odvody et al. (1973) report that no parasitic specialization has been found among the strains isolated in Nebraska. The international nurseries of the Texas A & M University (IDIN) and ICRISAT (ISLDN) should bring in very useful information on this subject.

Control Methods Other than Varietal Resistance

Even if genetic resistance proves to be the most effective method of disease control, other methods should not be overlooked. Some of these could in fact usefully complement control by genetic methods, especially since we are not at all certain of having completely resistant parents.

We have seen that *R. sorghi* survives from one year to another mainly through sclerotia on infected leaf debris. Odvody et al. (1973) recommend that cropping techniques, where infected leaves are allowed to remain on the soil, should be avoided. Crop residues should be destroyed. Crop rotation is also recomended by Edmunds and Zummo (1975).

Infection of grains by *R. sorghi* was indicated by Tarr (1962). Seeds should therefore be disinfected, but we have no indication of work on this aspect of control.

Chemical treatment during growth could be somewhat effective in limiting or stopping disease development. Sundaram (1977) indicates that in India several fungicide sprays used to control various leaf diseases have increased sorghum yields. Zineb was most effective followed by thiram and captan. In Senegal, Girard (1978) obtained good control by weekly spraying of captafol or methylthiophanate when the first lesions appeared (Table 2).

In fact, the purpose of these experiments was to gain more information on this disease and not chemical control.

Research Priorities

In this paper I have repeatedly stressed that

Table 2. Effect of chemical treatment on disease development and grain yield of sorghum.

Treatment	Scoring (1–5 scale)	Grain production (g/plot)
Nontreated check	3.67	2158
Captafol	1.83	2310
Methylthiophanate	1.08	2170
L. S. D. 5%	0.47	–

certain aspects of the study on *R. sorghi* need further investigation. One of the first problems to be solved concerns yield losses due to *R. sorghi*. Can this fungus significantly reduce yields? If so, it would be useful to intensify research on this parasite. If not, a major research effort on *R. sorghi* would be superfluous, as other disease problems should be given priority. However, even with this hypothesis, an effort should be made to maintain or even improve resistance, not only because varieties spotted by too many lesions would make a bad impression on the farmer even if yields are excellent, but also to avoid developing highly susceptible varieties, which could then be seriously damaged by the disease.

The second important gap in our knowledge of *R. sorghi* concerns the development of the disease during the same cropping season. This point must be studied further to enable the development of rational and reliable screening tests for identification of resistance.

At present there are still no indications of physiological races of *R. sorghi*, and not much is known about stability of resistance to this parasite. It is absolutely necessary to obtain more specific information and to ensure that the sources of resistance used are stable.

As already mentioned in the beginning of this paper, identification of sources of resistance should continue, and this resistance should be incorporated in high-yielding sorghum varieties.

It would be useful to note that a young scientist from Senegal, Marcel Galiba, is now completing his thesis at the Laval University in Quebec on the variation and inheritance of the response of sorghum on *R. sorghi*. This work should be a very important contribution to research on this disease.

Rough Leaf Spot

Rough leaf spot (*Ascochyta sorghina*) was first reported in 1878 by Saccardo on sorghum in Italy. It seems to be restricted to *Sorghum* spp. It has been reported from Italy, USA, Africa, and India (Tarr 1962).

Symptoms

The first symptom observed on leaf blades is a slight chlorosis, on and around which groups of round black pycnidia develop. The pycnidia protrude above the leaf surface so that, when rubbed, they give the leaf a characteristically rough texture. Subsequently, the tissues within the infected area become necrotic, and light-colored circular to oval lesions with darker margins develop. The lesions are surrounded by chlorotic haloes, and are covered with the black pycnidia. Lesions may coalesce to form large irregular necrotic areas and whole leaves may be killed.

Description of the Pathogen

The fruiting bodies of this fungus are globose, depressed, papillate pycnidia. Depending on the author, their diameter may vary from 140 to almost 300 μ (Luttrell 1950; Saccas 1954). They contain numerous hyaline, two-celled pycnospores.

Damage

Ascochyta sorghina is generally considered as causing little damage (Edmunds and Zurnmo 1975). However, Weimer et al. (1937) indicate that it is capable of causing considerable damage in Georgia.

Biology and Epidemiology

Little is known about the biology and epidemiology of this disease. It certainly survives the off-season on crop residues. Seed transmission has been suggested, but not demonstrated. During the crop season, *A. sorghina* is probably spread by airborne pycnospores (Tarr 1962).

Control

Crop rotation, crop sanitation, selection of seed from healthy fields, seed treatments, and use of resistant varieties have been proposed for controlling this disease. Mathur and Prakash (1975) consider the following sorghum lines resistant to *A. sorghina*, 15–84, 305, 329, 558, 604–2, 610, C-5, CSH-1, CSH-2 and CSH-4.

Remarks

Another *Ascochyta* species, *Ascochyta sorghi* Saccardo, can also cause rough leaf spot. Ac-

cording to Sprague and Johnson (1950), *A. sorghi* is synonymous with *A. graminicola* and *A. elymni*, which indicates a wide host range among grasses. *A. sorgh* been reported mainly from cool temperate areas of North America and Europe, but also from India and Tanzania. The main morphological difference from *A. sorghina* is that the latter has larger pycnidia with coarser pycnospores than *A. sorghi* (Tarr 1962).

Zonate Leaf Spot

Zonate leaf spot was first observed in 1940 on sweet sorghum in Louisiana. Its pathogen was erroneously taken as that of sooty stripe. After several attempts to identify it, Bain and Edgerton (1943) proposed to call this new parasite *Gloeocercospora sorghi*, hence creating the genus *Gloeocercospora* to be classified in the family of Tuberculariaceae.

Deighton (1971) made the Latin diagnosis, because the initial description by Bain and Edgerton had been made in English.

Symptoms

The characteristic zonate leaf spot lesions are roughly circular (or semicircular if they originate near the edge of the leaf) with alternating bands of dark purple or red color and tan or straw color, to give a concentric or zonate appearance. Initially the lesions appear as small reddish brown water-soaked spots, sometimes with a narrow green halo. They enlarge, become dark red, and tend to elongate, initially parallel with the veins, and eventually spread across the leaf, developing the zonations to attain their characteristic mature appearance. Under warm humid conditions the fungus produces large pinkish gelatinous fruiting bodies (conidiophores and conidia), visible with the naked eye on and around the necrotic areas of the lesions. On heavily infected leaves, lesions may coalesce over a large proportion of the leaf surface. Black sclerotia may form in mature lesions.

Description of the Pathogen (after Bain and Edgerton 1943)

The fruiting bodies of the fungus are sporodochia which are formed on the leaf surface from hyphae emerging from the stomata. There is no substomatal stroma, as there is with *Ramulispora sorghi*. Sporodochia produce conidia often aggregated within a salmon-colored jelly. Conidia are hyaline, often filiform, multiseptate, varying in length from 20 to 19 μm and a maximal width of 3 μm. Black sclerotia are formed within the tissues of old lesions.

Geographical Distribution

Zonate leaf spot has been reported from most tropical and subtropical countries where sorghum is grown.

Damage

Unlike *Ramulispora sorghi*, which attacks only *Sorghum* spp; *G. sorghi* can infect other gramineae such as maize, pearl millet, sugarcane, bentgrass (*Eragrostis* spp), bermudagrass (*Cynodon dactylon*) (Tarr 1962), and *Vetiveria zizanioides* (Puranik 1966). On sorghum and on bentgrass damage is sometimes of economic importance (Tarr 1962).

On sorghum, damage varies according to varieties, climatic conditions, growth stage of host plant at infection etc. According to Odvody et al. (1974) the incidence of zonate leaf spot in Nebraska was 50%, with 5 to 10% of the total foliage area completely destroyed. Saccas (1954) mentions very severe infections in the north of Oubangui and on the mid-Chari, leading, in some cases to complete destruction of the plants. Zummo (1971) writes that *G. sorghi* may cause sweet sorghum plants to die when there is a high incidence of the disease at seedling stage. Severe infection at a later stage may result in premature defoliation and a reduction of the yield of the stalks and the sugar content of the juice.

Biology and Epidemiology

In many aspects, *G. sorghi* resembles *R. sorghi*. Dean (1968) has shown that the sclerotia of *G. sorghi* overwinter on leaf debris not buried in the soil and produce conidia when plated on lima-bean agar. Therefore sclerotia probably play a major role in the survival and spread of

the disease. Zonate leaf spot is probably induced by the germination of sclerotia and then spread in the field by rain and wind (Odvody et al. 1974). Seed transmission of the disease has also been reported. Cicarrone (1949) suggests that zonate leaf spot was introduced into Venezuela by infected seeds from the United States. Alternate hosts might play a role in disease survival, as the fungus will infect several grass species, but this aspect has not been investigated.

There are some indications of the existence of specialized forms of the fungus, e.g., an isolate of *G. sorghi* from sorghum caused severe infection of sorghum, but only slight infection of millet, and an isolate from millet caused only moderate infection of either sorghum or millet (Luttrell 1954).

The fungus can grow on ordinary culture media (Bain and Edgerton 1943; Dean 1968; Rawla 1973).

It appears that *G. sorghi* can modify the chlorophyll and carotenoid content of sorghum leaves (Chiranjeevi and Tripathi 1976).

Control Measures

Resistance

Luttrell (1950) showed that there were differences in varietal susceptibility to zonate leaf spot, but there were too many variations from one location to another to permit a ranking of the varieties. Texas Milo appeared to be the most resistant variety.

Dean (1966) found no resistant variety among the 1509 lines that had been artificially inoculated in a greenhouse.

Sundaram et al. (1965) have recorded the reaction to zonate leaf spot of 658 entries from the world collection. Almost 30% were resistant to this disease, and these varieties belonged to the races Conspicuum, Roxburghii, Nigricans, Caudatum, zera zera, and Cernuum.

Zummo (1971) wrote that highly resistant varieties were not available, but that breeding lines with a high degree of mature tissue and field resistance were being used in the breeding programs.

Therefore, as in the case of *R. sorghi*, it is not certain whether immune lines are available, but it seems that parents with a high degree of field resistance may be found.

Other Control Measures

Saccas (1954) proposed the following preventive control methods:

1. Incineration of stalks and leaves after harvest to destroy spores and sclerotia;
2. Crop rotation;
3. Seed disinfection.

Keil (1946) has found that three fungicides gave good control of *G. sorghi* on turf: Puraturf (phenyl mercuri thiethanol ammonium lactate) (1/10 000), Puratized 177 (1/5000), and Zerlate (zinc dimethyl dithiocarbamate) (1.5/100 gal).

From laboratory and field experiments by Agnihotri and Pandey (1976), acidulated Benomyl and Bavistin (1000 parts per million appeared to be the most effective for controlling zonate leaf spot of sorghum.

Research Priorities

Research priorities for zonate leaf spot should be more or less the same as those for sooty stripe:

- Determination of economic effects of the disease (yield losses);
- Epidemiology: development of the disease during the same cropping season;
- Development of reliable screening techniques for identification of resistance;
- Search for stable resistance.

Oval Leaf Spot

Oval leaf spot, *Ramulispora sorghicola*, was first described by Harris (1960) in Nigeria. Although it has been reported from other countries in Africa and in Asia, it is less common than *Ramulispora sorghi*.

The symptoms first appear as small water-soaked spots. These develop into small roughly circular (2 to 4 mm in dia) lesions with dark red to brown margins and lighter centers, in which small black sclerotia can be produced. These symptoms resemble and can be confused with the leaf anthracnose symptoms. The two diseases can be distinguished with the aid of a hand lens, for the fruiting bodies of oval leaf spot do not possess protruding black setae.

Description of the Pathogen

The pathogen resembles *R. sorghi*. The clearest

differences between the two species is provided by the sclerotia — those of *R. sorghi* are tuberculate, glabrous, and very profuse, while those of *R. sorghicola* are fewer and bear small septate setae on the surface (Harris 1960).

Geographical Distribution

Oval leaf spot has been reported from the following countries in Africa: Nigeria, Sudan, Malawi, Ghana, Uganda (Harris 1960), Upper Volta (Delassus 1964), Niger (Jouan and Delassus 1971), and Senegal (Girard and Delassus 1976). In Niger, Jouan and Delassus (1971) consider it as the most frequent disease of sorghum. In other regions it has been reported from Pakistan (Harris 1960) and India (Nagarajan et al. 1972), and Hawaii (Harris 1960).

Damage

Oval leaf spot generally causes little damage, although it has been reported that in Nigeria up to 25% of the leaf area may be affected (Harris 1960).

Biology and Epidemiology

Very little is known about the biology and the epidemiolcgy of the fungus. It can be grown on cornmeal agar, and it sporulates abundantly during the first few days (Harris 1960). Nagarajan et al. (1971) grew it on potato-dextrose agar and sorghum-meal agar and obtained better sporulation on the latter medium.

While sclerotia can produce conidia on their surface, they are not necessary for the seasonal carryover of the disease, the infected leaf fragments being able to sporulate after having spent the dry season on the ground surface (Harris 1960).

Control Measures

Very few attempts have been made to screen for resistance to *R. sorghicola*. Nagarajan et al. (1971) tried to evaluate the reactions of 7850 sorghum lines to *R. sorghi* and *R. sorghicola*, but only 198 lines were found to be infected by oval leaf spot. Most of the highly susceptible lines were "Durra" types from India.

Varieties have been screened for resistance to the different foliage diseases, including oval leaf spot in Nigeria. These data are available at the Institute for Agricultural Research at Samaru (Zummo, personal communication).

Although oval leaf spot is not generally considered a potentially damaging disease, plant breeders should discard highly susceptible lines, particularly in the areas where this disease is very common (Niger and Nigeria).

Crop rotations could be recommended in case of severe infection (Harris 1960), as the pathogen spends the dry season on leaf debris on the soil.

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Sorghum Rust

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Rust (*Puccinia purpurea* Cooke) is widely distributed and occurs in almost all sorghum-growing areas of the world, particularly East Africa, India, and South and Central America. For all practical purposes, rust attacks only sorghum species. The symptoms of rust are so well known that they will not be reviewed in this paper, other than to point out that rust of stem tissue — particularly sorghum peduncles, rachis, branches, and glumes — is quite common in environments under which rust is an economically important disease.

Tarr (1962) indicates that rust rarely causes severe losses, other than the occasional desiccation of leaves. Unfortunately there is little evidence to the contrary. The problem is, I am convinced, that sorghum rust under some conditions is a major yield inhibitor, and its presence predisposes sorghum to other major disease problems — such as the *Fusarium* stalk rots, occasionally charcoal rot, small seed, and possibly grain molding. Dr. Jose Amador, plant pathologist of the Texas Agricultural Extension Service at Weslaco, estimated that in 1975 sorghum rust may have caused losses up to 1500 kg of grain per ha in the Rio Grande Valley of Texas. These differences in yield were obtained by sorghum cultivars planted at the same rates, at the same location, approximately 2 weeks apart. The environment itself was not strikingly different, other than late summer rains, and cooler than normal weather permitted a favorable environment for the development of rust. The earlier planted sorghums essentially escaped the disease, whereas those planted later were devastated.

In certain regions of central Mexico (note: Country Reports: Mexico, by J. Betancourt), yields approaching 14 000 kg/ha are not uncommon. In this area, rusts may cause extensive yield losses. In the area near Lake Chapala, very few hybrids have been successful. Hybrids grown in the region initially escaped the disease, perhaps because of low inoculum concentration. In time, the original hybrids were severely damaged by rust and, at times, by other foliage diseases. These were replaced by commercial hybrids developed in the USA, which possessed a surprisingly high degree of "slow rusting." It is my opinion that slow rusting is the key characteristic of hybrids still popular in these regions; in those hybrids in which rust develops early, there is a preponderance of reduced seed size, lodging caused by *Fusarium* sp desiccation of the leaves, and, at times, deterioration of the grain (which is related in part to the small grain size). Rust has been very important in Argentina, and I have seen severe rust in Brazil. It is a damaging disease in Puerto Rico, where death of the foliage is not uncommon in the more susceptible cultivars. Bergquist (1971) clearly indicated that rust would be a major deterrent to the ratooning of sorghum in Hawaii, as did Hill (1969) for the New Guinea islands. Consequently, where grain sorghum is grown in the cooler humid tropical regions of the world, we must contend with rust. It needs to be controlled.

Rust Control

Frederiksen and Rosenow (1972) argued that in North America rust is a naturally stabilized disease. They argued this simply on the basis of the fact that cultivars and/or hybrids with moderate levels of resistance respond similarly in many locations throughout the Americas. There have been no significant physiologic differences in the pathogen population that differentiated the intermediate levels of resistance. Consequently, generalized resistance, based on the past 10 years of observations, has been found very stable. One of the first slow-rusting sources found in the Texas Conversion Program was released in a variety known as TAM-

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428. TAM-428, as a rust-resistant pollinator for a variety of different hybrids, has moderate to excellent levels of resistance, even in the severe rust regions of Mexico. This level of resistance is adequate for the southern USA and for Puerto Rico. Frederiksen and Rosenow also contended that rust is naturally stabilized, because survival pressure is placed on the pathogen in cooler, more humid environments, such as the lower Rio Grande Valley or northern Mexico during the winter months. These observations are consistent with some earlier reports on origins of inoculum in the Americas. Since sorghum matures and develops during the hot dry growing season on the Great Plains, rust — because of its adaptation to the more humid tropics — in all probability will not become a major disease problem on the Great Plains. This may also be true in a number of temperate regions. However, in the tropical areas, particularly where elevation tends to keep temperatures relatively cool, rust can be and is potentially a major problem.

The Pathogen and Its Infection Process

The last published original work on the taxonomy of the *Puccinia purpurea* was done by Pavgi (1972) who worked with Dickson at the University of Wisconsin. One of the questions dealt with was whether or not *P. purpurea* was closely related to *P. sorghi* or *P. polysora.* In all cases, Pavgi concluded that the species was valid and morphologically distinct from the other species of rust that attack maize. Dalmacio (1969), who studied the infection process in the Philippines, found that germination takes place in a matter of a few hours; pustules appear in 10 to 14 days, appearing more rapidly on seedlings than on mature plants. Not surprisingly, pustules appear on the undersides of the leaves. Dalmacio believed that this was due in part to the thicker epidermal layers of the upper leaf surfaces. This observation has not been confirmed. We have observed in our work that there are, fundamentally, three types of reaction to rust in the sorghums. There are those that are essentially free from rust — except for an occasional large pustule appearing near a midvein — under all conditions. There are those groups of sorghums that characteristically show either smaller or fewer pustules. SC-175, for example, is a line that develops small pustules, TAM-428 develops fewer pustules, more slowly than fully susceptible types. The other reaction class is that of the fully susceptible types. However, there are differences — expressed primarily in numbers of infection foci on the leaf — among these lines. Miller and Cruzado (1969) demonstrated different allelic interactions with rust at the Pu locus. In some instances the heterozygote became susceptible to rust as the season matured, whereas the homozygous resistant materials had consistently low levels of disease.

Bergquist (1974) described two races of *Puccinia sorghi* in the (Hawaiian) Islands. IS 2814-TSC was capable of separating the population into two races, while Rio (SC-599) was resistant to both races. Differentials with generalized resistance were undesirable. Bergquist (1971) showed that rust resistance was inherited as a dominant trait. In India at the same time, Rana et al. (1976) argued that susceptibility is a dominant character among the parental lines that they had used. It is interesting to note that they also reported that there is a linkage of rust resistance with tan plant color, which may account for observations by other sorghum workers who have noted additional leaf disease resistance in tan plants. Rust resistance to sorghum is and has been described for a number of sorghum lines (Anonymous 1974, 1975, 1976; Bergquist 1971, 1974: Broadhead and Coleman 1974; Lopez et al. 1975). A method for improving the levels of rust resistance and/or screening for resistance has been described and consequently host resistance may well be the most reasonable means for controlling rust (Frederiksen and Rosenow 1974; Patil Kulkarni et al. 1972). Fortunately, systemic fungicides are available with the potential for controlling rust, although in North America these fungicides are not labeled for use (Agarwal and Kotasthane 1973).

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Sorghum Leaf Blight

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In 1962, Tarr stated that foliage diseases do not usually kill the plant, unless seedlings or young plants are exposed to prolonged attacks. He further stated that by destroying green photosynthetic tissue, and in some cases causing premature wilting and leaf death, these diseases reduce or delay plant growth and development and as a consequence reduce yield of both grain and fodder. Leaf blight, caused by *Exserohilum turcicum* (Pass.) Leonard and Suggs, is one of the two most widely distributed and at times damaging foliage pathogens of the sorghums.

Distribution and Symptoms

Leaf blight has been found or observed in all of the major sorghum-growing areas of the world (Tarumoto et al. 1977). Severe damage caused by this disease has been observed in the United States, Argentina, Mexico, and Israel, and globally it may be the most important foliar disease of sorghum (Edmunds and Zummo 1975; Parodi et al. 1977; Prakash et al. 1975; Robert and Findley 1952; Tarr 1962; Tuleen 1975). Fortunately, the economic importance or losses caused by leaf blight appear to be minor (Olson and Santos 1976; Sundaram et al. 1972). Leaf blight, like a number of foliar diseases of sorghum, is quite conspicuous. Consequently, it may frequently be the first disease identified during a cursory examination of a sorghum field. Leaf blight differs from the symptoms caused by the other foliar pathogens, although it can be quite similar to sooty stripe. Lesions on mature susceptible leaves are often fusiform, 1 to 3 cm wide by several cm long, with pigmented edges and tan or grey centers which darken during sporulation. Under low magnification the conidiophores are seen to rise at random, and they may develop from aerial hyphae on material kept in an aqueous saturated atmosphere. A distinctive feature of leaf blight is the timing of the appearance of symptoms. Small flecks appear, usually 3 or 4 days after a favorable infection period. These small lesions can be seen with a hand lens, but the large distinctive lesions do not appear until about 2 weeks later. The plugging of nearby vessels causes a localized wilt within the leaf tissue. During successive favorable periods, the fungus will continue to colonize a leaf, leaving bands or characteristic zones within the leaf. When infections are abundant, total leaf wilting is not uncommon.

Nomenclature of the Pathogen

In 1876, Passerini described the northern leaf blight species as *Helminthosporium turcicum*. In 1958, Luttrell found a way to induce formation of a few ascocarps in culture and described the perfect stage of this fungus as *Trichometasphaeria* (Luttrell 1958). In 1974, Leonard and Suggs redescribed the perfect stage as *Setosphaeria turcica*. The ascocarps of this group were described by Holm (1957). Regarding the imperfect stage, Drechsler (1934) described the *Helminthosporium* conidial stages as those having the true *Helminthosporium* characters, including *H. turcicum*, and the cylindro-*Helminthosporium*. Apparently this was not acceptable to Shoemaker (1959), who proposed a new genus for the graminicola species with *Bipolaris turcica* as the type species (sloane et al.). Leonard and Suggs (1974) removed from *Bipolaris* the species having a protuberant conidial hylum, and established *Exserohilum turcicum* (Pass.). Because of this last work, we now believe that the generic name *Exserohilum* will probably become the preferred name for the pathogen that causes leaf blight of sorghum.

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Host Range

E. turcicum is a common pathogen of sorghum, teosinte, *Paspalum*, and *Zea* in nature. In addition, *Triticum, Hordeum, Avena, Saccharum,* and *Oryza* are susceptible to *E. turcicum* when artificially inoculated.

Numerous workers have examined the host range of isolates of *E. turcicum* from maize, sorghum, and johnsongrass. There is a strong tendency for isolates from one species to infect that same species. Isolates from nature that were homocaryons were pathogenic to the monospecies, whereas species that were heterocaryons were capable of attacking two or more species (Bergquist and Masias 1973, 1974). Bergquist and Masias 1974; Bhowmik and Prasada 1970; Hamid and Aragaki 1975; Masias and Bergquist 1974). These workers also distinguished races of *E. turcicum,* using isolates capable of differentiating between maize lines with and without the H_1–resistance gene and those races which attacked both maize and sorghum (Masias and Bergquist 1974). Hamid and Aragaki (1975) showed that virulence to maize and sorghum was inherited independently, as indicated by a one-to-one-to-one segregation for virulence to both, from a cross between a maize-specific isolate and an isolate virulent to both sorghum and johnsongrass. They proposed that the common occurrence of field isolates with virulence to both maize and sorghum should be treated as a third specialized form, and suggested the trinomial *Setophaeria turcica* forma speciales *complexa*.

Disease Development and Host Reaction

The conidium of *Exserohilum* is unique, not only in appearance but in function. The conidia are known to thicken their walls and become conidiospores as an overwintering or overseasoning spore. Spores or conidia germinate by the formation of a germ tube which may or may not form an appressorium on the surface of the leaf. Beneath the appressorium a peg will penetrate through the cuticle and form hyphae within the host cells. Most individual penetrations result in the appearance of the hypersensitive fleck (Tuleen and Frederiksen 1977; Ullstrup 1978). Frequency of infection foci, resulting in colonization of the host, can be used as a tool to differentiate levels of nonspecific resistance or generalized resistance among cultivars. Infection hyphae slowly pass through living cells with scant disturbance initially forming rudimentary appressoria as each wall is encountered. Cells of resistant sorghum hosts may form pigments at this stage. Maize with monogenic resistance has a chloronemic halo around the infection site — in the jargon of maize workers, "the typical H_1 reaction." A similar response has been observed occasionally in some of the more resistant sorghum cultivars. In the absence of resistance, hyphae encounter a vascular bundle, enter a vessel, begin to absorb nutrients, and proliferate. Damage is assumed to result from the mycelial plugging of the vessel. Leaf blight was accurately described as a local or localized wilt (Jennings and Ullstrup 1957). It is possible that the wilting is actually due to the tyloses or complexes with polysaccharides released by digestion of the vessel lumen, rather than to the actual physical plugging by the hyphae. Pectin plugs have not been found in maize. Toxins may be partially responsible for the death and collapse of host cells (Tuleen and Frederiksen 1977). Following the development of a major lesion, pathogen fruiting begins. Hyphae fill the substomal cavity or epidermal cell and produce a stroma. The condiophores of *E. turcicum* develop from the stroma. The condiophore has about three cells and produces a conidium at the apex. If the humidity is high, the conidiophore curves. When humidity declines, conidiophore cells begin to dry, bubbles appear inside them, and the condiophore snaps to an upright position, throwing the conidium away and out of the boundary layer. Not surprisingly, most conidia are released on days following rainfall (40% are discharged between 0800 and 1200 hr, as the morning sun dries the foliage, Meredith 1965). Mycelia within the vessels continue to advance almost on a daily cycle, as indicated by the daily boundaries or borders of the lesion. The mycelium will utilize a crossover vessel to widen the lesion and form a larger area of colonization (Jennings and Ullstrup 1957). The conidiophore is capable of emitting additional conidia on subsequent days. Tuleen 1975, using five different sorghum differentials at six maturity stages, found that plants in the imma-

ture stages of plant growth were more susceptible than more mature plants. Even plants of the highly resistant TAM-2572 developed some lesions when inoculated in the early seedling stages of growth. Other plants developed resistant reactions as they matured. The most susceptible were more susceptible through more mature stages (Table 1). Most inoculation sites result in the appearance of hypersensitive flecks. These fleck responses develop even on plants of the youngest maturity stage (Table 2). The ability of the host to form these flecks decreased not only with susceptibility, but with the stage of maturity at inoculation for each cultivar. Since cultivars decrease in susceptibility as they mature, the frequency of hypersensitivity merely appears to be the normal response of sorghum to infection by *E. turcicum*. Tuleen and Frederiksen (1977) also presented evidence to suggest that toxic substances produced by *E. turcicum* were somewhat specific and could differentiate between relative levels of resistance to *E. turcicum*. This work in part has been supported by workers from other areas (Karve et al. 1977).

Control of Leaf Blight

Leaf blight is not as devastating in sorghum as it could be if it were not for the relatively high

Table 1. Leaf blight reactions on host differentials at six maturity stages 14 days after inoculation.

Host differential	disease reactions[a] for maturity stages:[b]					
	1	2	3	4	5	6
TAM-2572	MR[c]	MR[c]	R	R	R	R
TAM-2566	MS	MS	R	R	R	R
Tx-3197	S	MS	R	R	R	R
Tx-7078	S	S	S	MS	MS	d
IS-2403C	S	S	S	S	R	R

a. Qualitative disease reactions represent means for three replications of two to four plants for each maturity stage; each replication was inoculated and incubated separately at 22°C for 38 hours. R = resistant; MR = moderately resistant; MS = moderately susceptible; S = susceptible.
b. Plant maturity stages as defined by Vanderlip (1972).
c. only one lesion observed on one of eight plants.
d. Missing sample.

Table 2. Frequency of hypersensitive flecking with *Exserohilum turcicum* appressoria on host differentials at six maturity stages 72 hours after inoculation.

Host differential	Percent appressoria surrounded by flecks for maturity stages[a]					
	1	2	3	4	5	6
TAM-2572	100	50	50	50	50	0[b]
TAM-2566	100	25	25	33	0	0
Tx-3197	100	40	0	10	–[c]	0
Tx-7078	90	50	10	0	0	–[c]
IS-2403C	100	50	0	0	0	0

a. Flecks associated with 20 appressoria on 2-cm² leaf tissue were counted from a plant of each of the maturity stages defined by Vanderlip (1972).
b. No spores were found to adhere to leaf surfaces in Stage 6.
c. Missing sample.

levels of host resistance available in sorghum (Edmunds and Zummo 1975). Consciously or unconsciously, breeders have selected sorghum cultivars with surprisingly high levels of resistance to leaf blight, particularly on plant tissues approaching maturity (Drolsom 1957; Fletcher et al. 1975). In 1977 in Egypt, all entries in the Texas International Disease and Insect Nursery were classified as resistant to *E. turcicum* when compared to the local varieties grown there. Consequently, we find ourselves in the comfortable situation of having essentially a broad-based level of resistance in many of our sorghum cultivars, to which we were merely adding additional genes for resistance. Both monogenic and polygenic sources of resistance have been reported for maize (Jenkins and Robert 1961; Warren 1975), and it is apparent that similar systems exist for sorghum. The host-specific genes conditioning the hyper-sensitivity have been described by several workers (Bergquist and Masias 1973; Frederiksen et al. 1975; and Tuleen and Frederiksen 1977; Ullstrup 1978) and Rosenow and Frederiksen (unpublished data) have provided some evidence to indicate that there are two major genes and some maternal factors conditioning resistance to leaf blight in sorghum.

Incorporation of leaf blight resistance requires a relatively simple breeding and selection procedure. Several "techniques" are available for the evaluation of host resistance, both under field and laboratory conditions (Hilu and Hooker 1964, 1965). The variability of *E. turcicum* has been examined critically by several authors (Bhowmik and Prasada 1970; Hilu and Hooker 1965; Masias and Bergquist 1974; Misra and Mishra 1971; Nelson et al. 1965). These authors suggest that the organism tends to be quite stable; discreetly different physiologic races of the pathogen have however, been described (Bergquist and Masias 1973; Bhowmik and Prasada 1970). The utilization of the generalized form of resistance would tend to suggest that this disease can be adequately and easily controlled by host resistance. Most of the work on chemical control has been done with maize (Berger 1970); several isolated studies on the control of leaf blight have indicated that it is easily controlled by common fungicides (Sundaram et al. 1972). The major problem in interpreting the usefulness of fungicides has been the lack of feasibility data in conjunction with their applications. At present it would appear that chemical control of leaf blight is an unnecessary practice, unless for some reason it is necessary to grow blight-susceptible materials.

Future Research Needs with *E. turcicum*

Currently, the principal strategy for controlling *E. turcicum* is the use and deployment of host resistance. This program has been successful in areas where E. turcicum causes moderately severe disease. However, in areas where the disease causes extensive damage, higher levels of resistance (such as exists in TAM-2572), when available to the professional or commercial plant breeders, should reduce leaf blight losses. With the incorporation of these high levels of resistance, it is conceivable that the disease will be even less threatening. In view of the variability of the pathogen, monitoring for potential shifts of the pathogen must be continued. In addition, information is needed on the dissemination and survival of *E. turcicum*, particularly in sorghum. As indicated in the sources of leaf disease resistance by Frederiksen and Franklin in these proceedings, distribution of *E. turcicum* in the field is directly proportional to either the presence of susceptible cultivars or initial inoculum from initial infection foci. However, knowledge of the distribution and occurrence of the pathogen in sorghum fields during vulnerable stages is critical, and epidemiological data similar to that known for maize does not exist for sorghum (Meredith 1966; Robert 1964; Saccas 1954).

Acknowledgment

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Screening of Sorghum for Leaf-Disease Resistance in India

H. C. Sharma*

Importance of Leaf Diseases

Sorghum is an important food and fodder crop in India. It occupies about 17.5 million ha with a production of 8.5 million metric tons (tonnes). The crop is subject to several leaf diseases of bacterial, viral, and fungal etiology, which cause losses in grain yield (Ramakrishna 1963; Harris and Fisher 1974; Sharma and Jain 1975). With increasing understanding of leaf diseases — their wide distribution, frequent occurrence, and capacity to cause losses — their importance has been properly recognized in the sorghum-improvement program in India.

Distribution of Leaf Disease

The leaf diseases of bacterial and viral origin at present are not important in India (Anonymous 1977). Amongst the diseases of fungal origin, anthracnose (*Colletotrichum graminicola*), rust (*Puccinia purpurea*), grey leaf spot (*Cercospora sorghi*), zonate leaf spot (*Gloeocercospora sorghi*), leaf blight (*Exserohilum turcicum*), sooty stripe (*Ramulispora sorghi*), and rough leaf spot (*Ascochyta sorghi*) are prevalent in all sorghum-growing states of India. Anthracnose has been reported with greater severity from northern India — Delhi, Udaipur (Rajasthan), Madhya Pradesh, and parts of Maharashtra — whereas rust and grey leaf spot occur in a greater intensity in the south (Karnataka and Tamil Nadu). Rough leaf spot is severe in Madhya Pradesh. Leaf blight, sooty stripe, and zonate leaf spot are commonly present in varying intensities throughout the sorghum-growing areas. Zonate leaf spot was reported to be severe in Akola and Parbhani (Maharashtra) in seasons with high rainfall.

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Losses in grain yields in sorghum caused by leaf spot diseases have been discussed by Sharma and Jain (1975). The losses caused by rust (*Puccinia purpurea*) and leaf blight (*Exserohilum turcicum*) were estimated to be around 50 and 45% respectively at Dharwar and Hyderabad. The losses were estimated by comparing the yields of protected and unprotected plots, for several leaf diseases at Indore following the method of Chester (1959) (Table 1). Estimated losses range from 32 to 60%.

Capacity to Cause Epidemics

Disease intensities were recorded on a 0 to 5 scale for rust, anthracnose, rough leaf spot, zonate leaf spot, sooty stripe, and grey leaf spot 75 days after planting (DAP). All leaves were evaluated for each disease.

The disease most often developing in epidemics was anthracnose, followed by rust, grey leaf spot, sooty stripe, rough leaf spot, and zonate leaf spot. Sorghum grain yields can be enhanced by breeding varieties resistant to leaf diseases. This requires reliable and practical screening methods. Before considering currently used screening methods or developing

Table 1. Estimated yield losses in sorghum due to several diseases, at Indore, India.

Disease	Variety	Estimated losses (%)
Rust	1188	50
Anthracnose	PJ 4K	48
	Local	41
	V 60-1	60
Rough spot	555	35
Zonate leaf spot	302	32
Sooty stripe	370	36
Gray leaf spot	CK 60-B	–

a new one, it will be worthwhile to know the intricacies involved in the process of breeding for disease resistance with particular reference to sorghum: (a) sorghum leaf diseases are caused by species of several genera of fungi, (b) pathogenic variability can occur within species, (c) our knowledge regarding races, strains, and biotypes is meager, (d) sorghum (a rainy-season crop) completed its life-cycle in varying environments, (e) mutual interference e.g., grey leaf spot and rough leaf spot were observed to interfere with each other's development on varieties 36B and G-1B (Table 2); Similarly, there was mutual interference in the development of anthracnose and rust on varieties VZM 2B and Giza (Table 3). To effectively screen for resistance we must overcome these problems. But do we know enough about the biology and epidemiology of these diseases to do this?

Table 2. Mutual interference of gray spot and rough spot — disease indices recorded for mixed infections at Indore, Madhya Pradesh, India.

Variety 36 B		Variety G-1 B	
Rough spot	Gray spot	Rough spot	Gray spot
2.00	0.42	1.13	0.86
2.35	0.35	1.15	0.55
1.55	1.94	0.53	1.60
1.97	1.43	1.25	1.38
1.47	2.10	0.40	1.50
2.13	1.00	0.85	1.03
1.25	2.32	1.00	0.53
0.78	2.25	1.17	0.35
1.55	1.45	1.10	0.90
1.25	2.00	1.23	1.69

Biology of Pathogens

Rana et al. (1976), suggest the existence of races of sorghum rust (*Puccinia purpurea*) in India. Bergquist (1974) reported two races of rust from Hawaii. Information on the prevalence of races of other leaf pathogens has not been reported. Variations in reaction types have been detected in India for anthracnose, rust, and grey leaf spot (Figs. 1, 2, 3). In the absence of sufficient knowledge regarding races and strains of a pathogen, it is worthwhile to collect from different locations infective material from varieties showing different host reactions. Varieties showing varying symptoms should be used as "infector" and "indicator" rows in the screening plot of a disease, so that different races and strains are probably included in the screening plot. The infector varieties used by the author for leaf diseases at Indore and listed in Table 4.

Table 3. Mutual interference of anthracnose and rust — disease indices recorded for mixed infection at Indore, Madhya Pradesh, India.

Variety VZM-2 B		Variety Giza	
Anthracnose	Rust	Anthracnose	Rust
2.10	1.80	0.90	1.40
2.15	1.40	0.60	1.70
0.74	2.00	1.60	0.10
0.65	2.05	1.75	0.15
2.07	1.20	0.50	1.93
2.36	1.05	1.10	1.20
2.63	0.60	0.50	1.44
2.35	0.80	1.10	0.48
1.30	1.58	0.50	1.33
1.88	1.88	1.45	0.69

Table 4. Susceptible selections used as "infector" and "indicator" rows in screening plots for various sorghum leaf diseases at Indore (M. P.), India.

S. No.	Isolines	Leaf Diseases
1	Selections from 654, 364 (locals)	anthracnose
2	Selections from 3660B, Nag B, 322 and IS 857	grey leaf spot
3	Selections from 1188 and E 302	rust
4	Selections from 555 and 914	rough leaf spot
5	Selections from 97 (local) and IS 338	zonate leaf spot
6	Selections from CSV-3, 290 (local) and IS 643	sooty stripe

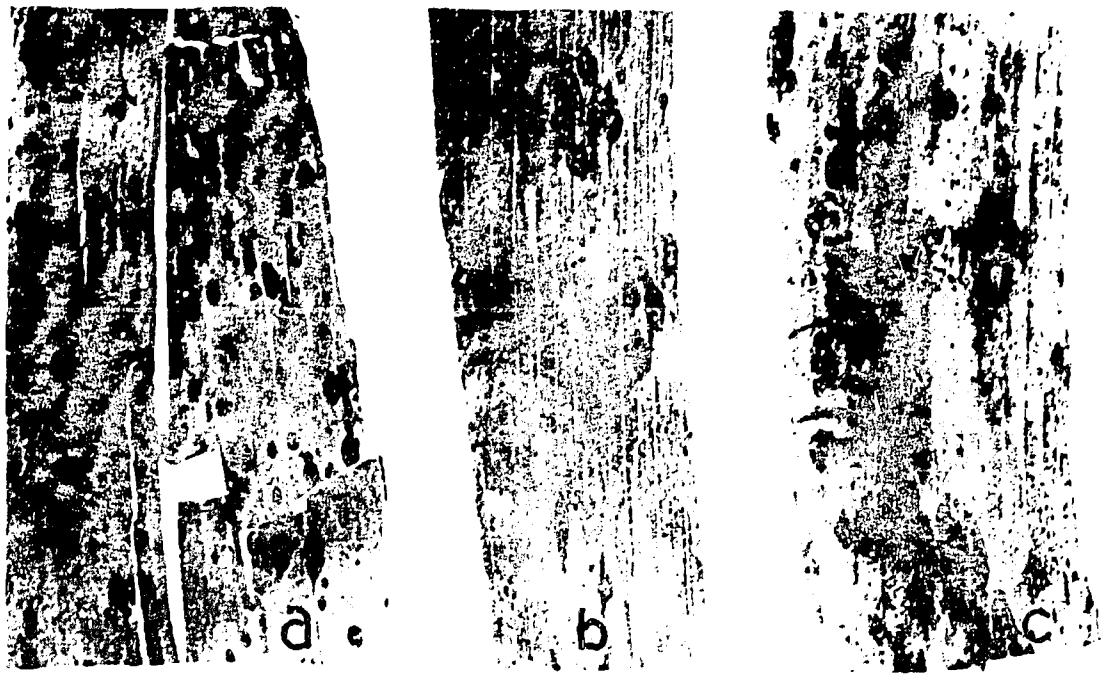
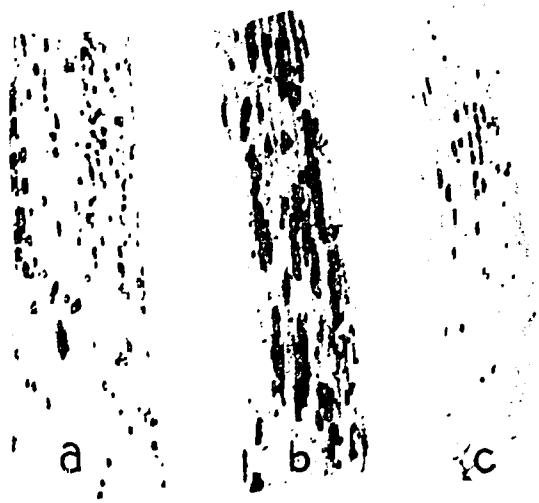


Figure 1. Variation in symptoms of anthracnose (Colletotrichum graminicola) *observed in different or the same variety: (a) small limited spots, (b) big limited spots, and (c) big diffusing spots.*

Figure 2. Variation in size of rust (Puccinia purpurea) *pustules on different or the same variety: (a) small, and (b) large pustules.*



GREY SPOT

Figure 3. Variation in symptoms of gray spot (Cercospora sorghi) *observed on different varieties of sorghum: (a) small round or circular spots with gray centers; (b) large elongated irregular spots; and (c) large elongated rectangular spots.*

Creation of Epidemics

Abundant development of a disease or diseases in the screening plot is a prerequisite to differentiating disease resistance from disease escapes (Sharma and Jain 1977). The facts that crowding the susceptible host plants promotes epidemic development, and that in general, leaf diseases spread much faster in the direction of the wind, were satisfactorily used by the author in creation of epidemics in the screening plots. The epidemics were regulated by growing a thick population of susceptible lines across the wind direction (Figs. 4, 10). Plants in the infector rows are inoculated with the infective material. The inoculum multiplies on the infector rows and serves as an inoculum source for disease spread in to the screening plot. Indicator rows are grown perpendicular to the "infector" rows, with a separation of 1 meter between them (Fig. 4). The indicator rows are repeated at 5-m intervals so as to measure the epidemic force of the disease in the screening plot. Disease intensity is measured as percent disease-affected area according to a standard disease scale (Fig. 5–8), and epidemic force of the disease is computed according to the details in Figure 9. Sufficient epidemic force of a disease develops in the screening plot to differentiate between resistance sources and escapes (Figs. 10, 11, 12, and 13).

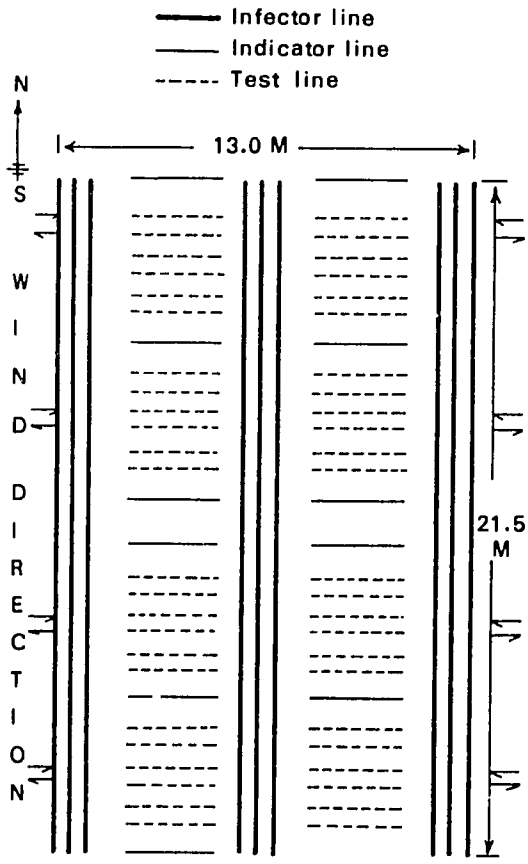


Figure 4. Layout of screening plot.

Screening Methods

Screening in Multilocation Varietal, Hybrid, and Parental Trials under Natural Conditions

Disease intensity is recorded according to a standard disease scale under natural infection in the field. Varieties are compared on the basis of disease intensity. Sources of error are: (a) the method does not assure the presence of pathogen, and if present there is no guarantee that all the varieties are uniformly exposed to it, and (b) a high population of advanced plant materials does not permit a smooth multiplication and spread of the disease in the experimental plot, and thus chances of escapes are great.

Screening in Greenhouse or Screenhouse

The optimum conditions of temperature, humidity, and light favorable to disease development may be regulated in green or screen houses. Plants are predisposed by incubating under optimum conditions and are then inoculated with cultures of the pathogens. This method saves time and gives quick results, However, there are limitations: (a) it is not possible to handle a large number of breeding materials, (b) one cannot be sure of the pathogenicity of pathogens (pure cultures maintained on artificial media are usually used and the virulence of pathogens is likely to be reduced or altered), (c) the variety under predisposed conditions does not get an opportunity for adjustment and for expression of post-infection resistance (Allen 1959), and this may lead to rejection of potentially valuable material as susceptible.

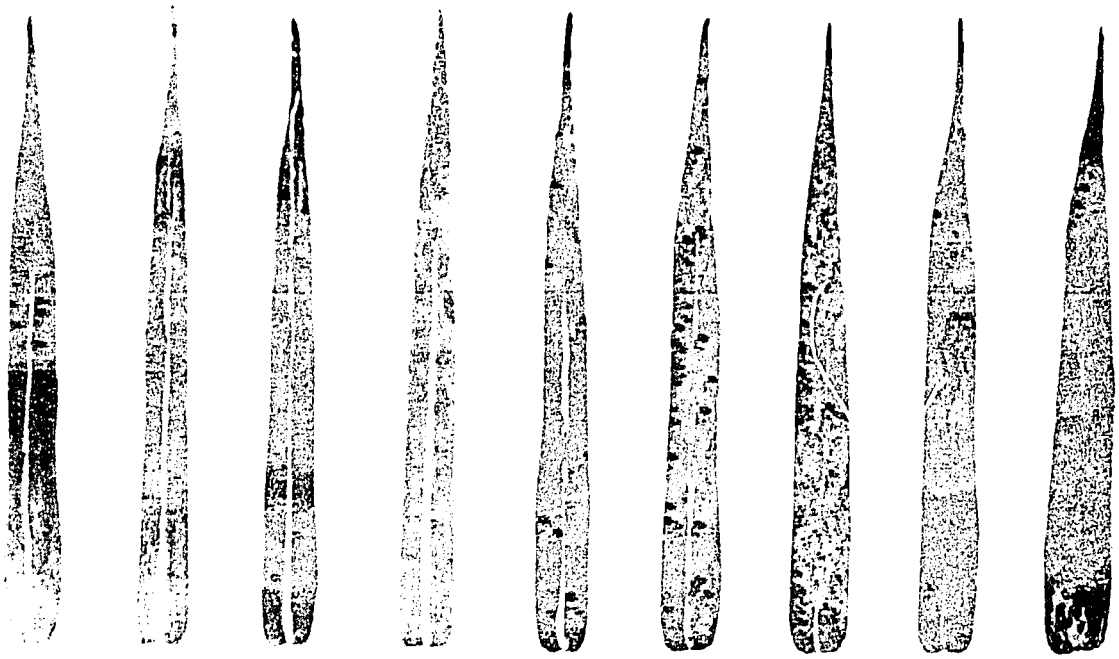
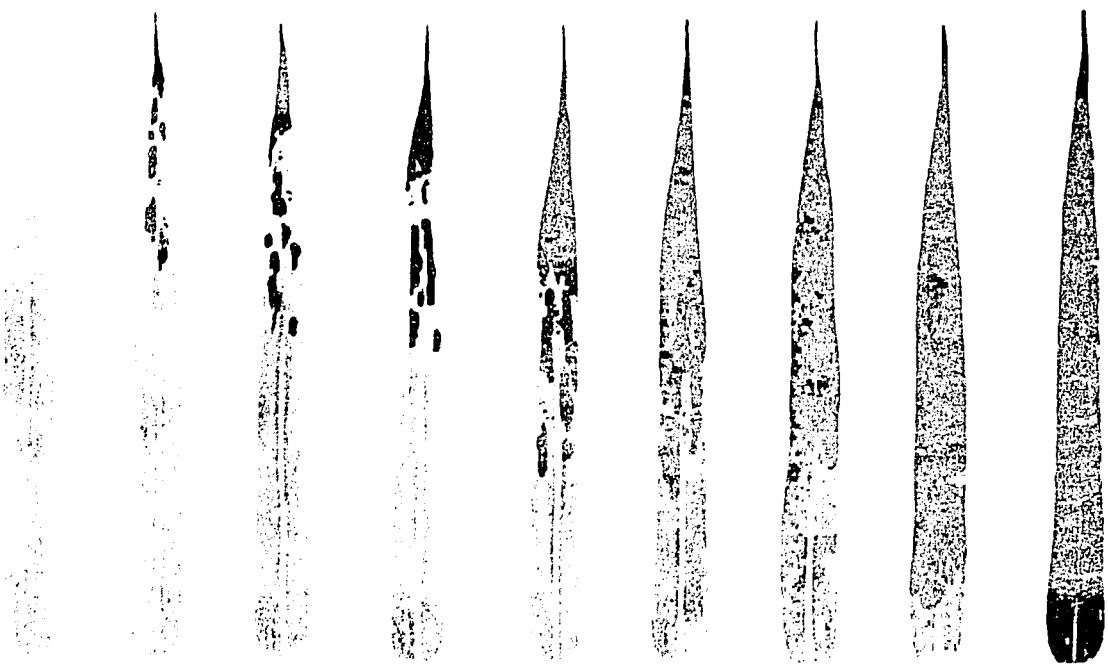


Figure 5. *Anthracnose* (Colletotrichum graminicola).

Standard visual ratings for scoring approximate percentage of leaf area affected by leaf diseases. Leaf on left shows no area affected and is rated '1' for 0%. Leaf on right is almost totally affected, and is rated '9' for 100%. Intermediate ratings, left to right, are '2' (2.5%), '3' (5%), '4' (10%), '5' (20%), '6' (35%), '7' (50%), and '8' (75%).

Figure 6. *Gray spot* (Cercospora sorghi).



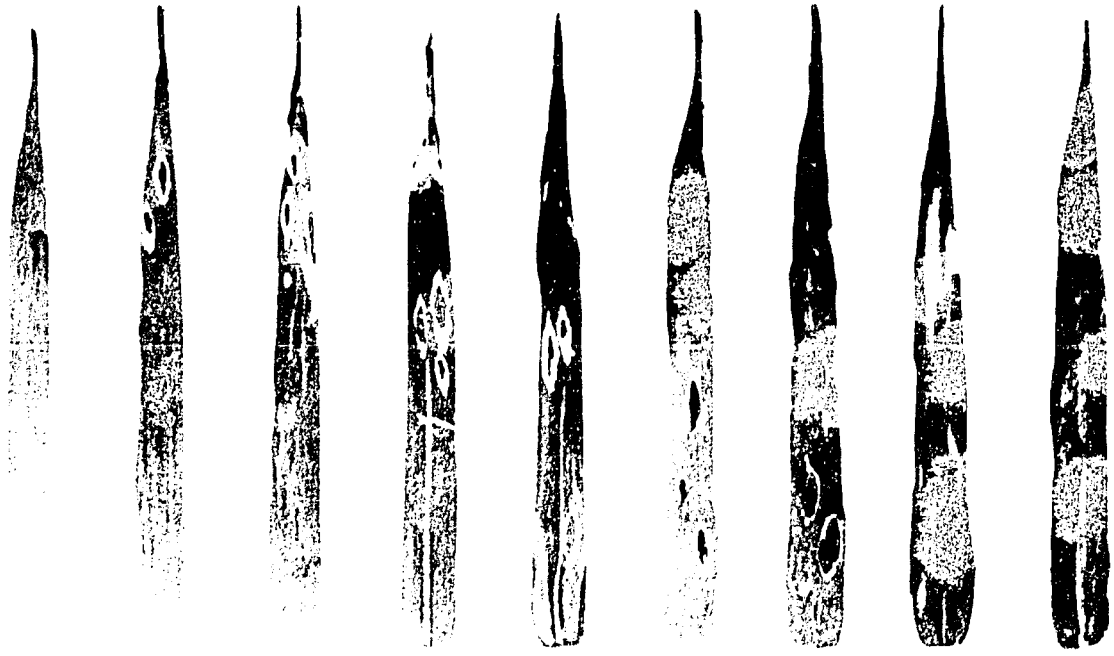
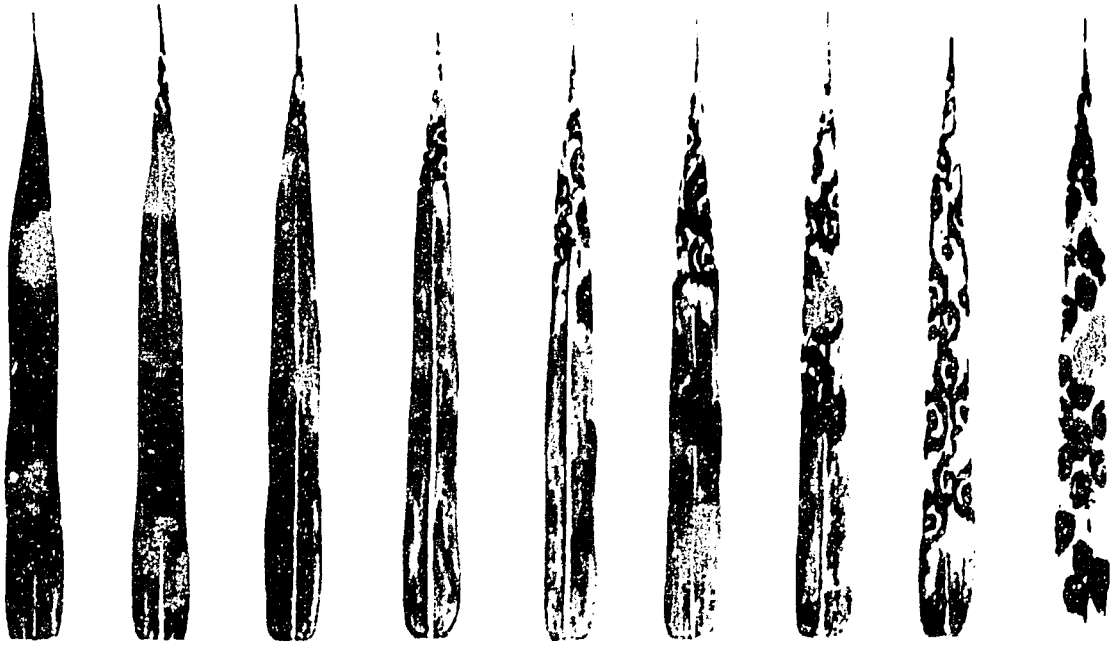


Figure 7. *Sooty Stripe* (Ramulispora sorghi).

Standard visual ratings for scoring approximate percentage of leaf area affected by leaf diseases. Leaf on left shows no area affected and is rated '1' for 0%. Leaf on right is almost totally affected, and is rated '9' for 100%. Intermediate ratings, left to right, are '2' (2.5%), '3' (5%), '4' (10%), '5' (20%), '6' (35%), '7' (50%), and '8' (75%).

Figure 8. *Zonate spot* (Gloeocercospora sorghi).



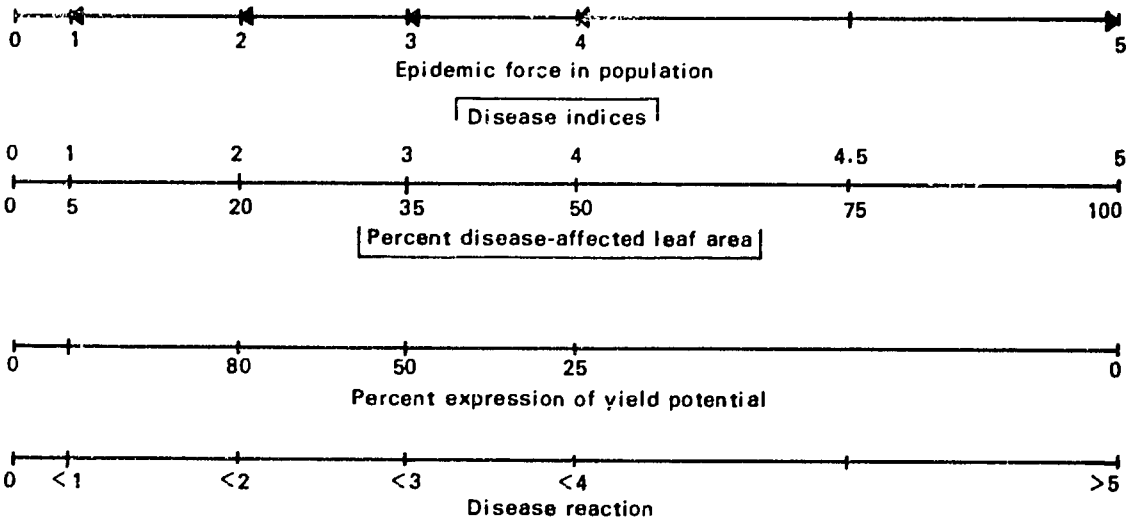


Figure 9. Relationship between disease intensity, epidemic force, expression of yield potential, and disease reaction.

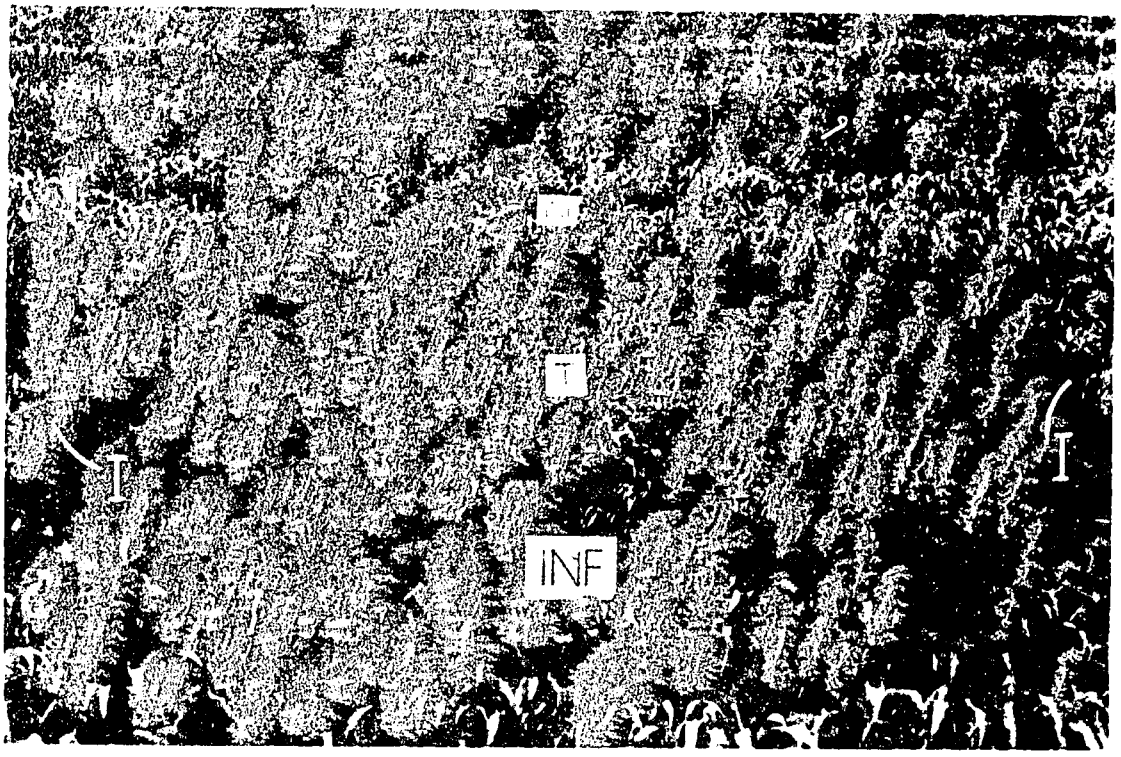


Figure 10. Sector of a screening plot 40 days old. INF indicates infector rows and T indicates test entries. Indicator rows are indicated by I.

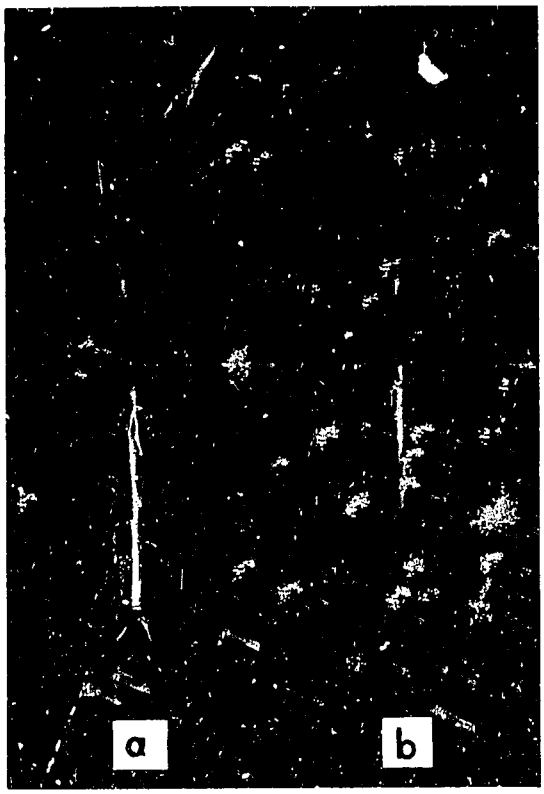


Figure 11. Anthracnose on the four basal leaves of CSH-5 (resistant) (a) and on a standard susceptible cultivar (b). Plants, 45 days old, were growing in a screening plot.

Screening in Local Disease Gardens

Disease gardens of all important pathogens and their races are maintained specifically for testing important parental material and hybrid lines. Diverse infective material is collected for use in creating artificial epidemics under conditions that favor maximum disease development. This is a desirable method, but has the following limitations: (a) it considers only the important pathogens, and determination of these is difficult, for sometimes inconspicuous and unimportant pathogens have become important on varieties that have developed resistance to the so-called important ones, and (b) it is not possible to catalog all races of pathogens at any given time.

Regional and Seasonal Disease Gardens

In this method, candidate entries are exposed to the full force of epidemics that can be produced by a potential pathogen or pathogens present under regional climatic conditions during the crop season (Sharma 1975; Sharma and Jain 1977, 1978). This method is based on the following assumptions: (a) susceptible varieties of a region harbor the pathogen population, including its races, strains, and biotypes, (b) ideal environment for screening sorghum germplasm for disease resistance is the environment of the crop season of the region. Isolines predominantly susceptible to a disease or diseases, along with infective material, are collected from several places and varieties to represent the prevalent disease races of the region. A disease epidemic is developed using infector rows as indicated above. The disease intensity is recorded according to a standard scale and the varieties are classified for their resistance (Fig. 5–9). Multilocation trials, or repeat of the trial for at least 3 consecutive years, are suggested to confirm behavior of a resistant variety to overcome the danger of variations in the expression of virulence of the pathogen or that of susceptibility of the variety by inhibitor genes or fluctuations in the environment. The advantages of this method are: (a) it facilitates exposure of the candidate entries to the full force that a potential pathogen may cause under actual climatic conditions (results obtained are directly applicable), (b) it assures abundant disease development in the screening plots, (c) it provides a tool "indicator" to measure intensity and distribution of a disease in the field, (d) screening is done under a standard epidemic level, (e) enables a constant watch on the fluctuations of host-parasite balance caused by new introductions (Mcknew 1960), (f) handles a large number of breeding materials, (g) permits satisfactory screening without elaborate laboratory and greenhouse facilities. The fact that results are specific and applicable to the regional conditions alone is considered to be a limitation.

Screening Scales and Techniques

Primarily, the problem of determining plant-disease resistance is based on measurement of

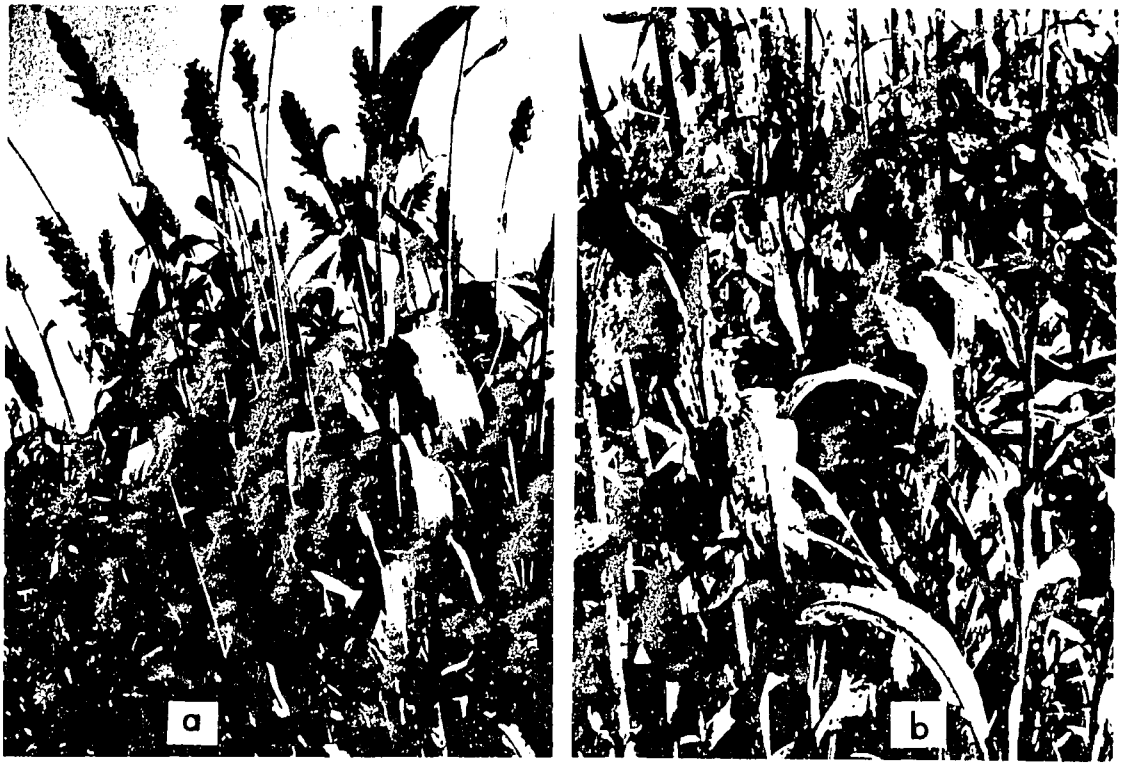


Figure 12. Disease development on CSH-5 (a) and a standard susceptible cultivar (b) in the screening plot for gray spot. The susceptible cultivar was used in the "Indicator" rows.

Figure 13. Infection of rust on the flag leaf; lower leaves have either no rust infection or a trace of rust infection.

the disease intensity and translating this into categories of resistance. The identification and detection of resistance would thus depend upon the accuracy of the disease scale. An ideal disease scale should be comprehensive, well defined, and properly demarcated at important disease intensities between the "no-disease" point at one end and the "maximum-disease" point at the other.

Disease scales presently used in scoring leaf diseases of sorghum include that used in the International Sorghum Leaf Disease Nursery, that suggested by AICSIP, and a scale (developed by the author) used at Indore.

Disease Scoring Scale Suggested by ICRISAT in ISLDN

ICRISAT recommends a 1–5 disease scale (Appendix Table 1). The disease rating "2" is descriptive and does not clearly demarcate between 2 and 3 as it does in the case of 3, 4, and 5. Disease rating "2" will vary and suffers from the human factor. The disease rating 5 is very

wide and does not specify the maximum disease point.

Disease Scoring Scale Recommended by AICSIP for Use in Leaf Disease Trials

The disease scale is similar to that suggested by ICRISAT (Appendix Table 2). However, it is based mainly on percent disease-affected leaf area. The disease rating "2" is very wide and ignores a marking at 5 %, a point recognized in general as "epidemic outbreak point" for leaf diseases (van der Plank 1960, 1963). The disease rating 5 suffers from the same drawback as in the case of disease scale suggested by ICRISAT.

The disease rating of a variety will vary with the maximum which in turn varies from one location to another, and from season to season (Walker 1965) and, therefore, it needs to be specified.

Disease Scoring Scale Used at AICSIP, Indore, for Measuring Leaf Diseases of Sorghum

An elaborate and descriptive scale for measuring intensity of foliar diseases of sorghum was developed at AICSIP, JNKVV, Indore. The scale is based on percent leaf area infected. It reads that disease in percentage leaf area affected with the help of visual standards 1–9 representing 0, 2.5, 5, 10, 20, 35, 50, 75, and 100 % disease-affected area (Fig. 5–8). Disease indices 0 to 5 are marked at 0, 5, 20, 35, 50, and 100% on the disease scale to denote important conversion point for disease reaction and epidemic force (Fig. 9). The important points: marked as disease index 1 denotes a point of epidemic outbreak in the population and disease indices 2, 3, and 4 are the points where a variety would yield approximately 80, 50, and 25% of its potential (Fig. 9), determined on the basis of overall results of correlation and regression studies, and disease index 5 is the maximum disease point.

Recording Data

Since severity is determined by visual observations the readings can not be absolutely accurate. The accuracy of recording observations will depend mostly on the individual worker. However, use of visual standards of percent diseased leaf area gives a satisfactory degree of accuracy and uniformity in recording data. Disease index is computed as follows:

$$\text{Infection index} = \frac{\Sigma\,\%\text{ values recorded}}{\text{No. of observations}}$$

Finally, the disease index of variety, plot, or field (as the case may be) is read as disease index on the percentage disease scale.

Time of Recording Data

A single observation will not give sufficient indication of the dynamics of disease development in a variety. Further, the sorghum crop grows tall, consequently basal and top leaves of the same plant lie in different environmental planes. Diseases that were severe on basal leaves may appear in traces on the top leaves and vice versa. It has been observed that rough spot is severe on basal leaves of the Vidisha 60–1, whereas anthracnose or rust are severe on top leaves. Recording periodic observations at vulnerable stages — grand growth period, floral primordial formation, boot leaf, and dough maturity stages — of the crop would be ideal. Since recording foliar diseases on sorghum is time-consuming and laborious (several leaf diseases are involved). AICSIP recommended three observations at 30, 60, and 75 days after crop emergence and the timings happened to correspond with the vulnerable stages of various sorghum varieties. Similarly, ICRISAT emphasizes disease recording at milk or dough stage of the crop. However, it appears that two observations, around boot-leaf stage, and around dough maturity stages of the crop, are necessary to get a clear picture of the leaf diseases in a variety. The correlation coefficients of grain yields with disease intensities recorded between 45 and 75 days were negative and significant in the case of CSV-3, a medium maturity variety (Fig. 14). The timings can accordingly be decided for early and late-maturing varieties. As far as possible, varieties to be compared should be measured at the same time, when they share the same weather conditions. However, timings of recording data can be suitably decided to represent a particular stage or stages at which the comparison is intended.

Evaluation of Varieties for Resistance

The "point of epidemic outbreak," i.e., 5%

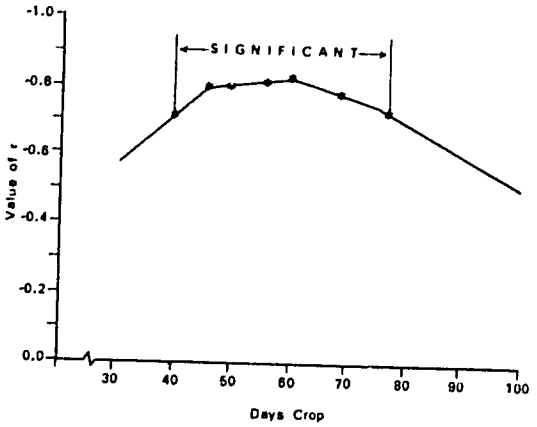


Figure 14. Significance of correlation coefficient of grain yields with disease intensities recorded at different timings. (Var. CSV-3) Mean of 3 years.

disease intensity, is taken into account for determining disease reactions. Varieties in which disease development is limited to this point during the crop season are designated as resistant (denoted by R). In determining further categories, results of studies on correlation and regression of grain yields on disease intensity carried out in different varieties and in different seasons were considered, and accordingly disease indices 2, 3 and 4, were marked. Varieties are evaluated with respect to the standard susceptible varieties. The varieties showing disease index 2 with respect to 3 of the standard susceptible are designated as moderately resistant, denoted by MR. In case of disease indices higher or lower than the standard susceptible variety the corresponding MR (moderately resistant) and R (resistant) points may be computed by multiplying the disease index by a factor 0.6 and 0.3 respectively. This is done so that valuable plant material is not rejected as susceptible (under unusually heavy epidemic force) and the escapes may not be selected as highly resistant under low epidemic force in the screening plots. It is desirable that disease indices, particularly beyond 4, are recorded in fractions to give an idea of maximum disease in the plot.

Source of Resistance

Sundaram (1972) enumerated the reaction of the world collection of sorghum to important diseases based on the results of field evaluation, mostly from India. Since then a large number of Indian × exotic derivative lines were tested for their yield potential and their reactions to important diseases of sorghum. A critical review of the data on reaction of the breeding material to important leaf diseases reported from various AICSIP centers (Anonymous 1971 to 1977) reveal that there is no consistency in the expression of resistance of a variety at different locations, particularly in case of rust and grey leaf spot. In general, higher ratings of these diseases were reported from Dharwar and Coimbatore in southern India. In the case of rust, varieties that showed a satisfactory degree of resistance at certain centers were susceptible or highly susceptible at Dharwar and Coimbatore. Whether it is due to variation in pathogen, the higher initial inoculum in the screening plots, or the environmental factors prevailing during the test period is not known. In spite of these difficulties some varieties, hybrids, and derivatives have shown a good degree of multiple and general resistance at most locations. Some of the recommended varieties, hybrids, Indian × exotic derivatives, and derivatives of multiple crosses of derivatives showing multiple resistance for a number of leaf disease or general resistance are CSV-4, CSV-5, CSH-5, CSH-6, SPV-1, SPV-3, SPV-4, SPV-5, SPV-9, SPV-23, SPV-29, SPV-33, SPV-34, SPV-35, SPV-59, SPV-69, and SPV-70. The reactions to important leaf diseases of the derivatives tested for three crop seasons at more than three locations in India, are given in Table 5.

A good degree of resistance to leaf diseases was observed in Indian × exotic derivatives (Table 6). The exotic parents IS-3687, IS-2954, and IS-3922 seems to have donated resistance to leaf diseases. In general the Indian parents showed susceptibility to leaf diseases (Anonymous 1971–1977). The exotic parents were from the USA. IS-3687 belongs to the Conspicuum group and IS-2954 and IS-3922 belong to the Caffrorum group; these were reported to be sources of resistance by Rachie (1970).

It is evident that the resistant derivatives are

Table 5. Disease reactions of Indian × Exotic derivatives in India.

Derivative	Anthracnose	Rust	Rough spot	Gray spot	Leaf blight	Zonate
SPV-1	MR	MR	MR	MR	R	MR
SPV-3	R	S	MR	MR	MR	MR
SPV-4	MR	S	MR	MR	MR	MR
SPV-5	R	MR	R	R	R	MR
SPV-9	R	MR	MR	S	R	MR
SPV-12	R	MR	R	MR	R	MR
SPV-13	R	S	MR	S	R	R
SPV-23	R	MR	MR	R	R	MR
SPV-29	MR	MR	MR	MR	R	MR
SPV-33	R	R	R	MR	R	R
SPV-34	R	R	MR	S	—	R
SPV-35	R	R	R	S	R	MR
SPV-59	MR	MR	MR	R	R	R
SPV-69	S	R	—	MR	MR	MR
SPV-70	MR	MR	—	MR	MR	R

R = Resistant
MR = Moderately resistant
— = Not tested

Table 6. Particulars of sorghum derivatives showing resistance to important leaf diseases in India.

Derivative	Pedigree	Origin
SPV-1	IS 3687 × Aispuri	AICSIP, Hyderabad
SPV-3	IS 3922 × Karad local	"
SPV-4	"	"
SPV-5	IS 3687 × Aispuri	"
SPV-9	Mutant derivative of GM1-5	AICSIP, UAS, Dharwar
SPV-12	IS 3922 × Aispuri	AICSIP, Hyderabad
SPV-13	IS 84 × Karad local	AICSIP, UAS, Dharwar
SPV-23	IS 2954 × BP-53	AICSIP, Hyderabad
SPV-29	CS 3687 × Vidisha 60-1	AICSIP, JNKVV, Indore
SPV-33	Mutant derivative of GM1 1-5	AICSIP, UAS, Dharwar
SPV-34	" "	"
SPV-35	Selection from Purdue base No. 954, source No. 166024 (S.B. 1066)	"
SPV-59	1 × 303	AICSIP, Hyderabad
SPV-69	IS 2954 × B.P.-53	"
SPV-70	"	"
Released Varieties		
CSV-4	IS 3675 × IS 3541	"
CSV-5	IS 3687 × Aispuri	"
Released Hybrids		
CSH-5	2077 A × CS 3541	"
CSH-6	2219 × CS 3541	"

predominantly from AICSIP Hyderabad and Dharwar.

Resistance Stability and Pathogen Variability

Not much information is available on these aspects. However, the released resistant varieties and hybrids i.e., CSV-4, CSV-5, and CSH-6 are maintaining their resistance in the field within tolerable limits of variations.

Suggested International Screening

The greatest single handicap to the development of resistant varieties has been the occurrence of physiologic races within species of most pathogens. This handicap is more so in breeding resistant varieties of sorghum for leaf diseases caused by several species of fungi.

The handicap can be overcome through international screening. International leaf disease nurseries should be located to represent different geographical and climatic zones in sorghum-growing areas. The locations within a zone should be selected at places where the disease or diseases in question are well established — so that it is easy to regulate epidemics in the screening plots.

Visual standards for measuring the disease intensity (Fig. 5–8) may be used to bring about uniformity in disease scoring. Colored figures would be more useful.

Evaluation of varieties with respect to a standard disease index is necessary, so that the data collected in different seasons and different locations in the international nurseries may be compared.

Screening should aim at general, horizontal and multiple resistance particularly in the case of these diseases that are favored by similar environmental conditions.

Control of Leaf Diseases by Means Other than Host-plant Resistance

Breeding for general resistance in sorghum is a long-term program. However, there are accepted improved varieties (i.e., CSV-4, CSV-5, and hybrids CSH-5 and CSH-6) that show a good degree of general resistance against important leaf diseases (Sharma and Jain 1977), but are moderately resistant or mildly susceptible to one or two leaf diseases under conditions of high epidemic force. Cultivar CSV-5 shows moderate resistance to rust at Indore but has been reported to be susceptible at Dharwar and Coimbatore. In the light of these variations a possibility of controlling leaf diseases by other means such as seed treatment with fungicides and by fungicidal sprays on the standing crop, were explored.

Seed Treatments

Seed treatment with fungicides helped improve the stand, and seedlings raised from treated seeds were healthier than those from untreated seeds. However, there is no direct evidence nor quantitative data to show that leaf diseases are controlled by seed treatment. The fungicidal seed treatment mainly protects seedlings from pathogens like *Fusarium* and *Curvularia*, which cause pre- and post-emergence rots. The result is an increased stand and more vigorous seedlings.

In India, seed treatment with Agroson GN, Thiram, and Captan gave satisfactory results (Table 7).

Application of Fungicides in the Standing Crop

Commonly available fungicides such as sulfur,

Table 7. Effect of fungicidal and insecticidal seed treatment on final stand and yield of sorghum variety 370. Seed sown @ 136 000 per ha.

Treatment	Final stand (1000 plants/ha)	Yield in (kg/ha)
Agroson GN 0.2%	53	3275
Captan	59	3437
Sulphur	46	2800
Thiram	60	3650
Agroson + Carbofuran	68	3475
Captan + Carbofuran	67	3687
Sulphur + Carbofuran	49	3275
Thiram	73	4225
Carbofuran	44	2962
Control	47	2787
C D at 5%	18.4	850

Captan, ziram, zineb, and Thiram were applied as foliar sprays (0.2%) to control anthracnose, rust, rough leaf spot, grey leaf spot, zonate leaf spot and sooty stripe on their respective susceptible host varieties. In general foliar applications of fungicides controlled the diseases (Table 8). The disease indices and grain yield varied with the fungicide. Zineb and ziram for the control of anthracnose and rust, wettable sulfur for the control of rough spot, and Captan and Thiram for the control of grey leaf spot, zonate leaf spot, and sooty stripe were superior to other fungicide, for in addition to lower values of disease indices, higher grain yields were recorded in these treatments.

If resistant varieties are not available, a judicious selection of variety with proper and timely spray of a suitable fungicide may be helpful.

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Table 8. Disease indices and grain yield recorded with different fungicidal sprays, after 35, 60, and 75 days (mean of 2 crop seasons).

Varieties	364(Local)		1188		555		CK 60 B		302	
Disease	Anthracnose		Rust		Rough Spot		Gray Spot		Zonate Spot	
Wettable Sulphur 0.2%	DI[a]	Y[b]	DI	Y	DI	Y	DI	Y	DI	Y
	3.04	2046	2.57	3995	2.40	4340	2.99	976	2.20	1693
Captan 0.2%	2.76	2181	2.43	3897	2.50	3941	2.10	1569	1.94	2100
Ziram 0.2%	2.15	2477	2.27	4660	2.33	3960	2.47	1188	1.99	1834
Zineb 0.2%	2.03	2597	2.55	4620	2.31	4010	2.64	1210	2.06	1760
Thiram 0.2%	2.55	2277	2.48	4015	2.46	4020	2.20	1505	2.05	2040
Control	3.42	1985	3.07	3440	3.07	3610	3.12	931	2.59	1400

a. DI = Disease Index
b. Y = Yield (Kg/ha)

Appendix Table 1. 1–5 disease-rating scale recommended by ICRISAT in ISLDN for various leaf diseases.

Rating value	area infected (%)	Description
1	–	No symptoms
2	–	Few scattered lesions/spots
3	Up to 25	Typical lesions developing on the leaves covering up to 25% leaf area
4	26 to 40	Coalescing spots covering about 26–40% leaf areas
5	40	Symptoms more severe, covering more than 40% leaf area

Appendix Table 2. 1–5 disease-index scale recommended by AICSIP for screening leaf diseases in Coordinated trials.

Rating value	area infected (%)	Description
1	–	Free from disease
2	Trace of 10	Slight symptoms
3	11–25	Moderate symptoms
4	26–50	Moderately severe
5	50	Very severe

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Sources of Resistance to Foliar Disease of Sorghum in the International Disease and Insect Nursery

R. A. Frederiksen and Denis Franklin*

Philosophy

The purpose of this paper is to describe the Texas program for improvement in leaf-disease resistance and the diseases involved in the program. Leaf-disease resistance has traditionally been secondary to smuts, downy mildew, virus diseases, and stalk rots, but with improvement in levels of resistance to these problems, more attention is being directed toward control of foliage pathogens. Currently we seek at least moderate levels of resistance to all foliar disease and, particularly, we seek to avoid high susceptibility.

Sorghums with diverse genetic backgrounds ranging from new introductions to partially converted or fully converted breeding lines are grown in extensive field nurseries in southern Texas. These nursery sites are selected in part because of their past histories of soilborne diseases and previous sorghum cropping. In these nurseries, mixtures of rust, grey leaf spot, zonate leaf spot and bacterial stripe frequently develop rapidly on maturing plants. The random occurrence of several different pathogens at periodic intervals in a naturally infected field nursery may be beset with difficulties. For example, only those plants with some levels of resistance to all the diseases stand out. This is an ideal situation, although severity of reaction to any one disease may interfere with evaluation of resistance to another. Frequently, if diseases of more mature hosts are present, reactions to earlier diseases are not observed. Leaf blight, which develops on younger plants, will be much less obvious on plants with serious grey leaf spot or zonate than on plants with higher levels of resistance to zonate. Consequently, leaf-disease reactions would be scored incorrectly. Mixtures of diseases also interfere positively. Zonate leaf spot develops more aggressively in fields with a higher incidence of downy mildew. Recently, we have learned of positive interactions with numerous other leaf diseases and downy mildew, including anthracnose and grey leaf spot. This may be due in part to lack of resistance expression in SDM-affected plants and, subsequently, increased availability of inoculum. In Texas, controlling downy mildew tends to reduce foliage problems. Because of these interactions of foliar diseases, one with another and with other diseases, it is better to evaluate host resistance to leaf diseases in environments where these problems develop one at a time.

Another factor interfering with interpretation of host resistance is the physiologic stage of the host at the time of appearance of inoculum. This has much to do with apparent levels of resistance. Generally, zonate leaf spot (*Gloeocercospora sorghi*) develops on plants approaching maturity and nearly always follows anthesis (exceptions include reactions of sorghum seedlings or ratooned sorghum). Sorghums differing in physiologic maturity appear to have different levels of resistance to diseases, when in fact there are no real differences in resistance *per se*. Grey leaf spot (*Cercospora sorghi*) also develops late. This, however, may be due more to the lack of early inoculum and less to host maturity. Leaf blight (*Exserohilum turcicum*) develops on young tissue. Mature, fully expanded leaves are much less likely than juvenile tissue to be invaded by this pathogen. Plant density may have either a positive or negative influence on disease development, as may plant height. Frequently, dense populations will have higher scores than will sparse plantings of the same material. Taller lines also often escape severe disease development when grown with dwarf varieties. But when grown with materials of comparable height, the levels of resistance are similar.

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Characters associated with leaf-disease resistance need attention. Tan plant color, thick waxy leaves, and erect leaves are but a few of these characters. Frequently, foliage diseases are much less apparent on tan plants because the discoloration in red and purple pigmented plants is more obvious. Nevertheless, there is a genuine belief that tan plants are more resistant (note N. G. P Rao's report on breeding programs in India). That belief is encouraged by the legitimate needs of the food industry for nonstained grains. Traits such as tan plant color need cautious evaluation. Possibly, different standards are necessary when evaluating their reactions (N. Zummo, personal communication).

These observations support a greater need for accuracy in controlling the environment, inoculum, and host maturity during evaluation for host resistance.

Methods

In the Texas program, we rely in most cases on natural inoculum. Unfortunately, natural inoculum may not be equally distributed in the nurseries. Leaf blight may spread from certain very susceptible cultivars such as sweet sudangrass and affect dwarf sorghums for as far as 4 to 5 m down the direction of the prevailing wind. Sometimes it may be useful to know which sorghums are extremely susceptible to some pathogens and omit them from the trial or place them at such a frequency that their distribution will minimize "position" effects in the nursery. It is necessary to plant in many different areas each year, because (a) each location will offer a different environment for disease expression, and (b) not all of the pathogens are found at any one location. Many times, a given location is excellent for the screening of a particular disease, whereas, in a more tropical region, many foliage diseases may be observed at a single location.

Ideally, all plants in a trial should receive equal amounts of inoculum at the same stage of plant growth and development in an environment favorable to disease development, while isolated from other pathogens. Artificial inoculation can be used in field nurseries to augment or control some of the variables in uncontrolled field plots. To date, we have successfully used the whorl-drop method for leaf blight, zonate leaf spot, and anthracnose. For each of these diseases, their respective pathogens are grown on sterile sorghum grain and dropped in the whorl of young seedlings. In that way, inoculum will remain in the whorl, often for weeks following deposition. The inoculum is present in young tissue, and there will nearly always be a favorable environment for initial infection. We prefer to inoculate only a fixed unit of plants at the beginning of the row. In this way, it may be possible to measure both the initial reaction to the inoculum and subsequent spread of the disease in the population of plants, thereby giving a measure of the generalized resistance of any entry.

It would appear that there are a few advantages to evaluation of sorghum diseases in populations of plants or among various sorghum entries within a field nursery. This, however, is not the case. To date, essentially all of the known sources of leaf-disease resistance have been found in open-field nurseries, often when there have been no inoculations or spreader rows. The technique of selecting individual plants within rows or whole rows from within families of related materials provides some confidence for selection of improved levels of resistance to any particular foliage disease or group of foliage diseases. Further testing tends to confirm the resistance of these particular entries. The justification for doing this in a disease nursery is supported by the need for agronomic adaptation and other cultural characteristics that may be evaluated concurrently with levels of leaf-disease resistance. Since there are always a variety of nurseries distributed specifically for agronomic adaptation, foliage disease evaluations made at these sites can confirm other observations. We recommend that, wherever possible, the types of foliage disease problems be anticipated and that they be encouraged, either through the use of inoculated spreader rows or inoculated individual plants within the nursery.

Reaction of Commercial Hybrids

In U.S. literature, it is frequently recommended that resistant hybrids be grown as a means of controlling a particular foliage disease in sorghum. The problem is that, to our knowledge, commercial hybrids have not been effectively scored for their reactions to foliage diseases.

During the past few years we have observed substantial differences among hybrids in their reaction to various foliage diseases in Texas, including rust, grey leaf spot, zonate leaf spot, and in experimental hybrids, leaf blight. Many of the early generation hybrids were resistant to bacterial stripe, and some parents involved in these hybrids were resistant to grey leaf spot.

Most sorghum is grown as hybrids; the F_1 reaction o disease resistance or heritability of the reaction of any leaf disease is therefore of tremendous value. With the advent of new sources of resistance to leaf diseases, attention focused on the heritability of this resistance. Consequently, we seek lines with resistance that is dominant or partially dominant rather than lines that have recessive genes for resistance, unless, of course, the same resistance is available in both inbreds. Occasionally there are rather striking interactions with certain parental combinations, and it is almost impossible at times to predict the reaction of hybrids. At other times, the reactions of the hybrids are very predictable. The F_1 heritability reactions of common foliar diseases are given in Table 1.

Part of our philosophy is to maintain adequate levels of as broad-based a resistance as possible, not only in a horizontal sense but in the classical sense of generalized resistance to a variety of different diseases. First, we are satisfied when we can reduce disease severity below the economic threshold. Often foliage diseases, under commercial conditions, occur late in the development of the crop, and (except in ratooned crops) partial resistance is adequate. This is particularly true for resistance to rust. Second, a cultivar would be unacceptable to us if it was susceptible to one of the common foliage diseases though resistant to all others. Some superior sources of resistance to the more common foliar diseases in Texas are summarized in Table 2. Foliar anthracnose is not listed, because under our conditions large numbers of entries have exceedingly high levels of resistance.

Table 1. F_1 heritability reactions of common foliar diseases.

Disease	F_1 Resistance[a]
Anthracnose	dominant
Bacterial Stripe	dominant
Grey leaf spot	recessive
Leaf blight	dominant
Rust	dominant
Zonate leaf spot	recessive

a. Based on naturally occurring outcrosses from resistant breeding lines, as determined by D. T. Rosenow.

Table 2. Sources of foliar-disease resistance from entries deployed in the Texas A & M University disease and insect nurseries.

IS Number or Derivation	Designation or Sorghum Conversion Number
Zonate Leaf Spot	
IS 2816 C	SC 120-14
IS 3758 der.	SC 326-6
IS 3955 C	SC 242-14
IS 12610 der.	SC 110-9
IS 12610 der.	TAM 428
IS 12664 der.	SC 173-9
IS 12666 der.	TAM 2566
IS 2816 × IS 410	(SC 326-6 × SC 103-12)
Leaf Blight	
IS 1335 C	SC 418-14
IS 2462 der.	SC 325-12
IS 3071 C	SC 237-14
IS 3574 C	SC 239-14
IS 3758 der.	SC 326-6
IS 6882 C	SC 320-14
IS 7064 der.	SC 426-12
IS 8337 der.	SC 574-6
IS 7254 C	
IS 12594 der.	SC 89-9
IS 12658 C	SC 167-14
–	Tx 430
–	NSA 935-6
Grey Leaf Spot	
IS 2816 C	SC 120-14
IS 3063 der.	SC 333-6
IS 3574 C	SC 239-14
IS 3758 der.	SC 326-6
IS 3955 C	SC 242-14
IS 5843 der.	SC 448-6
IS 2816 der × IS 410	(SC 120-6 × Tx 7000)
IS 2930 × IS 3922	77CSI

Continued.

Table 2. *Continued*

IS Number or Derivation	Designation or Sorghum Conversion Number
IS 12610 der. × IS 2816 der.	SC 110-9 × SC 120-6
–	NSA 935-6
–	TP2-76-3
–	TP4-2
Bacterial Stripe	
IS 2579 C	SC 423-14
IS 2816 C	SC 120-14
IS 3758 der.	SC 326-6
IS 3955 C	SC 242-14
IS 12608 C	SC 108-14
IS 12610 der.	SC 110-9
IS 2816 der. × IS 410	SC 120-6 × Tx 7000
IS 17459 × IS 12625	SC 599-6 × SC 134-6
	NSA 440-12
	BTx 3197
Rust	
IS 12610 C	SC 110-
IS 2816 C	SC 120-
IS 12539 C	SC 19
IS 12564 C	SC 48
IS 12666 C	SC 175
IS 530 C	SC 229
IS 3758	SC 326-6
IS 7809 C	SC 402
IS 1335 C	SC 418
IS 5530 C	SC 457
IS 17459	SC 599-6
–	NSA 935-6
–	TAM 2566

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The International Sorghum Leaf Disease Nursery

R. J. Williams, K. N. Rao, and S. R. S. Dange*

The International Sorghum Leaf Disease Nursery (ISLDN) program was initiated in 1976 with the following objectives:

1. To identify sources of stable resistance to the important sorghum leaf diseases;
2. To obtain information on the variability of the leaf disease pathogens; and
3. To promote the development of a communicating cooperating international network of scientists interested in sorghum leaf diseases.

In the 3 years 1976, 1977, and 1978, cooperators from 13 countries in Asia and Africa (Table 1) have participated in the ISLDN program.

There are several important leaf diseases and all do not occur at each location. The basic requirement of cooperators is that they should be able to expose the ISLDN test entries to sufficient pressure of at least one leaf disease to adequately test the reactions of the entries.

Selection of ISLDN Test Entries

In the 3 years of operation of the nursery, the entries consisted of sorghum lines reported to be resistant to one or more leaf diseases in various national programs, some lines identified as promising in germplasm evaluation at ICRISAT, and lines reported as susceptible to one or more leaf diseases to act as indicators of

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Table 1. Cooperators and locations in the International Sorghum Leaf Disease Nursery Program in 1976, 1977, and 1978.

Cooperator	Location	Year(s)
J. C. Girard	Nioro du Rip, Senegal	1977
J. A. Frowd	Saria and Kamboinse, Upper Volta	1977 and 1978
O. Sidibe	Tarna, Niger	1977 and 1978
S. O. Okiror	Samaru, Nigeria	1976
N. V. Sundaram	Samaru, Nigeria	1977 and 1978
B. D. A. Beck	Shire Valley, Malawi	1978
S. Z. Mukuru	Ilonga, Tanzania	1978
B. van Arkel and M. F. Vis	Nakuru, Kenya	1976, 1977 and 1978
Bhrane Gebrekidan and colleagues	Alemaya and Arsi Negele, Ethiopia	1977 and 1978
H. C. Sharma and N. K. Jain	Indore, India	1976, 1977 and 1978
S. M. Naik and K. Mathur	Udaipur, India	1977 and 1978
S. B. Mathur and K. S. Dhanraj	Delhi, India	1977 and 1978
T. B. Garud	Parbhani, India	1977 and 1978
H. L. Chauhan	Navsari, India	1978
K. N. Rao and S. R. S. Dange	ICRISAT Center, India	1976 1978
V. Sethuraman and K. Ramanujam	Kovilpatti, India	1976, 1977 and 1978
C. Pannabokke	Maha Illuppallama, Sri Lanka	1978
S. F. Hussan	Islamabad, Pakistan	1977
A. and S. Patanothai	Khon Kaen, Thailand	1977 and 1978

disease pressure. Entries were requested from sorghum workers, provided that the lines had clearly shown a good level of resistance to one or more leaf diseases at the home location.

Operation of the ISLDN Program

The seed of test entries is assembled and multiplied at ICRISAT Center. All cooperators receive seed from the same seed lot for each entry. This is important if erroneous information on pathogen variability is to be avoided.

A set of entries is sent to each cooperator with a book that includes information on the objectives of the trial; suggestions on planting, fertilization, inoculum provision, time and method of scoring; and data record sheets for climatic data and plant reaction data.

Cooperators are requested to return one copy of the data sheets to ICRISAT as soon as possible after completion of the trial. A report, containing the data from all cooperators with discussion of the important aspects of the results, is distributed to all the cooperators, in April of the following year.

ISLDN Results: 1976, 1977, and 1978

1976 Results

In this first year of operation we received results from three Indian locations — Indore for anthracnose, grey leaf spot, and zonate leaf spot; ICRISAT Center for anthracnose, rust, grey leaf spot, and rough leaf spot; and Kovilpatti for leaf blight, anthracnose, zonate leaf spot, and sooty stripe — and from two African locations — Nakuru, Kenya, for leaf blight, rust and bacterial leaf stripe, and Samaru, Nigeria, for anthracnose and grey leaf spot.

At Samaru, Indore, ICRISAT Center, and Kovilpatti

Test entries were evaluated for the severity of seven leaf diseases on a 1 to 5 scale where 1 indicates no disease and 5 indicates severe disease. Cooperators were given detailed descriptive scoring scales for each disease. Scores of 1 or 2 are regarded as resistant; a score of 3 is intermediate; and 4 and 5 are susceptible and highly susceptible respectively.

In Table 2 the numbers of lines with a maximum score of 1 or 2 across the four locations is given for each disease. Rough leaf spot was the least severe (52 resistant lines) and anthracnose was the most severe (2 resistant lines).

Entries resistant to more than two diseases across locations are listed, with the diseases to which they are resistant, in Table 3. One entry, IS-7254, was apparently resistant to all seven diseases at all four locations, and IS-4277 was apparently resistant to five diseases at all locations. Eight entries were resistant to four of the seven diseases and 17 entries were resistant to three of the seven diseases.

At Nakuru, Kenya

The results from Nakuru are given separately, as only 38 of the 157 sorghum lines tested there were included in the ISLDN. Dr. van Arkle requested lines before the ISLDN had been finalized, for a March planting, and Nakuru, at 1930 m above sea level, has a distinctly different environment from any of the other locations. Temperatures are low, particularly at night, and at Nakuru most of the sorghums take about 6 months to produce mature grain.

Table 2. The number of entries in the 1976 International Sorghum Leaf Disease Nursery with maximum scores of 1 or 2[a] for each of seven diseases across four locations in India and West Africa.

Disease	Number of entries[b]	
	Maximum score 1	Maximum score 2
Leaf blight	2	26
Grey leaf spot	0	5
Anthracnose	0	2
Zonate leaf spot	3	18
Rust	5	16
Rough leaf spot	12	40
Sooty stripe	7	17

a. On a 1 to 5 scale where 1 is absence of the disease, 2 only slight incidence, and 5 is severely diseased.
b. There were 57 entries in the 1976 ISLDN.

Table 3. Identity of entries apparently resistant to more than two diseases in tests at four locations in the 1976 International Sorghum Leaf Disease Nursery.

Entry	Diseases[a] to which the entry is apparently resistant[b]
IS-7254	LB, GLS, Anth, ZLS, Rust, RLS, SS
IS-4277	LB, ZLS, Rust, RLS, SS
IS-2419	LB, Rust, RLS, SS
IS-7322	LB, Rust, RLS, SS
IS-8171	LB, ZLS, Rust, RLS
IS-10325	LB, Rust, RLS, SS
Bulk Y×Entomology	LB, Rust, RLS, SS
IS-2223	LB, ZLS, Rust, RLS
TAM-428	LB, ZLS, Rust, RLS
IS-2550	LB, ZLS, RLS, SS
IS-4951	LB, ZLS, RLS
IS-6266	LB, ZLS, RLS
Bulk Y×Pickett	LB, ZLS, RLS
SC-110-14	LB, ZLS, RLS
IS-10180	LB, RLS, SS
IS-10264	LB, RLS, SS
IS-10564	LB, RLS, SS
IS-10568	LB, RLS, SS
IS-10870	LB, RLS, SS
IS-2042	LB, RLS, SS
IS 7188	LB, RLS, SS
PJ 739	GLS, ZLS, SS
IS 10740	GLS, Rust, RLS
Bulk Y×GPR-165	ZLS, Rust, RLS
IS 5345	Rust, RLS, SS
IS 4150	Rust, RLS, SS
IS 101	Rust, RLS, SS

a. LB – leaf blight; GLS – grey leaf spot; Anth – anthracnose; ZLS – zonate leaf spot; RLS – rough leaf spot; SS – sooty stripe.
b. With maximum scores of 1 or 2 on a 1–5 severity scale.

Of the 38 ISLDN lines planted at Nakuru, only one (IS-10242) was resistant to leaf blight and 18 were classed as intermediate. Five lines were free from rust and four lines were given a score of 2. Bacterial stripe was absent from 7 lines and occurred only slightly on 12 lines. Entries free from at least one disease are listed in Table 4. At the time the disease incidence notes were taken, there was great variability between entries in the physiologic stage of development. Some lines were only ankle high and completely vegetative, others were in the boot-leaf stage and at various stages of flowering and grain filling. This variability makes difficult the comparison of disease reaction between lines, and is a major problem in any program dealing with disease reactions in variable duration materials. Care must be taken in classing lines as resistant if they show little or no disease and are still in the vegetative or early flowering stages. In such a situation lines should be evaluated at specific physiological stages and not at a given chronological point of the crop.

1977 Results

There were two groups of entries under the ISLDN program in 1977. For those locations where main-season planting is carried out during the January through March period, an Early-Season ISLDN (ES-ISLDN) consisting of 25 entries was provided. For the majority of the cooperative locations in India and in West Africa, planting takes place during the June through July period and these locations were provided with a Main-Season (MS-ISLDN) 30-entry trial, 20 of which were also included in the ES-ISLDN. The results of these two groups of entries are reported separately in order to minimize confusion in comparisons.

Early-Season ISLDN

The ES-ISLDN was tested in Thailand, Ethiopia, and Kenya. Because of vastly different environments, different diseases encountered, and different screening procedures used, the results from each country are presented separately.

THAILAND—KHON KAEN UNIVERSITY. Grey leaf spot was the only leaf disease to occur with sufficient severity to meaningfully differentiate entry reactions (Table 5). IS-7254 was the only entry free from the disease. Six entries had no more than reaction 2, ten entries had a maximum score of 3, and seven entries had maximum scores of 4 or 5. A possible relationship between date of flowering and disease incidence is indicated, for the best six entries all flowered in more than 70 days, whereas five of the worst six entries flowered in less than 70 days. The best entry, IS-7254, was the latest to

Table 4. Disease reaction and stage of physiologic development of ISLDN entries at Nakuru, Kenya, free from at least one of the three diseases.

Entry	Physiologic stage	leaf blight	rust	bacterial stripe
IS 10242	Vegetative	1	2	2
IS 2384	Vegetative	2–3	1	?
IS 2550	Flowering	5	1	1
IS 5345	Vegetative	4–5	1	?
IS 7188	Vegetative	2–3	1	?
Bulk Y×GPR-165	Flowering	3	1	?
IS 4951	Vegetative	4	2–3	1
IS 10326	Vegetative	3	2–3	1
R 46943	Flowering	2–3	2–3	1
R 46945	Flowering	2–3	2–3	1
PR 50070	Flowering	5	2–3	1
IS 555	Vegetative	2–3	2	1

Table 5. Grey leaf spot reactions[a] of 23 Sorghum entries in the 1973 early season ISLDN at Khon Kaen, Thailand.

Entry	Rep. I	Rep. II	DTF[b]
IS-7254	1	1	106
IS-7322	2	2	96
BY × Entol.	2	2	71
IS-4951	2	2	81
IS-10180	2	2	71
IS-5345	2	2	94
TAM-428	3	2	71
IS-2550	3	2	69
IS-6266	3	2	92
BY × GPR 165	3	2	70
IS-2223	3	3	94
IS-8171	3	3	66
IS-4150	3	3	76
IS-10264	3	3	67
IS-10564	3	3	66
IS-856	3	3	70
IS-4277	4	3	66
BY × Pickett	4	3	69
SC-110-14	4	3	70
IS-643	3	4	64
H-112	4	3	68
CSV-2	4	3	66
IS-73	5	4	62

a. 1 = Free from symptoms. 5 = Severe symptoms.
b. Days to 50% flowering.

flower (106 days), whereas the worst entry, IS-73, was the earliest to flower (62 days).

ETHIOPIA—ARSI NEGELE AND ALEMAYA. In Ethiopia the entries were scored for overall leaf disease severity, sterility, and agronomic desirability. Results are summarized in Table 6. Entry IS-10264 had the least leaf disease, little sterility, and was moderately desirable. Other entries with only slight to moderate leaf disease were BY× GPR 165, IS-2550, IS-4150, IS-2223, IS-7254, and BY× Entol. Of these latter six entries, IS-7254 had the best combination of other characters with no sterility and 2/2.5 for desirability. Thirteen of the 23 entries had at least a 4 or 5 rating for leaf diseases. It will be interesting to determine the particular leaf disease involved. Data on flowering dates were not provided.

KENYA—LANET AND BUSIA. These are two contrasting locations in Kenya. Lanet is situated about 1°S of the Equator at approximately 1890 m elevation, whereas Busia is located approximately 1°N of the Equator at about 1200 m elevation. Mean days to 50% flowering at Lanet was 120, while at Busia mean days to 50% flowering was only 79. Leaf blight and rust occurred severely at both locations, while bacterial stripe occurred only at Lanet and grey leaf spot only at Busia. In addition to these leaf diseases, grain mold and ergot severity were

Table 6. Overall leaf disease severity ratings, sterility ratings and general agronomic desirability ratings of 23 entries in the 1977 ES-ISLDN at two locations in Ethiopia.

Entry	Overall leaf disease[a]		Sterility[a]		Desirability[a]	
	1	2	1	2	1	2
IS-10264	1	1	1	0	3	2.5
IS-7254	2	2.5	0	0	2	2.5
BY × GPR 165	1	2	3	3	5	5
IS-2550	1.5	2	2	3	5	5
IS-4150	1.5	2	0	0	2.5	5
IS-2223	2	2	2	1	4	4
BY × Entol	2.5	2	2	0	4	2
IS 10564	2.5	3	1	0	3	5
IS 10180	2.5	3	1	2	2.5	4
TAM 28	2.5	4	0	0	2.5	3
BY × Pickett	3	3	2	2	5	5
IS-4277	3	4	0	0	5	4
IS-4951	3	4	0	3	4	5
IS-5345	3.5	4	–	0	5	3
IS-6266	3	5	1	1	4	4
IS-7322	3	5	0	0	5	3
CSV-2	4	5	4	5	4	5
IS-8171	4	5	1	0	4	4
SC-110-14	4	4	3	1	5	5
H-112	5	5	4	4	5	5
IS-73	5	5	3	1	5	5
IS-643	5	5	2	2	5	5
IS-856	5	5	2	2	4	4

a. 0 = none 1 = Very little leaf disease, very little sterility and most desirable.
5 = Severe leaf disease, almost complete sterility and least desirable.

also recorded; data are summarized in Table 7.

No entry was rust free, and at Busia no entry recorded a score less than 3 for this disease. Six entries recorded a maximum score of 2 for rust at Lanet. Leaf blight was moderate to severe on almost all entries at both locations. Four entries scored 2 for grey leaf spot at Busia and only two entries had a maximum score of 2 for bacterial stripe at Lanet. Entries IS-7254, IS-4951, and IS-5345 had low grain mold incidence, and IS-5345 and IS-4150 were free from ergot.

The environment and disease pressures at Lanet and Busia are quite different from those of most other sites in the ISLDN, particularly in latitude, elevation, and associated parameters. The low night temperatures at Lanet delay flowering, and only at this location is bacterial stripe a problem. In view of the severe reactions to several diseases, none of these entries appeared promising for these locations.

Main-Season ISLDN

In order to provide a clear picture of which diseases occurred at which locations with sufficient severity to provide meaningful entry reactions, the numbers of entries in reaction categories 1–5 for seven leaf diseases at eight locations are given in Table 8. Unless at least one entry was in at least category 4 for a particular disease at a particular location, the location is considered as not having provided sufficient pressure for that disease. The disease-location relationships based on this criterion are summarized in Table 9. Samaru in Nigeria provided sufficient pressure for the

Table 7. Flowering data and disease severity records of 23 1977 ES-ISLDN entries at Lanet (L) and Busia (B) in Kenya.

	DTF[a]		Rust		Leaf blight		GLS	B. stripe	GM[b]	Ergot[b]
	L	B	L	B	L	B	B	L		
IS-7254	152	110	3	3	4	3	3	3	2	3
IS-4277	108	77	3	4	5	3	3	3	5	3
IS-7322	–	84	3	3	4	3	2	4	4	5
IS-8171	125	77	3	3	4	3	4	4	5	4
BY × Entol	152	83	3	3	4	4	2	–[c]	5	3
IS-2223	118	84	4	3	5	3	–	2	5	4
TAM-428	125	85	2	4	5	4	3	–	5	4
IS-2550	96	86	3	5	5	4	3	–	5	3
IS-4951	–	85	3	5	4	3	2	3	2	3
IS-6266	147	76	2	4	4	3	3	5	4	4
BY × Pickett	105	81	2	5	5	3	2	4	4	4
SC-110-14	105	81	2	4	5	4	3	4	4	2
IS-10180	118	84	3	3	5	3	4	4	5	3
BY × GPR-165	136	81	4	3	5	4	–	–	5	4
IS-5345	–	81	4	5	4	3	2	–	2	1
IS-4150	147	82	3	3	5	3	–	–	4	1
IS-10264	96	83	4	3	5	3	3	3	5	3
IS-10564	108	84	4	4	4	3	3	5	5	3
IS-643	121	82	2	3	5	3	3	4	5	3
H-112	125	79	2	–	5	–	–	3	4	3
IS-73	96	81	3	5	5	2	3	3	5	5
CSV-2	–	81	3	3	5	3	4	2	5	5
IS-856	133	84	4	5	4	3	3	4	5	4

a. Days to 50% flowering.
b. Maximum score from two locations.
c. No data provided.

greatest number of diseases (five) and sooty stripe was the disease occurring with sufficient pressure at most locations (six).

The reactions of the test entries to the seven diseases at those locations with sufficient pressure are presented in Tables 10 to 14.

SOOTY STRIPE. Six entries had maximum reactions of 1 or 2 at all locations; 12 entries had a maximum score of 3; 7 entries had a maximum score of 4; and 2 entries had a maximum score of 5. Entry IS-10262 was the best entry, free from symptoms at five of the six locations and a score of 2 at the sixth (Table 10).

ANTHRACNOSE. Only one entry, BY × Entomology, remained within the 1-2 categories across all locations, and three entries, IS-7322, 555, and BY × Pickett, had maximum scores of 3. There appear to be large differences among locations, e.g., BY × GPR-165-3 had scores of 1 or 2 at all locations except Delhi where it was given a 4. Conversely the highly susceptible H 112 had scores of 4 or 5 at all locations except Delhi, where it surprisingly received a 2. Some entries were susceptible at Indian locations but were free at the African locations for example, IS-2550, CSV-2, SC-110-14, TAM-428, and IS-643. These apparent differential reactions need further investigation (Table 11).

LEAF BLIGHT. There are four distinct groups of entries for leaf blight reactions. Group 1 entries (IS-3390, CS-3541, SC-110-14, IS-4277, and

Table 8. The number of entries in reaction categories 1–5[a] for seven leaf diseases at eight locations in the 1977 ISLDN.

		India					West Africa		
Disease	Cat.	Kovilpatti	Parbhani	Indore	Udaipur	Delhi	Samaru	Kamboinse	Niorodu Rip
Leaf blight	1	6	28	28	27	8	11	11	28
	2	12	0	0	0	12	8	3	0
	3	7	0	0	0	6	6	10	0
	4	3	0	0	0	2	1	3	0
	5	0	0	0	0	0	2	1	0
Grey Leaf Spot	1	8	28	21	19	0	0	23	25
	2	13	0	5	4	5	0	5	3
	3	0	0	2	4	11	1	0	0
	4	0	0	0	0	10	2	0	0
	5	0	0	0	0	2	25	0	0
Anthracnose	1	2	28	5	26	10	14	8	23
	2	17	0	7	1	2	3	8	4
	3	5	0	5	0	5	8	6	1
	4	3	0	6	0	11	3	5	0
	5	2	0	5	0	0	0	1	0
Zonate Leaf Spot	1	18	28	14	22	21	27	21	3
	2	6	0	7	5	6	0	5	14
	3	4	0	4	0	1	1	2	9
	4	1	0	3	0	0	0	0	1
	5	0	0	0	0	0	0	0	1
Rust	1	29	28	10	12	28	28	28	28
	2	0	0	7	7	0	0	0	0
	3	0	0	8	2	0	0	0	0
	4	0	0	1	4	0	0	0	0
	5	0	0	2	0	0	0	0	0
Rough Leaf Spot	1	18	26	27	27	18	24	25	28
	2	8	1	1	0	9	2	3	0
	3	3	1	0	0	1	1	0	0
	4	0	0	0	0	0	1	0	0
	5	0	0	0	0	0	0	0	0
Sooty stripe	1	5	18	27	27	24	9	18	4
	2	16	5	1	0	1	4	7	8
	3	6	3	0	0	2	10	2	13
	4	1	2	0	0	1	4	1	3
	5	1	0	0	0	0	1	0	0

a. 1 = Free from symptoms. 5 = Severe symptoms.

IS-223) had reactions 1 or 2 maximum at all four locations. Group 11 entry IS-10240 was free from blight at the two Indian locations but had moderately severe blight at the two West African locations. Conversely, Group III entries (IS-10262, IS-10264, R-55 DX, and Sel. 512) were free from blight at the two West African locations and had light to severe blight at the Indian

Table 9. Locations with sufficient (+) or insufficient (–) pressure for determining reactions of entries to seven leaf diseases[a] in the 1977 Main Season-ISLDN.

Location	RLS	RUST	GLS	ZLS	BLIGHT	ANTH	SOOTY	TOTAL +ve
Udaipur	–	+	–	–	–	–	–	1
Parbhani	–	–	–	–	–	–	+	1
Nioro du Rip	–	–	–	+	–	–	+	2
Indore	–	+	–	+	–	+	–	3
Kamboinse	–	–	–	–	+	+	+	3
Kovilpatti	–	–	–	+	+	+	+	4
Delhi	–	–	+	–	+	+	+	4
Samaru	+	–	+	–	+	+	+	5
Total +ve	1	2	2	3	4	5	6	

a. rough leaf spot; rust; grey leaf spot; zonate leaf spot; leaf blight; anthracnose; sooty stripe.

Table 10. Sooty stripe severity reactions[a] of 28 entries at six locations in the 1977 ISLDN.

	Location[b]							
Entry	1	2	3	4	5	6	Total	Range
IS 10262	2	1	1	1	1	1	7	1–2
IS 10240	2	2	1	1	1	1	8	1–2
IS 73	1	2	2	1	1	2	9	1–2
IS 3390	2	1	1	1	2	2	9	1–2
IS 7322	2	1	1	2	1	2	9	1–2
IS 4150	2	1	1	2	2	2	10	1–2
BY × GPR 165-2	1	1	1	3	1	1	8	1–3
TAM 428	2	1	1	1	1	3	9	1–3
IS 856	1	1	1	3	1	2	9	1–3
Sel. 512	2	1	1	2	1	3	10	1–3
CS 3541	1	1	1	3	1	3	10	1–3
IS 2223	1	1	1	4	2	1	10	1–4
BY × GPR 165-1	3	1	1	1	2	3	11	1–3
BY × Entol	2	1	1	4	1	2	11	1–4
BY × GPR 165-3	2	3	1	3	1	2	12	1–3
By × Pickett	3	1	1	3	1	3	12	1–3
R 55 DX	3	1	1	3	1	3	12	1–3
IS 7254	2	1	1	3	2	3	12	1–3
SC-110-14	3	1	1	2	2	3	12	1–3
IS 2550	2	1	1	4	1	3	12	1–4
IS 8171	2	2	1	3	1	4	13	1–4
IS 4277	2	2	1	3	3	3	14	1–3
IS 10264	2	3	3	1	3	2	14	1–3
IS 6838	3	1	3	4	1	3	14	1–4
IS 643	4	4	1	1	1	4	15	1–4
CSV-2	2	3	4	1	2	3	15	1–4
H-112	5	4	1	3	1	4	18	1–5
555	3	2	1	5	4	3	18	1–5

a. The greater of two reps. 1 = No symptoms. 5 = Severe symptoms.
b. 1. Kovilpatti; 2. Parbhani; 3. Delhi; 4. Samaru; 5. Kamboinse; 6. Nioro du Rip.

Table 11. Anthracnose severity reactions[a] of 28 entries at five locations in the 1977 ISLDN.

Entry	Location[b] 1	2	3	4	5	Total	Range
By × Entol.	2	1	1	1	2	77	1–2
IS-7322	2	1	3	1	2	9	1–3
555	2	3	1	2	1	9	1–3
BY × Pickett	2	2	3	3	2	12	1–3
BY × GPR 165-3	1	2	4	1	2	10	1–4
BY × GPR 165-1	3	1	1	1	4	10	1–4
IS-2550	2	2	4	1	1	10	1–4
Sel. 512	2	1	2	5	1	11	1–5
IS-4277	2	5	1	2	1	11	1–5
CS-3541	1	4	4	2	1	12	1–4
IS-8171	2	2	4	1	3	12	1–4
TAM 428	4	3	3	1	1	12	1–4
SC-110-14	4	2	4	1	1	12	1–4
IS-10264	2	4	1	3	2	12	1–4
CSV-2	3	4	3	1	1	12	1–4
IS-856	2	1	4	1	4	12	1–4
IS-643	5	2	3	1	1	12	1–5
R 55 DX	2	3	1	4	3	13	1–4
IS-7254	2	1	4	3	3	13	1–4
IS-3390	2	5	1	3	2	13	1–5
IS-10262	2	5	2	1	4	14	1–5
BY × GPR 165-2	2	5	1	3	3	14	1–5
IS-2223	3	5	1	3	2	14	1–5
IS-10240	2	5	1	3	4	15	1–5
IS-6838	5	2	4	1	3	15	1–5
IS-4150	3	4	4	4	2	17	1–4
IS-73	2	4	4	3	4	17	1–4
H-112	4	4	2	4	5	19	1–5

a. The greater of two reps. 1 = No symptoms; 5 = Severe symptoms.
b. 1. Kovilpatti; 2. Indore; 3. Delhi; 4. Samaru; 5. Kamboinse.

locations. In Group IV there was no distinct Indian vs. West African differential reaction and 10 entries in this group had maximum scores of 3, five entries maximum scores of 4, and three entries maximum scores of 5 (Table 12).

ZONATE LEAF SPOT. Three entries (CS-3541, IS-4150 and 555) were free from zonate leaf spot (ZLS) at the three locations. Ten entries had maximum scores of 2, 11 entries had maximum scores of 3, 3 entries had maximum scores of 4, and the most susceptible entry was IS-73 with scores of 4, 4, and 5 at Kovilpatti, Indore, and Nioro du Rip, respectively. Six entries free from ZLS at the two Indian locations had scores of 2 or 3 at Nioro du Rip (Table 13).

GREY LEAF SPOT. At Delhi in India and Samaru in Nigeria no entry was free from grey leaf spot. At Delhi five entries had a maximum score of 2. At Samaru the minimum score was 3 (one entry), two entries recorded 4, and the remainder 5. This large scale severe grey leaf spot reaction at Samaru is surprising and needs to be thoroughly investigated in subsequent trials (Table 14).

RUST. Udaipur was the only location in the

Table 12. Leaf blight severity reactions[a] of 28 entries at four locations in the 1977 ISLDN.

Entry	Location[b] 1	2	3	4	Total	Range
Group I						
IS-3390	1	1	2	1	5	1–2
IS-7254	1	1	2	1	5	1–2
SC-110-14	1	2	1	1	5	1–2
IS-4277	2	1	2	1	6	1–2
IS-2223	2	2	1	2	7	1–2
Group II						
IS 10240	1	1	3	3	8	1–3
Group III						
IS-10262	2	2	1	1	6	1–2
IS-10264	3	3	1	1	8	1–3
R 55 DX	4	3	1	1	9	1–4
Sel. 512	3	4	1	1	9	1–4
Group IV						
IS-8171	3	1	1	1	6	1–3
CSV-2	2	1	1	3	7	1–3
555	1	2	3	1	7	1–3
BY × GPR 165-1	1	3	1	2	7	1–3
IS-4150	2	2	3	1	8	1–3
BY × GPR 165-3	3	2	1	3	9	1–3
CS 3541	2	2	3	3	10	2–3
IS-6838	3	2	2	3	10	2–3
IS-7322	2	2	2	4	10	2–4
IS-73	2	1	5	2	10	1–5
BY × GPR 165-2	2	4	1	4	11	1–4
BY × Pickett	2	3	3	3	11	2–3
IS 643	3	3	2	3	11	2–3
BY × Entol.	2	2	4	3	11	1–4
TAM-428	4	2	2	3	11	2–4
IS-656	2	1	5	3	11	1–5
IS-2550	3	3	3	4	13	3–4
H-112	4	2	2	5	13	2–5

a. The greater of two reps. 1 = No symptoms; 5 = Severe symptoms.
b. Kovilpatti; Delhi; Samaru; Kamboinse.

ISLDN with sufficient rust pressure to evaluate the entries. Here 12 entries were free from rust, 7 entries had a maximum score of 2, 2 entries had maximum scores of 3, 4 entries scored 4, and 2 entries scored 5 (Table 14).

ROUGH LEAF SPOT. At Samaru, 24 entries were free from rough leaf spot, 2 entries scored 2, 1 entry scored 3, and 1 entry scored 4 (Table 14).

OVERALL REACTIONS. In order to evaluate entries across the seven diseases, the maximum score for each entry-disease combination is given in Table 15. Entry IS-7254 is the best entry with two 1s, two 2s, two 3s, and one 4. Another five entries (IS-10240, IS-10262, IS-4150, IS-2223,

Table 13. Zonate leaf spot severity reactions[a] of 28 entries at three locations in the 1977 ISLDN.

Entry	Kovilpatti	Indore	Nioro du Rip	Range
CS-3541	1	1	1	1
IS-4150	1	1	1	1
555	1	1	1	1
IS-7254	1	1	2	1–2
IS-2223	1	1	2	1–2
IS-2550	1	1	2	1–2
R 55 DX	1	1	3	1–3
Sel. 512	1	1	3	1–3
IS-7322	1	1	3	1–3
BY x GPR 165-2	1	2	2	1–2
IS-8171	1	2	2	1–2
BY x Entol.	1	2	2	1–2
TAM-428	1	2	2	1–2
IS-10264	1	2	2	1–2
IS-643	2	1	2	1–2
IS-10240	2	2	2	2
SC-110-14	1	3	2	1–3
BY x GPR 165-1	2	1	3	1–3
H-112	3	1	2	1–3
CSV-2	1	2	3	1–3
IS-3390	3	1	3	1–3
BY x Pickett	3	1	3	1–3
BY x GPR 165-3	1	4	2	1–4
IS-10262	2	3	3	2–3
IS-4277	3	3	2	2–3
IS-856	2	4	3	2–4
IS-6838	2	3	4	2–4
IS-73	4	4	5	4–5

a. Greater of two reps. 1 = No symptoms; 5 = Severe symptoms.

and BY x Entol.) had maximum scores of 1 or 2 to four of the seven diseases.

The locations of the 1977 ISLDN were diverse in distance, elevation, latitude, and disease pressure. The high-elevation locations in Ethiopia and Kenya require a different set of entries compared with the locations in India and West Africa, and in subsequent years a high altitude trial should be assembled for the Ethiopian and Kenyan locations.

In India and West Africa sooty stripe, anthracnose, blight, and zonate leaf spot were the most common diseases and "hot-spot" locations were identified. The apparent differential reactions for blight and anthracnose need further examination.

The entry IS-7254 performed well at most locations including Khon Kaen and the Ethiopian locations, and was also the best entry in the 1976 ISLDN. However, it was somewhat later flowering than most of the entries (Table 12). On the other hand IS-10240 and IS-10262 were relatively early flowering and they were close to IS-7254 in overall superior performance.

1978 Results

At the time of preparation of this paper ISLDN results have been received from four locations: Navsari and Indore in India, Khon Kaen in Thailand and Farako-Bá in Upper Volta. As for the 1977 analysis, a location is regarded as

Table 14. Grey leaf spot, rust and rough leaf spot severity reactions[a] of 28 entries at two, one, and one location respectively in the 1977 ISLDN.

Entry	Grey leaf spot		Rust	Rough leaf spot
	Delhi	Samaru	Udaipur	Samaru
IS-10240	2	5	1	1
R-55-DX	3	5	1	1
IS-10262	4	5	1	1
BY x GPR 165-2	3	5	1	1
IS-7254	4	3	1	1
IS-2223	3	4	1	1
TAM-428	4	5	1	1
IS-2550	3	5	1	1
IS-10264	4	5	1	1
CSV-2	3	5	1	1
IS-4150	2	5	1	2
IS-3390	2	5	1	4
Sel. 512	2	5	2	1
CS-3541	3	4	2	1
BY x Entol.	2	5	2	1
BY x Pickett	4	5	2	1
SC-110-14	4	5	2	1
BY x GPR 165-1	3	5	2	1
IS-7322	4	5	2	2
IS-4277	3	5	3	1
IS-73	5	5	3	1
BY x GPR 165-3	3	5	4	1
IS-643	3	5	4	1
H-112	4	5	4	1
555	3	5	4	3
IS-856	4	5	5	1
IS-6838	4	5	5	1
IS-8171	5	5	NR[b]	1

a. Greatest of two reps. 1 = No symptoms. 5 = Severe symptoms.
b. No record.

having provided sufficient pressure for a particular disease only if at least one entry developed a 4-type reaction there to that disease. By this criterion, Indore provided sufficient pressure for rough leaf spot and rust; Navsari sufficient pressure for leaf blight, grey leaf spot, anthracnose, zonate leaf spot, rust, and rough leaf spot; Khon Kaen sufficient pressure for grey leaf spot, anthracnose, and zonate leaf spot; and Farako-Bá sufficient pressure for grey leaf spot, anthracnose, zonate leaf spot, and sooty stripe.

A full analysis of these data will be made when the majority of the 1978 ISLDN data sheets have been returned. For this paper we have summarized the reactions of some of the best lines for particular diseases, and the best lines from 1977.

Three entries gave an excellent performance with scores of 1 or 2 only for each "severe" disease at each Asian location (Tables 16, 17). CS-3541 was free from grey leaf spot, anthracnose, zonate leaf spot, rust, and blight at all locations, and scored a 2 for rough leaf spot at

Table 18. **Flowering data and maximum reactions of 28 entries to seven leaf diseases at various locations in the 1977 ISLDN.**

Entry	MDTF[a]	Rough	Rust	Zonate	Sooty	Blight	Anth.	Grey	No. ≤ 2
IS-7254	84	1	1	2	3	2	4	3	(4)
IS-10240	64	1	1	2	2	3	5	5	(4)
IS-10262	58	1	1	3	2	2	5	5	(4)
IS-4150	72	2	1	1	2	3	4	5	(4)
IS-2223	69	1	1	2	4	2	5	4	(4)
BY x Entol.	69	1	2	2	4	4	2	5	(4)
CS-3541	65	1	2	1	3	3	4	4	(3)
IS-10264	60	1	1	2	3	3	4	5	(3)
IS-3390	57	4	1	3	2	5	2	5	(3)
BY x GPR 165-2	61	1	1	2	3	4	5	5	(3)
TAM-428	64	1	1	2	3	4	4	5	(3)
IS-2550	66	1	1	2	4	3	4	5	(3)
SC-110-14	61	1	2	3	3	2	4	5	(3)
IS-7322	85	2	2	3	2	4	3	5	(3)
R 55 DX	55	1	1	3	3	4	4	5	(2)
Sel. 512	70	1	2	3	3	4	5	5	(2)
IS-4277	55	1	3	3	3	2	5	5	(2)
IS-8171	71	1	–[b]	2	4	3	4	5	(2)
BY x Pickett	61	1	2	3	3	3	3	5	(2)
BY x GPR 165-1	61	1	2	3	3	3	4	5	(2)
IS-643	55	1	4	2	4	3	5	5	(2)
IS-73	55	1	3	5	2	5	4	5	(2)
CSV-2	63	1	1	3	4	3	4	5	(2)
BY x GPR 165-3	61	1	4	4	3	3	4	5	(1)
555	74	3	4	1	5	3	3	5	(1)
IS-856	59	1	5	4	3	5	4	5	(1)
IS-6848	58	1	5	4	4	3	5	5	(1)
H-112	58	1	4	3	5	5	5	5	(1)

a. Mean number of days to 50% flowering.
b. No data available.

Navsari and Indore. IS-2276 and IS-4150 were almost as good with no more than a score of 2 for any disease at any of the locations listed.

The anthracnose reactions of IS-10240, IS-10262, and IS-223 at Navsari, Khon Kaen, and Farako-Bâ indicate the possibility of different pathotypes of the anthracnose organism at these locations.

The Farako-Bâ reactions of the selected entries are given in Table 18. IS-4150 was free from all the major diseases there. CS-3541 also performed well.

Overall Performance

As the ISLDN locations are diverse in distance and variable in elevation, latitude, and important local pathogens, it is perhaps not as relevant to look for overall performance of the ISLDN entries as it is for the ISDMN or ISGMN entries. Nevertheless, we should examine whether there are multiple resistant entries. In the 1977 MS-ISLDN, which is the largest and most detailed data set, six entries scored 2 or less for four diseases and an additional eight entries

Table 16. Reactions to grey leaf spot, anthracnose, and zonate leaf spot of selected entries from the 1978 ISLDN trial at Navsari, India, and Khon Kaen, Thailand.

	Grey Leaf Spot				Anthracnose				Zonate Leaf Spot			
	Navsari		Khon Kaen		Navsari		Khon Kaen		Navsari		Khon Kaen	
Entry	R_1	R_2	R_1	R_2	R_1	R_2	R_1	R_2	R_1	R_2	R_1	R_2
IS-2276	1	1	1	1	1	2	1	1	1	1	1	1
CS-3541	1	1	1	1	1	1	1	1	1	1	1	1
IS-4150	1	–	2	2	1	–	1	1	1	–	1	1
IS-10240	4	3	3	3	4	3	1	1	4	3	1	1
IS-10262	1	3	3	3	4	4	1	1	2	2	3	3
IS-2223	1	1	3	1	2	2	5	5	2	3	1	1
IS-73	1	3	4	5	2	3	1	1	3	3	4	4

Table 17. Reactions to rust, rough leaf spot and blight of selected entries from the 1978 ISLDN trial at Navsari and Indore, India.

	Rust				Rough Leaf Spot				Blight	
	Navsari		Indore		Navasari		Indore		Navsari	
Entry	R_1	R_2	R_1	R_2	R_1	R_2	R_1	R_2	R_1	R_2
IS-2276	1	1	2	2	1	1	2	1	1	1
CS-3541	1	1	1	1	1	2	2	2	1	1
IS-4150	1	–	1	1	2	–	2	2	2	–
IS-10240	1	1	1	1	3	1	1	2	2	1
IS-10262	1	1	1	1	2	1	1	3	1	2
IS-2223	1	1	1	2	4	2	2	2	4	1

Table 18. Reactions at Farako-Ba, Upper Volta, to grey leaf spot, anthracnose, zonate leaf spot and sooty stripe of selected entries from the 1978 ISLDN.

	Grey leaf spot		Anthracnose		Zonate leaf spot		Sooty stripe	
Entry	R_1	R_2	R_1	R_2	R_1	R_2	R_1	R_2
IS-2276	2	1	1	1	1	1	3	2
CS-3541	1	1	2	2	1	1	2	1
IS-4150	1	1	1	1	1	1	1	1
IS-10240	4	2	1	1	1	1	1	1
IS-10262	2	2	1	1	3	2	1	1

scored 2 or less for three diseases. Of these 14 entries CS-3541 and IS-4150 have performed very well at the four locations from which the 1978 data are available. Unfortunately data on IS-7254 were not included in the three 1978 ISLDN data sheets returned and we have not yet learned what the problem was with this entry. It gave a good stand and grew well at ICRISAT Center.

Discussion

The ISLDN entries are tested against several different pathogen species in a wide range of environments. No entry has been resistant to all diseases at all locations, but some entries have shown apparent resistance to three or four diseases at several locations.

The 1977 and 1978 anthracnose data and 1977 leaf blight data (Tables 11, 12, and 16) indicate distinct differential reactions to these diseases by some entries and stable resistance to them in others.

The ISLDN to date has provided a good beginning to the international effort to locate sources of stable and multiple resistance to leaf diseases, but it is only the beginning. The 3-year data will need careful analysis to properly assess the validity and consistency of specific cultivar-location-disease reactions, the possibility of the existence of races of the pathogens, and the existence of multiple stable sources of resistance.

Questions

1. The first and most important question is whether the ISLDN should continue as a composite nursery for all leaf diseases for all locations, or whether we should now develop separate nurseries for individual diseases, groups of diseases, and environments. There are strong arguments for and against splitting up the ISLDN, and we would like participants to come up with a consensus answer to this important question.
2. A second question somewhat tied to the first is if and how the investigations of indications of races proceed? We can develop tentative differential sets based on the evidence to date and send these to the apparently differentiating locations. Is this going to provide useful information? Should it begin in 1979?
3. As yet we have no cooperators in the Americas. We feel that this is a major gap in the ISLDN program. Who would be able to participate in the ISLDN from the Americas?

Acknowledgment

We are indebted to all the cooperators in the ISLDN program who have given their time, energy, and resources in order to produce the results reported in this paper.

Leaf Disease Discussion Session

General

Brhane:
What is the relationship between the stage of development of the sorghum plant and its reaction to leaf diseases? In Ethiopia, the leaf diseases seem to come mainly after flowering.

Frederiksen:
Generally, sorghum becomes more susceptible to foliage pathogens after anthesis; however, *Exserohilum turcicum* is more damaging on young plants. Rust and grey leaf spot attack susceptible cultivars equally well at all crop stages.

Tyagi:
Why do sorghum plants with systemic SDM infection lose their resistance to foliar diseases?

Frederiksen:
This is a very interesting phenomenon, and we do not know why it occurs.

Selvaraj:
Dr. Girard has tried foliar application of chemicals for the control of sooty stripe. Dr. Sharma has also conducted experiments with foilar sprays. These are not economical for the farmers in the SAT countries. So, would you please enlighten me on the objectives of these studies?

Girard:
The purpose was not to see if it was economical; we wanted to see if a large-scale application of chemicals was able to control the disease. We also wanted to see if, with good control of the diseases, we would have an impact on yield. Disease control did not seem to have an impact on yield.

Sharma:
We have some high-yielding lines that are resistant to some diseases, but susceptible to one particular disease. By chemical control of this one disease, we can increase yield.

K. N. Rao:
In resistance screening, should we have trials on separate leaf diseases, or should we work with several together?

Doggett:
For a plant breeder it is far more useful to have several diseases in one trial than to have to deal with them one by one.

Frederiksen:
I agree with Dr. Doggett. For resistance-screening work, we want to locate broad-based resistance to several leaf pathogens.

Balasubramanian:
What about the use of seed treatment and sanitation on leaf diseases?

Sharma:
In our studies, a combination of fungicide and carbofuran increased the plant population and yield of sorghum. I don't know how much was contributed by the fungicide. Seed treatment is effective in controlling diseases in the early stages of development.

Teyssandier:
It is important to determine the correct stages of growth at which to score for leaf-disease reactions.

Frederiksen:
There are several possible ways of scoring leaf diseases of sorghum, ranging from detailed plant by plant examination to rapid whole plot estimates. More than one rating is necessary. If possible make three ratings at weekly intervals. You have to get to know the diseases and use common sense.

Denis:
Which leaf diseases increase in importance

with increases in crop density and/or increases in fertilizers?

Frederiksen:
In general, diseases that are endemic and spread from within a crop increase with plant density. Some diseases are more important in succulent plants and thus nitrogen levels should affect them. We need more information on these factors.

Rust

Mengistu:
Do you think the hyperparasite *Darluca* can reduce the problems of sorghum rust? I ask this because we see less rust than we used to, and *Darluca* is always present.

Frederiksen:
The most *Darluca* I have seen is where there is the most rust. *Darluca* actively colonizes rust pustules in some environments. Unfortunately the experimental evidence does not support the hypothesis that it can be used for rust control. It is probably a case of too little too late.

Leaf Blight

Tyagi:
In view of some disagreement among mycologists on the matter, should we change the name of *Helminthosporium turcicum* once more and call it *Exserohilum turcicum*?

Frederiksen:
The perfect stage was described as a new species. The work was done by a competent mycologist and geneticist. I think *Exserohilum turcicum* is a valid species name for the imperfect stage.

Oval Leaf Spot

Anahosur:
Are the slides you showed of *Ramulispora sorghi* and *R. sorghicola* taken from natural conditions or from cultures? CMI reports *R. sorghi* is branched in natural and artificial conditions, and *R. sorghicola* unbranched in natural conditions.

Girard:
I have observed branched conidia of *R. sorghicola* from natural conditions, from Senegal. *R. sorghi* may have branched conidia in the proportion of 60%, and *R. sorghicola* only up to 10%.

Sorghum
Head Blights and Stalk Rots

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Sorghum Anthracnose

M. A. Pastor-Corrales and R. A. Frederiksen*

Sorghum anthracnose, also known as red stalk rot, is caused by the fungus *Colletotrichum graminicola* (Cesati) Wilson. The disease was first reported separately from North Carolina and Texas in 1911 and 1912, despite the fact that anthracnose in sorghum had been known for sometime in India and elsewhere (Heald 1912; Stevens and Hall 1911, Tarr 1962). The disease has since been reported from most areas where sorghum is grown; however, it is more prevalent and important in warm, humid regions of the world (Bergquist 1973; Jouan and Delassus 1971; Lebeau et al. 1951; Muntanola 1952; Noble 1937; Porter 1976; Saccas 1954; Sundaram et al. 1972).

Since its introduction to the Americas, grain sorghum has become increasingly important. It is the second most important feed grain in the United States and in Latin America and has had a very rapid acreage increase in the last few years. Grain sorghum in Latin America is a very important crop from the nutritional and economic point of view, and the acreage appears to be still increasing — particularly in Mexico, Guatemala, Venezuela, Colombia, Brazil, Argentina, and Uruguay. With this increase diseases have also become an important factor in sorghum production. Anthracnose has become of economic importance in Latin America, where it is the most serious disease of grain sorghum, and in some areas it is one of the main yield-limiting factors, specifically in Brazil, Venezuela, and Guatemala (Maunder 1975; Sharvelle 1975). In the USA, anthracnose is an important disease in the southeast. The devastating effects of this disease on sorghum in Georgia and other humid areas are well documented (Harris and Fisher 1973; Luekel et al. 1951; Lohman and Stokes 1944). It also has been a destructive disease in some years on grain sorghum and broomcorn in Texas and has caused serious loss in broomcorn in Illinois (Koehler 1943; Toler and Frederiksen 1970).

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The Pathogen

C. graminicola is characterized by dark acervuli, the disc-shaped fruiting structures present in the necrotic parts of the lesions. In sorghum, acervuli can be found on both surfaces of the leaf, on the leaf midrib, peduncle, rachis branches, and grain. Typically, an acervulus is small erumpment, and has dark spines or setae at the edge or among the conidiophores. The conidia are terminal, sickle-shaped, and borne singly. The conidia, which generally have one or two oil drops present, are held together in an orange-to salmon to pinkish gelatinous matrix. When rain and splashing water strike this matrix, the spores are disseminated through the air. When these conidial land on host tissue, they produce one or two germ tubes, which in turn form appressoria. The appressoria give rise to infection pegs that penetrate directly through the epidermis and cuticle of the host.

Symptoms

The characteristic symptoms caused by *C. graminicola* in sorghum will not be discussed here since they have been adequately described elsewhere (Dickson 1956; LeBeau et al. 1951; Tarr 1962; Williams et al. 1978). Anthracnose symptoms vary with genotype and are generally more evident on maturing plants; however, infection can occur on young plants.

C. graminicola infects leaves, stems, peduncles, heads, and the grain, either separately or all together. Often these different aspects of anthracnose will be manifested as apparently different diseases. For this reason it is not uncommon to find in the literature discussions of the leaf symptoms separate from the discussion of the peduncle and head symptoms, which are referred to as the red-rot aspect of the disease. Red rot, a term borrowed from anthracnose in sugarcane, is not very descriptive of the symptoms on the stem, peduncle, and head, and leads to confusion, since other pathogens, i.e., *Fusarium moniliforme, Mac-*

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rophomina phaseolina and some insects can incite red-color symptoms also. For this reason, the use of "red rot" to describe the anthracnose symptoms of the stem should be avoided.

We have divided anthracnose symptoms into three groups: leaf, peduncle, and head symptoms. Very often only one type of symptom is present or predominates over the other two. In Georgia, particularly in 1977, we found the leaf symptom to be the most common; in many entries, plants were found to show only leaf infection. Less often they had either only peduncle infection or only head infection. In 1978, the leaf symptom was almost nonexistent in the Corpus Christi (Texas) nursery, even on those genotypes with very severe anthracnose symptoms on the peduncle and head.

Many times, anthracnose symptoms are found in all three or only two plant parts. When anthracnose is found in only two plant parts, it more commonly infects the head and peduncle rather than the leaf.

Considering this selective tendency, more studies appear necessary to elucidate the causative factors in infection of one plant part and not another. The separation in symptom expression may be controlled by the environment or by host genotype.

Association of Anthracnose with Other Diseases

Anthracnose is favored by warm, humid weather and wind-blown rains, which aid in spore dispersal from one plant to another. These conditions are also most favorable for other sorghum foliar pathogens. *Gloeocercospora sorghi (zonate leaf spot) and Cercospora sorghi* (grey leaf spot). When resistance to anthracnose is present in one genotype, often one or both of these pathogens attack the anthracnose-resistant genotype. Occasionally, anthracnose-resistant lines are also resistant to one or both of the other pathogens. Unfortunately, the same genes do not condition resistance to the three pathogens.

We also have some evidence indicating a relationship in the susceptibility of sorghum to anthracnose and sorghum downy mildew caused by *Peronosclerospora sorghi*. We have observed that sorghum varieties or hybrids which are susceptible to downy mildew are severely infected by *C. graminicola*. We have also noted that anthracnose-resistant genotypes after infection with downy mildew become susceptible to the anthracnose fungus. This observation is a preliminary one and needs further confirmation.

Host Range and Pathogenicity

When Wilson published his work (1914) on the identity of anthracnose of grasses in the USA, he combined many species of *Colletotrichum* with falcate conidia into a single species which he erected as *Colletotrichum graminicola* (Cesati) Wilson 1914. In this new broad species, he included: *Colletotrichum lineola* Corda, *C. bromi* Jennings, *C. cereale* Manns, *Dicladium graminearum* Cesati, *Psilonia apalospora* Berk and Curt., *Vermicularia culmigera* Cooke, *V. holci* Sydon, *V. melicae* Fuckel, *V. sanguinea* Ellis and Halsted, *V. graminicola* Westend, *V. affinis* Socc. and Briard.

C. graminicola has a wide host range among cultivated and wild species of cereals and grasses (Tarr 1962) including barley, corn, oats, rye, sorghum, sudangrass, johnsongrass, wheat, and a large number of grasses (Bruehl and Dickson 1950; Luke and Sechler 1963; Sanford 1935; Sprague 1950; Shurtleff 1973; Wiese 1977). However, isolates from one host species do not necessarily infect other species. The capacity of an anthracnose fungus isolate to infect several species is not a subject of general agreement. Selby and Manns (1909) were able to produce typical anthracnose symptoms on wheat and emmer in the field, using spore suspensions obtained from these two hosts. In addition, these authors reported the infection of emmer with the isolate from wheat. Edgerton (1911), using spores of *C. lineola* obtained from wheat and other grains, and spores of *C. cereale*, obtained from johnsongrass and broomcorn, failed to develop infection on sugarcane. These two *Colletotrichum* species were later grouped as *C. graminicola*, which suggests that Edgerton was indeed working with two different isolates of the same fungus (Wilson 1914). Similarly, Sanford (1935), using an isolate from oats, was unable to develop infection on wheat, barley or flax. Chowdhury (1936) in India reported that an isolate of *C. graminicola* from maize readily caused high infections on sor-

ghum and infected to a lesser degree other hosts, including finger millet, Italian millet, teosinte, barley, wheat, oats, *Panicum frumentaceum, Pennisetum typhoideum*, and *Paspalum scrobiculatum*. He also reported the infection of maize with an isolate from sorghum. Lohman and Stokes (1944) described stem anthracnose and red rot of sorghum caused by *Colletotrichum* occurring in Mississippi. They pointed out the close similarity between the causal agent of the sorghum disease and *C. falcatum*, the agent responsible for red rot in sugarcane. They also report that *C. falcatum* from sugarcane produced the red rot symptoms in sorghum. LeBeau (1950) conducted pathogenicity studies with *Colletotrichum* from different hosts on sorghum and sugarcane (the isolates were from sugarcane, sorghum, johnsongrass, sudangrass, broomcorn, *Erianthus* and 18 grass species). Isolates from sugarcane were highly pathogenic to sugarcane and rarely pathogenic to sorghum; isolates from sorghum, johnsongrass, sudangrass, broomcorn, and *Erianthus* were nonpathogenic on sugarcane but, for the most part, were highly pathogenic to sorghum. Bruehl and Dickson (1950) found host specificity among isolates of *C. graminicola* from different cereals and grasses, with symptoms produced only by the isolates from the same or closely related species. LeBeau et al. (1951) suggested that *C. graminicola* consisted of more than one pathogenic race, and that the isolates from sorghum, johnsongrass, sudangrass, broomcorn and *Erianthus* appear to form one or more races characterized by their high pathogenicity to sorghum, whereas isolates from 14 other grass species appear to belong to one or more races which do not attack sorghum. Tarr (1962) did not find anthracnose in maize or other activated crops in the Sudan, even though the fungus was found commonly on sorghum species. Williams and Willis (1963), using a local isolate associated with maize anthracnose in Ohio, were unable to infect wounded leaves of wheat, oats, or barley. Similarly, Dale (1963) reported that a maize isolate from Arkansas failed to infect sorghum, and an isolate from sorghum did not infect maize. Wheeler et al. (1974) reported that 30 isolates of *C. graminicola* from six states (USA) readily infected maize and all members of the genus *Sorghum* tested (*S. bicolor, S. sudanense* and *S. halepense*). They also reported that isolates from *Avena, Medicago, Hordeum, Bromus, Triticum, Calamagrostis, Festuca, Sorghum,* and *Danthonia* were nonpathogenic on maize. Nicholson (1974) on the other hand, reported that a *C. graminicola* isolate from Indiana maize failed to infect wild cane, johnsongrass, sorghum, fall panicum and six different foxtails. Similarly, isolates from maize in Illinois were pathogenic to maize but not to sorghum (Hooker 1974).

To aid control of anthracnose in sorghum or any other cereal or grass, the host range of the causal organism should be known. This is particularly important in areas such as Texas or Central America where sorghum and maize are grown side by side. The literature seems to indicate that certain isolates of maize anthracnose are capable of infecting sorghum in the glasshouse (Muntanola 1952). More information is needed on the ability of different geographical anthracnose isolates to cross-infect from one host species to another.

Pathogen Variability and Physiologic Specialization

A physiologic race, as defined by Stakman and Harrar (1957) is a biotype or group of biotypes that can be distinguished with reasonable certainty and facility by physiologic characters including pathogenicity.

Harris and Johnson (1967) suggested that pathogenic races may exist in *C. graminicola*. Similarly, Frederiksen and Rosenow (1971) noted specific differences in reactions among selected sorghum lines in Texas, Mississippi and Georgia; however, they pointed out that the majority of sorghum lines tested as resistant or susceptible in one area of the United States tended to react similarly in another.

To study the virulence of *C. graminicola*, and to monitor the possible existence or appearance of different or new physiological races of the pathogen, an International Sorghum Anthracnose Virulence Nursery (ISAVN) was started by the Texas Agricultural Experiment Station in 1975). Since its inception, this nursery has been grown in countries in North, Central and South America, the Caribbean region, Asia, and Africa. The information so far obtained has been most useful in formulating

some generalizations about the anthracnose fungus. The data obtained in 1975 strongly suggested that physiologic races exist within the isolates of *C. graminicola* that infect sorghum (Texas Agricultural Experiment Station 1975). The variety Wiley, which is highly resistant elsewhere, was infected by *C. graminicola* in Venezuela. In addition the varieties Mn-960 and TAM-428 were highly susceptible to the anthracnose fungus in Nigeria whereas they were resistant in the Americas. Conversely, BTx 398, Pioneer Brand 846, and C-424, which are highly susceptible in the Americas, showed less infection in Nigeria.

During 1976, ISAVN nursery results were obtained only from a few locations. In that year, the variety TAM-428, resistant in the USA, was infected by *C. graminicola* in Puerto Rico.

The results obtained in 1975 and 1976 warranted the initiation of an anthracnose nursery for converted lines; thus, the Converted Sorghum Lines Anthracnose Test (CLAT) was established and the seed distributed in 1977 and 1978 in different locations of the Americas.

Control

Of all possible disease control methods for anthracnose of sorghum, the use of disease-resistant material offers the most satisfactory means of reducing losses. A number of commercial sorghum hybrids with good degrees of resistance have been developed in the last few years. Since the disease is most severe in warm, humid areas, the need for avoidance of susceptible genotypes where the disease is known to occur cannot be overemphasized. In areas where the environment is not conducive to the development of the disease, a low level of resistance to the pathogen will be adequate. Although breeding for anthracnose resistance has been successful, the possible existence of physiologic races, requires screening of sorghum genotypes with many of the suspected physiologic races, in order to generate usefully resistant material.

Cultural practices that include the elimination of susceptible (reservoir) weeds such as johnsongrass, plowing under of infected crop debris, and crop rotation should help control the disease by reducing the primary inoculum and thus preventing early severe infection. As the disease is most damaging following physiological maturity of sorghum, delay in harvest should be avoided. Sorghum also should be planted so that maturity will not coincide with rainy weather, since this condition is most conducive to the dissemination of inoculum.

Chemical control of anthracnose in sorghum is not practiced, and little information is available. Seed treatment might increase stand and decrease seedling blight when very susceptible genotypes, such as broomcorn, are planted. The use of systemic fungicides during seedset also offers some good possibilities; however, the cost may be prohibitive.

Future Research Needs

More complete study is needed of:

1. The pathogenicity and host range of sorghum isolates of *C. graminicola*, particularly in areas where anthracnose, sorghum, and other cultivated and noncultivated hosts occur together.
2. The possible existence of physiologic races, with studies conducted on a worldwide basis.
3. The inheritance of resistance. Some authors have suggested that different dominant genes control resistance to leaf and stalk anthracnose. This point has not been proven.
4. The type of resistance in sorghum to anthracnose, related to
   a. Reduction in total number of infections;
   b. Reduction in sporulation;
   c. Lengthening the latent period;
   d. Reduction of spore deposition;
   e. Shortening of infection period;
   f. High infection threshold;
   g. Alteration of resistance by climate, nutrition, and other factors.
5. Sorghum seedling reaction to anthracnose, correlating the findings with field results. Does the perfect stage of the fungus exist? Do the isolates from leaf, stem, and head differ? These and other related questions also need further study.

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Anthracnose of Sorghum in Brazil

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In the early seventies, when sorghum began to expand in Brazil, it was noted that a relatively large number of diseases damaged the crop. In order to establish research priorities in the area of sorghum pathology, a disease survey was made of all Brazil, using a National Disease Nursery and other national trials. Foliar anthracnose was the principal sorghum disease, because of its frequency and intensity in most areas of the country. Anthracnose damage was most intense in Central Brazil, particularly in the area of Ribeirao Preto, São Paulo. As a result of the survey the plant-breeding program at the National Maize and Sorghum Research Center (CNPMS) was directed to obtain breeding lines resistant to anthracnose. Selections were made under field conditions in Ribeirao Preto and in Sete Lagoas, Minas Gerais.

Research at the University of Sao Paulo in Piracicaba aimed at selecting plants resistant to anthracnose in the greenhouse, is the topic of several thesis problems. As sorghum was a new crop in Brazil, it was necessary to import advanced breeding materials to begin the breeding program. Many breeding lines introduced from Texas A & M University in the USA were considered to be elite with good disease resistance, including resistance to the pathogen *Colletotrichum graminicola* (TX-2536, SC-170, SC-173, SC-110, SC-599-6-10, TAM-428, TAM-430). But many of these lines have not shown resistance to foliar anthracnose under our conditions. These lines were probably evaluated and selected for resistance to red rot (an important disease in the USA), and not to foliar anthracnose. Other breeding lines, such as TAM-2566, SC-175-14, P-721, TX-623 and TX-399, have been highly susceptible under our conditions.

Actually, the number of lines resistant to anthracnose in our breeding program is small; they include SC-326-6 and derivatives of SC-326-6, SC-234, SC-239-14, Brandes, and an early selection from a late-maturing forage variety known as Santa Elisa. In order to increase this limited genetic base, 400 breeding lines — including many sorghum conversion entries — were evaluated last year in Ribeirao Preto and Sete Lagoas. Some entries have shown excellent levels of resistance and are being evaluated again this year. Unfortunately, last year was dry in central and southern Brazil and the level of anthracnose development was below normal. Other material in our germplasm bank is in the process of being increased and will be evaluated for anthracnose in the near future.

The pathogen has been identified by its lesion type and its morphological characteristics (presence of setae, and shape of conidia) as *Colletotrichum graminicola*. However, elongated lesions have also been encountered, which could indicate the existence of other species of *Colletotrichum* such as *C. falcatum*.

Anthracnose lesions frequently occur on the peduncle and stalk, in the panicle, and on the grain. In the latter case *C. graminicola* may be contributing to reduced germination and seedling vigor. Tests with seed fungicides are being initiated this year.

The inheritance of anthracnose resistance is being studied. F_1s and F_2s of crosses between resistant lines such as SC-326-6, SC-283, and Brandes lines, with varying degrees of susceptibility such as Wheatland (TX-399), CK-60 (TX-3197), Redlan (TX-378), TX-623, and a male-sterile line BR-007 are being evaluated this year. Preliminary results of last year's work indicate that incomplete dominance is involved.

The National Maize and Sorghum Research Center has worked in cooperation with other institutions, principally with Texas A & M University in the USA, to study the problems of anthracnose more closely and would like to expand these activities.

Our sorghum-breeding program released several grain-sorghum hybrids, forage-

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sorghum hybrids, and sweet-sorghum varieties with good agronomic characteristics and excellent resistance to diseases during 1978.

Summary

A survey of sorghum diseases in Brazil has shown anthracnose to be the principal disease of this crop because of its frequency and intensity. The greatest damage from this disease has occurred in the region of Ribeirao Preto. Selection for resistance to this disease is being conducted by the National Maize and Sorghum Research Center, in Sete Lagoas and Ribeirao Preto. Greenhouse studies on resistance are being conducted by the University of Sao Paulo at Piracicaba. Breeding stocks imported from the USA as sources of anthracnose resistance have reacted as susceptible to foliar anthracnose in our conditions.

The number of resistant lines currently utilized in our breeding program is limited. New sources of resistance are being identified from our germplasm bank and from new introductions.

The pathogen has been identified as *Colletotrichum graminicola*, though it is possible that *C. falcatum* could also be involved. Lesions can be found on all plant parts, including the seed. Preliminary field observations have indicated that the inheritance of resistance to this pathogen is partially dominant.

Commercial grain and forage hybrids and sweet-sorghum varieties resistant to anthracnose were released for growing in farmers' fields in 1978.

Fusarium Disease Complex of Sorghum in West Africa[1]

N. Zummo*

Introduction

A *Fusarium* disease complex affecting sorghum, millet, and maize was observed in Nigeria and other parts of West Africa in 1974 and 1975 and on sorghum and sugarcane in the United States from 1966 to the present. A portion of this disease complex has long been known on sugarcane as pokkah boeng or twisted top (Bolle 1927, 1930; North 1932; Zummo 1970) characterized by deformed and/or discolored leaves near the top of the plant.

On sorghum and millet, *F. moniliforme* Sheldon affects plants at all stages of growth and can cause seedling blight, root and stalk rot, pokkah boeng or twisted top, seed mold, and head blight (Saccas 1954; Tarr 1962). In West Africa, young sorghum plants in the one- to three-leaf stage are often severely affected by *F. moniliforme* during periods of prolonged cloudy humid weather. The first symptom on these plants are variously colored (tan, brown, red, purple, black) irregular lesions on the leaves. The tips of the leaves wither and later the entire leaf blades die. If conditions are favorable for plant growth, the sorghum plants will outgrow the disease. Where the disease is severe and the environment remains unfavorable for rapid seedling growth, infected plants are killed and the crop may have to be replanted.

Fusarium Root and Stalk Rot

Fusarium moniliforme has become increasingly common in recent years as a root and stalk rot pathogen of sorghum in many areas of West Africa.

In the United States, the disease is generally found in the same areas where charcoal rot occurs, particularly on the High Plains from Texas to Kansas (Edmunds and Zummo 1975). Like charcoal rot, *Fusarium* stalk rot apparently requires some predisposing conditions for disease development as plants approach maturity. Unlike charcoal rot, which does most injury during periods of moisture stress, *Fusarium* stalk rot usually is most damaging during cool wet weather following hot dry weather.

Fusarium stalk rot is usually accompanied by extensive root damage. Under irrigation and heavy nitrogen fertilization, root damage may not cause noticeable change in the crop's above ground appearance before the stalks begin to rot. Stalk rot may reduce seed filling, resulting in seed weight losses up to 60%.

Root damage typically involves first the cortical tissues, then the vascular tissues of all roots. Newly formed roots may exhibit distinct lesions of various sizes and shapes. The rot is progressive; older roots often are destroyed, leaving little plant anchorage. When such a rot is extensive, plants are easily uprooted.

Fusarium stalk rot can usually be distinguished from charcoal rot because of the less pronounced pigmentation and disintegration of pith tissues and slower rot rate of the *Fusarium*. Charcoal rot may destroy a field of sorghum in 2 to 3 days; *Fusarium* stalk rot may require 2 to 3 weeks to do so.

Coincident with increased *Fusarium* disease incidence are several cultural practices suspected of contributing to the increase of the disease. These include minimum tillage, high nitrogen fertilization, high plant populations, and continuous cropping. The fungus persists in the soil, on crop refuse, and on weed hosts. Sorghum varieties completely resistant to *Fusarium* root and stalk rot are not available. Avoiding conditions that may predispose the crop to the disease may help reduce losses.

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1. Cooperative investigations of the USDA, SEA-FR, Sugar Crops Field Station, Rt. 10, Box 152, Meridian, MS 39301, and Mississippi Agriculture and Forestry Experiment Station, Mississippi State University, MS 39762.

Pokkah Boeng, or Twisted Top

Pokkah boeng, or twisted top, is incited by the soilborne fungus *Fusarium moniliforme*, which is present in all sorghum areas where high humidity is prevalent. Although the disease may be conspicuous on some sorghum varieties, overall losses usually are small.

Pokkah boeng is characterized by deformed or discolored leaves near the top of the plant. In some cases, the leaves become so wrinkled they are unable to unfold properly, resulting in a plant with a ladder-like appearance. In extreme cases, infection may move from the leaves and sheath into the stalks, causing death of the tops. In mild cases, symptoms often resemble that of the mosaic symptom caused by virus.

Pokkah boeng can be differentiated from mosaic by the wrinkled bases and numerous small transverse cuts in the margins of the leaves of infected plants. Sometimes the disease causes stalks to bend, and sometimes the stalks display "knifecut" symptoms (narrow, uniform, transverse cuts in the rind that give the impression the tissue has been removed with a sharp knife). Because they are covered by the leaf sheaths, those lesions may not be apparent when pokkah boeng leaf symptoms are present. Under physical stress such as windstorms, the stalks may break along the "knife-cut" lesions.

During prolonged wet weather, *F. moniliforme* grows upward on the outside of sorghum stalks and the fungus temporarily may become established behind the leaf sheaths or in the whorls. Metabolites produced by the fungus incite distortions in the plants (Zummo 1972). When the wet weather subsides, the fungus dries and the plant resumes normal growth. There is some evidence that the fungus may be transmitted by seed. Pokkah boeng also affects maize, johnsongrass, sugarcane, broomcorn, and sudangrass.

Fusarium Head Blight

Fusarium head blight incited by *Fusarium moniliforme* (Zummo and Frederiksen 1973) can be a serious problem on some of the exotic dwarf, short-season sorghums in West Africa, especially if they flower during extended periods of heavy rain and high humidity. The disease is characterized by death of several to all of the florets in seed heads. When the disease is severe, the entire seed head may be covered with a copious fungal growth, cream to pinkish tan in color. In extremely severe cases, all heads in a field may be killed. When the panicle is split lengthwise, a red-brown to black discoloration is evident in the upper portion of the peduncle and extends into the branches of the head. Sometimes the discoloration may extend throughout the peduncle and into the upper internodes of the stalk, in which case the rind may also be discolored. In severe cases, breakover of peduncles may occur.

Fusarium head blight can be distinguished from red rot and peduncle breakage incited by *Colletotrichum graminicola* (Cesati) Wilson, because the discoloration in head blight is uniform throughout. In red rot, the discoloration is interspersed with discrete white areas. In some sorghum varieties, individual red rot lesions in the peduncle may be easily identified by their distinct lenticular shape.

The mechanism of infection and penetration by the fungus is not fully understood. It is suspected that, during extended periods of wet weather, mycelium of the fungus, which lives in the soil, grows up on the waxy bloom along the outside of the stalk. Alternatively, the fungus may infect the head through airborne conidia. Penetration may occur through cracks or insect wounds in the rind of the peduncle, rachis, or panicle branches. Sorghum varieties with dense, compact heads are more prone to attack by head blight than are varieties with loose, open heads. Most of the tall local sorghum varieties show resistance to *Fusarium* head blight. Some of the early exotic sorghum varieties show excellent resistance to head blight and should when possible be grown where short-season sorghums are recommended.

Fusarium Seed Mold

Fusarium seed mold is an important disease on short-season, nonphotosensitive sorghum varieties that mature before the end of the rainy season in West Africa. Because sorghum seeds are produced in rather compact panicles with large portions of the seed directly exposed to the environment, they provide an ideal site for fungal growth, especially if humid conditions prevail as the grain matures. In normal years, most local photosensitive sorghum varieties,

such as "Fara Fara" and "Short Kaura," develop their grain after the rains end and the grain remains free from seed mold. However, during the recent drought period in the Sahel from (1969 through 1973), some of the long-season photosensitive varieties failed to produce satisfactory crop yields because the rains ended too soon. Short-season nonphotosensitive varieties that mature grain before the end of the normal rainy season were thus introduced. These short, exotic varieties were most responsive to higher plant densities and fertility levels. Some were very susceptible to *Fusarium* head blight and seed mold. When seed from plants infected with head blight and seed mold is sown, *Fusarium* seedling blight may be increased.

At Samaro, Nigeria, Manzo (personal communication 1975) showed an association of several seed-borne fungi with seed of selected sorghum varieties planted in the same field. When three varieties were compared, *Phoma sorghina* was isolated from 36 percent of the "Short Kaura" seed, 37 percent of the "Samaru 2123" seed, and 90 percent of "Roma" seed. *Fusarium moniliforme* was isolated from 2 percent of the "Roma" seed, 4 percent of the "Samaru 2123" seed, and 37 percent of the "Short Kaura" seed.

The action and interaction of *F. moniliforme* on seed in storage is not fully known. It can be assumed, however, that under conditions of relatively high humidity, it can cause seed rot, a decrease in food quality of the grain, and reduced germination.

Summary

A *Fusarium moniliforme* disease complex of sorghum is reported from West Africa. The *Fusarium* attacks sorghum plants at all stages of growth and can cause seedling blight, root and stalk rot, Pokkah boeng or twisted top, seed mold, and head blight. The disease, which can also affect maize, millet, and sugarcane, is most severe when cloudy humid weather persists for an extended time.

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The Photosynthetic Stress-Translocation Balance Concept of Sorghum Stalk Rots

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Root and stalk rots of sorghum can cause severe grain losses; yet resistance has been evasive and difficult to find. A genotype may appear resistant in one year in one location, but may lodge badly during the next season in the same field. Even within a row of several plants that are genetically identical, not all may have stalk rot. This distribution frequently makes selection of resistance difficult and discouraging. There is an obvious need to understand the dynamics of the environment-host-pathogen interactions before constructing stalk-rot resistance selection methods, or recommending cultural practices to minimize stalk rot of sorghum.

Very few stalk-rot organisms, on only a few genotypes, cause significant rot on sorghum before flowering. *Periconia circinata* (Mang.) Sacc. caused early rots in susceptible genotypes in the southwestern USA. This disease was overcome by simply inherited resistance now present in all U.S. varieties. *Colletotrichum graminicola* (Cesati) Wilson also can cause early rot to a few susceptible genotypes of sorghum.

In the USA, however, most stalk rot problems are associated with *Macrophomina phaseolina* (Tassi) Goid. (*Sclerotium bataticola* Taub.), *Gibberella fujikuroi* Saw., (*Fusarium moniliforme* Sheldon), and *Glomerella graminicola* Politis (*Colletotrichum graminicola* [Cesati] Wilson). These are commonly called charcoal rot, Fusarium stalk rot, and anthracnose or red rot, respectively. These fungi, ubiquitous on plant debris of many crops, are not aggressive pathogens capable of attacking vigorous plant tissue, but are able to overcome senescing tissue. Each of these stalk rots is associated with environmental stresses, rotting roots, and approaching plant maturity.

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Charcoal rot is generally associated with high temperature drought stresses, and senescence (Rosenow et al. 1977). The stress on the plant is a predisposing factor to invasion by *M. phaseolina* (Edmunds 1964; Edmunds et al. 1965, Hsi 1961), the fungus that apparently has competitive advantages over other potential pathogens in this environment. Invaded lower-stalk pith tissue has intense black or red pigmentation, with sclerotia on the vascular bundles. Frequently, badly affected stalks only show the bundles covered with sclerotia; there is no remaining pith tissue. As with the other stalk rots, other fungi may be isolated from the rotted tissue.

Fusarium stalk rot is associated with several environmental stresses, such as fertilizer imbalance, high plant populations, and root damage (Edmunds and Zummo 1975; Tarr 1962). The fungus is often found growing from excised leaves of most mature maize plants and probably on other grasses as well. The fungus does not give a distinctive discoloration to the stalk. It appears to be a quick invader of senescing tissue and a rapid grower from infected plant parts, making isolation easy. It may be that this ease of isolation from dead stalks causes pathologists to identify common stalk rots as Fusarium stalk rot, although other fungi may be the primary producers of decay in the dead tissue.

Stalks infected with *C. graminicola* have a distinctive red discoloration interspersed with whiter tissue, especially in upper-stalk tissue. Frequently the infection becomes limited to the peduncle and upper stalk, the area of the plant in which pith senescence first occurs. High humidity appears to favor this fungus, which can invade senescing tissue of both maize and sorghum. In either host species, most genotypes have resistance until after flowering.

The occurrence of stalk rots is associated with senescence of plant tissue (Katsanos and Papel-

lis 1965; 1966b, 1969) and the lack of nonstructural carbohydrates in the senescing tissue (Eschie et al. 1977). Furthermore, this type of tissue in maize has been shown to produce less DIMBOA, one of the metabolites involved in resistance to microorganisms, apparently accounting for the ability of weak pathogens to invade the predisposed plants.

Resistance to maturity-related stalk rots has complex inheritance patterns linked to environmental and physiological interactions in plants. Consequently, selection of genotypes that will reliably maintain healthy stalks and yet have high yields over many environments is difficult. A better understanding and elucidation of the nature of the interactions is a prerequisite to selection of genotypes and to recommending cultural practices for more stable total crop performance.

These interactions are explained by the photosynthetic stress-translocation balance concept of predisposition to root and stalk rots (Dodd 1977). According to this theory, root and stalk rot predisposition begins with senescence of root tissue because of an insufficient supply of carbohydrate for normal metabolic function. The senescing cells, apparently unable to produce normal resistance metabolites, are invaded by microorganisms that are only weakly pathogenic on vigorous cells. As more root tissue is destroyed, the ability of the plant to obtain water from the soil is reduced. The plant eventually reaches the point where transpiration rates exceed water uptake and consequently permanent wilting occurs. Death of all tissue occurs after wilting. Several microorganisms now invade and digest the remaining stalk structure, eventually resulting in lodging. The predisposition, therefore, involves factors affecting the carbohydrate supply to roots, i.e., the rate of photosynthesis and the rate of translocation of carbohydrates to the roots. Various stresses reduce the rate and amount of photosynthesis. The translocation rate to roots is largely influenced by supply (photosynthetic rate) and competition with the grain.

Photosynthetic Stresses Influencing Stalk Rot of Sorghum

Because of the translocation-balance component, the stresses significant to stalk rot occur after flowering during grain fill. Likewise, the level of stress necessary to induce stalk rot predisposition is dependent upon the size of the carbohydrate sink of the developing grain. Photosynthetic stresses include water deficit, destruction of leaf tissue, light reduction, and mineral deficiency.

Water Deficits

Water shortages affect photosynthetic rate in sorghum in nearly a threshold manner. Shearman et al. (1972) found no depression of CO_2 exchange at −18.6 atmospheres water potential, but a 50% reduction at −20.3 atmospheres. A severe stress of −26.3 atmospheres inhibited photosynthesis to a near-compensation point. The severely stressed plants never completely recovered after watering to the full carbon exchange rate. These experiments were conducted under high light intensity, simulating field conditions, whereas other studies under different conditions showed inhibition of photosynthesis in sorghum at −10 atmospheres leaf water potential (Beadle et al. 1973).

Water deficits affect photosynthesis in plants by causing stomatal closure, changes in chloroplast activity, reduction in leaf growth, and senescence of leaves (Boyer 1976; Kozlowski 1976; McCree & Davis 1974). Senescence of lower leaves was also induced presumably because of water deficit, by cutting roots (Katsanos and Papellis 1966a; Papellis & Katsanos 1966).

Charcoal rot is the stalk rot most commonly associated with water deficits. Water stresses occurring after flowering increased charcoal rot by up to 90% (Edmunds 1964; Edmunds et al. 1964, 1965). Odvody and Dunkle (1979) showed that *Macrophomina phaseolina* only penetrated host cells after application of stress. Katsanos and Pappelis (1966a) found a similar reaction with *Colletotrichum graminicola*, the fungus only penetrating dead cells after water stress was induced by cutting roots. Predisposition of plants by the effect of water deficit on photosynthesis probably leads to any of the maturity-related stalk rots.

Leaf Destruction

Any reduction in actively photosynthesizing

leaf tissue reduces the amount of carbohydrate available to the plant for cell maintenance and storage in grains. Artificial removal of the distal halves of all leaves increases the rate of cell death in sorghum (Papellis & Katsanos 1966) as it does in maize (Papellis 1963). *Colletotrichum graminicola* invaded only the dead cells (Katsanos & Papellis 1969). Leaf diseases, such as that caused by *Helminthosporium turcicum* Pass., are associated with stalk rot, apparently because they reduce photosynthesis. Yield losses commonly associated with these diseases are probably the result of incomplete grain fill because of the root-rot premature-death syndrome. Reduction of productive leaf area by insect feeding or hail can also predispose plants to root and stalk rots.

Light Reduction

In species such as maize and sorghum with the C_4-photosynthetic pathways, light intensity is extremely important, because carbon dioxide is not generally a limiting factor to photosynthesis in the field. Light intensity is directly correlated with photosynthetic rate up to full sunlight. Cloudy weather, however, frequently reduces light intensity by 20 to 70%, and the photosynthetic rate drops accordingly. A prolonged period of cloudy skies should, therefore, create photosynthetic stress, predisposing sorghum to root rot. Light reduction also occurs through plant competition from narrow rows, or crowded adjacent plants within a row. Mortimore and Gates (1969) have shown that, when adequate water is available, stalk rot increases in maize planted at higher densities, because of the shading effect.

Mineral Deficiencies

The mineral requirements considered optimum for grain yield can be expected, in general, to increase photosynthetic rate. Potassium availability appears to be particularly important in stalk rot interactions. In maize, low potassium and high nitrogen availability is associated with stalk rot (Josephson 1962). Addition of potassium to potassium-deficient soils decreased stalk rot and lodging in sorghum (Murphy 1975). Potassium is known to be involved with senescence and apparently acts on carbohydrate production (Liebhardt 1968).

Translocation Balance and Predisposition to Stalk Rots

Removal of the grain head in sorghum reduces the rate of senescence in stalk pith tissue (Papellis & Katsanos 1966) and reduces the spread of *Colletotrichum graminicola* from the site of inoculation in the stalk (Katsanos and Papellis 1969). *Macrophomina phaseolina* does not spread in sterile sorghum plants, but does in fertile plants treated with the same postflowering stresses (Edmunds & Voigt 1966; Odvody & Dunkle 1979). Edmunds and Voigt (1966) also found that plants with incomplete pollination had an intermediate level of charcoal rot. Dodd (1977) found, in comparisons of 110 pairs of maize plants, that the prematurely dead plants had 20.6% more kernels than did adjacent live plants. Similar comparisons with sorghum show the same relationship (J. Dodd unpublished). Clearly, filling of the grain head is involved with predisposition to root and stalk-rot development, and factors influencing the size of the grain sink need to be considered.

Studies with maize (Johnson & Tanner 1972) indicate that the number of pollinated ovules is critical in determining the amount of carbohydrate deposited in the grain of a given genotype. It appears that each grain pulls carbohydrate at a particular genetically determined rate per day, almost regardless of kernel number or stress on the plant, until normal abscission layer development or plant death. This translocation pattern is implied in maize (Johnson and Tanner 1972), wheat (Wardlaw 1967), and sorghum (Kaigama et al. 1977).

Kaigama et al. (1977) showed that dry-matter accumulation in sorghum panicles remained constant from bloom until black-layer formation, but dry-matter accumulation in roots, stems, and leaves dropped. Nonirrigated plants, however, had a greater reduction in dry-matter accumulation in these tissues and an earlier completion of dry-matter accumulation in the panicle, resulting in less weight per kernel. Possibly, translocation to the panicle stopped sooner because of premature death (wilting) from root rot in the nonirrigated plot. Wardlaw (1967) found nearly the same result with wheat, in which translocation to the grain remained the same in stressed as in nonstressed plants, but lower internode and tiller

weights dropped. The root and crown translocation was reduced by 50% when stressed during this postflowering period.

The number of kernels multiplied by rate of translocation to each kernel determines the size of total grain sink. Although both of these characters are genetically influenced, kernel number is more greatly influenced by environment. A genotype appears to have a rather narrow range in rate of fill per kernel, but a wide range of kernel number capacity. The preflowering environment greatly influences kernel number, and therefore influences the translocation balance and predisposition to root and stalk rot.

Differentiation and the beginning of establishment of the panicle occurs about 36 days after seedling emergence (Vanderlip 1972). The environment from that time through bloom establishes the number of kernels per panicle. Sorghum is most vulnerable to yield loss when undergoing water stress during the boot to bloom stage (Lewis et al. 1974; Salter and Goode 1967). This is apparently due to reduction in kernel numbers, rather than reduced weight per head. Pepper and Prine (1972) found that artificially shading sorghum plants 2 wk prior to 50% anthesis decreased kernel numbers by 40 to 60%.

Therefore, cloudy weather during this time could have a major effect on establishing kernel number and the size of grain sink. Water availability, light, and other preflowering environmental factors interact with each genotype's physiology to establish the final grain-sink size.

The predisposition of sorghum plants to root and stalk rots is influenced by preflowering and postflowering environments. Genotypes will respond to these environments in different ways. Some varieties will react to excellent preflowering environment by establishing an exceptional grain sink that can be supplied only by excellent postflowering conditions, producing adequate carbohydrate to maintain the roots, and still meet the demands of the grain sink. Such a variety will probably exhibit outstanding yields in this situation. However, if this genotype is unable to meet these grain sink demands because of postflowering stress, root rot and consequent loss of yield from light grain weight and lodging will result. In the latter situation, a genotype that does not establish as large a grain sink in response to the excellent preflowering environment may stand better and have more yield because of greater fill per kernel under the postflowering environmental stress.

The interaction between a genotype's ability to establish high yield potential (i.e., large grain sink), and its consequent vulnerability to root and stalk rots presents an enigma that sorghum breeders must consider. Protection against some stresses can be relatively easily obtained through resistance to leaf diseases, viruses, downy mildew, and leaf-feeding insects. Genotypes vary in their ability to cope with low soil-water potential with differences in root size, leaf structure, and cellular physiology. We need to identify these differences and find practical means of screening for genotypes that are less affected by drought stress.

The combination of high yield potential and resistance to root and stalk rots of sorghum will come from genotypes that are most energy efficient. These genotypes will combine higher rates of photosynthesis per unit area of leaf, more effective leaf area per plant, complete leaf canopy, efficient use of maintenance energy, large grain sinks, and efficient root systems. This obviously multigenically inherited system will be difficult to select, but probably will be constructed over many years through cooperative contributions from many disciplines.

Meanwhile, pathologists and breeders need to utilize schemes such as that used by Texas A & M University (Rosenow 1977) for artificially applying stress and identifying those genotypes that manage the most yield and least stalk rot. Other schemes will also be devised, but they must be consistent with the kinds of environmental stresses in the farmers' fields. Results of these screening methods should be evaluated in a manner consistent with the photosynthetic stress-translocation balance concept.

Cultural Control of Stalk Rots

Cultural practices can make important contributions to the reduction of root and stalk rots of sorghum. Irrigation practices, if available, can greatly influence the stalk rot pattern. A large preflowering watering and poor postflowering watering should be avoided. Plants should have adequate water for photosynthesis until normal

abscission layer formation 30 to 40 days after half bloom. Water-retention capabilities can be increased by certain cultural practices, which therefore reduce the severity of water-stress climates. Fertilizer imbalances, particularly with high nitrogen and low potassium, should be avoided.

Implications for International Sorghum Breeding Programs

1. Resistance breeding must be carried out in basic total-performance breeding programs.
2. Care must be taken to select for both high yield and good stalk maintenance.
3. Evaluations must be undertaken in environments that are most likely to occur in the farmers' fields.
4. Inoculations may be useful but are of little use if the grain development, maturity, or stresses are ignored.
5. Wet preflowering and stressful postflowering environments promote the most stalk rots.
6. Single plant selections are difficult.
7. Inbred reactions may not indicate hybrid reactions; both must be evaluated.
8. Inheritance of resistance involves inheritance of stress resistance, morphological and physiological factors affecting photosynthetic rates, and panicle development.
9. Visual evaluation of senescence may be the most efficient means of resistance selection, but must be related to maturity of grain development.
10. As attempts to improve yield continue by genetics or cultural practices, vulnerability to stalk rots will need to be screened.

Sorghum pathologists can make large contributions to breeders' and farmers' efforts to reduce damage from root and stalk rots by identifying the specific stresses predisposing sorghum plants to the rots and developing methods to overcome these stresses.

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Stalk Rot Resistance Breeding in Texas

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Stalk rots are a serious disease problem in sorghum. Stalks weakened by rots lodge easily, with loss in harvestable grain. Also, stalk rots may cause premature plant death before grain is physiologically mature, curtailing grain yields. Stalk rots are often associated with environmental and pest stresses, such as those caused by drought, greenbugs, and mites. These factors are common and are thus a threat in the sorghum-producing areas of Texas and other locations in the USA.

Since lodging and stalk rots are often related, the term lodging needs further explanation. The terms "stalk strength," "stalk quality," and "lodging resistance" have all been used in the literature and are basically identical. The term "lodging" refers to any bending or breaking of any portion of the stalk in such a way as to interfere with normal harvest, or cause loss of grain, or both. Lodging can be the result of one or more plant, environmental, pest, or disease factors.

Lodging in sorghum is often associated with stalk rots. Considerable research is reported on the causal factors and the relationship between stalk rots and lodging — especially with respect to charcoal rot (Edmunds 1964a, 1964b; Edmunds et al. 1965, 1970, 1973; Edmunds and Zummo 1975; Hoffmaster and Tullis 1944; Hsi 1961; Malm and Hsi 1965; Voight and Edmunds 1970). The major stalk rots of the Great Plains are charcoal rot (*Macrophomina phaseolina* (Tassi) Goid.) and *Fusarium* stalk rot (*Fusarium moniliforme* Sheld). In the humid southern areas, red rot (*Colletotrichum graminicola* (Cesati) Wilson) is important, although *Fusarium* stalk rot can also be severe. Research has shown that charcoal rot develops only in plants that have been predisposed by moisture stress during the late stage of grain development and is especially severe when moisture stress is accompanied by high temperatures (Edmunds et al. 1965; Edmunds 1964b). Techniques to screen for charcoal rot were developed by Hsi (1961), Malm and Hsi (1965), and Edmunds (1964a; 1964b; Edmunds et al. 1965). The techniques involve either artificial or natural (dry-climate) moisture stress during the grain-development stage, combined with infected-toothpick inoculation of the stalk. Conditions favoring *Fusarium* stalk rot are less well understood. It is usually most severe when cool wet weather follows hot dry weather (Edmunds and Zummo 1975).

Root rots are also involved in the stalk rot-lodging problem (Edmunds et al. 1973; Johnson et al. 1968). *Pythium* sp appeared to cause extensive lodging and serious grain loss in northwestern Texas in 1971 (Edmunds et al. 1973). Weak neck is generally considered to be a nonparasitic disease associated with a weakness at the base of the peduncle (Edmunds and Zummo 1975). However, Frederiksen et al. (1973) and Zummo and Frederiksen (1973) reported that when *Fusarium* head blight is severe and the rot progresses down the stalk, it can result in weak neck and stalk lodging.

Insects such as the greenbug, are important in predisposing sorghum plants to stalk rots, but little research has been done on this aspect. Teetes et al. (1973) showed that charcoal rot was more severe following toothpick inoculation in greenbug-infested plots. In 1978, Johnson (personal communication) found that under natural conditions more charcoal rot developed in greenbug-susceptible hybrids than in resistant hybrids under high greenbug numbers and under moisture stress. The Banks grass mite and the sugarcane root stalk weevil are also thought to contribute to stalk rot.

Several older reports indicate differences among sorghums in resistance to stalk rots (Edmunds et al. 1965; Frederiksen and Rosenow 1971; Malm and Hsi 1965; Tarr 1962; Voight and Edmunds 1970). However, none of the lines possessed a sufficiently high level of resistance to contribute substantially to im-

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proved stalk rot-resistant types. New Mexico-31 was the first sorghum line developed and released primarily for its charcoal rot resistance.

In maize, Zuber (1973) reported generally positive results in improving stalk strength through the use of various stalk-strength selection techniques.

Research on stalk-strength measurements of sorghum was first reported by Al-Tayar (1974) and Schertz et al. (1978). These workers evaluated crushing strength, penetration pressure, shearing strength, and bending pressure of stalks at four positions, and related them to field lodging. They found that the second internode above the ground and the base of the peduncle were the best positions to estimate stalk strength. Bending of dry plants and green stalk penetration appeared to be the most promising, although some significant correlations were obtained from each of the testing metnods. Bashford et al. (1976) studied mechanical properties affecting lodging in sorghum. They found that genotypes classified as lodging-resistant required more force to cause lodging and were generally shorter and stockier. They also showed that leaf sheaths provided considerable support, and that plants acquired maximum lodging resistance just prior to physiological maturity. Esechie et al. (1977) found that lodging resistance was associated with larger diameter of basal internodes, shorter peduncles, shorter plant height, higher weight of basal stalk and peduncle sections, and a thicker rind. Resistant lines were later maturing, appeared to be more perennial in habit, and contained higher total nonstructural carbohydrates but lower stalk potassium and protein.

Anatomical variation in sorghum stalk internodes was studied by Schertz and Rosenow (1977). Large differences were found in the number of cells with lignified walls and in wall thickness. Differences were also present in the degree of lignification in the epidermis, subepidermis, and vascular bundles. Although no attempt was made to correlate the differences with lodging resistance, it appeared that resistant lines generally had more lignification than susceptible ones.

In 1972, Rosenow (1972) reported promising preliminary results in selecting for lodging and charcoal rot resistance. In 1977, reports by Rosenow (1977) and Rosenow et al. (1977) indicated excellent progress in selecting for resistance to charcoal rot and lodging in Texas. The use of ratings on the degree of plant nonsenescence (made on plants under moisture stress during the late grain-development stage) to predict subsequent lodging and charcoal rot was discussed by Rosenow et al. (1977). They found significant correlations between nonsenescence, lodging resistance, and charcoal rot resistance. Duncan (1977) described some characteristics of nonsenescence and found that nonsenescing lines had higher leaf-blade chlorophyll content and 26% higher leaf-area duration than did senescing lines. Dodd (1977) reported a photosynthetic stress-translocation balance concept of corn stalk rot. Katsanos and Pappelis (1965) discussed the relationship of senescing tissue and stalk rots. These concepts appear quite compatible with our observations regarding the relationship between nonsenescence and stalk rot resistance in sorghum.

Screening and Evaluation

The screening and evaluation techniques we use at the Texas Agricultural Experiment Station have proven effective in improving resistance to several types of stalk rot. The major features of the program are: (a) initial identification of potential lodging resistance by any worker in any nursery, (b) initial screening in single-row observation plots in a lodging nursery left standing over winter, or allowing an entire breeding nursery to stand over winter, (c) screening in replicated trials at several locations for charcoal rot as well as for several types of lodging.

The initial screening phase is primarily for resistance to after-freeze stalk breakage and weak neck resulting from strong winds (often exceeding 80 kmph) during the winter months. Lines or hybrids with good resistance to this type of lodging are then entered in replicated trials throughout Texas, where they are exposed to root-lodging pressure, moisture stress, stalk or root rots, and any other natural diseases or insect pests. They are also planted in charcoal rot-inoculated screening nurseries in West Texas. In these charcoal-rot lodging nurseries, we use basically the procedure of Hsi (1961), Edmunds (1964a, 1964b), and Edmunds et al. (1965). Ideal growing conditions are main-

tained during early plant growth, especially regarding moisture availability. However, as the plants near flowering, irrigation is withheld in an attempt to induce moisture stress during the late grain-development stage. Stress during this period predisposes plants to charcoal rot. Toothpicks infected with the causal organism, *M. phaseolina*, are inserted into an internode area of the stalk, usually 2.5 to 5 cm above the soil surface. We try to inoculate 2 weeks after flowering, but the timing is apparently not critical as long as it is after flowering and before physiological maturity. We usually inoculate five plants in each of three replications. After 3 to 4 weeks or later, inoculated stalks are split and the stalk disintegration or charcoal rot invasion is rated on a 1-to-5 scale, where <1 = less than one internode affected, 1 = one internode invaded, but rot does not pass through any nodal area, 2 = two internodes, 3 = more than two, 4 = more than three internodes invaded, sometimes with sclerotia, 5 = extensive invasion, shredding, death, and sclerotia.

Data taken on the replicated tests include flowering date, plant height, head exsertion, and desirability (an estimate of yield). Lodging notes, recorded as percentage of lodged plants, are taken periodically through the season whenever significant lodging occurs. A leaf and plant death or senescence rating is also recorded when plants are under significant moisture stress during the late grain-development stage. Under these conditions, nonsenescence has been found to be highly correlated with resistance to both lodging and charcoal rot. Two new screening techniques that were tried the past 2 years are infrared aerial photography using the technique described by Blum et al. (1978), and puncture pressure at base of green stalks in the field.

Results and Discussion

Breeding Lines

Excellent progress has been made in breeding for sorghum lines with improved charcoal rot and lodging resistance. Data from the 1975 and 1976 Statewide Lodging Test (SLT) are summarized in Table 1. In addition to the vast improvement in lodging resistance in the 20 resistant lines compared with the five standards, the average charcoal rot rating was also much lower —1.3 compared to 3.3. Note that average flowering dates are not sufficiently different to account for such dramatic differences in lodging percentage and charcoal rot ratings.

Lodging and charcoal rot ratings for 13 of the best source lines for resistance to lodging and charcoal rot, along with five check varieties, are presented in Table 2. The line New Mexico-31 was released as a charcoal rot-resistant line (Malm and Hsi 1965). However, all of the 13 lines had lower charcoal rot ratings than New Mexico-31. All but one of the 13 lines were derived either directly or indirectly from lines developed in the sorghum-conversion program (Stephens et al. 1967). Several reports name sorghums with some degree of charcoal rot resistance (Edmunds et al. 1965; Frederiksen and Rosenow 1971; Malm and Hsi 1965; Tarr 1962; Voight and Edmunds 1970), but their

Table 1. Summary of agronomic, lodging, and charcoal rot data from the Statewide Lodging Test, 1975–1976.

	1975				1976	
					Lodging (%)[a]	
Entries	Date of 50% bloom	Lodging[a] (%)	Charcoal rating[a]	LPD rating[a]	2/10	3/8
Research lines (20)	8/14	9.3	1.3	2.8	0.5	13.8
Standard (5)	8/13	64.6	3.3	3.4	68.1	90.7

a. Flowering data, charcoal, and LPD from Halfway, Lodging from Lubbock and is total lodging with data taken late winter or on date indicated.

Table 2. Charcoal rot and lodging of selected sorghum lines, Lubbock and Halfway, Texas, 1973–1977.

Designation	Charcoal rot rating[a]					Lodging (%)[b]
	1973 Lubbock	1975 Lubbock	1975 Halfway	1976 Halfway	Avg.	1973–1977 Lubbock
SC35-6 (IS12555 der.)	0.8	1.1	2.7	1.5	1.5	2.8
SC56-6 (IS12568 der.)	0.6	0.7	2.2	1.9	1.4	5.6
SC56-14 (IS12568C)	0.4	0.6	0.7	–	0.6	2.8
SC170-6-17 (IS12661 der.)	0.6	0.7	1.7	1.2	1.1	39.4
SC-599-6(9188)(IS17459 der.)	0.8	0.6	1.8	1.4	1.2	34.6
SC-599-6(9247)(IS17459 der.)	0.6	0.7	0.8	1.0	0.8	11.0
NSA-440 (SS Kaf der.)	0.9	1.4	1.3	0.3	1.0	3.0
1790E (56 × 33) der.	–	0.9	2.4	1.4	1.6	19.3
1790L (56 × 33) der.	1.0	0.8	0.8	2.1	1.2	3.6
1778 (56 × 33) der.	0.9	0.4	0.6	–	0.6	10.2
SC-326-6 (IS-3758 der.)	0.4	0.5	1.7	–	0.9	1.8
B-4R (B406 × Rio) der.	0.5	0.5	1.9	0.9	1.0	3.8
R-T584 (56 × 170) der.	0.6	0.8	0.8	–	0.8	2.0
New Mexico-31	1.7	0.9	2.7	1.4	1.7	54.0
B-Tx-378 (Redlan)(IS413)	2.4	1.3	3.9	1.5	2.2	89.8
Tx-7000 (Caprock)(IS410)	–	2.5	3.3	2.2	2.7	90.3
B-Tx-399 (Wheatland)(IS896)	1.6	1.1	2.5	1.7	1.7	65.2
TAM-428 (IS12610 der.)	1.8	3.0	3.5	–	2.6	86.4

a. Rated on 1–5 scale: <1 = < one internode, 1 = one internode, 4 = more than three internodes, 5 = death.
b. Lodging rating taken late in winter following exposure to strong winds.

resistance has not been sufficiently high to be very useful. It appears that several lines listed in Table 2 have charcoal rot resistance superior to any previously reported lines. The 13 lines either have been released or are in the process of being released as germplasm-source lines for use in breeding for charcoal rot and lodging resistance.

Although these lines were selected for resistance to charcoal rot and various types of lodging, we believe they also possess resistance to *Fusarium* stalk rot and other stalk- and root-rotting organisms associated with stress in Texas. Confirmatory evidence for this is lacking, but our conclusion is based on the overall lack of any stalk or root rot or deterioration in these charcoal rot-resistant lines in the various lodging nurseries.

In our screening process, most newly identified lodging-resistant lines are first entered in the Preliminary Lodging Test (PLT) grown only on the High Plains. Entries with sufficient promise are then moved to the Advanced Lodging Test (ALT), which is also grown at a few other key locations over Texas—such as at Chillicothe and Beeville. The Statewide Lodging Test (SLT) contains only the most promising and well-tested lines, and is grown at approximately 15 locations over Texas. These three tests contain only lines or varieties, not hybrids.

Hybrids

Observation of hybrids in screening tests has shown that hybrids are more susceptible to charcoal rot and lodging than are breeding lines. It also appears that resistance is recessive in nature. This means that a hybrid with good stalk quality must have resistance in both parents. As potentially desirable lodging- and stalk rot-resistant lines are identified, they are crossed to male steriles to determine their fertility-restoration reaction. If they are nonrestorers (B-lines), they are backcrossed until a

suitable A-line counterpart has been developed. Several male-sterile lines with charcoal rot resistance have been developed. Hybrids where both parents have resistance were first evaluated in the Hybrid Lodging Test (HLT) in 1975. The HLT is grown in the lodging-screening nurseries on the High Plains as well as at other key Texas locations.

Data on selected hybrids for 1975 and 1976 are presented in Table 3 and Table 4, respectively. Hybrids with one or more resistant parents generally had less lodging, lower charcoal rot ratings, and lower senescence (LPD) ratings than did the standards (the last four entries in Table 3 and the last five in Table 4). However, differences were less than between lines in the SLT. Based on lodging, charcoal rot, and senescence ratings, many hybrids with only one resistant parent perform much like the susceptible parent. However, in a few lines — such as SC-35-6 and 1790 — resistance appears to have more dominance. Excessive height and head exsertion contributed to lodging problems of some hybrids. The maturity and yield of these hybrids is comparable to standard commercial hybrids.

Nonsenescence

Senescence (leaf and plant death — LPD) ratings made on plants under moisture stress during the late grain-development stages appear to be a good indicator of subsequent lodging and charcoal rot. Rating is on a 1 to 5 scale, where 1 = completely green, and 5 = dead. Highly significant positive correlation coefficients of about r = 0.7 were obtained between such ratings and both lodging and charcoal rot (Table 5, 6). The low correlation between LPD and lodging in the 1976 SLT at Lubbock indicates that the relationship is not good if the nursery is not under sufficient moisture stress. Senescence ratings can be a valuable breeding tool in any breeding nursery under late-season moisture stress. They are quick and eliminate the necessity of leaving a nursery stand for long periods, or for time consuming charcoal rot inoculations. Such selection is, in fact, selecting for late-season drought tolerance. One of the most highly nonsenescing lines, SC-599-6, a Rio derivative, also has the highest level of *Fusarium* head blight resistance (Frederiksen et al. 1973). Non-

Table 3. Agronomic, lodging, and charcoal rot data from the Hybrid Lodging Test, Lubbock and Halfway, Texas, 1975.

Hybrid		Date of 50% bloom	Plant height (cm)	LPD rating[a] Half.	Charcoal rating[a] Half.	Lodging (%)[b] Lub.	Yield (kg/ha) Lub.
A599 X SC56-14	RxR[c]	8/7	140	3.4	2.8	67	7120
A618 X SC56-14	SxR	8/4	151	3.0	1.9	92	7430
A599 X NSA440	RxR	8/11	125	3.3	3.8	61	6690
A1887 X NSA440	RxR	8/11	112	3.1	3.3	26	6690
A618 X NSA440	SxR	8/6	126	3.5	4.6	79	7430
A618 X SC35-6	SxR	8/5	149	2.9	3.5	91	7120
A618 X 1790L	SxR	8/6	138	3.0	2.3	47	6910
A618 X SC599-6	SxR	8/3	137	4.2	4.8	94	6590
A599 X Tx7000	RxS	8/4	130	4.5	4.3	95	6120
A599 X Tx2536	RxS	8/3	130	4.7	4.0	69	6800
RS671	SxS	8/3	128	4.1	4.0	99	7010
TAM 680	SxS	8/3	152	4.3	3.1	100	7320
A399 X Tx2536	SxS	8/3	115	4.3	4.3	98	5960
A378 X Tx2536	SxS	8/4	130	3.5	4.4	100	6180

a. Rated on 1–5 scale: 1 = none, 5 = dead.
b. Lodging data taken Feb. 1976.
c. Parental lines rated as R = lodging resistant, S = lodging susceptible.

Table 4. Agronomic, lodging, and charcoal rot data from the Hybrid Lodging Test, Lubbock and Halfway, Texas, 1976.

Hybrid		Days to 50% bloom	Plant height (cm)	LPD rating[a] Half.	Charcoal rating[a] Half.	Lodging (%)[b] Lub.	Yield (kg/ha) Lub.
A35 X SC56-14	RxR[c]	70	115	1.9	1.1	2	5910
A599 X SC56-14	RxR	69	135	1.8	1.0	73	6940
A399 X SC56-14	SxR	69	123	1.9	1.6	65	5010
A618 X SC56-14	SxR	65	165	2.1	0.9	96	6040
A35 X SC599-[illegible]8)	RxR	69	129	2.[illegible]	1.0	13	6050
A618 X SC599-6(88)	SxR	66	144	3.3	1.9	88	5500
A1778 X SC599-6(47)	RxR	70	116	1.9	1.0	15	5940
A1778 X SC170-6-17	RxR	69	126	2.1	0.7	58	4950
A599 X NSA440	RxR	70	122	2.2	1.1	22	5910
A4R X NSA440	RxR	70	102	2.0	0.9	3	5770
A618 X NSA440	SxR	69	124	2.2	1.5	72	5500
A399 X 1790L	SxR	72	123	1.6	0.8	5	3990
A399 X 1790E	SxR	65	119	2.0	1.1	42	6600
A599 X SC326-6	RxR	70	125	2.0	0.7	20	5770
A599 X TAM428	RxS	69	130	3.0	2.8	100	6190
Commercial		68	117	2.8	2.0	98	5910
TAM 680	SxS	69	110	3.7	3.0	96	4950
A399 X Tx2536	SxS	68	108	2.7	1.7	99	5220
A378 X Tx2536	SxS	66	143	2.7	2.7	100	5360
A378 X Tx7000	SxS	71	133	3.2	2.1	100	6590

a. Rated on 1–5 scale: 1 = none, 5 = dead.
b. Lodging data taken Feb. 1977.
c. Parental line rating of R = lodging resistant, S = lodging susceptible.

Table 5. Correlation coefficients of senescence (LPD) rating with lodging and charcoal rot.

Test, year, and location[a]	Lodging Halfway	Charcoal Halfway	Lodging Lubbock	Charcoal Lubbock
HLT-75-H-LPD	0.74**	0.78**	–	–
HLT-75-L-LPD	–	0.68**	0.55**	0.70**
HLT-76-H-LPD	0.78**	0.73**	0.73**	–
SLT-75-H-LPD	–	0.55**	0.66**	–
SLT-76-H-LPD	–	–	0.70**	–
SLT-76-L-LPD	–	–	0.37	–

*, ** significant at 0.05 and 0.01, respectively.
a. HLT = Hybrid Lodging Test; SLT = Statewide Lodging Test. 75 = 1975, 76 = 1976. LPD = Leaf and plant death rating.

senescing lines also have rather good Bank's grass mite resistance (Foster et al. 1977).

Infrared Aerial Photography

Correlation coefficients in Table 6 show that infrared ratings that measure differences in plant canopy temperatures on individual plots were not associated with senescence or lodging.

Puncture Pressure

In 1976, plants at physiological maturity in a random-mated population were punctured with a 1/16-inch bit in a hand-held penetrometer. The puncture was in the first obvious internode, or

Table 6. Correlation coefficients among six characteristics, Statewide Lodging Test—1976.

Trait	Days to 50% bloom	Plant height	Infrared	Leaf, plant death	Puncture pressure	Lodging (%)
Days	–	−.47*	−.23	−.47*	.64**	−.60**
Height		–	.22	.40	−.29	.60**
Infra red			–	.18	.08	.20
Plant death				–	−.52*	.70**
Puncture pressure					–	−.71**
Lodging (%)						–

* *and* ** significant at 0.05 and 0.01, respectively.

about 2.5 to 5 cm above the soil line. Seven percent of plants with the highest pressure were selected and random mated. Comparisons in 1977 indicate that the selected population exceeded the original population by an average of 0.9 kg of pressure to penetrate each stalk (11.16 to 10.26).

Also in 1976, we punctured each line in the Statewide Lodging Test and obtained a highly significant negative correlation ($r = -.71$) with lodging (Table 6). The average pressure for the five check lines was 8.91 kg, while that of the 20 lodging-resistant lines was 10.85 kg.

Insect Damage

The greenbug and Bank's grass mite often create serious lodging and stalk rot problems. Resistance to these pests has been found, and can increase the level of overall stalk rot resistance.

Inheritance of Resistance

We have not done inheritance studies on stalk rot resistance. F_1 data in (Table 3, 4) indicate that in most lines resistance tends to be recessive, while in a few lines it is more dominant. In F_2 populations the influence of lodging-resistant parents is very obvious. In either F_2 populations in each of three groups, classified by their parental reactions (Res × Res × Sus, and Sus × Sus), the average lodging was 3.5, 16.8, and 49.4%, respectively. Lodging resistance as we measure it is indeed heritable, but not by a single gene as Coleman and Stokes (1958) reported in sorgo.

Maturity and Height

Plant height and maturity are important considerations in breeding for stalk rot and lodging resistance, since resistance is often related to short stature and late maturity (Table 6). By plotting a curve, selection could be done only within heights or maturities. We have not done this, but we attempt to subjectively exert selection pressure for earliness and away from extremely short plants with little head exsertion.

Summary

Although stalk rot resistance is a complex phenomena, much progress has been made in breeding for higher levels of resistance using proper screening techniques. Our technique of first selecting for resistance to after-freeze stalk breakage, followed by screening for moisture stress-charcoal rot type lodging resistance has worked well in breeding for charcoal rot resistance. I believe we have made progress in two areas: we have selected for anatomically stronger plants, and we have selected for plants that have a different physiological response under moisture stress. These plants do not become predisposed to stalk rot susceptibility by moisture stress as easily as common sorghums. Such lines, in fact, have tolerance to late-season drought stress.

Portions of this program could well be adopted by other sorghum breeders. A promising selection technique that can be used anywhere moisture stress is common is the use of leaf and plant death (nonsenescence) ratings.

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The ICRISAT Charcoal Rot Resistance Program

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Charcoal rot caused by *Macrophomina phaseolina* (Tassi) Goid. is a potentially important disease of sorghum in many parts of the world. It is severe on sorghum that is filling grain during hot dry weather, particularly if the crop is subjected to moisture stress. There is evidence that the incidence of the disease is related to stress factors connected with the translocation of carbohydrates from the stalks to the grain, and for this reason the disease appears to be more important in high-yielding grain sorghums than in lower yielding materials. Although charcoal rot was acknowledged as an important disease of sorghum, research activity on its control did not begin until 1977, as grain mold problems kept the limited staff busy until that time.

Objectives

The objectives of the project at ICRISAT are to:

1. survey the extent of yield losses and the affected areas in the SAT;
2. understand the factors influencing the disease;
3. standardize field-screening techniques;
4. identify sources of resistance in the germplasm and other breeding lines;
5. breed for broad-spectrum stable resistant lines in elite agronomic backgrounds; and
6. investigate the inheritance of charcoal rot resistance.

Geographical Distribution

The charcoal rot organism is widely distributed in tropical soils and is known to infect a wide variety of crop species (Dhingra and Sinclair 1977). Charcoal rot is most severe in susceptible cultivars when grain filling coincides with periods of high soil temperatures (35°C) and moisture stress (<25% available soil moisture) (Hsi 1967; Edmunds 1962).

Symptoms

The most common symptoms in sorghum are poor grain filling, premature leaf senescence, and crop lodging. Internally the stem pith of infected plants becomes disintegrated; the separated fibro-vascular bundles are covered with the small black sclerotial bodies of the fungus which give the stem a blackened appearance, hence the name "charcoal rot." The only feasible means of control of this disease in the SAT is the use of resistant cultivars.

Screening Methods

The basic requirement of any resistance-identification program is a meaningful and effective screening technique. Screening methods and programs for charcoal rot have been reported by Uppal et al. 1936; Hoffmaster and Tullis 1944; Karper 1949, 1953; Edmunds 1962, 1964; Malm and Hsi 1964; Edmunds and Voigt 1966; Hsi 1967; and Anonymous 1977. In this paper we give an account of the program for charcoal rot resistance at ICRISAT, in which we present details of the wide range of cultivar reactions encountered in our field-screening programs using toothpick inoculations and induced drought stress, and describe the nature of breeding material generated from the beginning of the program in postrainy season 1977.

Resistance Screening at ICRISAT Center

Inoculum Preparation

The pathogen was cultured on wooden toothpicks in honey-peptone medium (peptone 1 g, honey 5 ml, distilled water 94 ml). Toothpicks

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were packed, pointed ends up, into wide-mouthed screw-capped bottles, and were sterilized at 15 psi for 20 min. Two loops of a mycelial-sclerotial suspension made from stock cultures of *M. phaseolina* were seeded into each 100 ml of sterilized cooled honey-peptone medium. The medium was shaken thoroughly to allow even inoculum distribution and poured under aseptic conditions (using a laminarflow clean-air chamber) into the wide-mouthed bottles (about 20 ml/bottle), containing the sterile toothpicks so that the level of medium in the bottle covered about one-third of the length of the toothpicks. The bottles were incubated at 35°C for 7 days at which time the toothpicks were covered with mycelia and sclerotia of the charcoal rot fungus and ready for use in inoculation.

Field-inoculation Procedure

Plants were inoculated 2 to 4 weeks after 50% flowering. Irrigation was withheld when the majority of the cultivars were at the boot-leaf stage (this was during the postrainy season, and moisture stress was easily controlled by withholding or applying irrigation). A fungus-infected toothpick was inserted obliquely into each stalk at its second internode; the toothpick was inserted into a hole punched into the stalk with a thin iron needle. Care was taken to ensure that the toothpick did not emerge through the other side of the stem, for this would promote rapid drying of the inoculum. Between 20 and 34 plants of each entry, in a single plot with no replication, were inoculated.

Cultivars Tested

A total of 517 sorghum elite cultivars, hybrids, and male-sterile maintainers — based on their eliteness to various characters such as yield and resistance to drought, disease, and insects — were selected for testing.

Evaluation of Reactions and the Results

Cultivars were evaluated for resistance at physiological maturity. The stalk of each plant of each cultivar was first squeezed to determine if the internal structure was destroyed, as would be indicated by a soft stalk. The stems were then split open so that the extent of fungal colonization of the stem, as indicated by the number of nodes crossed and the total length of visible fungal colonization, could be measured.

As indicated, three infection parameters were measured on each plant of each entry — presence or absence of soft stalk, number of nodes crossed, and distance of spread of disease from the toothpick. Lines that had no plant with soft stalk, no plant with nodes crossed, and a mean symptom spread of no more than 5 cm were classed as highly resistant.

In Table 1 the charcoal rot reactions and seed color are given for the best 27 lines together with those of five highly susceptible lines. Nineteen of the 27 highly resistant lines originated from the populations developed by the late Dr. R. E. Karper of Texas A & M University in the USA. The downy mildew — and rust-resistant sorghum SC-120-14 is among those lines highly resistant to charcoal rot.

None of the 15 B-lines tested, were in the highly resistant category (Table 2), but several appeared moderately resistant when compared with VZM 2-B, Maldandi-B, and 2077-B.

This is the first large-scale field screening for charcoal rot resistance at ICRISAT Center. Because of the influence of soil moisture and temperature on sorghum susceptibility to charcoal rot (Hsi 1967; Edmunds 1962, 1964), it will be important to sufficiently replicate entries in advanced screening of selected lines.

Although there is a reasonable correlation between the three infection parameters (Table 3), it seems useful when dealing with elite material to continue to record all three infection parameters and to assess susceptibility on the combination of the three.

Sorghum cultivars already reported resistant to charcoal rot are Rice kafir, Tall red kafir No. 7, Atlas sorgo 899, Wild amber sorgo, Kansas orange, African millet, Sumac 35038, Sumac 1712, Tall white sorghum 787, Corneous sorghum 6166 (Wadsworth and Sieglinger 1950); Texas black kafir (Karper 1953); and New Mexico-31 (Malm and Hsi 1964).

As with several other sorghum diseases, care must be taken to avoid the confounding of different flowering dates. For charcoal rot screening, lines of similar maturity dates should be grouped together to facilitate initiation of moisture stress at the correct physiological stage (boot-leaf).

Table 4. Charcoal rot reaction parameters and seed color of selected entries in the postrainy season 1977 field-screening trial.

Entry	Days to 50% flowering	Soft stalk (%)	Mean Nodes crossed/ plant	Mean length of sprout (cm)	Seed color
21-76	61	0	0	1.3	Yellow
25-98	61	0	0	1.4	Yellow
1-52	64	0	0	1.8	Yellow
1-30	61	0	0	1.8	Yellow
SC-120-14	62	0	0	1.8	White
4-45	64	0	0	1.9	Yellow
6-39	65	0	0	1.9	Dark cream
20-67	70	0	0	2.2	Yellow
8-55	55	0	0	2.4	Yellow
2-86	69	0	0	2.5	White
5-33	64	0	0	2.5	Yellow
4-20	62	0	0	2.6	Dark cream
(954063×CS 3541)-30	82	0	0	2.8	Dark cream
21-82	65	0	0	2.8	Yellow
(954068×CS 3541)-64	82	0	0	2.8	Pearly white
20-87	61	0	0	3.3	Yellow
IS-410	72	0	0	3.4	Yellow
18-10	70	0	0	3.6	Yellow
4-22	62	0	0	3.7	Dark cream
IS-1266-C	48	0	0	3.9	Brown
15-36	56	0	0	4.2	Yellow
IS-121	44	0	0	4.4	White black subcoat
23-94	63	0	0	4.4	Cream
(954068×CS 3541)-11	70	0	0	4.7	Cream
IS-1235	56	0	0	4.9	Dark brown
SC-120	70	0	0	5.0	Dark brown
(954068×CS 3541)-70	71	0	0	5.0	Chalky white
2-34	76	100	4.9	10.3	Yellow
IS-5622×WABC 1121	70	100	2.4	17.5	Pearly white
A-2268	77	100	4.8	22.5	Cream white
VZM-2B	76	100	2.6	29.1	Pearly white
Maladandi-B	76	100	3.0	32.2	Pearly white

Table 2. Charcoal rot reactions of 15 sorghum B-lines screened at ICRISAT Center, postrainy season 1977

Entry	Days to 50% flowering	Soft stalk (%)	Mean nodes crossed/plant	Mean length of spread (cm)
10449-B	61	9.4	0.2	5.3
10248-B	57	10.0	0.2	2.0
2219-B	63	10.0	0.2	4.9
1202-B	72	14.3	0.5	4.6
1253-B	70	16.7	0.7	5.3
10511-B	69	26.9	0.7	4.6
30-B	76	33.3	1.8	13.8
1036-B	69	42.3	1.3	8.0
10460-B	70	42.9	2.5	7.6
CK-60-B	69	48.3	2.0	12.2
3659-B	65	57.1	1.7	12.3
534-B	58	75.9	0.1	7.6
VZM-2B	76	100	2.6	29.1
Maladandi-B	76	100	3.0	32.2
2077-B	78	100	7.8	35.7

Table 3. Correlation coefficients between three charcoal rot reaction parameters.

Soft stalk value	1	1
Nodes crossed (mean no./plant)	0.76	1
Mean length of spread (cm)	0.80	0.70

The Breeding Program — A Projection

The charcoal rot resistance utilization program will involve screening and selection following the crossing in a cyclic manner. Lines successfully emerging from the initial screening will be funnelled through advanced screening in replicated multilocation trials for exposure to various populations of the pathogen, under different environments. In order to strengthen the source material, the different sources will be intercrossed, assuming that the resistant genes in various lines are different. In F_2 selection will be for agronomic traits, and the F_3 and F_4 lines will be screened against charcoal rot by inoculation. In F_5, replicated trials with inoculations will be performed. The final selections will be included in the international trials tested over many locations throughout the SAT. This forms Unit-1 activity (Fig. 1), where emphasis is on developing and strengthening the source material.

The crosses between the sources and other agronomically good lines will be handled similarly; for the most part. In the F_5, preliminary yield trials will be conducted in India. The selections will be assessed for their yield potential. Selections from this test, after seed increase, will be tested in international trials throughout the SAT. (Unit 2 activity, Fig 1).

In Unit 3, the charcoal rot resistance sources will be crossed to lines resistant to other diseases or pests. In F_3 and F_4, the lines will be screened for as many traits as possible.

Assuming that the gene number involved in the resistance is not large and the effects of genes are mostly additive, the breeding method envisaged in the scheme is pedigree. However, the breeding procedures will be appropriately modified, depending on the information we obtain on gene number, gene effects, pathogen variability, etc.

Breeding Activity

In the 1977–1978 season, lines reported to be

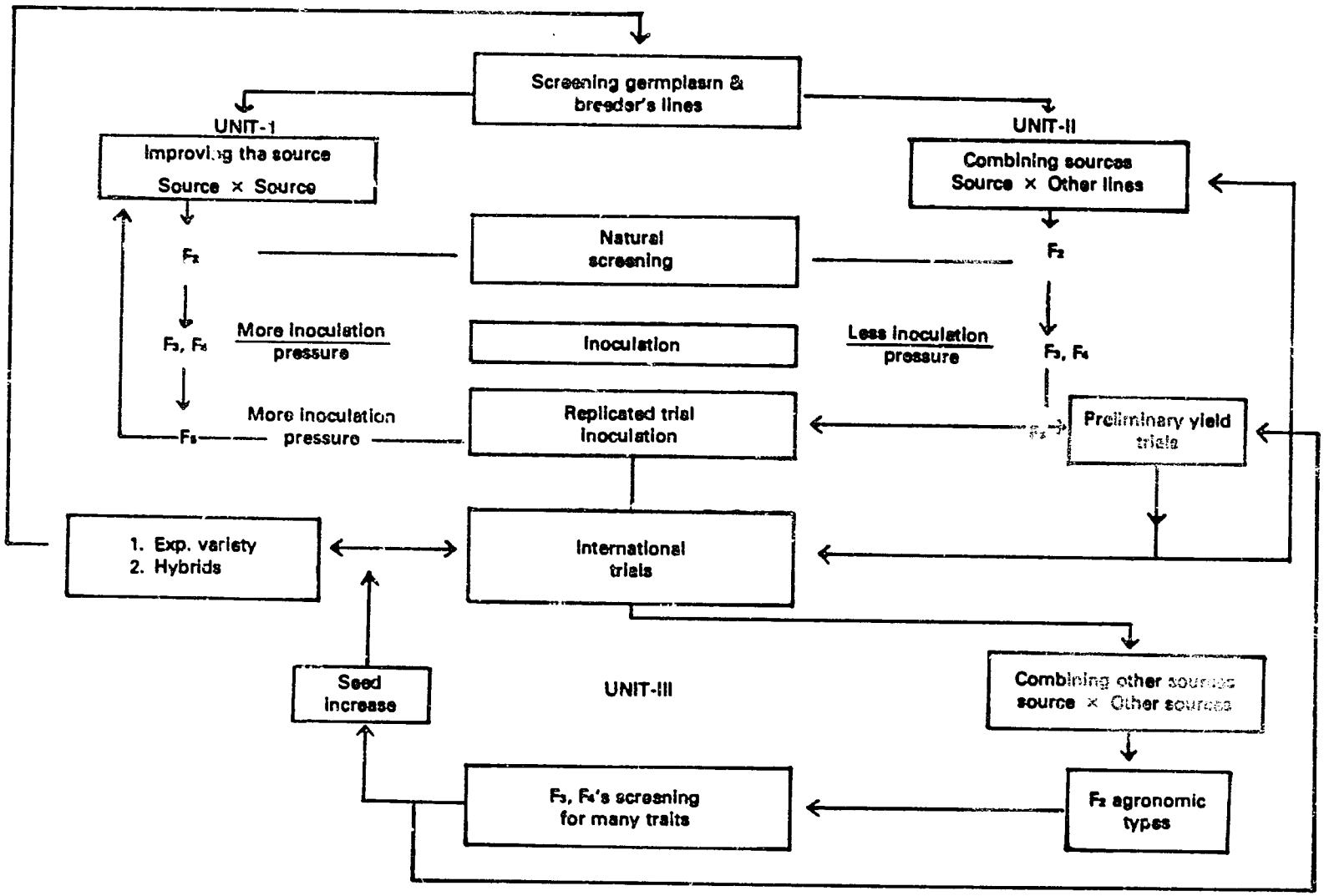

Figure 1. The projected charcoal rot resistance program at ICRISAT.

resistant in other programs were crossed. The F_1s were advanced in the summer season at Bhavanisagar (11°27'N). The F_2s were planted at ICRISAT Center, and Dharwar in the 1978 rainy season. The population size in F_2s ranged from 600 to 1000. Inoculation was not carried out. Charcoal rot did not develop, (because rains were good throughout the growing season), so selections were confined to agronomic traits. About 998 F_3 selections were made from the ICRISAT Center planting. Parents involved in these F_3s are listed in Table 4. In order to avoid the rains and encourage charcoal rot, late (Aug) plantings were made at Dharwar. However, these plantings suffered from head molds, and charcoal rot was observed only in pockets. Nearly 300 agronomically good types were recovered from this material.

In the present plantings (postrainy 1978) all the F_3s were planted in two 4-m rows with 40 plants per row. Inoculations will be carried out at the appropriate stage and the selection made for less susceptible plants among and within families.

The crosses obtained from the Bhavanisagar summer sowings were advanced to F_2 by planting at ICRISAT Center in the 1978 rainy season. Very little selection pressure was applied among the F_1s. About 472 F_2s are now planted at Dharwar as well as the ICRISAT Center. The population size is the same as the rainy season plantings. In 40 of these F_2s, both parents are less susceptible to charcoal rot. In the remaining F_2s, the second parent is drawn from across the disciplines.

The F_1s (1300) made in the postrainy season 1978 are now being advanced. The parents involved in these are listed in Table 4.

Summary

The importance of charcoal rot in high yielding dryland sorghum is increasing. The factors affecting the causal organism are briefly discussed. The objectives of the program at ICRISAT are outlined and the program for utilization of charcoal rot resistance is projected. Inoculum preparation and field inoculation procedures are described. The postrainy season 1977–78 screening results are presented. The breeding material generated from the beginning of the project in 1977–78 postrainy season is described.

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Table 4. Parents[a] involved in generating the variability planted in postrainy season 1978.

Parental traits	No. involved		
	F_3	F_2	F_1
Less susceptible to charcoal rot	35	27	51
Less susceptible to other diseases	11	7	16
Less susceptible to pests	17	11	15
Less susceptible to *Striga*	3	3	4
All India Coordinated Sorghum Improvement Project	15	13	14
Population derivatives	20	16	4
Good grain quality	5	12	3
Reverted diploids from tetraploids	3	3	3
Karper's nursery	24	16	67
Others	98	67	104

a. Some of the parents are common.

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Sorghum Stalk Rots in West Africa

J. A. Frowd*

The stalk rots of sorghum observed in West Africa can be classed into two types, according to their causal pathogen — charcoal rot, caused by the fungus *Macrophomina phaseolina*, and soft rot, caused by the fungus *Fusarium moniliforme*. Both diseases are known throughout West Africa, in a region extending from Senegal to Chad and the Central African Empire.

The recent upsurge in introduction of exotic sorghums into West Africa has been associated with observations of stalk rots. The continuing development of genetic resources to identify materials adapted to specific subregional environments in West Africa, together with a high rate of germplasm introductions, has necessitated a program of monitoring susceptibility to stalk rots. Of the two stalk rots, charcoal rot is the more important, and its discussion will form the main part of this paper.

In the present study, carried out at Kamboinse in Upper Volta, the occurrence of charcoal rot on a major scale was first noted in 1976. CSH-6 growing on a shallow lateritic soil was severely attacked; the disease appeared to be most severe on the upper parts of gently sloping toposequences; that is, where fluctuations in soil moisture are the most rapid between increasingly spaced rain-showers at the end of the rainy season. Further observations in 1977 confirmed the susceptibility of CSH-6; additionally, CSH-1 and SPV-13 were found susceptible. Conversely, resistance appeared to be present in S-29 (an improved local variety developed in Upper Volta by IRAT), in EC-64734–2 (now known as VS-701), and in 2KX-2E-21 (now known as VS-703). Field observations in 1978 gave further information on the susceptibility of certain lines, notably SPV-35, 926, and four F_2 hybrids (SC-108–3 × IS-9333, SC-108–4–8 ×SPV-9, SC-108–4–8 ×SPV-35, and SC 108–4–8 ×IS-3962). These observations were made in naturally infested plots, but many were confirmed and characterized in detail in artificial-inoculation tests.

Experimentation

Critical testing for charcoal rot resistance was carried out in two 1978 experiments grown at Kamboinse: (a) The International Sorghum Charcoal Rot Nursery (30 entries) assembled and distributed from ICRISAT Center (Hyderabad), and (b) high-yielding advanced varieties (17 entries) adapted to the Sudano-Sahelian zone of West Africa.

The objectives of the testing program were (a) identification of sources of resistance to the charcoal rot fungus found in West Africa, and (b) the ascertainment of the resistance or susceptibility of advanced materials with potential of being extended to farmers.

Materials and Methods

Inoculations were carried out using the toothpick method. Sclerotia of the fungus, collected from stem tissues of infected plants in 1977, were seeded onto a medium containing 15 g agar, 5 g honey, and 1 g potassium nitrate per liter. Toothpicks were inserted into the seeded agar; after 4 days at 32°C they were infested with mycelium. Toothpicks thus infested were inserted into the stems of 20 plants per two-row plot, 2 weeks after flowering. Plants were observed for lodging and soft stems at physiological maturity; stems were then sectioned longitudinally and the number of nodes crossed by the fungus was recorded.

Results and Discussion

Principal results are presented in Tables 1, 2, and 3. Good sources of resistance to charcoal

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Table 1. Charcoal Rot Nursery entries showing high resistance at Kamboinse.

Entry	Soft stalks (%)	Nodes crossed[a] (no)
2-86	0	0.1
4-45	0	0.2
8-55	0	0.3
1-30	0	0.3
CSV-4	0	0.4

a. Mean of 20 observations

Table 2. Entries in the advanced varieties test showing high resistance to charcoal rot at Kamboinse.

Entry	Soft stalks[a] (%)	Nodes crossed[a] (no)
NES-1077	0	0.1
SC-108-4-8	0	0.1
2219 B	0	0.1
65/30	0	0.1
CSV-1	0	0.1
VS-701	0	0.1

a. Mean of 20 observations

Table 3. Entries susceptible to charcoal rot in inoculated trials at Kamboinsé.

Entry	Soft stalks[a] (%)	Nodes crossed[a] (no)
SPV-35	45	1.9
A-2268	35	4.0
H-410	35	1.8
CSH-6	35	1.1
(IS-954068 × CS-3541)-11	25	0.7

a. Mean of 20 observations

rot — e.g., 2-86, CSV-1, and VS-701 — are available.

The extreme susceptibility of CSH-6 and SPV-35 has been confirmed under experimental conditions, in support of earlier field observations. The value of these two cultivars to farmers is in doubt. Rao and Williams (1978) have confirmed the extreme susceptibility of A-2268, whereas eight lines showed no soft-stalk symptoms at ICRISAT Center or in our studies at Kamboinse. These were 25–98, 1–52, 1–30, 4–45, 8–55, 2–86, 23–94, and (IS-954068 × CS-3541)–64. However, two lines — H-410 and (IS-954068 × CS-3541)–11 — which appeared resistant at ICRISAT Center, were among the five most susceptible lines tested at Kamboinse.

The performance of existing "elite" lines is more problematic (Table 4). In the case of SC-108–4–8, field observations have suggested that it is susceptible to charcoal rot, and when crossed with other susceptible types, e.g., SPV-35, this susceptibility appears to increase, possibly by complementary gene action. The results of toothpick screening would suggest, however, that SC-108–4–8 carries resistance to charcoal rot at Kamboinse. Certainly, further testing is required to resolve this divergence of results, taking into account the environmental conditions of the test.

Soft Rots

Fusarium stalk rot of sorghum is similar to charcoal rot, in that it requires predisposing conditions for the onset of infection. At Kamboinsé, VS-703 was observed infected by a stem rot at soil level in a field with a high water table; the field had received a heavy dose of nitrogenous fertilizer. This observation, in early August, was characterized by a soft white water-soaked pith rot, spreading upwards to the shoot tip; at the time, it appeared that inflorescence development would be inhibited.

Table 4. Comparison of charcoal rot resistances in three elite lines at Kamboinse and at ICRISAT Center.

	Kamboinse		ICRISAT Center	
Entry	Soft stalks (%)	Nodes crossed (no)	Soft stalks (%)	Nodes crossed (no)
SPV-35	45	1.9	11.5	0.5
CSH-6	35	1.1	63.3	3.5
SC 108-4-8	0	0.1	56.5	1.7

In the light of this single observation, it would be advisable to recognize the existence of *F. moniliforme* and take steps to avoid its increase in the course of normal cultural practices.

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Sorghum Head Blights and Stalk Rots Discussion Session

Anthracnose

Balasubramanian:

What happens when both *Colletotrichum graminicola* and *C. falcatum* are inoculated together onto sorghum? Is there any suppression of one by the other? Does the close proximity of sugarcane affect *C. falcatum* incidence in sorghum?

Frederiksen:

There is information in the literature on the susceptibility of sorghum to *C. falcatum.* Some workers maintain that *C. falcatum* is just a strain of *C. graminicola*. I know of no work indicating antagonism between these species, nor of sugarcane being a source of inoculum for sorghum, though it would not surprise me. These are areas of major concern to us in our research and they are receiving attention from Mr. Pastor.

Sundaram:

There are three distinct types of symptoms of foliar anthracnose on sorghum in Nigeria. When varietal reactions are reported, the symptom type should be specified. We probably need further studies on this aspect.

Frederiksen:

Variation in symptom type is important and certainly an area that needs active investigation. We are looking at the relationships between isolates and symptoms produced.

Sharma:

At Indore in India the atypical diffuse lesions with several acervuli are observed. Lines resistant to isolates producing typical symptoms can show slight incidence of the atypical symptoms. The atypical symptoms have not been observed in farmers' fields.

Brhane:

What are the different methods of transmission of anthracnose, especially from season to season? Is there a reliable method of inoculation of anthracnose to get disease reaction before flowering?

Frederiksen:

Most inoculum survives with plant debris or wild collateral hosts such as johnsongrass or other *Sorghum* spp. There are many published inoculation techniques: (a) use of leaf debris from previous year's or season's crop; (b) artificially infested grain dropped in whorls of young plants; (c) hypodermic placement of conidia in whorls; (d) toothpicks inserted into stalks, a technique that will work in dry environments.

Mengistu:

I am of the view that seeds are good means for disease distribution. In our experience in Ethiopia, most of the diseases are confined to research stations, and newer diseases, not seen earlier in farmers' fields, are prominent at these stations.

Frederiksen:

The environment and genotypes at experiment stations are generally quite different from farmers' fields. Most sorghum pathogens are widely distributed. The severe disease reactions at experiment stations are reported on new unadapted sorghums and do not necessarily reflect importation of new pathogens. Most research seed is free from those few pathogens not already distributed worldwide.

Williams:

I fully agree with Dr. Frederiksen. We must remember that susceptibility is also seedborne. When you introduce an exotic genotype to pathogen populations for the first time, you may observe very severe disease. So it is natural that in a nursery of newly introduced cultivars you can get high levels of a disease that is unimportant in local cultivars. In this case, it is the suscep-

tibility that is seed-borne and not the pathogen.

Fusarium Diseases

Girard:

There are two forms of *Fusarium moniliforme*—*F. moniliforme sensu stricto*—producing microconidia in long chains, and *Fusarium moniliforme* var. *subglutinans*—producing microconidia in clumps (more or less like *F. oxysporum*). *F. moniliforme* var. *subglutinans* is associated with pokkah boeng. Can it also be associated with root rot, seedling blight, head blight, stalk rot, or grain molds? Are the relationships between the different diseases of the "*Fusarium* complex" known or suspected? Are the perfect stages *Gibberella fujikuroi* and *G. moniliformis* also found in nature associated with the *Fusarium* complex?

Selvaraj:

Fusarium moniliforme var. *subglutinans* is not only morphologically different from *F. moniliforme sensu stricto*, as you have rightly pointed out, but also pathologically different. Experiments conducted by us at Kano, Nigeria, have confirmed that the former causes pokka boeng but does not cause head blight or stalk rot, even though it is also found in the grain-mold complex and is seed transmitted. Head blight and stalk rot are caused by the same pathogen —*F. moniliforme sensu stricto*.

This pathogen is not able to incite pokkah boeng, even though it often causes head mold. Both *Fusaria* cause seed rot and seedling blight. The perfect stages of these pathogens were not recorded in the diseased host.

Charcoal Rot

Girard:

Macrophomina phaseolina can attack a wide range of plants, such as maize, sorghum, groundnut, cotton, beans, cowpea, and soybean. Are there specialized forms of this fungus, each being adapted to a specific host plant?

Generally the sterile form of the fungus called *Rhizoctonia bataticola* is found. In Senegal, under drought conditions, we find it on groundnut and cowpea. But under very humid conditions, the pycnidial stage *Macrophomina phaseolina* can be found. In fact we do not know if it is the same pathogen. Does anybody know if the pycnidial stage can be associated with sorghum stalk rot?

K. N. Rao:

I know of no evidence for host specialization. We see the sclerotial stage with sorghum charcoal rot.

Ravindranath:

Rhizoctonia bataticola and *Macrophomina phaseolina* attack many crops in the semi-arid tropics, such as peanut, castorbean, sunflower, sesamum etc. Work in India has shown that strains and physiological races do not exist. Slight morphological differences exist. In the last *rabi* (postrainy) season, CSH-6 was raised on some soils of Andhra Pradesh for the first time, and suffered heavily from charcoal rot. This incidence also supports the view that strain variation is not important.

Sundaram:

The mode of infection of the charcoal rot fungus in nature is through soil and roots, and the mechanism of resistance is therefore likely to be specific to roots. Are we justified in labeling plants susceptible if they develop symptoms following toothpick inoculation, because this does not simulate natural conditions?

Frederiksen:

Many stalk rotting fungi first invade roots and the charcoal rot pathogen is no exception. However, most inoculations involve artificial conditions, possibly bypassing some host resistance. They are useful insofar as they identify host genes conditioning levels of resistance.

Dodd:

I believe that when you inoculate directly into stalks you measure the degree of senescence of the tissue. The spread of the

pathogen provides the measure. I dislike toothpick inoculation, not because it is not valid, but because it is so labor intensive, which restricts the number of locations and replications that can be used.

Balasubramanian:
Do the grains of M35-1 shrivel following stalk deterioration in your trials?

K. N. Rao:
The grain of M35-1 was normal following severe stalk disintegration.

Balasubramanian:
Are erect-leaved sorghums more resistant to charcoal rot? Can we control charcoal rot by differential leaf clipping, thus changing carbohydrate movement?

Dodd:
In maize there is less stalk rot when lower leaves are clipped than when upper leaves are clipped. The effect is different if only alternate plants are clipped because of different shading effects.

Denis:
Is there any relationship between root mass or distribution and charcoal rot susceptibility?

Dodd:
Attraction of carbohydrate is toward meristematic tissue and therefore the number of root meristems could be important in the movement of carbohydrates towards the roots. This should be examined, but it might be difficult to bring such information into use for field screening.

Rosenow:
Among our stalk rot-resistant lines there is variability in root mass from large to small.

Dodd:
It would be important to look at root mass early in the season and not wait until plant death.

Rosenow:
Nonsenescent sorghums tend to have a small root system early in the season but maintain actively growing roots right up to the late stages of grain filling. Some of the highly susceptible lines have a large root system early in the season which dies before the completion of grain filling.

Selvaraj:
In northern Nigeria, root rot, stalk rot, and head blight all seem to be related.

Brhane:
It has been stated that amount of stalk rot (charcoal rot) and sink capacity are positively correlated. We have some lines with very small heads and thus low sink capacity, but they are stalk rot susceptible. If, in general, the relationship between sink size and stalk rot susceptibility is positive, how should breeders go about combining high yield and charcoal rot resistance?

Dodd:
This is an important question. You have to select for yield, not just kernel number, and stalk quality (by selecting for nonsenescence) in many environments.

Rosenow:
The generality is valid, but it is not absolute. Lines with the same yield potential can vary greatly in stalk rot susceptibility. Breeding for high yield should be continued in coordination with screening for stalk rot susceptibility. It is possible to get high yield and stalk rot resistance into the same plant.

Williams:
I have heard the charcoal rot pathogen referred to as *Sclerotium bataticola*. Does any one have any comment on the use of *S. bataticola?*

Ravindranath:
There should be no confusion on this matter. The pathogen was originally described as *Sclerotium bataticola* based on the sterile mycelial stage found by Taubenhaus in 1913. This was changed to *Rhizoctonia bataticola* (Taub.) Butler in 1925, because of the obvious and abundant rhizoid hyphae supporting and interconnecting the sclerotia. Thus the hyphal characteristics separate it from the genus *Sclerotium*.

Sorghum Smuts

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A World Review of Sorghum Smuts

J. A. Frowd*

Sorghum is one of the most important of the tropical grain crops. It is frequently referred to as guinea-corn in Anglophone West Africa and as jowar, jola, juar, or cholam in India. World production of sorghum (1974) is reported to be 46.9 million tonne, of which the USA is the largest producer (15.9 million tonne), followed by India (8 million tonne). Nigeria produces 3.5 million tonne annually, and, among West African countries, Upper Volta and Niger are also important sorghum producers. Of an estimated total area of 42.4 million ha in sorghum cultivation, India has the greatest share of 17 million ha, followed by Nigeria and the USA, each with 5.6 million ha. Yields in India and West Africa are substantially below the world average of 1103 kg/ha, and it is in these areas that the greatest need for removal of constraints to sorghum production exists.

Many aspects of the importance of the smut diseases of sorghum have been, and remain, the subject of conflicting statements in the literature. Since the publication in 1962 of Tarr's book on sorghum diseases, much additional material has appeared on the biology and control of sorghum smuts. However, we still need more information on certain aspects of these diseases: for example, on their distribution and relative importance in various areas of the world, race characterization in the smut fungi, resting-spore longevity and the requisite conditions for their germination and host infection, and more efficient or more acceptable measures for control of smut diseases.

This review attempts to highlight some of the possible research priorities that need to be pursued in the light of recent progress on smuts of sorghum.

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Historical Background

Four distinct smut diseases of sorghum are recognized, and they are caused by fungi included in the order Ustilaginales of the class Basidiomycetes. Covered smut is the best known of these diseases, and extensive literature concerning it and its causal pathogen has appeared over the last 70 years. It is caused by a fungus described as *Sporisorium sorghi* Link 1825, although *Sphacelotheca sorghi* (Link) Clinton (1902) is the name now usually accepted. Loose smut is caused by *Sphacelotheca cruenta* (Kuhn) Potter (1912), originally described as *Ustilago cruenta* Kuhn 1872. Another *Ustilago* sp, *U. reiliana* Kuhn (1875), was described as the cause of head smut disease, though the name *Sphacelotheca reiliana* (Kuhn) Clinton (1902) is widely used. Long smut disease was attributed to the fungus described as *Sorosporium ehrenbergii* Kuhn (1887), though its generally accepted name since 1903 has been *Tolyposporium ehrenbergii* (Kuhn) Patouillard.

Geographical Distribution

Sorghum smuts are widespread throughout the world and all four species occur in the Indian subcontinent. Of these, covered smut has the widest distribution, coextensive with sorghum cultivation. It has been reported from Denmark and Canada (northernmost limits) to Chile and Australia (southernmost limits) — CMI Map No. 220 (1974), and from ten countries in West Africa. Its pattern of distribution probably reflects its seedborne mode of carryover. Since loose smut is borne in the same manner and its causal fungus is in many respects similar to that of covered smut, its distribution probably does not differ greatly from covered smut. There is little information on the distribution of loose smut in West Africa (King 1972), though it is mentioned as occurring in Chad (Saccas 1954),

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Niger (Jouan and Delassus 1971), and Cameroon (Foko 1975).

Head smut has been reported from many parts of the world, though records of occurrence of this soilborne disease are not as numerous as those of covered smut. Estonia and the USA are the northernmost limits of its distribution, and Chile and New Zealand are its southernmost limits (CMI Map No. 69, 1969). There are records from seven West African countries: Cameroon (Foko 1975), Chad (Saccas 1954), Ghana, Niger (Jouan and Delassus 1971), Nigeria, Senegal (Bouhot and mallamaire (1965) and Upper Volta (Delassus 1964).

Long smut appears to have a more restricted distribution than the other smuts and apart from those of the Indian subcontinent, reports of its occurrence are largely from East Africa and the Middle East (CMI Map No. 337, 1970). Five records are known from West African countries, i.e., Cameroon (Foko 1975), Ghana, Niger (Jouan and Delassus 1971), Nigeria and Senegal (Bouhot and Mallamaire 1965; Bouhot 1966), long smut has not been reported from the western hemisphere. The author has frequently noted its occurrence in Upper Volta.

Losses

Although well-proven chemical methods for the control of covered smut are known, it is still regarded by many as the most destructive disease of sorghum (Ranganathaiah and Govindu 1970) or, at least, the most destructive of the smuts (e.g., Ramakrishnan 1963). This is particularly true in the third world countries, where seed dressing is not practiced. Losses in West Africa average 5 to 10% (Keay et al. 1967); Mercer-quasline 1969; King 1972) and in parts of Africa up to 50% (Frederiksen, King, and Sundaram, personal communications). Under typical cultural conditions, Mathur and Dalela (1971) estimated yield losses of 4.5 and 1.7% in two crop years in Rajasthan, India, but the possible extent of seed dressing in the crop was not taken into account. A loss of 1 % typically cause yield reduction of 4.1 million kg in Rajasthan and a financial loss of 2.1 million rupees (1964 prices). In Nigeria, Harris (1963) estimated a 1.3% loss due to covered smut at £500 000, or the unrewarded cultivation of 70 000 acres. Losses of 20 to 60% have been reported in crops that did not receive seed treatment (Sundaram 1972); maximum losses under extreme conditions have been 50% in India (Delvi 1958) and 60% in Burma (Anon. 1943). Covered smut is now quite rare in the USA (Edmunds and Zummo 1975).

Head smut and long smut are generally regarded as secondary in importance to covered smut. In regions with an annual rainfall of less than 635mm, long smut has been described as the next most serious disease to covered smut, and losses may attain 10% (Frederiksen, King, and Sundaram, personal communications) to 20% (Bamdadian 1968). Selvaraj (personal communication) notes that long smut and head smut may be serious in northern Nigeria in drought years. Though overall losses due to head smut in Nigeria are less than 2% (King 1972), in Niger head smut has been termed the most serious and destructive smut disease of sorghum (Jouan and Delassus 1971). Head smut caused heavy losses in USA in the late 1950s and resistant hybrids subsequently developed have proved susceptible to new races of *S. reiliana* which have appeared since 1968 (Frederiksen, King, and Sundaram, personal communications).

The common occurrence of loose smut in sorghum-growing areas is rarely regarded as a problem, since the disease is localized and, like covered smut, readily controlled by seed treatments. Possible losses of 2% have been assessed in Niger and Nigeria (King 1972). In areas where ratooning of sorghum is practiced, loose smut may reach epidemic proportions.

The Pathogens

Within the Ustilaginales, the genera implicated in causing smut diseases are *Sphacelotheca* and *Tolyposporium*. In this order, much of the mycelium is converted at maturity into chlamydospores or teliospores which produce basidia after a period of rest. Sporidia are produced from the subdivided basidium.

Sphacelotheca is differentiated from *Ustilago* by its possession of an outer false membrane (peridium) covering the sorus, and a central columella of host tissue. The peridium is composed of fungus cells which are transformed into spores. In many cases, the peridium is evanescent and the smut is mistaken for a species of *Ustilago* (Thirumalachar 1966). The

spores of *Sphacelotheca* are single within the sorus, and become dusty at maturity.

Tolyposporium is characterized by the appearance of its spores in regular balls containing many spores. *Tolyposporium* has been differentiated from *Sorosporium* by the fact that its spores are held together by interconnected thickenings of the exospore walls, whereas *Sorosporium* spores are not intercalary within the ball. The permanency of the spore ball has no meaning in differentiating between the two species (Thirumalachar 1966), and Duran (1969) appears to be in partial agreement in stating that the spore balls of *Tolyposporium* "tend to be permanent or semipermanent" whereas those of *Sorosporium* "tend to disintegrate into individual spores." Thirumalachar (1966), in examining the type genus *Tolyposporium*, considers that it conforms to a *Sorosporium* species though he suggests that the name *Tolyposporium* be retained as it is well-established and another type-genus should be designated.

The fungi of the Ustilaginales produce no sex organs and almost any two compatible cells may combine, the environment determining their behavior with regard to sexual fusion. Compatibility within the smut fungi appears to be extremely complex; for example, Vaheeduddin (1942) reported some 66 "sex groups" (mating types) in 74 lines from 28 teliospores of *Sphacelotheca sorghi*.

Discussion of fungal taxonomy at the species level follows in four sections, each devoted to a single smut disease.

Sphacelotheca sorghi

Taxonomy

The smut sori are filled with dark powdery spores which vary in size. The spores, averaging 4 to 7μ in diameter, are dark brown in mass but olive brown singly (Tarr 1962).

The existence of races in *S. sorghi* became apparent in the 1920s. Five races were identified in the USA (Tisdale et al. 1927) and these have well characterized differences in color, length, and manner of rupture of sori. However, races cannot be separated only on these characters, study of their cultural characters revealed further points of distinction.

Five races have been reported on sorghum in India (Vaheeduddin 1951), two of which were similar to USA races; a third resembled a synthetic hybrid between two races of the U.S. origin. The other two were described as new races. Other workers have since corroborated the existence of more than one physiologic form of the fungus in other parts of India: Dasgupta and Narain (1960) distinguished three races in Uttar Pradesh, while Ranganathaiah (1969) observed two races in Karnataka. The efficacy of chemical control measures against *S. sorghi* is believed to be the factor removing pressures on host resistances and, presumably, has prevented the evolution of new races (Edmunds and Zummo 1975). Natural hybridization between *S. sorghi* and *S. cruenta* is thought to have given rise to a range of races, but hybrids show irregularities in sporidia formation and those produced frequently fail to develop hyphae. The existence of relationships between the U.S., South African, and Indian races has not yet been determined in detail. Two races have been distinguished in South Africa by their differential reactions on White Yolo and Hegari sorghums. Designated as SA1 and SA2, they have been incorporated in a modification of Vaheeduddin's race nomenclature (Gorter 1961). In the relationships of the races, it is doubtful whether race 1 and race 6 (Vaheeduddin 1951) are distinct and similarly whether race 4 and race SA2 are distinct.

Spore Survival and Germination

Spores of *S. sorghi* may remain viable for up to 13 years under dry conditions (Sundaram 1972) and spore germination can take place immediately after they are formed; no resting period is required. Viability of spores depends on time of collection and method of determining their germination potential.

Host Range

S. sorghi appears to be restricted to the genus *Sorghum* (Zundel 1953).

Sphacelotheca cruenta

Taxonomy

The chlamydospores of *S. cruenta* are larger and darker in color than those of *S. sorghi*,

averaging 7 to 8 μ in diameter. In contrast to *S. sorghi*, sori usually rupture before exertion, and release of spores occurs before harvest.

Two races are known to occur in the USA (Melchers 1933) and the existence of three races is now generally recognized (Rodenhiser 1934). These have probably arisen as a result of hybridization with *S. sorghi.*

Spore Survival and Germination

Chlamydospores, when stored dry, have been reported to retain their viability for 4 years (Takasugi and Akaishi 1937).

Host Range

S. cruenta is apparently restricted to the genus *Sorghum* though it has been reported on certain sugarcane varieties in which *Sorghum halepense* was one parent (Chona and Munjal 1951).

Sphacelotheca reiliana

Taxonomy

Spore balls are produced on the mycelium which is systemic throughout the aerial parts of the diseased plant. They are 60 to 180μ in diameter when young and contain papillate spores, which average 13μ in diameter at maturity.

The existence of races in *S. reiliana* was demonstrated by Al-Sohaily et al. (1963) who tested 18 head smut collections from the USA and India on five sorghum differentials and recognized four races. When maize cultivar North Star was included as a differential, a fifth race could be distinguished. The criteria used by these workers are too stringent for uncovering all possible races of the pathogen, since no account was taken of the degree of plant infection, and the number of plants used in their experiments was small. Total virulence of all compatible lines was determined in several chlamydospores produced from monosporidial lines (Mehta et al. 1967). These workers identified three races in covered smut collected at different locations, but use of all compatible combinations from single chlamydospores showed that five races existed. This is explained in terms of the heterozygous nature of pathogenicity in chlamydospores, giving rise to different pathogenic forms from the same chlamydospore, Edmunds and Zummo (1975) broadly group three races as follows: (a) a race attacking most sorghum hybrids grown before 1975, (b) a California race pathogenic to Early Hegari SA 281, and (c) a race virulent to resistance derived from Tx 09 combined with White Feterita (Frederiksen and Roderiguez-Campos 1969).

The introduction of smut-resistant hybrids since 1957 (Stewart and Reyes 1958) gave a temporary solution to continued head smut losses, and by 1968 a new population of smuts was established — necessitating introduction of new sources of genetic resistance (Frederiksen, King, and Sundaram, personal communication). A new race of head smut designated as race 4 was characterized by its ability to attack TAM 2571, a line resistant to race 3 (Frederiksen et al. 1975).

The appearance of new virulent genes in *S. reiliana* is of concern in the USA and elsewhere. A possible new race is thought to occur in Nigeria (King 1972), though whether this is due to race subdivision remains to be determined. The relationships of the races described in the USA, to the three obtained from India (Al-Sohaily et al. 1963) and the two described by Padaganur and Govindu (1971) in India also require elucidation.

Spore Survival and Germination

The reported maximum period of viability of *S. reiliana* spores is 8 years (Brefeld 1883 [cited by Fischer 1936]) and, more recently, it has been stated that they will survive for at least 7 years (Jouan and Delassus 1971), and at least 10 years in Texas (Reyes and Frederiksen, personal communication). This data contrasts with other estimates of 2 years (Ramakrishnan 1963), 18 months (Sundaram 1972) and 12 months (Sundaram 1955). From these reports, it seems probable that *S. reiliana* spores will remain viable for long periods, during which sporadic germination occurs when conditions become favorable. Another possible factor is contained in the results of Swearngin et al. (1966), who showed that naturally released teliospores grew sparingly, whereas immature teliospores will readily germinate. The low percentage germination of spores in water (2%) reported by

Sundaram (1955) probably indicates this point. Enhancement of germination to 8% in 2% sugar solution was interpreted as a stimulatory effect of the sugar (Siddiqui 1965), but in the absence of critical comparisons it is obvious that much further information is required on the behavior of teliospores of *S. reiliana*.

Host Range

S. reiliana occurs in two distinct forms, infecting maize and sorghum. Reports exist of infections in three other genera: *Andropogon* (Uppal et al. 1935; Rayss 1956) *Cleistachne* (Wiehe 1953) and *Euchlaena* (teosinte) (Zevada et al. 1955). The importance of sudangrass in field margins as an alternate host for the pathogen has been demonstrated (Halisky 1963).

Tolyposporium ehrenbergii

Taxonomy

Spores are united into balls, which are dark brown globose to oval bodies between 30 and 240μ in diameter. The spores at the periphery of the spore ball are dark brown and p[illegible]te on the free side, whilst internal spores are light brown and smooth-walled. They are usually globose but somewhat angular, and average 12μ in diameter. The existance of at least two races of long smut has been suggested by Tarr (1962), a conclusion based on differential infection patterns in *Sorghum purpureo-sericeum* and nearby *Sorghum bicolor* plants.

Spore Survival and Germination

Spores of *T. ehrenbergii* are reported to retain their viability for 2 years (Ramakrishnan 1963; Sundaram 1972).

Host Range

T. ehrenbergii occurs on pearl millet in addition to sorghum (Tarr 1962).

Smut Relationships

Hybridization studies have elucidated some aspects of the inheritance of characters of each of the smut fungi. Intraspecific and interspecific hybridizations give rise to new pathogenic smut types, each of varying pathogenicity. Sometimes these are termed "races."

Hybrids between *S. sorghi* and *S. cruenta* occur readily (Rodenhiser 1932), but the heterothallic interactions between them result in a high degree of sterility and the basidia fail to develop sporidia, or develop only a few.

Dikaryons from haploid lines of *S. sorghi* and *S. reiliana* were found to be differentially pathogenic in four varieties of sorghum. Sorus type was either *S. sorghi* type, or an intermediate, though spores from F_1 sori gave sori resembling *S. sorghi* and *S. reliana* in a 2:7 ratio when tested on nine varieties. It was concluded that the factors controlling *S. sorghi* characters appeared closely linked, or were inherited as a unit (Rodriguez and Frederiksen 1968).

Vaheeduddin (1942) crossed *S. reiliana* with *S. cruenta* and obtained a range of hybrids, in some of which the sori resembled those of *Tolyposporium ehrenbergii* and, in some, heterosis was evident.

A summary of the characters of the four smut fungi is given in Table 1.

Mode of Host Infection

Covered Smut

McKnight (1966) showed that the ideal position for infection on sorghum, using ungerminated teliospores, was on the testa at the time of seeding. Significant infections of sorghum variety Martin occurred when inoculation was delayed, up to coleoptile length of 5mm, but subsequent infection was rarely successful. In sorghum variety Pink Kafir, infections were obtained up to a coleoptile length of 20 mm, but never when the primary leaf was more than 20 mm through the coleoptile. Soil transmission of the pathogen does not occur.

Loose Smut

The causal pathogen is able to infect florets directly, although it is probable that most infection arises from meristematic invasion during or shortly after germination (King 1972). Infection of the seedlings generally arises from seed contaminated with spores. As they germinate readily in water, soil transmission is unimportant.

Table 1. Characteristics of four smut fungi.

Smut	Host		Sorus		Spores	
	Stunted	Heading	Site	Membrane	Surface	Diam.
S. sorghi (covered smut)	Yes	Premature	Ovary	Rather permanent	Apparently smooth	6–7μ
S. cruenta (loose smut)	No	Normal	Ovary	Ruptures easily	Minutely echinulate	7–8μ
S. reiliana (head smut)	No	Premature	Inflorescence	Ruptures easily	Conspicuously echinulate	9–12μ
T. ehrenbergii (long smut)		Normal	Ovaries	Ruptures	Free surface papillate	12μ

Source: Ainsworth (1965)

Head Smut

Although seedborne infection may occur head smut is typically soilborne, with infection at the seedling stage. Delassus (1964) indicated that artificial inoculation of seed had little effect on ultimate disease incidence. Siddiqui (1965) obtained 40% infection of plants when spores were mixed with soil into which the seed was planted. Eight percent of the plants became infected when spores were shaken with seed before planting in sterilized soil.

Long Smut

The mode of infection of sorghum by the long smut fungus has received considerable attention in recent years. Ragab and Mahdi (1966) obtained results which enabled them to refute the belief that long smut infection was derived only from airborne sporidia, and suggested that infection may arise from mycelium in the grain, spores in the soil, or airborne sporidia. They reported that up to 1% of sorghum seed examined contained mycelium, although it was not identified certainty as that of *T. ehrenbergii*.

Teliospores mixed with seeds resulted in 21.9% plant infection, and seed planted in soil containing teliospores gave rise to 14.3% infected plants. Spraying panicles with sporidial suspensions did not result in infection, and dusting spores on to the panicles with a brush resulted in 5% plant infection. Conflicting reports of teliospore treatment of seeds as an infection route have since appeared; Tamimi (1971) showed infection of 12 of 14 cultivars resulted from seed dusting, whereas Hassan et al. (1970) were unable to induce infection by this means, nor were they able to domonstrate infection resulting from spores sprayed on to the panicles. Recently, it has been found that long smut infection occurred only by sporidia or germinating teliospores, inoculated to host plants from the boot stage to not later than anthesis. Inoculations at the boot stage using germinating teliospores gave maximum infection. Bamdadian (1968) described long smut as seedborne, and Mabry and Lightfield (1974) claimed to have intercepted long smut on sorghum seed imported to the USA. The relative importance of the three possible infection routes has yet to be determined.

Screening Procedures

In any screening trial, the objective is to provide adequate infection pressure onto the recipient host in order to detect genetic resistance. This is accomplished by supplying inoculum under the most suitable conditions for infection, and if possible, by manipulating the environment to further enhance the success of any possible initiation and buildup of infection. These techniques correspond to the most prevalent route of infection determined for the particular pathogen and applied to the host at its most susceptible phase of growth, which does not

necessarily correspond with the phase of growth when it is infected in nature.

Covered Smut

Most of the covered smut screening procedures have used the technique of mixing teliospores with dry seed prior to planting. The proportions of inoculum to seed vary widely between individual tests and in many cases (e.g., Ranganathaiah and Govindu 1970) are not specified. Grewal and Vir (1961) inoculated seed at a rate of 0.5 w/w prior to planting in the field, and obtained ultimate disease incidence of 6.9, 24.5, and 21.2% over 3 years. The application of spores to seed enables several races of *S. sorghi* to be applied simultaneously; for example, Casady et al. (1962) applied a mixture of five races of the fungus when testing for smut resistance in a sorghum breeding program.

The investigation of differential susceptibilities is not necessarily most rigorous when carried out under field conditions (Adlakha and Manjal 1963). Three of four sorghum lines failed to develop infection in the field when the seed was mixed with smut teliospores and tested under prevailing high-temperature conditions.

Loose Smut

Hypodermic injection of spores into the growing point is a suitable alternative to the application of teliospores to seeds immediately before planting. Keay et al. (1969) showed that 64% of 3-to 4-week old sorghum plants became infected when plants were inoculated with a hypodermic syringe.

Head Smut

The combination of seed and soil previously infected with *S. reiliana* teliospores is the most widely used screening method. On a small scale, this is done by sowing seeds in soil in pots, incorporating the teliospores (Padaganur and Govindu 1971). On a somewhat larger scale, "sick plots" have been established by hoeing-in of teliospores, as described by King (1972). This procedure increased infection in Pink Kafir sorghum from nil to 6.6%, i.e., the greater infection pressure converted "resistant" sorghum into "susceptible" variety.

Halisky (1963) applied teliospore suspensions directly to rows of six varieties of sorghum. An increase from 20 ml to 120 ml of teliospore suspension per foot of row resulted in an increase in panicle infection from 33.2 to 61.7%. This increase in plant infection of 85.8% corresponds to the logarithmic increase in the amount of inoculum used (77.8%) and tends to support the finding of Al-Sohaily (1961), who proposed that the percentage of head-smutted plants in a trial was directly proportional to the logarithm of the number of teliospores in the soil. In the same study, Al-Sohaily determined that 800 spores per gram of soil constituted a threshold for plant infection. It should be remembered that the success of these spores in causing plant infection is dependent on environmental effects.

Where head smut prevalence is known to be high, varietal screening may be carried out on land with a previous history of infected sorghum. This has been done by Casady et al. (1962) and in multilocational tests for race determination in Texas, by Frederiksen et al (1969). There are obvious drawbacks to this method, and its use is possibly best restricted to multilocational testing (within a region) to obtain an indication of relative susceptibilities of lines.

Hypodermic syringe inoculation of 3-week old plants is a method employed to ensure that every plant in a trial receives a standardized amount of inoculum. Examples of its use are quoted by Keay et al. (1969) and King (1972) from Nigeria. King showed that Nigerian sorghum types inoculated by this method showed 71 to 90% infection, whereas exotic types were not infected. The technique, by reason of the control of inoculum achieved, has been used widely in race-characterization studies. Al-Sohaily et al. (1963) inoculated young plants with sporidia at or near their growing points; Frederiksen and Rodriguez-Campos (1969) injected teliospores and sporidia into 3-week old plants in a race-characterization study, while Wilson and Frederiksen (1970) injected compatible monosporidial lines in a demonstration of differences in histopathological responses to infection.

Long Smut

Little work has been carried out on varietal screening to this pathogen, and artificial infec-

tion studies with the object of determining its mode of infection (described previously) represent the extent of work carried out. When methods of infection have been elucidated, critical screening procedures will require development.

Disease Assessment in the Sorghum Smuts

Disease assessment in all the sorghum smut diseases is problematical, on account of the quasi-systemic nature of the pathogens involved. Quantitative measurements are not possible, since lesions of different size or number are not produced. In the absence of a ready method for estimation of the degree of plant infection (such as using height reduction as a measure of infection by covered smut, attempted by Ranganathaiah and Govindu [1970]), the usual measure of susceptibility is the percentage of plants that show disease symptoms. It is the reliance on this method of assessment that emphasizes importance of ensuring that each individual plant is exposed to an adequate amount of inoculum, e.g., by hypodermic syringe inoculation. In the case of head smut, the criterion of "none" against "some" infection as the distinction between resistant and susceptible types may be too stringent for uncovering all races of the pathogen (Al-Sohaily et al. 1963). Furthermore, where not all infected plants become diseased, i.e., 100% infection does not occur, major genes for resistance in the host may be modified by certain factors. These might be so important as to obscure the resistance inheritance pattern and represent another group of sources of resistance to disease (Mehta et al. 1967). Working with covered smut disease, Gorter (1961) pinpointed the most difficult part of a resistance susceptibility investigation as that which lay in the decision as to whether a variety was susceptible or resistant when the percentage of infection was low. Frequently, the conclusions of varietal assessment studies are presented with the candidate entries assigned to arbitrary categories (Table 2).

The use of such arbitrary assessment can give conflicting results, as exemplified by the studies carried out at the same laboratory (Table 3).

Table 2. Varietal assessment of sorghums for reaction to smuts.

	Mathur et al. (1964)	Singh and Yadav (1966)	Ranganathaiah and Govindu (1970)
Resistant	< 10%	< 10%	0–1%
Moderately resistant	< 5%	< 5%	1–5%
Moderately susceptible	< 10%		5–10%
Susceptible	< 15%	< 15%	> 10%
Very susceptible	> 15%	> 15%	

Table 3. Disease assessment of some sorghum lines (studied at the same laboratory) according to arbitrary categories assigned by workers.

Sorghum Line	Mathur et al. (1964)	Singh and Yadav (1966)	Ranganathaiah and Govindu (1970)
510-Z	Resistant	Mod. resistant	Mod. resistant
T3	Very susceptible	Resistant	Resistant
59/76	Very susceptible	Mod. resistant	Mod. resistant
59/65	Susceptible	Susceptible	Mod. resistant

Gorter (1961) has attempted to improve on an arbitrary choice of the point of distinction between susceptibility and resistance by drawing it at the point of 0.1 x percent infection, where x is the highest infection level obtained on the most susceptible line in the experiment. The formula may also be used to compare single season, single line results to other similar experiments, e.g., a line appearing relatively resistant of all tested in one season may show a higher percentage infection than 0.1 multiplied by the value of a universally accepted highly resistant type averaged over several seasons. Thus the line would be designated "susceptible."

Most reports of disease assessment refer to rating at the time of heading or shortly before harvest. The report of Harris (1963) is noteworthy in that heads were sampled and dissected when 11 to 20 spikelets had not attained flowering stage.

Influence of Environmental Factors on Smut Infection and Disease Development

Infection of the Host Plant

Covered Smut

Relatively cool conditions increase the frequency of occurrence of covered smut (Doggett 1970). In Nigeria, Harris (1963) noted that lower temperatures in May and June promoted smut infection. This was related to higher infection rates where mean temperatures were near 75°F (23.9°C) (e.g., the Plateau-Zaria regions), than at 80°F (26.7°C) (e.g., the Sokoto-Bornu regions), the temperature at which sorghum growth is favored but smut spores germinate less readily. The importance of temperature as a determinant of initial infection at sowing time was stressed by Adlakha and Munjal (1963) who showed that three out of four lines previously inoculated with smut spores did not become infected when sown in the field at 34 to 42°C, but all showed infection when germinated in soil and incubated at 22 to 29°C.

Soil moisture is equally important as a determinant of smut infection. Relatively dry soils have long been recognized as promoting smut infection, and less than 28% moisture has been frequently quoted as optimum for plant infection. In light of this evidence, it is difficult to interpret Doggett's observations on the prevalence of covered smut (25% infection) on an impeded-drainage lower slope, compared to less than 5% infection on a free-draining sand at the top of the same slope. The observation may not be unconnected with increased plant infection observed on slightly acid soils (Sundaram 1972).

Loose Smut

The causal fungus requires infection conditions similar to those of covered smut; these have been examined in detail by Shukla and Mishra (1969) who reported spore germination over a range of temperatures between 10 and 35°C (optimum 25°C), a figure in close agreement with the figure of 8 to 38°C (optimum 28 to 32°C) reported by Shih (1938). Infection occurs over a range of pH values from 3 to 9 (Shukla and Mishra 1969).

Head Smut

In 1926, Christensen showed that cool, dry soils were conducive to head smut infection of sorghum. Optimum temperatures for infection were 24°C at 15% soil moisture content and 28°C at 25% soil moisture content. The disease is more serious in "drought" years (Selvaraj, personal communication) in Nigeria, though some regard it as more common in "wet" than in "very dry" years (Frederiksen, King, and Sundaram, personal communications). The association of "dry" soil with high pH has been associated with high disease incidence in southern Texas (Frederiksen, personal communication). Kruger (1969b) similarly observed that spore germination was lower at higher pH values.

Pai and Pan (1964) were able to obtain a six-fold increase in germination of *S. reiliana* spores when they were held in moist conditions at 32 to 35°C for 35 days, and then transferred to a sucrose solution. In general, high temperatures were more effective in promoting germination under moist than under dry conditions, although extended treatment (190 days) caused a drop in spore germinability.

Cool night temperatures and moderate day temperatures were offered as an explanation for higher incidence of head smut in upland

parts of Georgia, USA, (Harris et al. 1971), than elsewhere in the state. Temperatures in the range 21 to 25°C promoted the occurrence of head smut.

Kruger (1969a) showed that spores of head smut germinated readily immediately after moistening dry soil, but after that initial period only a few spores (1 to 3%) germinated. If the soil was then dried and remoistened, a second peak of germination occurred. In a later report, Kruger (1969b) stated that plants growing on infested soil kept moist for several weeks before seeding showed decreased infection. Head smut incidence was high if spores were kept in dry soil. These results are at variance with those of Doshimov (1974), who reported that winter conditions (low temperature, frozen soil, snow cover, and rain) increased the pathogenicity of head smut spores in the soil.

It is difficult to differentiate between the effects on spore germination and disease development in the plant under distinct microenvironments. For example, King (1972) has referred to a "tree effect" on the prevalence of head smut in northern Nigeria; the disease was more prevalent near trees.

Long Smut

Of the smut diseases, much less is known of the factors influencing host infection by *T. ehrenbergii*. King (1972) stated that the disease is more prevalent where rainfall is less than 750 mm. In observations on the disease in Israel, Minz and Palti (1960) found that it was more common where mean relative humidity was 50 to 55% and minima were 35 to 40% in July and August, than where mean relative humidity was 70 to 80% and minima 50 to 55% in the same months.

Spread Within the Host Plant

For all the smut diseases caused by *Sphacelotheca* sp, the infection pattern of the main stem determines the type of host colonization which results. All three *Sphacelotheca* are known to initiate meristem infections during or shortly after seed germination. Infections are carried upwards in the meristematic tissue, causing very little colonization in the vegetative plants, but they rapidly grow in the floral parts where sori develop.

In covered smut, sorus production depends on the pattern of early infection, though the main head may escape infection if the uppermost cells of the meristem are not invaded (Melchers 1933). The number and distribution of smutted florets in a panicle varies, and there is great variability in the grain/sori ratio as well as in the color, shape, and size of the sori (King 1972). In good growing conditions, the apex of the plant is able to keep ahead of the smut, and the panicle will not become infected (Doggett 1970). Hence the disease is frequent in cooler conditions unfavorable for sorghum growth. Although one might infer that slow seed germination might promote infection, it does not follow that rapid seed germination will reduce infection (Ramakrishnan 1963). If an infected plant is cut back, smutted tillers are likely to develop (Doggett 1970).

Loose smut differs from covered smut in that the fungus causes plant stunting, and sidebranch production is stimulated. The disease is predominantly found on tillers and not on the primary heads of infected plants. Furthermore, all ovaries in a panicle are infected, unlike in covered smut (King 1972). Sorghum plants affected with loose smut or covered smut in Andhra Pradesh (India) have been reported to flower one month ahead of healthy controls (Dakshinamurthy and Rao 1965), though this phenomenon is more widely recognized as a characteristic of loose smut infection (e.g., King 1972). King investigated the possibility that isolates from main shoots and tillers of loose smut-infected sorghum plants were different from each other, but apparently reached no conclusions.

The determinants of sorus production in head smut have been examined in some detail by Wilson and Frederiksen (1968, 1970); as in covered smut and loose smut, the mycelium is carried by elongating cells to the floral primordium and is summarized in Table 4.

Sori are usually extensively formed on the tillers. Alagianagalingam and Soumini Rajagopalan (1972) observed multisori production on an infected plant and found that not only inflorescences were converted into sori, but also vegetative parts by proliferation of axillary buds (actually colonization of axillary buds).

Long smut infection is generally believed to

occur at the time of panicle exsertion its mode of infection is not well understood.

Table 4. Colonization by mycelium and sorus production in head smut.

Colonization by mycelium	Sorus produced
Widespread at beginning of elongation	Typical, smooth
Only in basal regions of meristem before elongation	Vegetative head
Carried into lowest regions of floral primordia	Small
Widespread but sparse at time of elongation	Resembles partially differentiated inflorescence.

Spread Within the Crop

The cultural practices associated with the production of sorghum might be identified as the nonclimatic influences on spread and disease development within the crop. Many of these can be directly related to observations made in the course of other studies affecting plant infection. Early seeding has been shown to result in higher rates of infection by covered smut (El Helaly 1939) and by head smut (Harris et al. 1971). This concurs with the observation that moderate temperatures favor infection, as noted previously. The association between deep sowing and increased severity of loose smut (Ramakrishnan 1963) is also a function of lower temperatures at greater soil depths, promoting growth of the fungus. Early seeding and increased disease is most likely to be associated with lack of rain (hence, temperature will be a determinant), whereas deep sowing may involve more actively the role of soil moisture immediately following germination.

The application of fertilizer to soils has produced some pronounced effects on smut incidence. The role of phosphates has received greatest attention in field-scale experiments. Increased phosphate levels are well known in reducing both covered smut and loose smut infection. In studies of *S. reiliana* on maize, phosphate was less effective than potassium in reducing head smut incidence. Reductions were 9% for P_2O_5, 34% for KCl, and 45% for NPK. Lack of potassium was also observed to cause higher incidence of head smut.

Trials of chemicals applied to soilborne spores, however, have not given consistent results. Soils treated with B, Mn, and Cu at 0.1% had lower inoculum potential than untreated soil, presumably by an acceleration of spore germination (Barakhtayanskaya 1964). On the contrary Kruger (1969a) showed that salts (N, P, K, Mg, Fe, and S) added to enriched or minimal agars had no influence on head smut spore germination, though when added to soil, spores did not germinate or germinated very little.

In the case of head smut, more work remains to be done on the importance of crop residues as sources of inoculum for infection of the following crop. For example, Kruger (1969a) noted that soil containing residues from sorghum resulted in more smutted maize, though this was not observed with maize residues. The incorporation of residues in this manner influences the C:N ratio.

Sources of Resistance

Every major sorghum group of economic importance contains resistance derived from at least one variety or line (i.e., Feterita, Milo, Hegari, Kafir). Resistance from lines in the Feterita group has been used most extensively, as it is derived from allelic genes for resistance. In contrast, inheritance of Hegari resistance to smuts appears to be complex and controlled by genes which are nonallelic.

Some sources of resistance to covered smut, head smut, and long smut located by several workers are shown in Table 5. Types showing less than 0.1% infection of susceptible entries, as reported by their respective workers, are included.

Although many of these lines may contain a measure of resistance to one or more of the smut pathogens, care should be exercised in predicting hybrid reactions from the parental reaction. This has been emphasized by Frederiksen (personal communication), who was unable to estimate hybrid reaction to head smut in this manner, indicating that the mode of inheritance is not strictly allelic. Further details of inheritance appear in the next section.

Table 5. Some sources of resistance to covered smut, head smut, and long smut.

Entry	Degree of resistance*	Reference
COVERED SMUT		
53/3 (Gwalior 1) 53/10 (EC-4593) 57/1 57/9 57/9 (b) 57/10 (b) 57/17 57/20 57/24 57/30 510-2 4106 4106 A	resistant, infection 1%	Mathur et al. (1964)
48/13 4102	Moderately resistant infection 1–5%	Mathur et al. (1964)
T3	Resistant (as above)	Singh and Yadav (1966)
510-2 59/76 59/106 60/22	Moderately resistant (as above)	Singh and Yadav (1966)
T29/1 P.J. 7K P.J. 23K Nandyal (IS 3814) Bilichigan	Resistant	Adlakha and Munjal (1963)
KB 2540	Some resistance	Casady et al. (1962)
Nandyal (IS 3814) IS-84 D-340 BH 4-1-4 BS 81-3 C7-1162	Resistant*	Ranganathaiah and Govindu (1970)
Framida 90 Days White	Immune	Gorter (1963)
T3 Hybrid TI	Resistant	Mathur et al. (1970)
60/22 T4 510-2 59/65 59/76	Moderately resistant	Mathur et al. (1970)
No. 54 BAN	The most resistant (of several)	Markov (1964)
Savasai Gandhi	Semi-resistant	Fatemi (1967)
Spur Feterita	Immune	Tamimi and Al-Sohaily (1967)
Redlan Reliance White Yolo	Highly resistant	Tamimi and Al-Sohaily (1967)
HEAD SMUT		
TAM (SC 110-9) IS-12664C (SC-173) Lahoma Sudan PI-48770 (White Kafir)	The most resistant (of several)	Frederiksen (private communication)
Most sweet sorghums	Highly resistant	Edmunds and Zummo (1975)
Milo types Feterita types Broom Corn types Kafir types	Fairly resistant	Sundaram (1972)
Feterita types	Resistant	Al-Sohaily et al (1963)
FC 811	Dominant form of resistance	Al-Sohaily (1961)
RS 630	Resistant	Al-Sohaily et al. (1963)
Nandyal (IS-3814) IS-84 Karad local M. 35-1 (IS-1054) D. 531-21B CSH 1	Resistant	Padaganur and Govindu (1971)

* Terms used are those of respective workers, except where marked with an asterisk (*).

Continued.

Table 5. *Contd.*

Entry	Degree of resistance	Reference
All Feteritas Feterita-derived lines Hegari (P.I. 34911) White Kafir Wheatland Dwarf Kaiang Dwarf Yellow Milo Ryer Milo Dwarf Yellow Durra Tx 414 Lahoma	Resistant	Mehta et al. (1967)
White Kafir Early Hegari (IS 106)	Resistant	Frederiksen and Rodriguez-Campos (1969)
Kafir E65L-5427-3 (BTAM 618)	Resistant	Wilson and Frederiksen (1970)
KS 652	Not completely resistant	Casady (1964)
LONG SMUT		
Redlan Regular Hegari	Resistant*	Tamimi (1971)
Durra Selection Pierce Kaferita European Dwarf Broomcorn D. Dwf. Feterita Piper Desert Bishop As 3749 Standard Feterita Dutch Boy	Resistant	Tyagi (personal communication)

Inheritance and Mechanism of Resistance

Not only is there good resistance to covered smut, but resistance to at least some races seems to confer resistance to loose smut also and quite probably to head smut. In West Africa, the development of covered smut resistance may be desirable (King 1972), because seed dressings are rarely used.

The inheritance of covered smut resistance has been comprehensively studied by Casady. Casady (1961) found that three resistance genes for the three races of *S. sorghi* appeared to be linked. A 1:3 inheritance of resistance to susceptibility against each race was established in a spur Feterita × Pink Kafir recombination. However, reaction of F_1 progenies of Combine Kafir 60 × Spur Feterita and Pink Kafir × Spur Feterita indicated that resistance was incompletely dominant, and data from F_3 lines of Dwarf Yellow Milo × Spur Feterita showed that the two parents possessed the same gene for resistance against race 1 of *S. sorghi*. Casady therefore suggested that Spur Feterita is not a true Feterita, but it is the only variety available resistant to known races of *S. sorghi*. The reaction of F_1 progenies of sorghum crosses to *S. sorghi* (race 1) showed that smutting was dominant to the blasting reaction (Casady 1963), as determined by a study of F_3 lines of Spur Feterita × Pink Kafir.

Earlier, Casady et al. (1962) had observed that nearly all developed sorghum lines with resistance to covered smut were also resistant to head smut and suggested the existence of a close linkage between covered smut resistance and head smut resistance. If one accepts Casady's interpretation of earlier studies by Marcy, one could postulate three-way linkages for the genes that govern reaction to *S. sorghi*, *S. cruenta*, and *S. reiliana*. Casady and coworkers were unable to locate complete resistance to head smut. KS-3 (Spur Blackhull-3 × Redbine-60) inbred was released as a result of their program, though its level of resistance was not as high as in the original source variety, KB-2540. When KS-3 was crossed with Combine Kafir 60, KS-652 was developed, which (while not completely resistant to head smut) reduced its incidence to 2.4% where Combine 7078 (a susceptible type) showed 22% infection.

The study of host-parasite interactions has been confined to the characterization of three types of head smut resistance by their compatibilities. An incompatible reaction, associated with mycelium disappearance and appearance of teliospore-like objects and not influenced by temperature (as found in Early Hegari and F_1s with Early Hegari as the male parent) indicated a complex form of inheritance influenced by environmental factors. Hyphal growth was present in inoculation wounds of Lahoma sudangrass, White Kafir P 148770, and B TAM 618, but there was no infection of susceptible entries, as reported by their growth in host tissue; the reaction was not influenced

by temperature. Resistance factors were presumed to be present in the meristem zone. In Tx09 and F_1 derivatives, there was complete inhibition of sporidial development, stable over a range of temperatures and interpreted as "major gene resistance."

Conclusion

The direction of future research can be considered under four separate headings: fungus biology, race characterization, screening and associated disease-assessment problems, and, long smut (including overlaps into the three above-mentioned headings).

In the development of head smut, information is required on the environmental factors favoring the initiation and development of plant infection; for example, on the longevity of survival of the spores and the microclimatic effects that influence infection. The reported differences in isolates of loose smut obtained from tillers and main shoots of infected plants requires clarification, as do further studies on the effects of soil minerals on the development of all the smut diseases.

The extent of hybridization of the races between covered smut, loose smut, and head smut is poorly defined in all areas where the diseases occur together. Indeed, the existence of races of covered smut and head smut in Africa has not been surveyed or described. Continuing research on the determination of races of *S. reiliana* is called for; their present total number is not known and their relationships demand clarification. For example, is race Texas 9 (Al-Sohaily et al. 1963) the same as race 4 (Frederiksen et al. 1975); King (1972) has referred to the possible subdivision of races of head smut, which implicates the launching of a fundamental study on the genetics of *S. reiliana*.

The most pertinent problems in sorghum varietal improvement hinge on assessment methods and the isolation of resistance to *S. reiliana*.

The need for standardized methods for disease evaluation, including the drawing up of agreed assessment scales, would be a major asset in comparative screening work. Such a system could be immediately applied in a future program of screening of sorghums for resistance to the head smut pathogen, necessary for race monitoring, and for the noting of varietal responses, especially in conjunction with a breeding program, since head smut reaction of progeny lines are not always predictable from parental reactions.

Long smut is a disease of importance in certain localized regions (Upper Volta is a case in point) and many questions on the disease and its causal fungus require elucidation. For example, neither the mode of host-plant infection nor the conditions influencing disease incidence are well understood. In these circumstances, artificial inoculations in screening work cannot be taken at optimum time, optimum inoculum concentration, or optimum inoculation method. It is evident that race characterization will be needed in any future program on the sources of resistance to long smut by intensive study of varietial reactions.

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Importance of Sorghum Smuts in African Countries

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Smut diseases of sorghum are widespread in African countries, and are economically important in most of the sorghum-growing areas. All four kinds of smuts belonging to two genera and four species have been recorded on cultivated sorghum in Africa. The most destructive and common of them is covered smut, caused by *Sphacelotheca sorghi*. This disease was first described by Link in Egypt in 1825. *Sphacelotheca reiliana* (Kuhn) Clinton, the causal organism of head smut of sorghum, and long smut caused by *Tolyposporium ehrenberghii*, were also recorded in Egypt by Kuhn in 1875 and 1887, respectively, though under different generic names (Tarr 1962).

The recorded distribution of these smuts in Africa are listed below:

Common name	*Causal organism*	*African countries where reported*
Covered smut or grain smut	*Sphacelotheca sorghi* (Link) Clinton	Egypt, Uganda, Kenya, Sudan, Nigeria, Niger, Cameroon, Upper Volta, Senegal, Mali, Tanzania, Ethiopia, Somalia, Gabon, Zaire, Congo, and South Africa
Loose smut	*S. cruenta* (Kuhn) Potter	South Africa, West Africa, Tanzania, Uganda, Kenya, Sudan, Gabon, Zaire, Congo, Malawi, and Zambia
Head smut	*S. reiliana* (Kuhn) Clinton	Probably in all sorghum-growing countries in Africa
Long smut	*Tolyposporium ehrenberghii* (Kuhn) Patouillard	Egypt, Sudan, Kenya, Tanzania, Tunisia, Malawi, Somalia, West African countries

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Economic Importance

Of the four smuts, covered smut is the most serious in most of those African countries where prophylactic control measures are not used. Infected heads are more or less completely smutted. Though there is no systematic assessment of grain loss in the sorghum-growing countries in Africa, from a survey conducted by the author in Nigeria and neighboring countries in 1977 estimated grain loss in farmers' fields at about 5%. Up to 30% infection was observed in "hot-spot" areas. According to Wallace in 1934, the infection in Tanzania varied from 8 to 43%, with an average of 25% (Tarr 1962). One native variety had 100% infected heads. Similar situations may be expected in most of the countries in Africa where control measures are not practiced.

The losses due to sorghum loose smut are relatively low, rarely exceeding 10%, even in "hot-spot" areas. However, unlike those afflicted by covered smut, the infected plants are nearly always barren. Therefore, the loss in yield is directly proportional to the number of plants infected.

Head smut incidence is comparatively high in all sorghum-growing areas in Africa and particularly in low-lying fields. In Nigeria, the disease is especially severe along the northern Sudan savanna belt. The situation in Niger in areas adjoining Nigeria is similar. In some fields up to 10% of the plants may be infected. But overall, infection does not exceed 1 to 2%, and it is considered to be of minor importance at this time.

Long smut is widespread in areas where the plants flower during humid warm periods. A large number of varieties, including landrace

and improved cultivars, are susceptible to long smut in Nigeria, Cameroon, Niger, and in other countries with similar climatic conditions. Though the number of infected spikelets in any infected head normally may not exceed 20 to 30%, under favorable conditions, infected spikelets may exceed 70% in very highly susceptible cultivars. This is less important in wet and cooler areas. Besides West Africa, the disease is widespread in Egypt (5 to 50% of the grains with an average of 15% in each affected head), but crop losses do not exceed 2% on the average.

Control Methods

Few records are available as to how widely seed treatment has been adopted for the control of smut diseases in African countries. In Sudan, grain smut was rarely found in areas where the seeds were treated before sowing. In West African countries, grain smut is widespread in local varieties not treated with fungicides. Chemical control measures for other smuts are not known.

Future Program of Research

Since nontoxic fungicidal seed treatments are inexpensive and effective in controlling covered and loose smuts of sorghum, further efforts are needed to encourage their employment. Future research should stress on locating resistant materials for head smut and long smut, which are becoming increasingly important in low rainfall areas. Favorable conditions for their epiphytotic occurrence are present in most SAT regions. Continuous screening of all breeding materials at appropriate stages, to eliminate the lines most susceptible to these two smuts, is essential. Standardization of techniques to create epiphytotics for screening for resistance in various zones to suit local conditions is essential. The possibility of seed treatment with systemic fungicides, especially to provide resistance to head smut in the early stage of growth of the plants, needs to be studied. The only method available at present for the control of long smut is breeding for resistance.

There is a great need for a systemic fungicide that would control both head and long smuts, since control methods other than host resistance have proved ineffective.

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Sorghum Smuts Research and Control in Nigeria

J. Cyril Selvaraj*

Sorghum (*Sorghum bicolor* [L.] Moench) is grown on about 5.6 million ha in Nigeria under subsistence mixed-farming conditions (Anon. 1977). It is the principal food crop in the Sudan and northern Guinea zones, and is an important cereal in the southern Guinea and the derived savanna zones of Nigeria (Fig. 1). The current annual production of sorghum is estimated to be only about 2.8 million tonne (Anon. 1975). Diseases constitute an important production constraint under both traditional and improved farming conditions. On the basis of disease surveys conducted during the 1975 to 1978 cropping seasons, the loss in sorghum due to diseases (excluding *Striga*) in Nigeria is estimated at about to 10 to 12%.

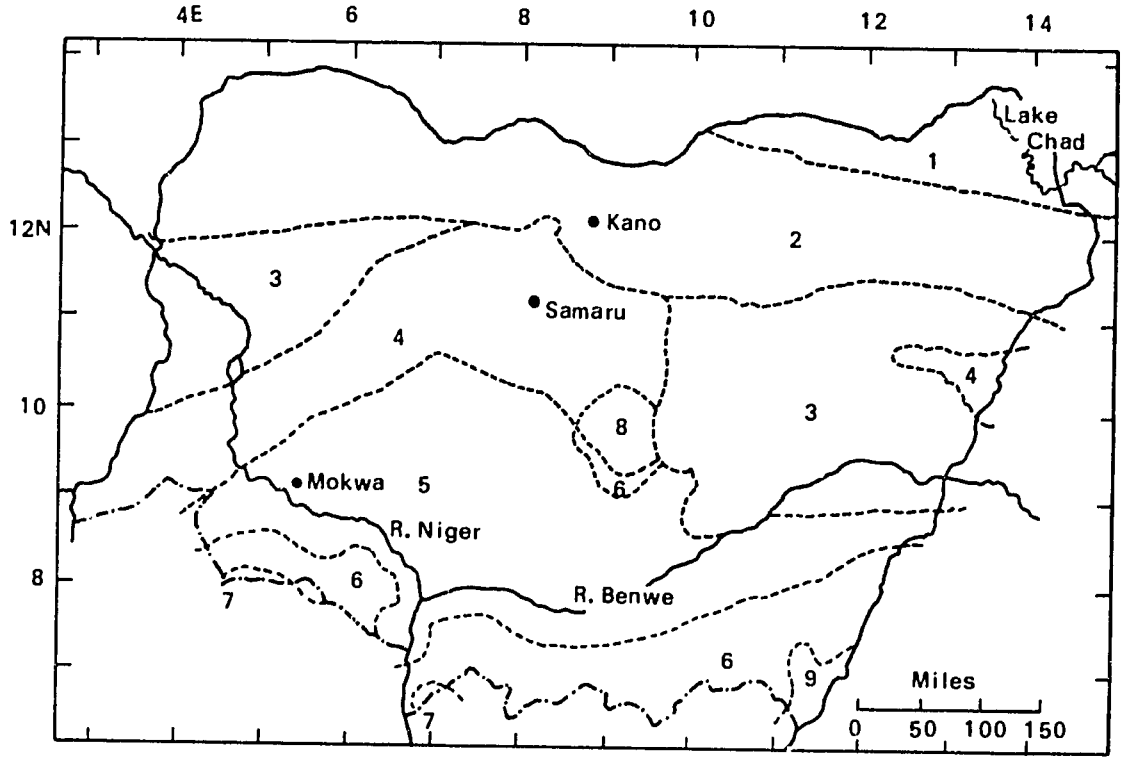

Figure 1. Vegetation zones in northern Nigeria (after Clayton, 1957). Vegetation zones: 1. Sahel savanna; 2. Sudan savanna; 3. Sub-Sudan savanna; 4. northern Guinea savanna; 5. southern Guinea savanna; 6. derived savanna; 7. forest; 8. plateau; 9. montane.

The smuts are the most important and widespread group of diseases in all sorghum-growing areas of Nigeria. They are more

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damaging to the traditional cultivars than to the improved cultivars. It was earlier estimated (King 1969, 1970b) that the smuts are responsible for 8 to 10% annual yield loss. The relative importance of the four smuts has changed considerably over the years. Detailed assessment of crop loss due to individual smuts is not available, but estimates based on the results of surveys from 1975 to 1978 are presented in Tables 1 and 2. A brief description of their past and present position, including general distribution, extent of damage, and the areas of severe incidence is presented below.

GRAIN SMUT. Grain or covered smut (*Sphacelotheca sorghi* [Link] Clinton) occurs throughout Nigeria. The disease was serious in the past, and occasional heavy attacks have been recorded (Harris 1962). In 1965 in Bauchi, Bornu, and Plateau provinces, the average loss due to this disease was 5%; however, a survey conducted in the Zaria province in 1967 revealed losses as great as 50% in some fields due to grain smut (Keay 1968). Harris (1963) examined 470 samples from the 12 northern provinces of Nigeria, and found that on average in each year about 1% of the spikelets were infected by grain smut. The disease has become less important, but in fields sown with non-treated seeds, the disease is common and sometimes severe. "Hot-spot" areas are Funtua, Samaru, Kaduna, Bauchi, Gombe, and Biu (Fig. 2) in the northern Guinea Savanna zone.

Table 1. Estimated sorghum crop loss due to smuts in Nigeria, 1975 to 1978 surveys.

Smuts	Yield loss (%)
Grain smut	1.0–1.5
Loose smut	0–0.5
Head smut	2.0–2.5
Long smut	2.0–2.5
Total	5.0–7.0

LOOSE SMUT. Loose smut (*Sphacelotheca cruenta* [Kuhn] Potter) is widespread, but severity is generally light and occurrence is sporadic. The yield loss did not exceed 2% (King 1970a), and the disease is not a problem at present. The disease is typically found on tillers of plants having healthy primary shoots. Fungicidal seed dressings effectively protect the crop from infection by smut spores adhering to the seed surface.

Table 2. Prevalence[a] and importance[b] of smuts in the various climatic zones in Nigeria.

Climatic zones	Year	Grain smut	Loose smut	Head smut	Long smut
Sudan savanna	1975	++2	+1	+++3	+++3
	1976	++1	+1	+++2	+++3
	1977	++2	+1	+++3	+++3
	1978	++1	+1	++2	+++2
Northern Guinea savanna	1975	++2	+1	++2	++2
	1976	++3	++1	+++3	++2
	1977	++2	+2	+++3	++2
	1978	++2	+1	++2	+1
Southern Guinea savanna	1975	+1	+1	++1	+1
	1976	++2	+1	++2	+1
	1977	++1	++1	++1	+1
	1978	++1	+1	++1	+1

a. Prevalence: – = not present; + = occasional; ++ = common; +++ = abundant.
b. Importance: 0 = no importance; 1 = minor; 2 = moderate; 3 = major.

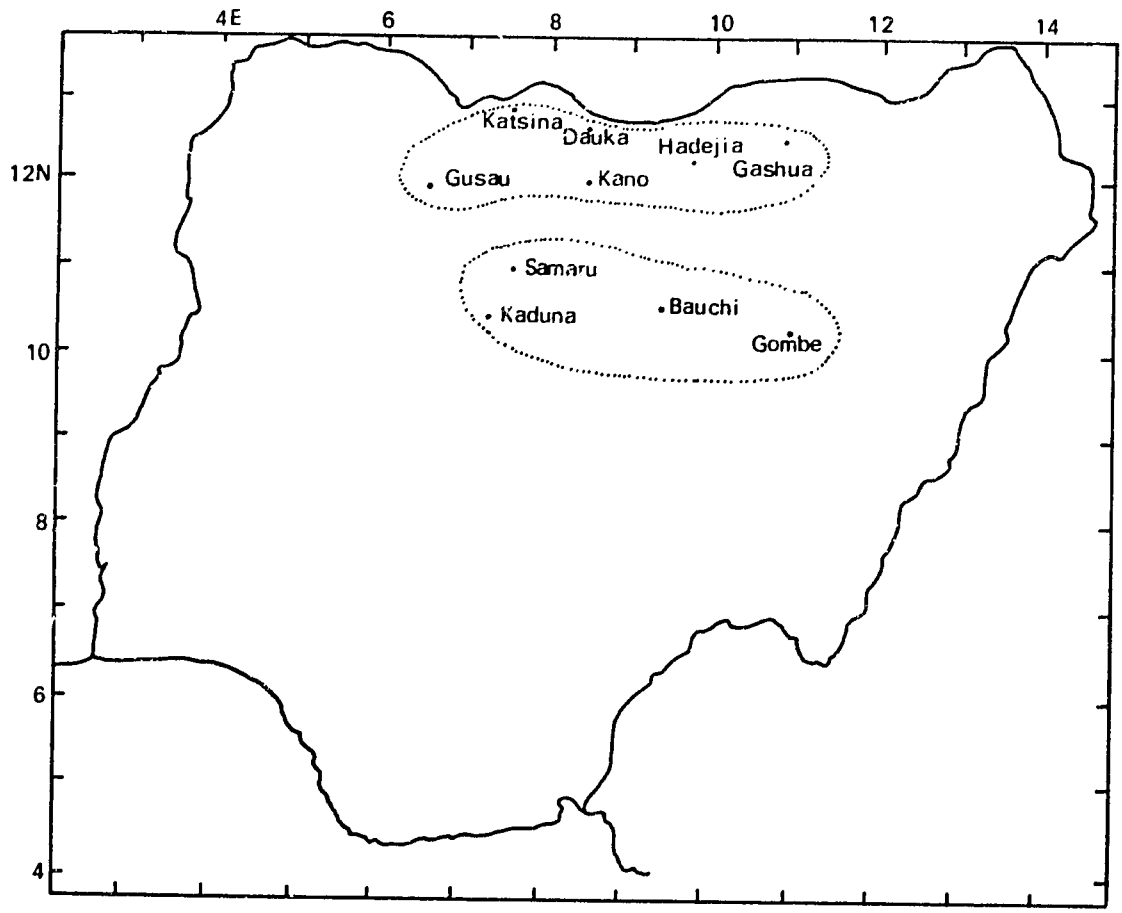

Figure 2. Areas of severe incidence of grain smut in Nigeria.

HEAD SMUT. Head smut (*Sphacelotheca reiliana* [Kuhn] Clinton) occurs sporadically in all sorghum-growing regions of Nigeria. It usually affects the late-maturing varieties, which are extensively grown by peasant farmers. Overall loss due to head smut was estimated at less than 2%. Infection is often greater in parts of the fields near trees. Though attacks were reported to be mild in the past (Keay 1968), in recent years the disease has increased considerably in the Sudan and northern Guinea savanna zones (Selvaraj 1977, 1978a, 1978d). "Hot-spot" areas are Samaru, Kaduna, Bauchi, Gombe, Gusau, Katsina, Daura, Kano, Hadejia, and Gashua (Fig. 3).

LONG SMUT. Long smut (*Tolyposporium ehrenbergii* [Kuhn] Potter) occurs annually and is severe in the drier Sudan and Sahel savanna zones. Annual yield loss attributed to the disease is about 1% (Manzo 1975). Long smut has caused severe damage during drought years (1971 to 1973), and has also been observed in a severe form in the past few years. It is particularly severe near trees. The mechanism of the tree-effect is not clearly understood. "Hot-spot" areas are located in the dry regions (Fig. 4).

Review of Past Research

Research on sorghum smuts in Nigeria has been in progress since 1957 (Harris 1963). Initially, studies were restricted to routine diagnosis of diseases, recording outbreaks, and advising farmers on field sanitation and other cultural methods of control. Grain smut was the most destructive disease and caused consider-

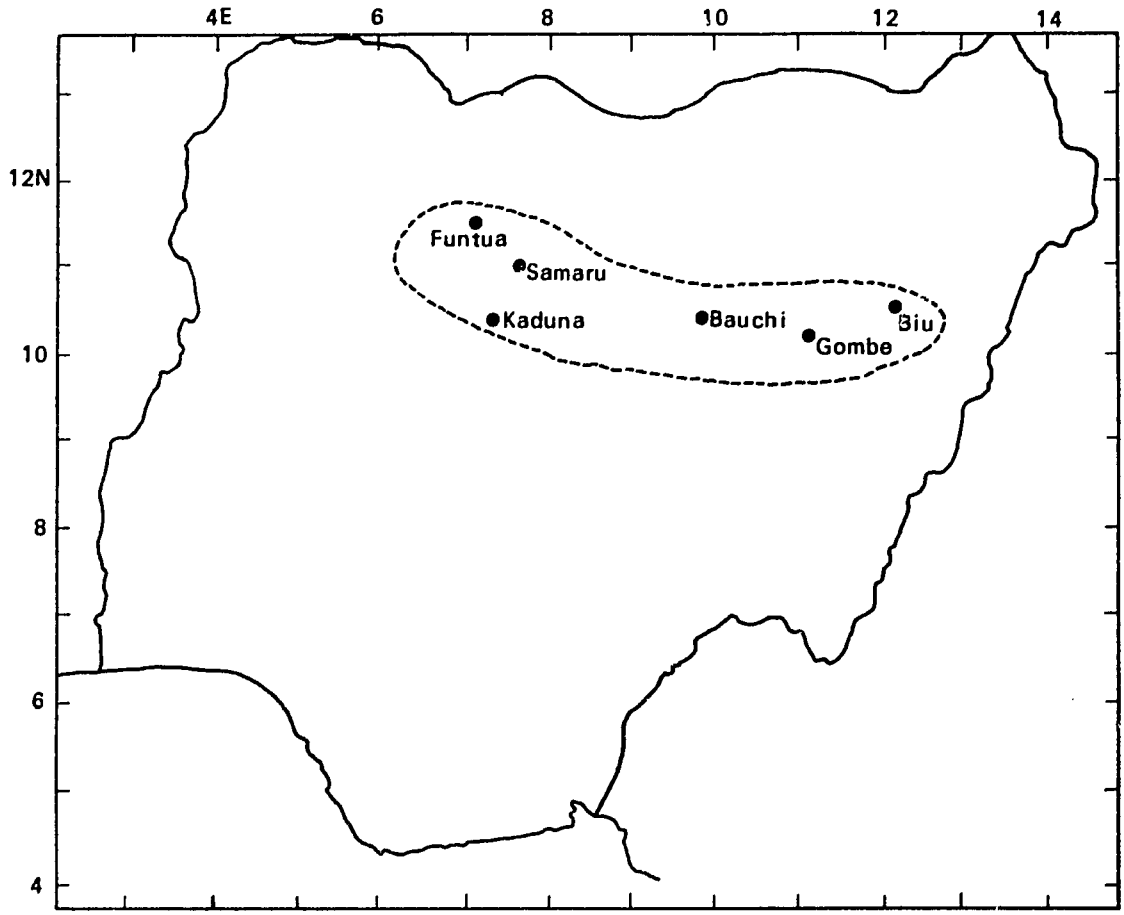

Figure 3. Areas of severe incidence of head smut.

able crop loss. The other smuts were relatively less damaging. Chemical seed treatment with organomercurials, which were later replaced by formulations containing thiram, was recommended to the farmers.

Studies on the physiologic races of head smut, grain smut, and loose smut were undertaken in later years, while the breeders were attempting to incorporate resistance to smuts in the new sorghum varieties. Few physiologic races of smuts exist in Nigeria. King (1969, 1970a, 1972) reported that only two races (race 2 and race 4) of grain smut exist on rainfed sorghum. He also reported that an unknown race, different from either of the above two, exists on sorghum grown during the dry season in the Lake Chad region. Variations in the grain-to-sori ratio of infected heads — as well as in the color, shape, and size of sori — are not associated with the existence of different physiologic races. The presence of a race similar to race 2 of loose smut has been demonstrated, and there are good indications of the presence of a new undescribed race of the loose smut fungus. It is likely that the Nigerian race of S. *reiliana* differs from any of the races described elsewhere. Investigations of the physiological races of long smut have not been made in Nigeria. The presence of only a few physiologic races among sorghum smuts might be due to lack of selection pressure on the smut fungi. This situation might change as more farmers follow improved agronomic practices.

No attempt was made to identify host resistance to grain smut. However, head smut reaction of Nigerian local materials was assessed.

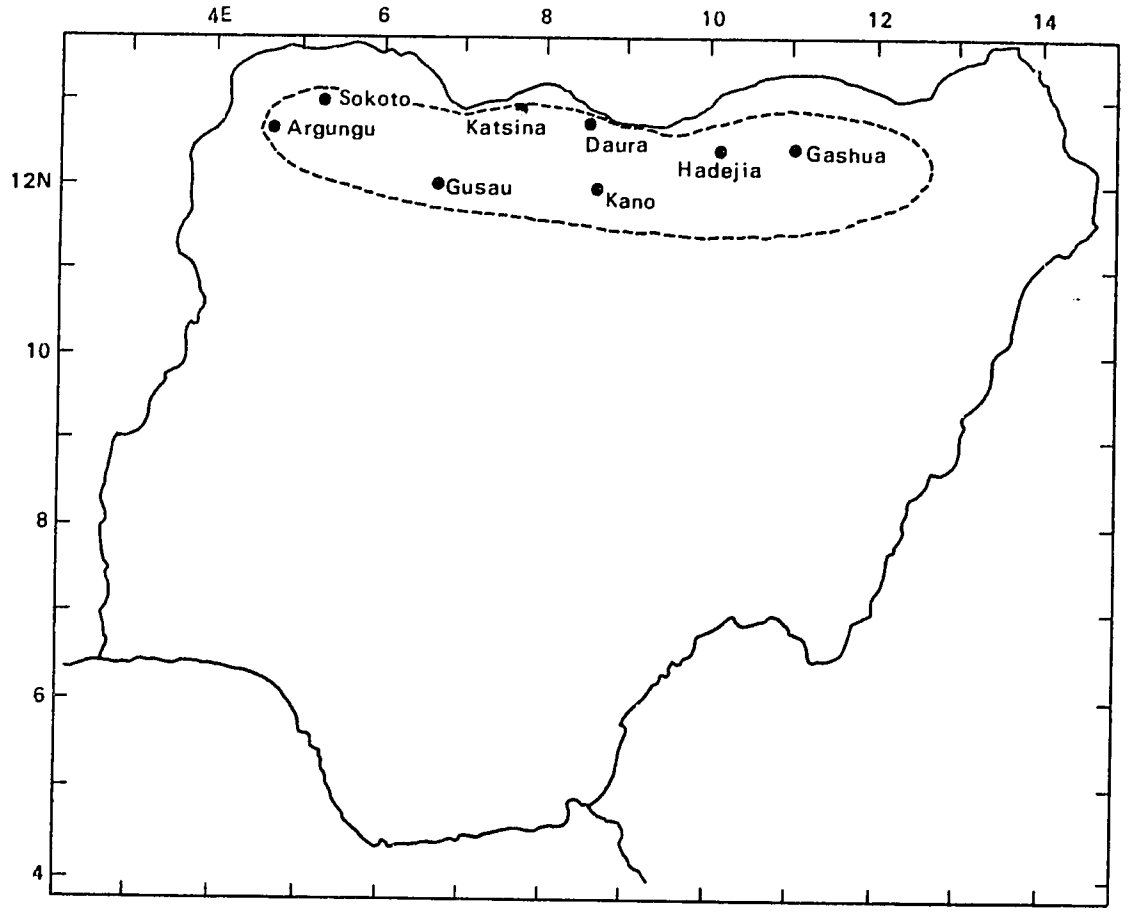

Figure 4. Areas of severe incidence of long smut.

Injection of head smut sporidial suspension into the growing point of seedlings produced satisfactory head smut development. The Nigerian sorghums SK-5912, YG-5760, L-2123, RZ 1-69, and FFBL 3-1-6 were very susceptible, while most exotic types showed very little infection.

The mode of infection of the long smut pathogen was studied in relation to environmental conditions (Manzo 1973, 1976). Infection results from airborne spores picked up from the soil and deposited on the panicle. Long smut spores have been detected on sorghum flag leaves at the early boot stage. Several inoculation techniques, including soil and seed application of sporidia, were compared. Good infection was obtained by injecting a suspension of sporidia or germinating teliospores into the boot with a hypodermic syringe. Infection decreased in inoculations (spraying of sporidia or germinated teliospores) performed in the pre- and early-anthesis stages. No infection was obtained from inoculations conducted at the early seed-set stage.

Techniques of Screening Germplasm and Breeding Material for Smuts Resistance

At the Kano Agricultural Research Station (Field C4B) germplasm collections and breeders' promising lines were screened for disease re-

sistance in the sorghum all-disease (including *Striga*) nursery. The nursery was developed and maintained by planting disease-susceptible materials and incorporating them into the soil towards the end of each growing season. However, materials from the "Breeding program for the Sudan savanna zone" were screened under natural epiphytotic conditions at the Kano breeding nursery. As smuts incidence is sporadic and not uniform, even in the all disease nursery, inoculations were made to create epiphytotic conditions.

Artificial Inoculation Techniques

Grain Smut

Two techniques have been evaluated:

SEED APPLICATION. Seeds were mixed with smut spores at the rate of 0.5 g/kg, and sown in cool soil. Severe infection occurred in fields sown under cold and wet soil conditions. This simple technique was followed for the evaluation of germplasm collections and breeding materials.

PARTIAL VACUUM INOCULATION OF SEED. Seeds were mixed with teliospores (0.2% by weight) by adding sufficient water to just cover the seed. The mixture was placed under partial vacuum for 2 hr, during which time the vacuum was broken at half-hour intervals. Seeds were spread to dry and the procedure repeated the following day. After the second smut application was dry, 0.2% of smut spores were dusted onto the seed surface. Good grain smut incidence was obtained by this technique. Seed-dressing chemicals and formulations were evaluated with seeds contaminated by this technique.

Long Smut

Inoculation of germinating teliospores in the boot stage (Manzo 1976) is currently used to screen limited numbers of germplasm and promising lines. Five grams of teliospores were germinated (germination percentage was 10 to 20%) in water, and made up to 1 liter. An injection of 25 ml of this suspension was forced into the boot with a hypodermic syringe equipped with a stout needle. This high level of inoculum was found to be necessary for satisfactory infection. Usually, ten heads were inoculated in each entry.

Head Smut

Several inoculation techniques, described below, were compared.

DISEASE NURSERY. A separate head smut nursery was developed at Kano (Field C4A). Smut spores (teliospores) collected in the previous year from locally grown sorghums were hoed into the upper 5 cm of soil at the rate of 5 g/m². Susceptible local selections (Fara-fara, SK-5912, and S-2123) were planted. The nursery was ready for screening in 1977. Germplasm and breeders' promising lines were screened in this nursery.

POT STUDIES. Plants were grown in 5 kg capacity pots in which head smut spores (0.5 g) were incorporated a few weeks prior to planting. This technique produced about 80% infection in susceptible materials, and is useful for screening small number of entries, particularly in the dry season.

APPLICATION OF GERMINATING TELIOSPORES TO SEED. Seeds were mixed with germinating teliospores (0.2% by weight) and sown immediately in cool soil.

PLACING GERMINATING TELIOSPORES IN THE PLANTING HOLE. Just before planting, a quantity (0.1 g approx.) of germinating teliospores was placed in the planting hole (5 to 7 cm depth). This technique has produced results comparable to those obtained in the smut nursery. However, it is followed only in certain limited seed-dressing trials, as a large quantity of smut spores is required for inoculation.

HYPODERMIC INJECTION TECHNIQUE. Smut spores collected in the previous year were stored in a dry cool place. Spores (0.5 g) were germinated in one liter of water and mixed in a blender for half a minute. Hypodermic injections were made twice, 20 and 40 days after planting. In the past, King (1969) employed this technique for screening sorghum collections. At present, it is used only in certain off-season screenings.

Procedure for Disease Rating

Grain Smut and Long Smut

For general evaluation of materials, disease on individual heads was assessed on a 0 to 5 scale, based on the estimated percentage of infected spikelets (0 = no disease; 1 = only a few seeds per head diseased; 2 = 1 to 2% of seeds per head diseased; 3 = 2 to 5% diseased; 4 = 5 to 10%, and 5 = more than 10% diseased).

In certain detailed studies with long smut, the actual number of sori in each head was counted and the average recorded.

Head Smut

The percentage of heads infected was recorded. In the screening trials, about 100 heads were counted to obtain reliable information on disease reaction.

Reaction of Germplasm and Breeders' Promising Lines to Smuts

Germplasm collections and breeding materials were evaluated for smut resistance, using some of the techniques described above.

Reaction of Grain Mold- and *Striga*-resistant Lines

Grain mold- and *Striga*-resistant lines, identified from germplasm collections by the IAR/JP 26 program, were tested for reaction to grain smut, head smut, and long smut.

All lines were susceptible to grain smut, some (SRN-4882 A, sart, SRN-4027, SRN-4481) had considerable resistance to head smut, and many of them had a certain degree of resistance to long smut (Table 3).

Reaction of Sorghum Varieties and Promising Lines

The sorghum breeding program at the Institute for Agricultural Research (I.A.R.), Samaru, is developing varieties adapted to the various climatic zones in northern Nigeria. Lines promising for the various zones were evaluated for long smut and head smut resistance.

Northern Sudan Savanna

The early nonphotosensitive materials are adapted to this zone, where the growing period ranges between 90 to 100 days. Reaction of early sorghum varieties to head smut and long smut under natural and artificial conditions of infection is presented in Table 4. Entries L 137/62 and G-59 are resistant to long smut, and P-147 and IS-12610C are resistant to head smut. Moreover, in the Kano breeding nursery, the early hybrids have only mild infections of the two smuts.

Sudan Savanna

The growing period for this zone is suited to photosensitive materials that flower after 100 days. Table 5 presents smut reactions of the promising lines. Line RZ-1 is very susceptible to long smut, and the other lines are moderately susceptible. KL 538-75, KL-2, and KL-5 have shown considerable resistance to head smut.

Northern Guinea Savanna

High-yielding semidwarf cultivars and hybrids are adapted to this zone. Reactions of promising lines and hybrids under natural conditions at Samaru and the promising lines under artificial inoculation at Kano are presented in Table 6.

At Samaru, under natural conditions, the incidence of head smut was mild and sporadic, while long smut occurred only in traces. Most of the lines are susceptible to head smut. The lines SL-187, SL 203/75, SL 1359/73, and SL 75/76 showed considerable resistance to long smut. However, as these lines are developed for the climatic zone where long smut is not a problem, resistance to this disease is not of primary importance.

Reaction of a Local Collection of Sorghum

A local collection of sorghum (late-flowering) consisting of 39 entries was screened for 2 successive years (1977 and 1978 crop seasons) for head smut and long smut resistance. Among these, six entries were identified to have a certain degree of resistance to head smut, and 14 entries were considered to be resistant to long smut.

Table 3. Reaction of selected grain mold- and *Striga*-resistant lines to smuts under artificial conditions of inoculation in Nigeria.

Lines	Reaction to[a]		Reaction to		
	Grain mold	*Striga*	Grain smut (0–5 scale)	Head smut (%)	Long smut (0–5 scale)
SRN-4841	R[b]	R	4.21	37.5	2.34
SRN-6838 A	R	R	3.96	19.4	1.92
SRN-4882 A	R	R	4.34	8.3	3.26
SRN-6496	R	R	4.27	42.4	3.13
Biu seed mold resistant	R	R	4.71	56.2	2.91
Tafirkit (Niger)	R	R	4.63	21.4	2.84
Sart	R	R	4.17	12.9	3.05
SRN-6788	R	MR	4.05	29.4	2.70
SRN-2123	R	MR	3.92	43.8	2.91
SRN-4310 B	MR[b]	MR	4.33	38.3	3.40
SRN-4627	MR	MR	4.64	16.4	3.36
SRN-4423 B	MR	MR	3.62	31.2	4.13
SRN-44881	MR	MR	3.83	14.6	2.82
SRN-4882 B	MR	MR	4.15	45.4	3.64

a. Reaction to grain mold and *Striga* is based on the evaluation made by IAR/JP. 26 program.
b. R = Resistant; MR = Moderately resistant.

Table 4. Reaction of early sorghum lines, promising for the Northern Sudan savanna, to smuts.

Entries	Natural incidence[a]		Artificial inoculation[b]	
	Head smut (%)	Long smut (0–5 scale)	Head smut (%)	Long smut (0–5 scale)
Early varieties				
CK-60B	4.4	2.2	32.5	4.5
HP-3	3.6	1.8	25.6	3.8
HP-8	5.7	1.0	43.7	1.7
P-147	0	0	12.9	2.0
IS-12610C	0	0.2	15.3	2.1
Ex-Bauchi	3.9	1.2	59.3	2.6
L-137/62	5.6	0	22.4	0.8
G-59	8.2	0.4	48.4	1.2
Early hybrids				
CK-60A × HP-3	2.9	1.3		
CK-60A × HP-8	1.4	0.9		
CK-60 × P-147	0	0.7		
CK-612A × HP-3	0.6	0.5		
CSH-5 (India)	1.1	0		
CSH-6 (India)	0	0.6		

a. Natural incidence recorded in the sorghum-breeding nursery, Kano, 1977.
b. Reaction of early hybrids was not assessed under artificial conditions of inoculation.

Table 5. Reaction of promising Sudan savanna lines to smuts at Kano.

Lines	Natural incidence		Artificial inoculation	
	Head smut (%)	Long smut (0–5 scale)	Head smut (%)	Long smut (0–5 scale)
RZ-1 (check)	2.1	2.9	29.8	4.6
YG-5760 (check)	4.9	1.1	37.3	2.4
KL-408-75	2.8	1.0	42.1	3.6
KL-538-75	1.0	0.8	12.4	2.5
L (94 × SK)	2.9	0.3	31.5	2.6
KL-1	4.6	1.2	29.3	2.1
KL-2	1.3	0.5	8.6	2.7
KL-3	5.3	1.4	52.1	3.1
KL-4	4.7	1.5	45.2	2.5
KL-5	1.2	1.0	14.7	2.3
KL-6	3.8	1.2	25.3	3.5
Kano bulk line	4.9	1.5	32.6	3.1
IS-1287	3.1	0.3	29.7	2.8

Table 6. Reaction of promising Northern Guinea lines and hybrids to smuts.

Entries	Natural incidence at Samaru, 1976		Artificial inoculation[a] at Kano, 1977	
	Head smut (%)	Long smut (0–5 scale)	Head smut (%)	Long smut (0–5 scale)
Lines and varieties				
SK-5912 (SSV-3)	1.2	0	54.3	1.2
Var. 2123 (SSV-4)	0.9	0.6	61.5	1.5
Var. 2141 (SSV-5)	1.1	0	49.2	1.9
SL-181 (SSV-8)	3.6	0	51.4	2.3
SL-187 (SSV-6)	4.3	0	32.5	0.6
SL-201/75	2.5	0.2	43.1	1.2
SL-203/75	1.5	0	26.4	0.54
SL-1202/73	4.2	0.6	35.3	1.8
SL-1266/73	3.5	0	41.7	2.4
SL-1389/73	1.1	0	25.6	0.48
SL-1499/73 (SSV-7)	1.4	0.4	54.2	1.7
SL-1621/73	2.5	0.6	18.5	1.5
SL-75/76	2.1	0.0	33.2	0.45
FFBL (Tall sorghum)	3.6	0.1	48.6	2.4
Hybrids				
RCFA × L-187 (SSH-1)	5.4	0		
RCFA × SK-5912 (SSH-2)	3.3	0.2		
ISNIA × L-187	1.1	0		
ISNIA × L-1499	0.5	0		
ISNIA × L-1266/73	0.8	0.3		
ISINIA × L-1389/73	2.1	0.3		
Kurgi A × SK-5912	1.0	0.2		

a. Reaction of the hybrids to smuts was not assessed under artificial conditions of inoculation.

Farming Practices on Smut Development

Sowing Date

It is the practice among farmers in Nigeria to sow sorghum as soon as the rains are established. Early plantings support good seed germination and seedling establishment, and in most cases, have given better yield. But early plantings have severe long smut incidence. Improved early varieties sown at this time mature before the rainy season ends, leading to molding of the heads. The natural incidence of long smut and other diseases in HP-3 sown at different dates was studied in the experiments planted by the Institute of Agricultural Research (I.A.R.) agronomist at Kano, 1976 to 1978.

Early plantings had good seed germination and seedling establishment, and generally gave good yield (Kaigama 1977–1978). Long smut incidence was negligible in the 1978 trial. Results (Table 7) show that in both 1976 and 1977 long smut was observed only in the early sowings.

Soil Fertility

In farmers' fields, plants grown on poor soils and poorly maintained can have severe grain smut.

The effect of various levels of nitrogen, applied as urea in a single dose before planting, on grain smut incidence in a susceptible inoculated local Fara-fara selection was studied at Kano in 1977. Grain smut was severe in low nitrogen levels (Table 8).

Table 8. Effect of nitrogen application on the development of grain smut in a susceptible sorghum at Kano, 1977.

Application rate (kg/ha)	Grain smut (0–5 scale)
0	4.12
25	3.07
50	1.95
100	1.18

Smut Control by Chemical Seed Treatment

Use of resistant varieties has been the primary means of disease control in sorghum. Fungicidal application is not economical. Seed treatment — inexpensive, safe, and easy to apply — is the only chemical method recommended to farmers in Nigeria. The objective of seed treatment is to improve seedling establishment and to control grain smut. Studies were made to increase the efficiency of seed and seedling protection, and to control grain smut.

Routine Seed Dressing Practiced by Farmers in Nigeria

Seed treatment with formulations containing thiram (thiram/aldrin[1], thiram/lindane[2]) is an

1. Aldrex T : Trademark of National (Shell) Ltd.
2. Femasan D: Trademark of ICI Plant Protection Ltd.

Table 7. Effect of sowing dates on the development of long smut[a] and other diseases in HP-3 sorghum at Kano, 1976 and 1977.

1976			1977		
Date	Long smut	Anthracnose	Date	Long smut	Grey leaf spot
1 July	1.32	1.23	21 June	1.89	1.78
10 July	1.01	1.83	29 June	1.46	2.64
20 July	0	2.75	7 July	0.32	3.42
30 July	0	3.46	15 July	0	3.95

a. Disease ratings are on 0 to 5 scale.

effective practice, which gives almost 100% control of grain smut in routine seed-dressing trials. An effort was made to understand the effectiveness of this practice in the farmers' fields. Prevalence of grain smut in adjacent fields planted with treated and untreated seeds was surveyed (Table 9). Most of the treated fields had less than 1% infection while the untreated fields had higher infection levels.

Table 9. **Prevalence of grain smut in farmers' fields sown with treated or untreated seeds in Nigeria.**

Location	Seed sown	No. of fields examined	No. of fields with various levels of smut incidence			
			0	0.1–1.0%	1.1–5.0%	above 5.0%
Ungogo	Treated	21	17	3	1	0
	Untreated	18	6	4	5	3
Rano	Treated	14	10	2	2	0
	Untreated	12	5	1	4	2
Birnin Kudu	Treated	17	13	3	1	0
	Untreated	19	8	3	3	5
Wudil	Treated	8	5	1	2	0
	Untreated	6	2	1	1	2
Bichi	Treated	24	18	6	0	0
	Untreated	16	6	3	4	3

Seed treatment is popular among the progressive farmers in Nigeria. A bench-mark survey (Williams et al. 1976) of selected locations in the Kano State of Nigeria revealed that in 1976 about 36% of the farmers applied chemical dressings to their seed before planting (Table 10). The survey did not indicate the percentage of area sown with treated seed, and it is not easy to estimate the percentage of the sorghum-growing area sown with treated seeds (Selvaraj 1978b). The National Seeds Service at Samaru is supplying farmers with improved seeds which are already treated with formulations containing thiram. The area sown with such seed is still less than 5%. However, as the traditional farmers are now also able to appreciate the importance of seed treatment, it is foreseen that in the future more area will be sown with treated seeds.

Nonsystemic Chemicals — Seedling Emergence and Grain Smut Control

Comparison of Chemicals

Medium-quality seeds were artificially contaminated with grain-smut spores, following the partial-vacuum method. Treatments were randomized in five replications with 200 seeds in each treatment (Plot size: 2 × 7.5 m). Seedling emergence counts were taken 15 days after planting and grain smut was recorded in the crop-maturing stage.

Thiram, maneb, captan, mancozeb (Dithane M-45), quintozene (Brassicol), thiram/aldrin (Aldrex-T), and thiram/lindane (Fernasan-D) were studied. Seeds in all chemical treatments produced significantly greater stand counts than the control seeds; the treatments also gave satisfactory control of grain smut.

Comparison of Good and Poor Quality Seeds

The effect of treating good and poor quality seeds on the incidence of grain smut was

Table 10. Percentage of farmers applying seed-dressing chemicals at selected locations in the Kano State, Nigeria, 1976.

Location	Percentage
Karaye	42
Gazarawa	40
Dawakin Kudu	19
Birnin Kudu	27
Roni	67
Gwaram	29
Average of all samples	36

Source: Williams et al. (1976).

studied (Table 11). Improved germination of poor quality seed was obtained by thiram treatment, but better control of grain smut was obtained in good quality seeds than in the poor ones. Perhaps poor quality seeds might be more vulnerable to attack by the grain smut pathogen. A high rate of thiram might be required for better control of grain smut in seeds of poor quality.

Systemic Chemicals—Seedling Emergence and Grain Smut Control

Though conventional formulations containing thiram have given satisfactory control of grain smut and favored seedling establishment, seedborne fungi associated with moldy grains, such as *Fusarium* sp, *Curvularia* sp, *Helminthosporium* sp, and *Phoma* sp, and soil-inhabiting *Fusarium* spp are not controlled by present seed-treatment practices (Selvaraj 1978b). Hence a broad approach to seed dressing was tested, and chemicals and mixtures of chemicals with a wide spectrum of activity were introduced to increase the efficiency of crop protection.

Poor-quality Seed

The effect of seed treatment on fungal infection, seed germination, and smut control was studied in severely molded poor-quality sorghum (var Ex-Bauchi). One lot of the treated and untreated seeds was germinated in petri dishes containing PDA. Each treatment had 60 seeds and was replicated four times. After 10 to 12 days, the percentage of germination and infection by various fungi was recorded. Another seed lot was artificially contaminated with grain smut spores and planted in the field.

In the laboratory (agar plates) and field tests, systemic fungicides remarkably increased germination and reduced the percentage of seeds from which *Fusarium* could be recovered (Table 12). The higher germination could be the direct effect of the chemicals on the fungal flora of the seeds. Thiabendazole and guazatine are very effective against *Fusarium* spp and gave the highest percentages of germination. *Fusarium* sp are the important fungi responsi-

Table 11. Effect of treatment (Thiram) and no treatment of good and poor sorghum seed on seed germination and grain smut incidence in some promising cultivars

Variety	Seed quality	Germination (%)		Grain smut (0–5 scale)	
		Not treated	Treated	Not treated	Treated
Ex-Bauchi	Good	82.3	82.6	4.14	0.68
	Poor	50.6	69.4	4.32	1.75
HP-3	Good	91.5	92.3	3.96	0.84
	Poor	42.5	73.5	4.12	2.13
S-7706	Good	79.2	80.4	3.20	0.73
	Poor	41.4	68.9	3.68	1.84
CK-60	Good	85.6	86.0	3.61	0.93
	Poor	64.9	81.2	4.22	2.01

ble for the reduction in seed germination (Selvaraj 1978c).

Guazatine is not effective against grain smut. The other chemicals gave good smut control.

Medium-quality Seed

The field experiment (Table 13) was conducted in 1976. All chemicals except carboxin increased seedling emergence. Systemic fungicides, alone or in combination with the conventional chemicals, are comparable or slightly superior to thiram/aldrin or thiram/lindane combinations. Except for the *Fusarium* spp, most of the fungi were found on the seed surfaces. Hence, even if the internal fungi are

Table 12. Effect of chemical treatment of moldy sorghum grains on fungal infection, seed germination, and grain smut incidence in Nigeria.

			PDA Plates		Field	
Chemical	a.i. (%)	a.i. (g/kg)	Germination (%)	*Fusarium* sp (%)	Germination (%)	Grain smut (0–5 scale)
Thiram	50	1.5	51.2	55.2	63.1	1.86
Maneb	80	2.0	50.7	58.9	61.9	1.98
Benomyl	50	1.0	68.5	37.3	60.2	0.92
Carbendazim	50	1.0	69.3	35.4	60.8	1.12
Carboxin	75	1.5	47.2	60.2	65.4	0.35
Thiophanate methyl	70	1.4	65.6	36.5	61.9	1.03
Thiabendazole	32	0.64	75.4	28.3	62.5	0.94
Guazatine	35	0.88	74.2	31.1	62.3	2.95
Untreated control	–	–	45.4	64.2	53.2	4.22
SE ±			1.83	1.62	1.54	

Table 13. Effect of seed treatment on seedling emergence and control of grain smut, 1976.

Chemical/formulation	a.i. (%)	a.i. (g/kg)	Seedling emergence (%)	Grain smut (0–5 scale)
Reference				
Thiram/aldrin (Aldrex T)	50/25	1.5/0.75	68.5	0.87
Systemic				
Benomyl (Benlate)	50	1.0	63.1	0.63
Carbendazim (Bavistin)	50	1.0	59.1	0.51
Thiabendazole (Tecto)	32	0.64	58.8	0.97
Carboxin (Vitavax)	75	1.5	51.4	0.32
Thiophanate methyl (Topsin)	70	1.4	61.2	0.74
Combinations				
Carbendazim/maneb (Bavistin M)	50/30	1.5/0.9	64.3	0.59
Thiophanate methyl/thiram (Homai)	50/30	1.0/0.6	75.2	0.61
Thiophanate methyl/maneb (Peltar)	25/25	0.5/0.5	65.8	0.73
Untreated control	–	–	52.7	4.27
SE ±			1.41	

controlled by systemic fungicides, results in terms of improved seedling emergence may not be striking.

All the chemicals gave satisfactory smut control. Carboxin is the most effective chemical against grain smut, though it has not improved seedling emergence.

Effect of Systemic Chemicals on Head Smut

Seed dressing with systemic chemicals was attempted as a new approach to head smut control.

In preliminary studies, various inoculation techniques were compared, and the application of germinating teliospores (teliospores + sporidia) in the planting hole or injection of the same in the growing point of seedlings gave the best results. Inoculation with ungerminated teliospores was less effective.

Comparison of Seed-Dressing Chemicals

The efficiency of three chemicals was compared, following the inoculation technique of placing the teliospores and sporidia in the planting hole. Results (Table 14) show a general reduction of head smut in seed-treated plots. Fenfuram is superior to Vitavax and pyracarbolid.

Comparison of Inoculation Techniques

The experiment was repeated using different inoculation techniques. Fara-fara sorghum, which had the highest level of infection in the previous year's trial, was used for the study. Results are presented in Table 15.

In inoculations, the chemicals generally reduced the percentage of head smut incidence. But Vitavax was not effective where technique C was employed. The results obtained with technique B confirm the previous year's results (Table 14). The protection offered by the chemicals is only partial. Perhaps the infection of seedlings might have occurred over an extended period of time which exceeded the period of protection offered by the chemicals. However, the results should be interpreted cautiously as the experiments were conducted only once at one location.

Summary and Future Line of Work

Of the four smuts recorded on sorghum in Nigeria, grain smut, head smut, and long smut are economically important. However, the prevalence and importance of the various smuts will be monitored along with other diseases to determine research priorities.

The identification of disease-resistant materials with a view to their utilization in resistance breeding is the main approach to the control of long and head smuts. Suitable artificial-inoculation techniques standardized at the I.A.R. were successfully employed to screen germplasm and breeding materials. Future efforts will be made to identify resistance from local collections. Only a few physiological races of the smuts have been reported in Nigeria.

Table 14. Effect of seed dressing on the incidence of head smut in four sorghum varieties, 1977.

Treatment	Rate per kg	Percentage of head smut on			
		SK-5912	Fara-fara	RZ-1	YG-5760
Nontreated check	–	1.8	0.7	1.2	0.9
Inoculated check	–	38.4	58.3	45.2	49.6
Vitavax 75, inoculated	2.0 g	25.1	39.3	27.6	30.5
Fenfuram (Panoram 35), inoculated	15.0 ml	18.3	26.4	20.3	16.9
Pyracarbolid 25, inoculated	2.5 g	22.5	33.6	25.2	28.2
SE ±		1.55	1.92	1.61	1.44

Table 15. Effect of inoculation techniques and systemic seed dressings on the percentage of incidence of head smut.

Seed treatments	Inoculation techniques[a]		
	A	B	C
Check 1 (no dressing, not inoculated)	0.6	1.5	1.1
Check 2 (no dressing, inoculated)	13.3	58.2	52.7
Vitavax 75, inoculated	5.6	39.4	48.3
Fenfuram (Panoram 35), inoculated	2.4	24.7	30.9
Pyracarbolid 25, inoculated	3.8	30.5	26.8
SE ±	1.07	1.93	1.76

a. A = Seed application of teliospores and sporidia.
B = Placing teliospores and sporidia in the planting hole.
C = Head smut sick plot incorporated with abundant teliospores.

Studies on the physiological races of head smut and long smut will be taken up again to determine if any new race has developed in recent years.

Investigations were made to improve chemical seed treatment, an economical and practical way of disease control on sorghum. Though formulations containing thiram have satisfactorily controlled grain smut, an overall approach to the control of sorghum diseases including the smuts (grain and head smuts) was resorted to, using chemicals with wide-spectrum activity. Evaluation of new chemicals will be continued to find more effective seed dressings.

Farming practices, like sowing date and soil fertility, influence the incidence of long smut and grain smut. Further studies are required to determine the role of soil moisture and other soil conditions on grain smut and head smut, and the influence of atmospheric humidity and temperature on long smut infection.

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The Head Smut Program at Texas A & M

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History of Head Smut in Texas

From 1890, when head smut was first reported in the USA, through the 1920s when reports of its distribution and occurrence were noted in Texas (Potter 1914; Reed et al. 1927), little attention was given to the disease. The key report by Stewart and Reyes (1958 established the severity of the disease, the use of disease-screening nurseries, and the possibility of physiologic races. Head smut, in fact, was a key disease which eventually led to the development of the sorghum disease resistance program at Texas A & M (Texas Agri Exp Sta 1974–76).

During the 1950s, RS-610 (ATx 3197 × Tx 7078), one of the early sorghum hybrids, was widely distributed in southern Texas. It, along with other hybrids (pedigrees similar to ATx 399 × Tx 7078), transformed this region into a grain sorghum growing area and at the same time promoted the appearance of head smut (Reyes et al, 1964). Rosenow (1963) used Combine White Feterita, Tx 09, as a source of resistance to head smut and developed a smut-resistant version of Combine 7078 known as Tx 414. Rs-626 (ATx 3197 × Tx 414) was an example of this first generation smut-resistant hybrid. While Rosenow cautioned about possible new races and found evidence of different genes for resistance, he was cautiously optimistic that with the new sources of resistance, when deployed through hybrids, "the disease should be brought under control, and head smut should again become a minor sorghum disease."

As early as 1965, shortly after the deployment of RS-626 and other head smut resistant lines, smut was found in resistant hybrids (this was reported by L. E. Clark and R. A. Frederiksen as a seed mixture). In 1967 near Berclair, Texas, we observed smut in commercial fields of several different commercial hybrids advertised as smut-resistant. In 1968, we grew a set of selected sorghum lines in one of these fields. These sorghum differential varieties were selected from the work reported by Mehta et al. (1967) and Wilson (1969). These sorghum lines subsequently became the base for the Uniform Head Smut Nursery, and in their initial deployment assisted in the description of a key race (race 3) of *Sphacelotheca reiliana* (Frederiksen, et al. 1968). In 1975, we also reported the occurrence of yet another population of smut, designated as race 4. (Frederiksen et al. 1975) This population, which is still increasing in prevalence in Texas, developed following the deployment of the first-generation downy mildew (*Peronosclerospora sorghi*) resistant hybrids. Currently, head smut is a potentially serious disease in most of the sorghum-growing areas of Texas. Major losses were observed in selected fields in 1976 and 1977 in both southern and northern growing regions of the state. This, coupled with head smut in maize, has required a high priority research effort in Texas (Frederiksen 1977; Frederiksen et al. 1976).

Background Research

Manis (1971) reconfirmed the nature of physiologic variability in *S. reiliana*. He demonstrated that potentially different races could be obtained from single teliospore progeny; clearly, races of *S. reiliana* represent populations and mixtures of populations. Mehta et al. (1967) established the basis for the Uniform Head Smut Nursery by finding certain sorghum entries resistant to all known isolates of the pathogen. Of his original list of resistant sorghums, only Lahoma remains. Rodriguez (1968) undertook to determine the role of interspecific hybridization in variability of sorghum smuts. Quite remarkably, he learned that *S. sorghi* and *S. reiliana*, while genetically related, quickly

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dissociate into their respective types. Certain physical traits of the respective pathogens appear to be closely linked. Spore marking, sporidia production, percentage germination, and infection type were all linked and quantitatively inherited. Transgressive segregation for virulence was not found from any of the crosses. Head smut teliospores were effective inoculum in soil mixtures, but not effective on seed. Consequently, these and related traits tend to skew populations toward the parent smut pathogens. Wilson and Frederiksen (1970a; 1970b) made studies on the histopathology of compatible and incompatible host-parasite interactions. In these studies, characteristics of pathogen differentiation were defined, as were characteristics of developing sori. In compatible systems, infection hyphae proceed intracellularly until they reach differentiating or partially differentiated meristem, when they develop intercellularly. Total colonization of partially differentiated panicles leads to the classical head smut sorus, while localization of the parasite results in partially smutted panicles. Only traces of the smut pathogen are present in blasted or sterile panicles. Wilson also found that in resistant varities of sorghum or with avirulent cultures of the pathogen, initial colonization of the host was restricted. In some cases, sporidia failed to fuse, and infection hyphae did not develop. By implication, Wilson's work indicated that there might be four initial states which might result in incompatible host-parasite interaction. These are: (a) failure to germinate in presence of the host, (b) failure to form a dikaryon, (c) failure to penetrate, and (d) failure to establish intercellular mycelium. Manis (1971) attempted to characterize the nature of host resistance to head smut. He noted physiologic changes, particularly among peroxidase enzymes from compatible and incompatible host-parasite interaction and an accumulation of a flavonoid-like material.

More recently, Miller (1978) studied a variety of inoculation techniques, hoping to improve on the hypodermic or field methods already employed (Edmunds 1963). Unfortunately, he was unable to improve on these procedures. J. H. Foster has learned that certain soils are more conducive to teliospore infection efficiency than others (M. S. thesis, in preparation).

Effect of Head Smut on Yield of Grain Sorghum

Populations of hybrid grain-sorghum plants were inoculated at various frequencies at Victoria and College Station, Texas. The experiments were designed to establish various levels of head smut in a constant plant density. The plant densities were established at about 150 000 plants per hectare. In all three tests, the effect of incidence of smut on grain yield was linear and significantly negatively correlated with yield (Table 1).

From a combined analysis of all three tests, it is estimated that an increase of 1% head smut decreases yield by 44.5 ± 2.7 kg/ha. These effects on yield are much more striking than those caused by downy mildew.

Table 1. Relations between incidence of head smut and grain sorghum yields at Victoria and College Station, Texas, 1975.

Victoria		College Station: Early planting		College Station: Late planting	
Average smut incidence (%)	Yield (kg/ha)	Average smut incidence (%)	Yield (kg/ha)	Average smut incidence (%)	Yield (kg/ha)
66.7	1259	25.8	4176	34.5	2470
37.1	3937	29.8	3815	31.3	2294
26.3	3102	12.7	4338	15.9	3327
0.7	4648	0.0	4838	0.0	3310

Race Concepts

Currently, four races of *S. reiliana* (Table 2) are recognized in the USA. Uniquely different populations exist in Africa (King and Frederiksen unpublished data), and there are differences among populations of *S. reiliana* attacking sorghum and corn (Frederiksen 1977; Miller 1978). Any population of *S. reiliana* is a mixture. The ability, however, to attack host cultivars with various genes for resistance is ever present. Currently, we designate these races on the basis of one of four key differential varieties, but many combinations of these pathogen populations are possible (Table 3). The two groups most frequently isolated include races 1, 3 and 1, 3, 4. Occasionally we have found races 1, 2, 3, 4 and races 1, 4. Very little race 2 exists in Texas (Table 4).

The distribution of these populations is directly related to previous cropping histories. Growers along the upper-coast areas, while utilizing one popular downy mildew resistant hybrid, developed a unique race 4 population (Horne 1976). Similarly, areas along the lower coast, growing other popular hybrids, developed their population of race 4; this race could be partially differentiated by commercial hybrids, but not by the differential varieties. It suggested to us that similar or closely related genes for resistance were being used in commercial breeding (Frederiksen et al. 1970). Monitoring with the Uniform Head Smut Nursery is done extensively in areas with head smut problems (Texas Agri Exp Sta 1974–76, Table 4).

Table 2. Reaction of four differential sorghum cultivars to physiologic races of *Sphacelotheca reiliana*.

Differential variety	Reaction by Race			
	1	1, 2	1, 2, 3	1, 2, 3, 4
Tx 7078	S	S	S	S
SA-281 (Early Hegari)	R	S	S	S
Tx 414	R	R	S	S
TAM-2571 (SC 170-6-17)	R	R	R	S

S = susceptible, R = resistant

Table 3. Common head smut race groups and their distribution in Texas.

Differential variety	Reaction to Race				
Tx 7078	S	S	S	S	S
SA-281	R	R	R	S	R
Tx 414	S	S	R	S	R
TAM-2571	R	S	R	S	S
Race mixtures	1, 3	1, 3, 4	1	1, 2, 3, 4	1, 4
Common race designation	3	4	1	4	4
Where found	South and Central	South Texas, High Plains	South Texas	High Plains	Central Texas

S = susceptible; R = resistant

Host Resistance to Head Smut

Resistance to head smut is complex and must involve either many genes or complex genes, suggesting that simple backcross breeding and testing in a single location are not appropriate. Lines selected as sources of head smut resistance must be stable over a number of locations. Recently, we initiated a population study by pooling S_1s based on, resistance at three locations. A single cycle of selection and random mating produced a dramatic shift in the frequency of resistant individuals (Miller 1978). This approach can be easily managed, but the question is: will the appropriate genes be pooled? Will these sources of resistance for smut-resistant hybrids have longer lives? Equally interesting has been the reaction of hybrids and hybrid combinations. Occasionally, with smut-resistant pollinator lines, or with smut-resistant seed parents, the resistance acts recessively and the hybrid smuts; in other cases, it is incompletely dominant. Occasionally a smut-resistant pollinator and seed parent will produce a smut-susceptible hybrid (Texas Agri Exp Sta 1974–76). Some common sorghum inbreds such as Wheatlacarel, Caprock, and certain kafirs have had a moderate level of smut resistance over the years and in different environments, even though they have been used extensively. Consequently, we are optimistic that a higher degree of stability of resistance to head smut is possible, particularly as more hybrids with different smut-resistant genes in

Table 4. Average incidence of head smut among entries of the Uniform Head Smut Nursery at seven Texas locations and one Kansas location in 1976.

Designation	1		2	3	4	5	6	7	8
	Reps								
	1&2	3&4							
	Smut percentages								
SA-281	0	0	0	0	0	0	0	0	0
PI-48770	0	0	0.9	0	0	0.1	0	0	0
B TAM-618	0	0	0	0	0	15.3	0	12.5	2.2
Lahoma Sudan	0	0	0	0	0	0	0	0	0
Calif. #38	0	1.4	0.6	0	0	11.7	0	3.2	0
Tx 7078	0	57.2	21.7	35.2	50.5	40.6	21.3	0	24.4
Tx 414	27.1	48.3	18.9	32.1	31.2	15.3	10.6	0	15.6
IS-12664C (SC-173)	0	0	1.2	0	0	2.4	0	1.5	0
IS-2403C (SC-103)	4.0	0	0	0	9.9	0.7	2.5	4.3	0
SC 170-6-17 (sel-4267)	0	1.1	1.9	0	17.1	14.9	5.6	0	6.1
SC 170-6-8 (sel-4252)	0	0	1.3	0	10.8	6.9	0	0	0
SC 170-14	0	0	0	0	5.4	4.5	4.4	5.3	0
TAM-428 (SC 110–9)	0	0	0	0	0	1.1	0	0	0
Tx-2536	0	5.8	4.3	6.9	9.3	9.1	5.4	49.3	2.8
B-Tx-399	0	12.0	3.0	4.0	8.6	0	9.5	0	0.
B-Tx-3048	0	0	0	0	0	10.5	0	0	0
B-Tx-3197	8.0	7.0	0.9	7.7	29.6	8.6	12.1	0	2.8
SC-241-12E	43.0	59.5	58.6	97.5	58.3	85.8	65.3	0	[illegible]
8311 (Pioneer)	10.6	4.2	1.1	2.9	7.5	11.7	5.3	4.7	2.2
NK-333	0	0	0	0	15.0	17.0	9.1	12.1	0
Race or race groups in each nursery	1,3 tr 4		1,3	1,3	1,3,4	1,3,4	1,3,4	1,3 tr 4	1,3 tr 4

1. Berclair; 2. Bonnie View (Acco nursery); 3. Corpus Christi; 4. Edna; 5. Ganado (P-A-G nursery); 6. Halfway; 7. Robstown; 8. Plains, Kansas.

different genetic backgrounds become available.

Sources for resistance have been screened over the past decade in the program at Texas A & M (Texas Agri Exp Sta 1974–76; Horne et al. 1978). The old pedigree sorghums (Table 5) and recent coverted lines have withstood repeated screening in many locations in Texas. These probably represent most of the known sources of head smut resistance.

Acknowledgment

The following co-workers contributed the data reported here: J. Foster, F. Miller, R. Miller, D. Rosenow, and D. Tuleen. The research was supported in part by the U.S. Agency for International Development, ta-C 1092 and ta-C 1389.

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Table 5. Comparison of head smut percentages in 1976 and 1977 in several open-pedigreed sorghum varieties previously identified as resistant.

Variety or designation		1976 Berclair	1976 Edna	1976 Halfway	1977 Kress
		Smut percentages			
SA-1754	Dwarf Bonar Durra	2.4	3.0	0	1.5
CI-182	Feterita	0	0	1.0	0
CI-693	Red Feterita	2.9	0	2.5	2.2
FC-6601	Spur Feterita	2.0	0	1.0	0
PI-3491	Hegari	0	0	0	3.3
SA-281	Early Hegari	0	0	0	0.5
SA-79	Bonita	2.1	0	0	4.6
SA-391	Combine Bonita	0.7	2.2	1.0	5.4
SA-392	Combine Hegari	0	0	0	1.7
B TAM-618	SR Combine Kafir	0	0	0	3.6
A TAM-618	SR Combine Kafir	0	0	0	0
FC-13641	White Kafir	0	1.5	0	0
PI-48770	White Kafir	0	0	0	0
SA-360	Imperial Kafir	1.1	0	0	6.0
TS-23240	Wetland Dwf. Kaoliang	0	0	0	0
CI-332	Dwarf Yellow Milo	0	0	0	0
CI-352	Standard White Milo	0	1.0	0	0
FC-8927	Dwarf White Milo	0	0	0	0
FC-16219	Finney	2.5	0	0	2.8
SA-368	Texas Milo	0	1.0	2.0	0
SA-7088	Chinch Bug Res. Milo	0	1.0	0	0
TS-13352	Double Dwf. White Milo	0	0	0	0
FC-8963	Double Dwf. Yellow Milo	2.0	0	–	0
FC-32127	Lahoma Sudan	0	0	0	0
SA-1998	Piper Sudan	15.0	0	0	–
DA-3494	Day × Atlas	0.8	0	0	0
FC-8989	Desert Bishop	0	0	0	0
KS-3	Spur Kaf. × Redbine 60	0	0	0	2.2
B TX 3048	Redbine selection	0	0	10.7	0
B Tx 3047	Redbine selection	0	0	0	0
B Tx 3035	Redbine selection	3.1	1.0	0	0
Tx 7000	Caprock	0	0	3.3	0

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Smut Discussion Session

Anahosur:

When there is partial infection in head smut, the head produces no grain; whereas with partial infection of grain smut, the non-smutted florets produce grain. What is the explanation for this difference?

Frederiksen:

An interesting question. I have no idea of the answer. There is little evidence of mycelium in the noninfected part. There must be some sort of interference with or antagonism toward the flowering mechanism.

K. N. Rao:

As head smut infection involves the rachis and rachis branches, there may be interference with nutrients going to other parts of the head. This would not be the case with grain smut.

Balasubramanian:

Has Dr. Frederiksen any information on the type of germination of head smut spores in the exudates from roots of resistant and susceptible cultivars?

Frederiksen:

Jim Foster in our laboratory has found that exudates from different cultivars affect rates of growth of the fungus following germination, but not germination *per se*. We believe that this may be a factor involved when different reactions are obtained with hypodermic inoculations, compared with reactions when the cultivars are exposed to soilborne inoculum in the field. When we inoculate by hypodermic injection we probably bypass some resistance mechanisms.

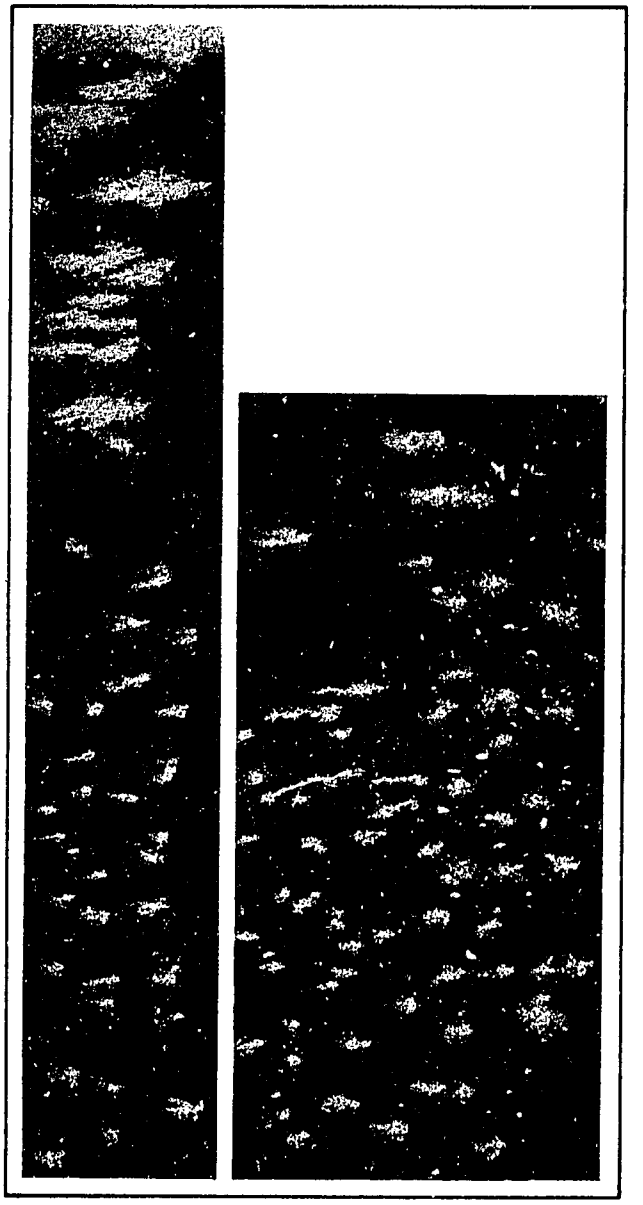
Doggett:

Does the population improvement approach offer a good opportunity for control of the variable smut populations? At ICRISAT we thought that if we could get our populations well established, as B and R populations, we might be able to move towards hybrids which were made from apparently homogenous material, but which would be genetically far more variable. This of course might be unsuitable in the USA where a high degree of uniformity is required, but it could work well in the developing world.

Frederiksen:

A heterogenous population should provide more stable resistance than a homogenous one. I think we have to be very careful about variability in maturity, not only because of problems of harvesting but also because of problems like midge — which increase with a spread of maturity. I think a mixture of different smut-resistant genotypes in a uniformly maturing background would be useful in controlling smut. Smuts are endemic, and thus the distribution of new genotypes occurs more slowly than with many other fungal pathogens, such as rusts.

Sorghum Ergot



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Sorghum Ergot

N. V. Sundaram*

Ergot, also known as sugary disease or "asali" disease of sorghum, was first collected by McRae in India (Madras State) in 1915. He described the pathogen in 1917 as *Sphacelia sorghi*, believed to be the conidial stage of an undetermined species of *Claviceps*. Kulkarni et al. (1976) described the perfect stage as *Claviceps*, but at present the name *Sphacelia sorghi* (based on the conidial stage), is in popular use.

Geographical Distribution, Host Range, and Economic Importance

Sorghum ergot has been reported from many sorghum growing countries (Tarr 1962; Molefe 1975).

The sorghum ergot pathogen has been reported to infect *Pennisetum americanum* and *Ischaemum pilosum* in India (Sundaram 1974; Sundaram and Singh 1975). Futrell and Webster (1966) reported successful infection of maize by *Sphacelia sorghi* from Nigeria. It was also found that the ergot from guinea grass *(Panicum maximum)* readily infected both sorghum and pearl millet. Reddy (1968) successfully infected pearl millet with sorghum ergot but Chinnadurai and Govindaswamy (1971) failed to get positive results. However, Reddy reported that the fungus from sorghum readily infected *Cenchrus ciliaris* and *C. setigerus* when artificially inoculated. Surprisingly, these two grass hosts were also reported by Ramakrishnan (1963) to be collateral hosts of *Claviceps microcephala (C. fusiformis)*, the millet ergot pathogen. Perhaps additional grass alternative hosts occur in other countries.

Ergot of rye *(Claviceps purpurea)* is well known for its alkaloid content, which is harmful to humans and cattle. In sorghum ergot, the sclerotia are not very well developed, in the sense that they are not so hard as those in ergot of rye or of other grasses; sorghum ergot has hollow locules containing conidia mixed with "honey secretion." This is perhaps the reason why harmful alkaloids are not always produced by the fungus. When mature sclerotia of ergot of rye are sectioned, no such hollow or honey secretion is observed. However, reports on the possible presence of alkaloids in sorghum ergot have come from Burma (Tarr 1962), India (Chinnadurai and Govindaswamy 1971), and Nigeria (Mantle and Waight 1968). The alkaloids varied from place to place. In Burma the alkaloids were undetermined, but caused fatal results when fed to cattle. In India ergotamine was found, while in Nigeria dihydro-ergosine was isolated.

Economic Importance

Ergot severity on sorghum depends mostly upon weather conditions at flowering. If days are cool (minimum of 19° ± 1°C), highly humid, and cloudy during the anthesis period, the spread of the disease is rapid and damage is severe. The male steriles become almost 100% infected. In India, many endemic pockets have been identified (Sundaram 1971). With the introduction of a hybrid program involving male steriles, the severity of te disease has increased, in some cases completely destroying seed setting. Estimation of yield loss is complex and dependent upon more than one factor. However, in Satara district of India's Maharashtra State during the 1975 wet season, extensive areas in seed production were severely damaged by this disease. I made similar observations in several seed-production plots elsewhere in India. Molefe (1975) reported Botswana's first epiphytotic of sorghum ergot during 1973–74. Late-planted sorghums were particularly affected.

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Resistance Screening Methods

Cool, wet, cloudy weather during anthesis of the crop favors rapid spread of the disease. Simulating these conditions successfully produced epiphytotic infections (Sundaram 1971). The method consists principally of spray-inoculating the spikelets with viable conidia when the stigmas are just emerging. Care is taken to direct the spray on all sides of the inflorescence so that the conidia are uniformly deposited. Immediately after spraying, the ears are covered with brown selfing bags. To assure inoculation, the spray is repeated. If the atmosphere is dry, it is essential to irrigate the field after spraying, preferably with sprinklers. Planting of the test entries should be timed so that flowering coincides with cool, wet weather. Best results are obtained when daytime atmospheric temperature remains between 19 and 28°C and there is good dew formation in the night.

Present screening methods appear to be quite effective and practical. To get the best results flowering must occur during cool wet weather — not always possible in rainfed semi-arid regions. In such cases it is difficult to achieve the desired results, unless we create humidity artificially during the incubation period of 6 to 10 days. If different types (photosensitive and nonphotosensitive) are screened, planting dates must be adjusted so that the plants will flower and can be inoculated at the same time.

With the scoring scales and techniques presently adopted, incidence on a crop basis is assessed by counting the infected and healthy ears, irrespective of the intensity of infection. Then, when normal grains mature, the intensity grade is worked out on a 1 to 5 or 1 to 10 scale as the case may be, with grade 1 being the least infected and grades 5 or 10 representing the most severely infected ears. The major drawback of this method is that the sugary secretion, which covers most of the healthy grains, makes it seem as if a much larger number of grains is infected. When sclerotia are formed, it is comparatively easy to assess the intensity.

Sources of Resistance

Few publications are available on sources of ergot resistance in sorghum. The pathogen is reported to infect ovaries up to 5 days following pollination. Resistant materials should be heavy pollen shedders, with the period from gynoecium to anthecium as short as possible.

Ajrekar (1926) tested numerous varieties grown at Poona and Dharwar, areas with high disease expression under natural conditions, and none was immune. Futrell and Webster (1966) reported that male sterility increases susceptibility, but lines that were female sterile showed a low degree of susceptibility. Twin-seeded varieties are also susceptible. A large-scale evaluation of 3680 varieties from the world collections of sorghum assembled by the Indian Council of Agricultural Research with the active cooperation of the Rockefeller Foundation was made at Coimbatore in late 1965 and 1966 (Sundaram 1971). Of 28 resistant lines identified, 15 were from Africa, eight from India, and the rest from the USA and Japan. An additional 76 entries were identified as somewhat resistant, with infection intensities of less than 10%. The resistant entries and the moderately resistant entries belonged to the major groups *caudatum* and *conspicuum,* and some of the moderately resistant entries were from the *durra* group. Periodic evaluation of released varieties and hybrids is done in India under the All-India Coordinated Sorghum Improvement Project.

No concentrated study appears to have been made on the variability of the pathogen, even though indications are that it is variable.

International Screening

To start with, locations could be selected in Sudan, Ethiopia, Kenya, Uganda, Botswana, Nigeria, Senegal, and Cameroon. In India, good locations would include Dharwar, Poona, Coimbatore, Indore, and Hyderabad.

Test entries may be limited to 50 in the first year of testing, and should include the 28 lines from the world collection already identified as resistant, ten to 12 of the elite varieties and hybrids from India. Released varieties and hybrids of the test locations could be included for comparison.

Research Priorities

Sources of Resistance

The facility to produce an artificial epiphytotic is

essential to evaluate test entries at any locality. All breeding materials and varieties resistant at one location should be evaluated in various centers.

Studies on Collateral Hosts

Further investigation is needed on the role of collateral hosts.

Fungicidal Control

Fungicidal control of this disease is not always feasible, but, in view of the intensity of the disease on male steriles commonly grown in hybrid programs, it is important to continue the study on the use of systemic fungicides for control of ergot.

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Ergot Discussion Session

Mengistu:
Ergot of sorghum occurs in Ethiopia, but sclerotia are not observed. Is sclerotial formation associated with certain ecological zones or other factors?

Sundaram:
It may be associated with ecological zones and also with cultivar. The factors influencing it are really not known.

Balasubramanian:
Sclerotia are closely associated with climate. Sclerotia may form in one year and not in another. Moreover, the appearance of ergot itself is greatly influenced by weather conditions, and given the same weather condition, the formation of sclerotia vary from one variety to another.

K. N. Rao:
I believe the environmental factors are of major importance and not the specific genotype.

Anahosur:
Two types of sclerotia are found in Dharwar, long curved and hard short ones. It is possible that two or more species may be involved.

Brhane:
How does the ergot organism perpetuate itself from season to season? In Ethiopia a higher incidence of the disease was recorded in the experimental farm than in farmers' fields. Inoculum may be building up under these conditions.

Sundaram:
Perpetuation is through sclerotia mixed with seeds and collateral hosts. Farmers' varieties probably flower during drier periods than do materials at the research station, and thus escape severe infection. There might be natural selection for resistance as well. In experimental fields, varieties flower at different times and thus some varieties may flower to coincide with favorable weather conditions, resulting in more ergot.

Zummo:
What part do insects play in the dissemination of ergot? Insects are attracted to the honeydew and will pick up the conidia and spread them throughout the crop.

Sundaram:
Insects spread the disease, from wild hosts to cultivated sorghum, as well as within the crop. In India 14 different insect species have been associated with field transmission of the pathogen.

K. N. Rao:
Are these rating scales for measuring ergot reaction?

Sundaram:
In the All India Coordinated Sorghum Improvement Project, we use incidence (which is crop infection %) and intensity of the disease on individual ears.

Balasubramanian:
Ergot is very common in the tropics, but is it prevalent in temperate sorghum?

Zummo:
The disease is not known in temperate sorghum-growing areas.

Bhat:
There are at least five areas of investigation needed for ergot toxins:

1. the chemical nature and biological effects of sorghum ergot sclerotia;
2. the carcinogenic nature of the alkaloids;
3. safe limits for human consumption;
4. the nature of ergot-induced diseases of cattle and man.

The staff of the National Institute of Nutri-

tion in India are willing to undertake studies of these problems, but they need a supply of materials.

House:

Time of flowering is effective in avoiding the disease in India. Is this also true of other countries, particularly in Africa?

Sundaram:

This appears to be generally true, but the critical flowering date varies with location.

Rosenow:

Is there a current list of resistance sources? Also, have elite breeding lines, containing wide adaptation and multiple disease resistance, been screened for ergot? Who has this information?

Sundaram:

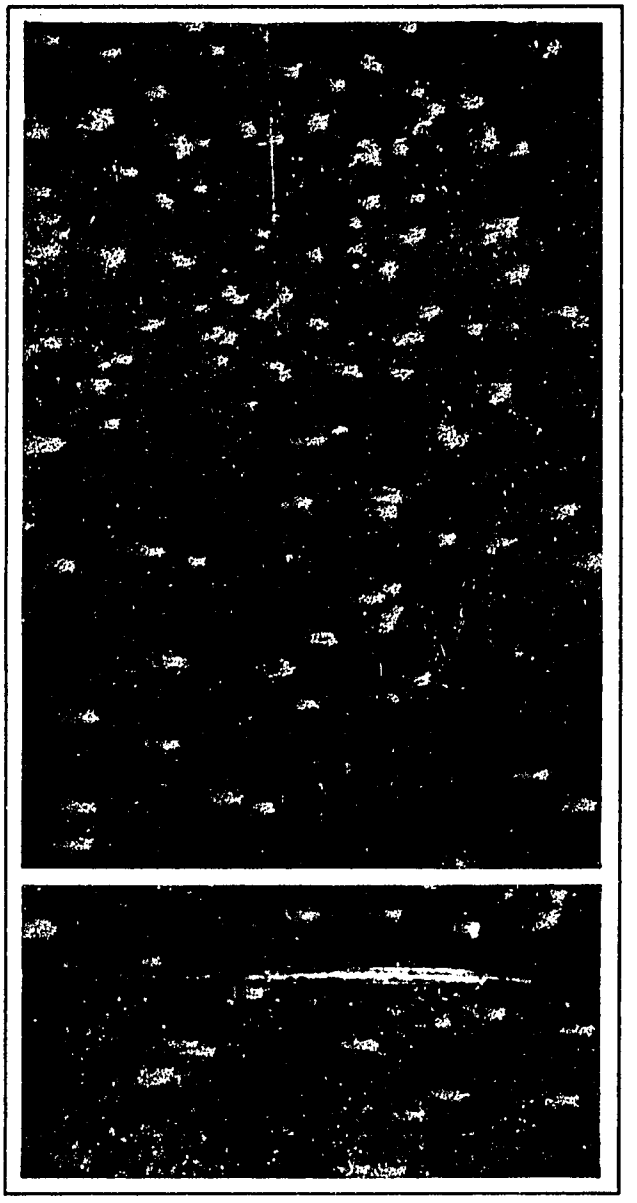
Two lists of sources of ergot resistance are available.

1. *Sorghum in the Seventies*, eds. Drs. N. G. P. Rao and L. R. House.
2. The Annual Reports of the All-India Coordinated Sorghum Improvement Project list the reaction of elite and released varieties and hybrids.

K. N. Rao:

At ICRISAT we are screening most of the elite materials this season, and we can assemble a preliminary ergot-resistance screening nursery for interested workers.

Sorghum
Bacterial Diseases



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Bacterial Diseases

N. V. Sundaram*

Bacterial diseases of sorghum, though they have been reported for a very long time, rarely cause epidemics in sorghum-growing areas. As many as ten bacterial pathogens, pathogenic either under field or experimental conditions, have been reported on sorghum from various parts of the world. Among these (Table 1), three have been frequently reported from many countries to be constantly associated with sorghum, causing leaf damage in the field under natural conditions.

Bacterial leaf diseases are especially important in rainfed sorghum, since continuous humidity with warm temperatures and rain splash help spread the pathogens. Occasionally bacterial pathogens are seedborne. Some pathogens have known alternative hosts, which include perennial grasses and cultivated crops. These also help the pathogens to survive adverse weather and other conditions.

Losses due to bacterial diseases have not been assessed, but there is no doubt that epidemics cause considerable damage to the crop. It is possible to mistake bacterial disease for deficiencies or rots, as at times saprophytes develop on leaf tissue destroyed by a bacterial pathogen, further confusing the symptoms.

Since the geographical distribution, capacity for epidemic development, screening methods, etc., vary with the types of bacterial diseases, they are dealt with separately in the following pages. Most of the information given is from Tarr's (1962) book.

Bacterial Leaf Stripe

Causal organism: *Pseudomonas andropogoni* (E. F. Smith) Stapp

Synonyms: *Bacterium andropogoni* E. F. Smith
Phytomonas andropogoni (E. F. Smith) Bergey et al.

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Bacterial stripe was one of the first bacterial diseases of sorghum to be described. The bacterium was isolated by Erwin F. Smith and his coworkers in the USA during the early twentieth century. Details of its taxonomy are given by Bradbury (1973).

Geographical Distribution and Economic Importance

Bacterial stripe has been reported throughout the sorghum belt of the United States, Argentina, Formosa, Australia, Nigeria, India, Japan, Sudan, and the USSR, and probably occurs in many other sorghum-growing countries.

In addition to grain sorghum, this disease occurs on sudangrass, broomcorn, johnsongrass, and *S. almum* under natural conditions. It is also known to affect maize and sugarcane under artificial inoculation.

Studies on the epidemiology and seasonal occurrence of the disease have been limited. Infection is established through the stomata and through leaf injuries. Among the disseminating agents are wind (carrying infected debris), rainfall, insects, and to some extent human beings passing through the field. The spread of the pathogen is favored by continued warm weather combined with high humidity. The spread of the disease is, however, arrested once dry weather occurs. Sources of infection include infected dry leaves left in the soil, seeds carrying the pathogen, and perhaps the perennial grass hosts around the field.

Screening Methods

Since detailed studies on the epidemiology of the pathogen have not been undertaken, no specific method for establishing artificial epiphytotics has been described. However, with the limited knowledge available on the various sources of inoculum and the method of entry of the bacterium into the host tissue, standardization of the technique to achieve effective

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methods of screening could be worked out. The main source of the inoculum could be infested debris spread on the whorls during optimum weather — high humidity and warm atmosphere. Sprinkler irrigation would help to establish the disease most effectively.

The scoring scales commonly used to score resistance to leaf diseases have been adopted

Table 1. Bacterial pathogens recorded on sorghum.

Name of the pathogen	Common name	First reported country	Author	Year
Pseudomonas andropogoni (E. F. Smith) Stapp	Bacterial leaf stripe	USA (Virginia)	E. F. Smith et al.	1911
Xanthomonas holcicola (C. Elliot) Starr and Burk.	Bacterial streak	USA (Texas)	Elliot	1929
Pseudomonas syringae Van Hall	Bacterial leaf spot	USA (Iowa)	Kendrick	1926
Bacterium sorghi (Burrill) Chester	Bacterial blight	USA (Illinois)	Burrill	1937
Pseudomonas fluorescens	Bacterial			
P. alboprecipitans Rosen	Leaf spot of Setaria	USA (Arkansas)	Rosen	1922
Pseudomonas rubrisubalbicans (Christopher and Edgerton) Kaasilnikov	Mottled stripe of sugarcane	USA	Christopher and Edgerton	1930
Xanthomonas albilineans (Ashby) Dowson	Sugarcane leaf scald		Ashby	1940
X. rubrilineans (Lee et al.) Starr and Burkholder	Bacterial stripe of sugarcane	USA (Hawaii)	Lee et al.	1925
X. rubrisubalbicans (Christopher and Edgerton) Savulescu	Mottled stripe of sugarcane	USA (Louisiana)	Christopher and Edgerton	1930
X. stewartii (E. F. Smith) Dowson	Bacterial wilt of maize	USA	E. F. Smith	
Pseudomonas lapsa (Ark) Starr and Burkholder	Bacterial stalk rot of maize	USA (California)	Ark	1940
Pseudomonas marginalis (Brown) Stevens	Leaf spot	Egypt	Melchers	1931
Doubtful pathogenicity				
Bacillus omelianski Serbinoff	Soft rot of sorghum	USSR	Serbinoff	1915
Bacterium termo Dujardin	Red stalk	France	Dujardin	1841
B. ovatum (Bruyning) Chester	Red blotches	Netherlands	Bruyning	1897

for use with bacterial diseases, hence, special scales are not required.

Sources of Resistance

Screening of varieties for their resistance to bacterial stripe has been mostly done under field conditions in almost all countries where the disease is present. In general, many workers have observed that "sorgos" as a group are more susceptible than grain sorghum or sudangrass. Elliot and Smith (1929) found that Silver Top, White (from India), Sumac Red Amber, and Early Amber were susceptible. Dwarf Early Sumac, Honey, Black Amber, Folger's Early Rose, and Sugar Drip were moderately resistant. Among the grain sorghums, only kafir (bird-proof) and chicken corn showed heavy infection; durras from India were resistant. In general, varieties from Sudan showed high resistance. On an average, about 40% of the grain sorghums tested in Virginia, USA, were highly resistant and 20% highly susceptible, the remainder being intermediate in reaction. Luttrell (1950), who made the evaluation in Georgia, USA, reported Hegari and Early Hegari to be highly susceptible, whereas Caprock, Double Dwarf Yellow Sooner, Double Dwarf White Sooner, Martin's Combine Milo, Texas Milo, Plainsman, and Imperial Kafir were slightly infected. Leoti sorgo, Cody, Shallu, and Tift, and sweet sudangrass are moderately resistant (Leukel et al. 1951). Broomcorn is severely affected in Argentina (Muntanola 1950). *P. andropogoni* is reported to infect *Sorghum caffrorum, S. bantuorum, S. durra, S. chinense,* and *S. saccharatum* in the USSR. (Yakushevskii et al. 1974).

The available literature on screening for resistance to bacterial stripe suggests possible sources of resistance, but no sorghum group was resistant. Continued evaluation of host resistances to bacterial stripe is essential.

Bacterial Streak

Causal organism: *Xanthomonas holcicola* (Elliot) Starr and Burkholder

Synonyms: *Bacterium holcicola* Elliot
Phytomonas holcicola (Elliot) Bergey et al.
Pseudomonas holcicola (Elliot) Stapp

Geographical Distribution and Economic Importance

This disease has been reported from the USA, Australia, South Africa, Argentina, New Zealand, Mexico, and perhaps from other sorghum-growing areas, especially the USSR, India, and the Philippines. Streak occurs in grain sorghums, johnsongrass, and sudangrass. Broomcorn is free from infection in the USA but is reported to be susceptible in Argentina.

There is no evidence to show the extent of crop loss due to this disease, but considering the leaf damage it can cause under favorable weather conditions, forage and grain losses could be considerable.

Epidemiology

Bacterial streak, like bacterial stripe, appears in warm, wet weather. Information on methods of creating epiphytotics is scant. Seed transmission cannot be overruled, since the infection is seen as early as the second-leaf stage in seedlings.

Sources of Resistance

Limited work suggests that sorghum cultivars differ in reaction to streak. In Kansas, Leoti Red sorghum, Barchet Kaoliang, Early White Milo, Buff durra, and certain kafirs were resistant under field conditions. Certain other varieties of kafirs and Freed sorghum were moderately resistant. Highly susceptible varieties were Wonder kafir, Farr's European milo Farr's Dwarf Hegari, and other Milo-Kafir-Hegari progenies. In general, kafirs were resistant while Leoti sorghum, Cody, Shallu, and Tift were moderately resistant.

Bacterial Leaf Spot

Causal organism: *Pseudomonas syringae* van Hall

Synonyms: *Bacterium holci* Kendrick
Phytomonas cerasi (Griffin) Bergey et al. in Clara, 1934
Phytomonas matthiolae

Pseudomonas holci (Kendrick) Bergey et al.
P. citraefaciens
P. citriputealis
P. trifoliorum
P. vignae
P. viridifaciens
P. prunicola
P. utiformica
P. hibisci
P. spongiosa

Geographical Distribution and Economic Importance

Since this bacterium has a wide host range, including monocots and dicots, its distribution is very wide. It is found in almost all sorghum-growing countries in the world.

Bacterial leaf spot on *Sorghum* spp has been reported from the USA, Argentina, China, Bulgaria, Yugoslavia, Rumania, the USSR, and probably is present in Italy, Hungary, Mexico, West Africa, and India. Although this disease is widespread, major crop loss has not been reported. However, epiphytotic outbreaks have occurred under favorable climatic conditions in Bulgaria on sudangrass, and caused severe leaf damage. Yakushevskii et al. (1974) reported this as a serious disease infecting almost all varieties of sorghum in the USSR.

Epidemiology

The organism causes leaf spotting on grass hosts. Spread of the disease is largely dependent upon wind and rain splash. Detailed information on the method of spread and conditions favorable to epidemic outbreak is lacking. Successful infection can be obtained by spraying a bacterial suspension on leaves, thereby implying that the bacterial pathogen gains entry through the stomata. The incubation period is very short, from 2 to 3 days. Initially bacteria move intercellularly, but soon cause the disintegration of the affected tissue and become intracellular. Sorghum and sudangrass are susceptible at all stages of their growth. Under low temperatures and wet weather, the pathogen spreads rapidly. By placing infected leaves on the soil surface at planting, Chumaevskaya and Nikolaeva (1975) obtained infection. Successive crops of sorghum favored higher infection.

The bacteria perpetuates on infected debris left in the soil, and is also seedborne. Seeds collected from infected plants and sown in sterile soil showed positive infection in most of the cases. The bacterium survives for at least 3 months on dry sorghum seeds.

Sources of Resistance

Systemic screening of varieties for reaction to bacterial spot has not been done in many countries. Recently, Chunaevskava and Nikolaeva (1975) tested 122 varieties of sorghum and found 42 resistant to *P. syringae*. Nikolaeva (1974) reported that of 153 cultivars screened, resistance was observed in the Negrityansku (African) group.

Other Bacterial Diseases of Minor Importance

Apart from the three major bacterial diseases reported on *Sorghum* spp, a number of bacterial pathogens have been reported from time to time. Among these the following are considered to be of some consequence.

Bacterial Blight

Bacterial blight is caused by *Bacterium sorghi* (Burrill) on sorghum and broomcorn.

Soft Rot

Caused by *Bacillus amelianski* Serbinoff, this disease was reported from Russia in 1915 as causing soft rot of sorghum stems, with the lower part of the stem darkening, softening, and emitting a butyric odor (Tarr 1962). However, no further report appears to have been made from any other country.

Bacterial Leaf Spot and Top Rot

Caused by *Pseudomonas alboprecipitans* Rosen. this disease was first reported from Arkansas, USA (Tarr 1962). It causes leaf spot on foxtail millet. Artificial inoculation of this bacterium was successful. On sorghum, the leaf spots are greyish green, bordered with red discoloration. Spraying a water suspension of the pathogen resulted in successful infection of

sorghum and sudangrass. Maize is also readily infected. Secondary infection of top rot caused by *Gibberella fujikuroi* var *subglutinans* is sometimes observed.

Bacterial Red Stripe of Sugarcane

It is caused by *Xanthomonas rubrilineans* (Lee et al.) Starr and Burkholder or *X. rubrilineans* (Lee et al.) Dowson. Under artificial inoculation, this pathogen was found to slightly infect sorghum in Hawaii, and was pathogenic on sorghum, broomcorn, sudangrass, johnsongrass, *Sorghum verticilliflorum, S. plumosum*, and *Zea mays* in Australia (Tarr 1962).

Mottled stripe disease of sugarcane caused by *Xanthomonas rubrisubalbicans* (Christopher and Edgerton) Savulescu infects sorghum under artificial inoculation. But the same does not appear on sorghum under natural conditions except in New Zealand, where it was reported to cause leaf stripe of sorghum by Hale and Wilkie (1972).

Bacterial wilt of maize caused by *Xanthomonas stewartii* (E. F. Smith) Dowson, which is very severe on maize, produces similar symptoms on sorghum when inoculated in the laboratory (Ivanoff 1935).

Sugarcane gummosis caused by *X. vasculorum* (Cobb) Dowson, readily infects sorghum, sudangrass, johnsongrass, and broomcorn under artificial inoculation only (Ivanoff 1935). Zummo and Freeman (1974) reported bacterial sunspot, a new disease of sugarcane and sweet sorghums, from Texas, USA. This disease causes abundant ooze on the undersides of the spots. Zummo (1969) also described a bacterial soft rot on sweet sorghum.

International Testing for Varietal Resistance

Since no systemic screening has been done in the past in most of the sorghum-growing countries, except perhaps to some extent in the USA and USSR, it may not be possible to immediately lay out an international screening program. Therefore, it is desirable to screen during the next sowing season in order to identify resistant materials from the local collections tested under artificial epiphytotic conditions. The resistant entries would then be included in future international tests.

Most of the sorghum bacterial diseases of economic importance have been known to be seedborne. Therefore, trials could be undertaken to evaluate effectiveness of seed treatments with bactericides. This could be done with local susceptible cultivars.

Research Priorities

There is little information in the literature on the extent of spread of the bacterial diseases, their economic importance, and the reaction of the local cultivars, etc. It is essential that basic data on the above be obtained by conducting a well-planned survey during the next few years. The identification of pathogens is of prime importance; to achieve it representative samples can be sent to a central organization, such as the Commonwealth Mycological Institute in the UK.

Simultaneously, screening of local cultivars can be undertaken, using standard techniques to create epiphytotic conditions. Commonly adopted methods of creating the condition consist of the following:

1. Seedborne pathogen: This can be achieved by treating the seed with the pathogen after harvest or, alternatively, by inoculating the pathogen on the ear when the seed starts maturing and preserving such seed for the next year's trial.
2. For others: Collect the leaf debris of a specific bacterial disease (especially from highly susceptible cultivars), dry well, and preserve in the laboratory for the next season. Spray-inoculate the debris on the young seedlings. The spray should fall on the soil and splash soil particles on the young leaves, simulating natural conditions. If possible, a more favorable environment for infection can be achieved by sprinkler irrigation, especially at noontime when the temperature will be high. Alternatively, inoculation of seedlings with finely powdered diseased debris could be done.

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Bacterial Diseases Discussion Session

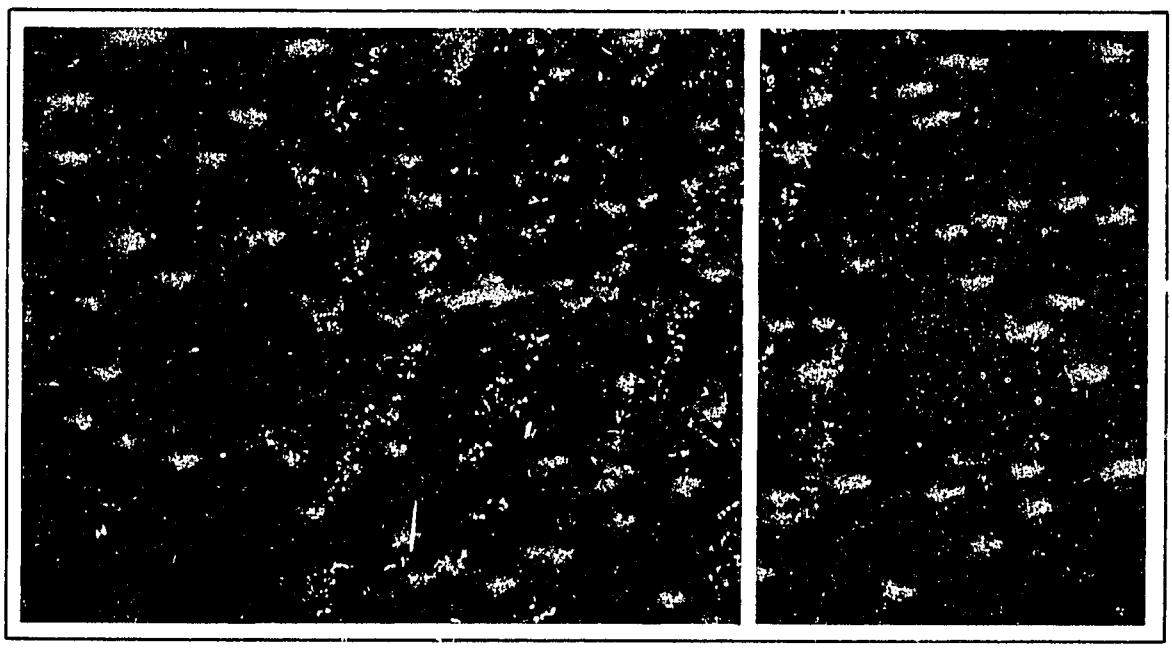
Dange: Should desiccated plant debris be used as a source of bacterial inoculum, or should bacterial cultures be used?

Sundaram: Desiccated plant debris can be stored over a period of months and the bacteria will remain viable. If you make several transfers of bacteria in culture, you may lose virulence.

Dange: The causal agent of bacterial red stripe of sugarcane is *Xanthomonas rubrilineans* (Lee et al) Starr and Burkholder.

C. Selvaraj: We need a detailed illustrated guide to aid in the difficult problems of identification of bacterial leaf diseases.
In Nigeria, bacterial streak (or stripe?) and yellow leaf blotch occur on sorghum every year.

Sorghum
Viral Diseases



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Viruses and Viral Diseases of Sorghum

R. W. Toler*

Tarr (1962) reviewed viral diseases of sorghum and reported that they caused surprisingly little damage. He identified sugarcane mosaic, maize mosaic, red stripe, Fiji disease, stunting disease (sugarcane ratoon stunting), bromegrass mosaic, oat pseudo-rosette, cucumber mosaic (celery mosaic), barley yellow dwarf, rice stripe, lucerne or alfalfa dwarf streak in Nicaragua, freckled yellow-stripe diseases, and maize streak as those diseases of sorghum occurring worldwide. Miller (1966) also published a list of virus diseases that infect sorghum.

The purpose of this paper is to present the current situation of sorghum viruses and viral diseases. Where appropriate, I have selectively extracted information with the intent of stimulating research on virus and virus-like diseases of sorghum.

Reddening or discoloration of the leaves, necrotic lesions, enations, galls, necrosis, and stunting are virus-disease symptoms of sorghum that are quite distinct from mosaic, yellows, and stripe-type diseases produced on sugarcane, maize, or other cereals. Of the 13 viral diseases listed by Tarr (1962), lucerne or alfalfa dwarf (Pierce's disease) (Freitag 1951) is now considered to be caused not by a virus, but by a rickettsia. Sugarcane ratoon stunt, another disease of sorghum reported by Tarr, is no longer considered to be caused by a virus, but has been recently shown to be incited by a bacterium.

The only sorghum disease definitely shown to have a mycoplasma agent as an incitant is yellow sorghum stunt (YSS). This disease was first observed in Mississippi on sweet sorghum (*Sorghum bicolor*) in 1966 by Zummo et al. (1975). The disease is of economic importance, and occurs in the sweet sorghum-growing areas of Mississippi, Alabama, Georgia, Kentucky, Ohio, and Texas. YSS was observed also on grain sorghum, but not on johnsongrass. Electron micrographs of diseased leaf sections revealed mycoplasma-like bodies in sieve elements of the phloem. The nonspiral bodies displayed characteristic mycoplasma ultrastructure, i.e., ribosomes, DNA-like fibers, and bounding unit membrane (Bradfute and Robertson 1974a; 1974b).

The symptoms include stunting, leaves bunched together at the upper part of the plant, and leaves that curl adaxially with puckering and undulating margins. The most conspicuous symptom is the yellow-tinged cream color of the upper leaves. Diseased plants rarely produce seed heads, and those that are produced are barren. The mode of transmission of the disease is unknown.

Zummo reports sweet sorghum cultivars MN1056, Roma, and Ramada to be very susceptible, while Rio, Brandes, and Sart, although exhibiting leaf symptoms, were not severely stunted (Zummo et al. 1975).

We evaluated grain sorghum accessions under natural infection to YSS in Texas; the reactions are shown in Table 1.

Sorghum was listed as susceptible to barley stripe mosaic virus by Smith (1972). After reviewing the original citations, this author found sorghum had been tested for susceptibility to BSMV by three workers. *Sorghum vulgare* Pers. hybrids, Modoc × Sooner (white-seeded) and Modoc × Sooner (brown-seeded) were nonsusceptible when mechanically inoculated by Slykhuis (1952). Two sorghum cultivars, Atlas and Westland, were found to be nonsusceptible by Sill and Hansing (1955). Singh et al. (1960) also found *Sorghum vulgare* var. *sudanense* (Piper) Hitch and *S. vulgare* Pers. cultivar Leota nonsusceptible to BSMV. No definitive report was found demonstrating sorghum as a susceptible host of BSMV.

Sorghum is considered susceptible to several viruses only under experimental transmission. Many of the following viruses have not been observed causing diseases of sorghum in na-

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Table 1. Reaction of *sorghum bicolor* (L.) Moench lines to yellow sorghum stunt (YSS) under natural infection at College Station, Texas.

Designation	Pedigree	Infected plants (%)
RTx2536		5.6
ATx378 × RT×2536		7.5
ATx399 × RT×2536		5.0
(75CS 5740)	(SC-0110-6-3-1-E₂×Tx2536)-8-2-3-2-1	30.0
ATx399 × (75CS-5740)		27.5
(75CS 5761)	(SC-170- × Tx2536)-40-1-1-1-x	25.0
ATx399 × (75CS-5761		37.5
(75CS 5769)	(SC-170- × Tx2536)-40-1-3-3-x	47.5
ATx398 × (75CS-5769)		52.5
(75CS-5822)	(SC0599-6 × SC0110-9-P1-1-1)-4-1-1-1-x	55.0
(75CS-5866)	(SC0599-6 × SC0110-9-P1-1-1)-4-1-3-2-x	60.0
(75CS-5866)	(SC0056-6-4-2-3 × SC0170-6-6-3)-4-4-6-3-6k	47.5
(75CS-5889)	(BTx3297 × SC0170-)-7-3-1-3-1-2	52.5
BTAM428		5.0
BTx414		2.5
Rio	Sweet sorghum	2.5
BTx3197	Combine Kafir-60	0
BTx378	Redlan	0
BTAM618	Smut Resistant Combine Kafir-60	2.5

ture. Although they may not be economically important at present, they are of significance as virus indicators, vector hosts, and in the identification of existing and unknown viruses. They are potentially important pathogens of sorghum.

Cucumber Mosaic Virus (CMV)

Wellman (1934a; 1934b) experimentally transmitted celery mosaic, which is a disease caused by cucumber mosaic virus, to *Sorghum vulgare* Pers. CMV was transmitted to sorghum from celery by the aphid *Aphis gossypii*. Sweet sorghum, milo, and kafir were susceptible, showing chlorotic lesions and stripes. CMV is characterized by Gibbs and Harrison in a summary published in 1970.

Panicum Mosaic Virus (PMV)

In a host-range study employing mechanical inoculation, Niblett and Paulson (1975; Niblett et al. 1977) infected and recovered PMV from *Sorghum bicolor* (L.) Moench. Small faint lesions were observed on the leaves of the inoculated plants. However, the St. Augustine decline strain of panicum mosaic virus (PMV-SAD) does not infect *S. bicolor* (Toler 1969), and cultivars Redlan and New Mexico 31 were not susceptible when mechanically inoculated with PMV-SAD. The virus was characterized by Niblett and Paulson (1975).

Rice Stripe Virus (RSV)

Experimentally, *Sorghum halepense* (L.) Pers. and *S. sudanense* (Piper) Stapf are hosts of RSV. Symptoms when the virus was transmitted by the leaf hopper *Laodelphax striatellus* Fallen include chlorosis and chlorotic striping (Lida 1967; Ling 1967; Yamada et al. 1956). Properties and transmission have been briefly discussed by Smith (1972).

Maize Rough Dwarf Virus

Maize rough dwarf virus was experimentally

transmitted to *Sorghum vulgare* Pers. by Vidauo et al. (1966). The virus is vectored by plant hoppers of the family Delphacidae, with the only known natural vector being *Laodelphax striatellus.* Lovisolo (1957; 1971) summarized the properties of the virus.

Barley Yellow Dwarf Virus (BYDV)

Barley yellow dwarf was first described in California by Oswald and Houston (1953a). Studies on the host range of the virus, reported by Houston (Oswald and Houston 1953b), included sorghum. Viruliferous apple-grain aphids (*Ropalisiphum prunifoliae*) were transferred to sorghum seedlings. After two 3-day feeding periods, the aphids were eliminated by application of an insecticide. The plants were grown for 1 month to allow symptom development evidence of virus multiplication and recovery was tested by feeding nonviruliferous apple-grain aphids for 3 days on the inoculated sorghum plants. After acquisition feeding, the aphids were allowed a transmission feeding period of 3 days on Blackhulles barley, the indicator host. Sudangrass, *Sorghum sudanense* (Piper) Stapf and *S. vulgare* cultivar double dwarf 38 milo were symptomless carriers. Cultivar double dwarf sooner milo was immune. The evidence suggests that if BYDV becomes important on sorghum, a source of immunity is available for use in control of BYDV in sorghum. Properties of BYDV were summarized by Rochow (1970). One possible occurrence on sorghum in Mississippi was reported by Summers and Bowman (1953).

Oat Pseudo-Rosette Virus (OPRV)

Soukhov (1938) and Soukhov and Vouk (1938) described the disease on Holocus sorghum, infected with OPRV when experimentally transmitted by the leaf hopper *Delphacodes striatella* Fall. Soukhov and Vouk (1938) found crystalline virus aggregates in infected cells. However, workers in Japan report two types of virus particles, and the virus is not clearly elucidated at this time. Transmission and properties were discussed by Smith (1972).

Peanut Clump Virus (PCV)

Sorghum arundinaceum (great millet) was found as a symptomless natural host of PCV in Upper Volta in 1976. The bioassay with *Chenopodium amaranticolor* was used to detect presence of the virus in symptomless plants (Dollet et al. 1976). In mechanical inoculations of *S. arundinaceum*, the plants could not be infected by leaf inoculation, but 13% of the plants were infected when roots were inoculated with PCV. Sorghum plants were also infected by planting in infested soil.

Maize Chlorotic Mottle Virus (MCMV)

Castillo and Hebert (1974) first described MCMV on maize in Peru, and reported experimental mechanical transmission and infection of *Sorghum vulgare* Pers. and *S. halepense* (L.) Pers. Symptoms on sorghum and johnsongrass included chlorosis and mottling (Loayza 1976). The virus was reported occurring in the USA by Niblett and coworkers (Niblett and Paulson 1975; Niblett and Claflin 1977; Niblett et al. 1977). The virus is synergistic with maize dwarf mosaic virus and wheat streak mosaic virus in corn. The isolate from Kansas produced local lesions on inoculated leaves of *Sorghum halepense* and *S. bicolor*. Based on serology and host reaction, two strains were described: Peru strain MCMV-P, and the Kansas strain MCMV-K (Niblett et al. 1977).

Sugarcane Fiji Disease Virus (SFDV)

Symptoms of Fiji disease, including elongated galls or enations on the under surface of leaves and on stems, were observed naturally occurring in the Philippines by Kunkel (1924) and later by Ocfemia and Celino (1955). The plant showed, in addition to leaf symptoms, shortened stalks with stiff malformed leaves, headless plants, or plants with poorly formed heads, and plants that turned prematurely brown. Harris (1958) observed a similar disease on sorghum in Nigeria in 1956. Hutchinson et al (1972; 1973) reported definitive transmission to *Sorghum bicolor* (L.) Moench cultivar R 525 Piper, by the leaf hopper *Perkinsiella saccharicida.* A

similar disease, called maize vein enation, has been observed on sorghum in India (Ashlawat and Raychaudhuri 1970), but the vector is reported to be *Cicadulina mbila*, that also vectors maize streak virus. SFDV is vectored by *Perkinsiella* spp and MRDV is transmitted by *Laodelpha striatellus.* Properties of the virus have been summarized by Hutchinson and Francki (1973).

Sugarcane Chlorotic Streak Virus

This disease was first reported in Louisiana. The unconfirmed vector is *Draeculacephala portola.* Soil transmission is reported, and a soil-inhabiting vector suggested (Abbott 1961). Transmission to *Sorghum verticilliflorum* was reported by Eagan (1965). On this host, pale yellow streaks, usually less than 5 cm in length, were observed on the second and third leaves. The streaks were irregular in width and not continuous.

Maize Streak Virus (MSV)

Maize streak (Storey 1929) has been reported in India (Seth et al. 1972b) and in Africa (Bock 1974; Bouriquet 1964). Three strains are known (a) maize (type) strain, (b) sugarcane strain, and (c) the guineagrass or panicum strain. Vectors are confined to the genus *Cicadulina* — i.e., *C. mbila, C. storeyi, C. bipunctella zea, C. latens,* and *C. parazeae* (Bock 1974). Damsteegt (1978; and personal communication 1977) observed four species, five subspecies, and one accession of sorghum to be susceptible to MSV in experimental transmissions with the vector *C. mbila. Sorghum plumosum, S. versicolor, S. verticilliflorum, and S. bicolor*, subspecies *miliaceum, nervosum, miliisorne, cernuum,* and *vulgare* all produced symptoms, including chlorotic streaks and stripes, when infected with MSV. One accession of *S. bicolor*, Tx-412, not only was susceptible but also exhibited intense chlorosis, striping, and stunting. Tx-412 might well serve as a good indicator host. Maize streak virus occurs in India on naturally infected sorghum, i.e., *Sorghum bicolor* cultivars Swarna and CH 5-2 (Raychaudhri et al. 1976). Properties of MSV were summarized by Bock (1974).

Brome Mosaic Virus (BMV)

BMV was described by McKinney et al. (1942). Sorghum, *Sorghum vulgare* Pers. johnsongrass, *Sorghum halepense*, and sudangrass *Sorghum vulgare* var. *sudanense* Hitchc. were found to be susceptible hosts when seedlings were mechanically inoculated (McKinney 1942; McKinney et al. 1942). An extended host-range study by Ford et al. (1970) revealed johnsongrass to be a nonsusceptible host, but not *Sorghum bicolor.* In 1975, sorghum plants with narrow interveinal chlorotic stripes with a mottled appearance were collected in fields of the Agricultural Experiment Station in Burelson county, Texas (Bush et al. 1976). Mechanical transmission studies of the host range of the virus from sorghum paralleled those of BMV, including lethal necrosis of sweetcorn and mottling of johnsongrass and sorghum cultivars Atlas, Rio, and Sart (Gordon personal communication). Electron microscopy, purification, and serology all demonstrated that the virus was brome mosaic. After observing natural occurrence of BMV in sorghum, Bush et al. (1976) evaluated the genetic vulnerability of the crop. The effects of BMV on a set of 12 sorghum differentials were studied in 1977; those susceptible to BMV included Redlan, Tx-414, Tx-7078, Tx-398, NM-31, Tx-412, Sumac, and Tx-3048. Resistant to BMV were Caprock, Combine White Feterida, Combine Kafir 60, and Combine Shallu. The materials classed as resistant did not develop symptoms. Sources of resistance are available in the sorghum genome, should BMV spread or become economically important. Lane (1977) has described and characterized the properties of BMV.

Maize Mosaic Virus (MMV)

Maize mosaic virus has not been reported occurring naturally on sorghum, but in experiments in Venezuela (Herold 1972) *S. bicolor* was infected with MMV when transmitted by *Peregrinus maidis.* Varma et al. (1975) and Bhargava and Shukla (1966) reported *P. maidis* vectored rhabdo virus (MMV) in India, but did not report host range other than *Zea mays*. Briton-Jones (1933) reported a possible natural occurrence in Trinidad. Properties and transmission of MMV were described by Herold (1972).

Maize Stripe Virus (MSV)

A stripe virus disease of sorghum and one called "freckled yellows" were described in India (Cherian and Kylasam 1937); they are reported to be transmitted by *Peregrinus maidis.* A pathogenic agent that causes chlorosis, early flowering, and excessive tillering in sorghum was described by Capoor et al. (1968), who identified it as maize stripe virus, characterized as a polyhedral virus vectored by *P. maidis.* It is considered to be different from maize streak virus, since streak is vectored by species in the genus *Cicadulina* (*Kulkarni* 1973). Chatterji (1971) described a stripe disease of maize occurring in Maharashtra; the disease was transmitted by *P. maidis*, but did not infect sorghum, pearl millet, wheat, or rice. Kulkarni's paper includes description and characterization of the disease.

Maize Chlorotic Dwarf Virus

Maize chlorotic dwarf infects sorghum, producing some slight stunting and tertiary vein chlorosis (stunting has been seen only in the field). Natural infection has been reported. The virus infects sudangrass, sweet sorghum, grain sorghum, and johnsongrass, but is not at present economically limiting on sorghum in the USA.

The leaf hopper vector is *Graminella nigrifrons* Forbes. *Sorghum bicolor* (cultivars Rio, Atlas, Sart, Sargo), *S. halepense*, and *S. vulgare* var *sudanense* were infected under experimental transmission by virulifirous *G. nigrifrons*, and the virus was recovered by leaf hopper transmission to indicator maize and by rate-zonal purification from infected tissue. Johnsongrass, *S. halepense*, is the only host that consistently occur in the same regions as the leaf nopper vector, and is considered to be the principal overwintering host for MCDV (Nault et al. 1976).

Sugarcane Mosaic Virus (SCMV)

SCMV was first described, according to Tarr (1962), by Brandes. It was identified on *Sorghum vulgare* Pers. by Brandes and Klapaak (1923) (see also Bockholt and Toler 1968). Kunkel (1924) reported tunisgrass, *Sorghum virgatum* (Hack.) Stapf, and johnsongrass, *S. halepense* (L.) Pers., as host plants of this virus. The only other report of SCMV infection in johnsongrass was by Abbot and Tippett (1966), who wrote that strains A, B, and D did not infect this species, but strain H produced a low percentage of infection. Host range of SCMV, including sorghums, is discussed by Matz (1938) and by Summers et al. (1948).

Maize dwarf mosaic virus (MDMV) is sufficiently serologically related to SCMV to be considered a strain of SCMV (Shepherd 1965). Some workers refer to this virus as the johnsongrass strain (SCMV-J or SCMV-JG) of the sugarcane mosaic virus (Teakle and Grylls 1973). In my discussion, it will be treated as a separate disease. It is important wherever sorghum is cultivated in the proximity of sugarcane.

Adsur (1950) reported using seedlings of the sorghum cultivar Hegari to determine some physical properties of SCMV in Puerto Rico. Dean and Coleman (1959) studied cultivar reaction of sorghum to SCMV strains B and D. Atlas, Sumac, Tracy, and Wiley exhibited a wide range of reaction and symptom types, and these workers concluded that sorghums were not ideal differential hosts for the SCMV strains studied. Abbot and Tippett (1966) studied the effects of strains A, B, and D on 18 sorghum cultivars; they failed to develop sorghum differentials, but reported resistance to SCMV in sorghum. Dean (1969; 1970) observed in 1964 that sorghum cultivars MN-1954, Sart, Rio, and Atlas produced differential reactions to strains A, B, D, E, and H. He also reported that sorghum cultivar Atlas was a local lesion host for SCMV strain E. The following year he found that Combine Kafir 60 was also a local lesion host for that particular strain. Zummo, writing about the new virulent strain L in 1974, reported that cultivar Sart is susceptible to the new strain. Soil transmission of SCMV-H to sorghum sudangrass hybrids Beef builder T and Lindsay 77 (*Sorghum vulgare* × *Sorghum sudanense*) was reported by Bond and Pirone (1970).

Resistance in sorghum to SCMV was evaluated by Dean and Coleman (1959), using 72 sweet sorghum accessions inoculated with SCMV strain B. Reactions of the hosts were classified as susceptible mottling and/or necrosis (red or tan) and resistant (delayed symptom expression and mild mottling); of

these 62 were classed susceptible and eight moderately resistant. Two were classified as very resistant on the grounds that mild mottling symptoms disappeared in the inoculated seedlings. Mottling symptoms in plants infected with SCMV have been observed to disappear or become masked as sorghum plants approach maturity (Brandes 1923; Brandes and Klapaak 1923; Dean and Coleman 1959; Dean 1969, 1970). Frazil et al. (1970) studied ten sorghum genotypes inoculated in the field with SCMV strains A, B, D, H, and I. Reaction varied from susceptible to resistant, and virulence of the strains on the ten genotypes were in decreasing order, B,H,A,D, and I. Susceptibility and resistance to the five strains included very susceptible cultivar New Mexico 31, Atlas, and Redlan. Cultivars of intermediate susceptibility to the strains were Tx-412, Rio, Tracy, Shallu, and Sumac; the resistant cultivars were Martin and Wiley. On a disease-severity index ranging from 1 to 9, cultivar Wiley rated 1.03 for strain SCMV-B; 1.05 for H; 1.04 for D; 1.02 for A; and 1.16 for strain I. For cultivar Martin, the ratings were 1.01 for strain SCMV-B; 1.45 for H; 1.15 for A; 1.45 for D; and 1.14 for strain I.

Infection of sorghum with SCMV strains delays flowering. Time to first flowering in inoculated plants, as compared with controls, was longer in the more susceptible accessions (12 days in cultivar Rio inoculated with SCMV-D), and shorter in the more resistant accessions (1 day in Martin). Plant height of the ten accessions was in general reduced because of the infections, but this characteristic varied with accession and with strain. Additional effects of accession × strain interactions included reduction of head length, threshing percentage, weight of seed, test weight, seed size, grain yield, and forage yield.

Transmission of SCMV strains to sorghum has been accomplished principally by mechanical means and, under natural conditions, by the aphids that vector SCMV to sugarcane. With one exception, aphid transmission of SCMV strains specific to sorghum has not been studied. The ambrosia aphid *Dactynotus ambrosiae*, when acquisition-fed in numbers of 1 to 15 on itchgrass, *Rottboellia exaltata*, multi-inoculated with SCMV-I and MDMV-A, acquired both strains. When transmission-feeding occurred on *Sorghum bicolor* cultivar Rio, some individual plants were infected with SCMV-I alone, others with MDMV-A alone, and some were infected with both (Koike 1970; Koike and Tippett 1971).

A number of diseases of millet are caused by mechanically transmissible and aphid-vectored viruses with properties similar to those of sugarcane mosaic virus. Sorghums can be naturally and artificially infected and listed as hosts of these viruses. Raychaudhuri et al. (1966; 1976) consider these to be related to SCMV — perhaps even strains of SCMV, as they are flexuous rods with many common hosts and vectors. They include ragi (*Eleusine coracana*) mosaic, described from New Delhi by Keshavmurthy and Yaraguntaiah (1969) and Batra et al. (1970), and from Mysore by Subbayya and Raychaudhuri (1970). Eleusine mosaic in the Deccan was described by Rao et al. (1965; 1967). Bajra (*Pennisetum typhoides* [Burm.] Stapf and Hubb.) mosaic was reported from Delhi (Seth 1967; Seth et al. 1972a, 1972b, 1972c; Seth and Singh 1975) and from Uttar Pradesh (Chaudhary and Singh 1976). Another virus disease with a sorghum host that does not infect sugarcane is called maize mosaic (Bhargava and Shukla 1966; Bhargava et al. 1971; 1972; Paliwali and Raychaudhuri 1965; Paliwali et al. 1968; Raychaudhuri et al. 1966; 1976), but is not to be confused with maize mosaic virus, which has a rhabdo particle. This viral disease is economically important on maize, sorghum, and millets in India, and is suspected to overwinter in grass and cereal stubble. It is related to, and is possibly a strain of, sugarcane mosaic virus, and somewhat resembles maize dwarf mosaic virus (SCMV-J). Narayan and Ram (1976) discussed sorghum red stripe disease, and listed SCMV as its causal virus in India.

Many strains of SCMV are described in the literature (Abbott 1961; Abott and Tippett 1966; Benda 1970; Bhargava et al. 1971; 1972; Chona and Rafay 1950; Edgerton and Taggert 1924; Khurana and Singh 1972; Koike and Tippett 1971; Litzenberger and Stevenson 1957; Matz 1933; 1938; Narayan and Ram 1976; Rishi and Ram 1967; Summers et al. 1948; Tippett and Abbott 1968; Vasudeva 1955; Wiehe 1948; 1953; Williams et al. 1976; and Zummo and Gordon 1971), but are not conclusively differentiated on sorghum.

Bhargava et al. described SCMV-A, B, F, and D from India (1971; 1972) and Khurana and Singh (1962; 1972) listed SCMV-E and C. New strains

in the USA include SCMV-I, K, L, and M (Koike and Gillaspie 1975; Tippett and Koike 1970; Zummo and Stokes 1973; Zummo 1974).

Sorghum red stripe was probably the first virus disease of economic importance naturally occurring on sorghum. It was first reported from Italy and a color photograph of the disease appears in Tarr (1962). It is considered by Lovisolo (1957) and by Grancini and Mariani (1974) to be a strain of SCMV.

Maize dwarf mosaic virus is serologically related to SCMV and is known by some workers as SCMV-J, or the johnsongrass strain of SCMV. In Australia, the sugarcane mosaic virus that infects johnsongrass is designated as SVMV-JG. Grancini and Mariani (1974) have summarized the work with sorghum. The disease has been reported from France, Bulgaria, Romania, Australia, South Africa, Yugoslavia, India, USA, Colombia, Venezuela, Peru, Thailand, Mexico, and the Philippines.

Globally, this virus has received more attention than any other sorghum disease-causing virus. MDMV was described in three papers by Dale (1964; 1965; 1966) and shown by Shepherd to be serologically related to SCMV. He suggested that it should be labeled the johnsongrass strain of SCMV; hence the designation SCMV-J (Shepherd 1965; Shepherd and Holdeman 1965). Variations of MDMV were revealed by Hill et al. (1973), who characterized and purified the non-johnsongrass isolate, the MDMV strain B. This is confusing, since technically if johnsongrass is not a host, it should not carry a MDMV designation or SCMV-J or SCMV-JG classification. The virus is seedborne in low percentage on maize, according to Shepherd. Soil transmission of the causative agent has been reported by Bond and Pirone (1970; 1971). Some 13 species of aphids are known to be vectors (Daniels and Toler 1969; Ingram and Summers 1936; Messieha 1967). Symptoms include stunting (Bockholt et al. 1968) mosaic reddening and striping of leaves (Bockholt and Toler 1968; Bockholt et al. 1968), delayed flowering, yellowing, mottling, small seed, and reduction in quality of seed, reduction in grain yield, and death of various genotypes. (Amador et al. 1969; Bocknolt and Toler 1968; Bockholt et al. 1968; Bockholt et al. 1969; Canerday and Guadauskas 1970; Ciferri 1955; Dale 1964; 1965; Damsteegt 1978; Daniels and Toler 1969; Ford et al. 1967; Gillaspie 1973; Gillaspie and Koike 1973; Haddan 1928; Hopkins 1958; Lastra 1976; Penrose 1974; Persley et al. 1972; Perseley et al. 1976; Persley et al. 1977; Snazella et al. 1971; Snow 1970; Teakle et al. 1970; Teakle and Grylls 1973; Toler et al. 1967; Toler 1968; Toler and Hobbs 1968).

Principal control is through host resistance (Table 2 to 7) (Conde et al. 1976). Mechanisms of resistance include tolerance (Bockholt and

Table 2. Maize Dwarf Mosaic Virus (MDMV) Strain A effect on sorghum indicators in Texas.

Sorghum entry	Symptoms	Disease Index 1–5	Reduction in yield (%)	Disease Reaction Rating
Tx-414	Mosaic	1.0–2.9	0.0	Tolerant
Tx-412	Mosaic	2.0–3.0	31.4	Susceptible
Tx-3042 (Redbine)	Mosaic	1.0	–	Tolerant
Tx-398 (Martin)	Mosaic	1.0–2.1	8.7	Tolerant
Sumac 6550	Stunting reddening	5.0	–	Tolerant
Tx-3197 (Combine Kafir-60)	Stunting reddening	4.5–5.0	47.6	Susceptible
New Mexico-31	Stunting reddening	5.0	100.0	Highly susceptible
Tx-3048 (Redbine)	Stunting necrosis	5.0	–	Susceptible
Tx-378 (Redlan)	Stunting necrosis	4.5–5.0	33.7	Highly susceptible
Tx-7000 (Caprock)	Stunting necrosis	4.8–5.0	–	Highly susceptible

Table 3. Tolerance to Maize Dwarf Mosaic Virus (MDMV) in grain sorghum lines and hybrids.

Entry		Days to flower	Yield (lb/acre)	Reduction in yield (%)
RS-621	I[a]	65.0	3600	
	H[b]	65.3	3500	0.0
Tx-414	I	68.3	2766	
	H	66.7	2766	0
RS-625	I	64.0	3866	
	H	64.0	4000	3.1
Martin	I	68.7	2800	
(Tx-398)	H	67.3	3066	8.7

a. Infected *b.* Healthy
Source: Toler and Bockholt 1967.

Table 4. Field resistance in grain sorghum to Maize Dwarf Mosaic Virus.

Entry	Disease Rating Index (1–5)	
	Mechanical Infection	Natural Infection
IS-2549C (SC 228)	2	1
IS-2816C (SC120)	2	1
IS-12612C (SC112)	2	1
IS-12666C (SC175)	2	1
Variety R10	3	1
TAM-2566	2.5	1

Source: Rosenow, Johnson, and Toler 1975).

Table 5. Immunity to maize dwarf mosaic virus in grain sorghum accessions

Source	Entry	Infection (%)
Teakle and Pritchard 1971	Krish QL-1	0
Perseley et al. 1972	Q7539(IS7596)-3 to 7 by exII-97-H74	0
Toler and Miller 1976	SC-0120-14-E 73-CS-31, 32	0

Table 6. Resistance to maize dwarf mosaic virus (low levels of infection) in grain sorghum accessions

Source	Entry	Infection (%)
Smith and Toler 1976	NM-31(M3)76-380-13	7.4
	NM-31(M3)76-369-2	6.0
Perseley et al. 1976	Q-7539(IS7596)-3Aex 80G w/h-74	5.0
Toler and Miller 1976	SC-0534(IS7596)PR	10.0
Toler and Miller 1976	SC-0097-14 73-CS-271, 72	5.0

Table 7. Reaction of grain sorghum virus indicators to maize dwarf mosaic virus strains A and B (Ford 1976).

Entry		Infection	
		MDMV-A (%)	MDMV-B (%)
NM-31	Weskan × Redbine	57.0	0.0
Tx-412	(Tx 09×Tx 403)	43.0	63.0
BTx-3197	Comb. kafir 60	23.0	0.0
SA-394	Combine shallu	37.0	21.0
PI-35038	Sumac	25.0	10.0
BTx-3048	Redbine Sel.	30.0	0.0
BTx-378	Redlan	24.0	0.0
SA-7000	Caprock	24.0	0.0
Tx-414	7078 Der.	37.0	0.0
SA-7078	7078	47.0	0.0
BTx-398	Martin	46.0	0.0
Tx309	Comb. Wh. Fet.	5.0	5.0

Toler 1968; Bockholt et al. 1968), immunity, and resistance to infection. Sorghum differentials for identification of MDSV and other SCMV strains have been suggested by Bockholt et al. (1968, 1969) and by Grancini and Mariani (1974). With modification and additions, an international virus disease nursery is proposed with a common set of differentials available worldwide. This would constitute an important

aid to uniform identification of viruses causing diseases of sorghum.

Summary

Viruses are important etiological agents of sorghum in the world. The first viral disease reported on sorghum was sugarcane mosaic, caused by sugarcane mosaic virus (SCMV) described by Brandes in 1923. Currently some 13 strains of SCMV are known to infect sorghum. The most important viral disease in regard to distribution and economic importance today is maize dwarf mosaic virus (MDMV), which is serologically related to SCMV and often called the J, JG_1, or johnsongrass strain of SCMV. This virus has been observed in Europe, India, and Australia as well as in North, Central, and South America. Fifteen other viruses have been identified as causing diseases of sorghum. These viruses vary in their economic effects in different countries. Viral disease control is primarily through host resistance. An International Uniform Virus Nursery is proposed with specific indicator genotypes to aid in virus and virus strain identification.

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The Cause and Control of Sorghum Viral Diseases in Australia

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Sorghums Present in Australia and Their Natural Infection by Viruses

The genus *Sorghum* is represented in tropical and warm-temperate Australia by species occurring as native plants, introduced weeds, or grain and forage crop plants. Native sorghums occur in tropical Australia where they are of minor importance as forage for cattle. They have not been found infected by viruses in the field, but three species — *S. laxiflorum, S. macrospermum*, and *S. stipoideum* — became naturally infected by the Australian johnsongrass strain of sugarcane mosaic virus (SCMV-JG) when grown in a glasshouse with aphid-infested, virus-infected sorghums (Teakle and Grylls 1973). Thus at least these species are potential field hosts of the virus.

Weed sorghums include *S. halepense* (johnsongrass) and *S. verticilliflorum* (wild sorghum). *S. halepense* has spread rapidly in Australia since 1970, and is regarded as a major weed (Monaghan 1978). Both species are important reservoirs of SCMV-JG, while *S. verticilliflorum* is also a major reservoir of the Australian maize stripe virus.

Commercial sorghums include *S. bicolor* (grain and forage sorghums), *S. almum* and *S. sudanense* (sudangrass). Figures for the 1976–1977 production year show that grain sorghum occupied 86% of the 618 000 ha sown to sorghums, and that the grain yield was 956 000 tonnes, worth about $A80 million. The main growing areas are southern and central Queensland and northern New South Wales, where commercial sorghums regularly become infected by SCMV-JG and occasionally by the Australian maize stripe virus.

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Importance of Sorghum Viral Diseases in Australia

The Australian Maize Stripe Agent

Diseases caused by this suspected virus were first recorded in maize by F. W. Blackford in 1943 and in *Sorghum verticilliflorum, S. bicolor*, and *S. sudanense* by R. S. Greber in 1960–62 (Simmonds 1966). These workers showed that the vector was the plant hopper, *Peregrinus maidis*, and that the acquisition and inoculation threshold periods were approximately 1 hr each. The nature of the presumed virus and its relationship with viruses causing similar diseases elsewhere have not been established (R. S. Greber, personal communication 1978). The agent causes broad yellow stripes on the foliage of maize and sorghums, and the yield of infected plants is greatly reduced. Infection incidence may be high in sweet corn or maize grown in coastal districts of Queensland, where the plant hopper vector and the major reservoir host, *S. verticilliflorum*, are common. It is rare in the major inland agricultural areas, where the resistant *S. halepense* is the prevalent weed sorghum and the vector is uncommon. Because this agent is unimportant in commercial sorghums in Australia, it will not be discussed further.

Sugarcane Mosaic Virus (SCMV)

Although four strains of SCMV occur in Australia (Teakle and Grylls 1973), only the Australian johnsongrass strain (SCMV-JG) has been found infecting sorghums naturally. This strain is widespread in central and southern Queensland and northern New South Wales, where it causes considerable losses in susceptible cultivars of maize and sorghum (Grogan and Teakle 1969; Teakle et al. 1970; Penrose 1974; Persley et al. 1976; Henzell et al. 1979). Its

occurrence in sweet corn in Victoria and in sorghum in the Northern Territory is confined to single records and there is as yet no evidence that it is permanently established in these states.

SCMV-JG has probably been present in Australia for more than 30 years. Symptoms now attributable to SCMV-JG were first recorded in maize by T. McKnight in 1948, in *S. almum* by A. J. Pritchard in 1960, and in *S. bicolor* and *S. halepense* by the author in 1966 (Simmonds 1966). Its identity as a strain different from the Australian sugarcane and American johnsongrass (MDMV-A) strains was established by Taylor and Pares (1968).

Sorghum Diseases Caused by SCMV/JG

Symptoms

Three distinct diseases caused by the SCMV-JG strain are recognized, and are called mosaic, red stripe, and red leaf. The genotype of the host determines the mosaic and red stripe reactions (Teakle et al. 1970), whereas genotype and temperature control the red leaf reaction (Persley et al. 1977). Plants containing Krish resistance are immune to systemic infection (Conde et al. 1976). Table 1 gives the genotypes of susceptible and resistant sorghums and an estimate of the yield losses which can occur. The K and N genes are either at the same locus or very closely linked (R. G. Henzell and D. S. Teakle, unpublished), whereas the *rlf* gene is independently inherited (Persley et al. 1977).

Crop Losses and Capacity for Epidemic Development

Forage sorghums often have 100% incidence of infection by the end of the first growing season. With grain sorghums, incidence of infection at maturity may be 50 to 100%. Incidence is usually higher in mosaic-reacting sorghums than in red stripe-reacting sorghums, but this is partially offset by the greater tolerance of many mosaic-reacting sorghums (Teakle and Moore 1972).

Epidemics occur when there is an abundant source of SCMV-JG in nearby weed or cultivated sorghums and, presumably, the movement of large numbers of aphids, such as *Rhopalosiphum maidis, Aphis craccivora*, and *A. gossypii* (Teakle and Grylls 1973). Cool weather promotes more severe symptom development, in particular the red leaf reaction (Persley et al. 1977).

Screening for Resistance to Infection

This can be done using any of three environments or three inoculation methods, each of which has its advantages and disadvantages (Table 2). In practice Queensland workers have mainly used natural (aphid) or airblast inoculation when working in the field, and leaf-rub inoculation for glasshouse or growth-cabinet work. Any environment or inoculation method is suitable when testing for the "strong" monogenic resistance of Krish sorghum, but natural (aphid) inoculation in the field has given best results with the "weak" multigenic field resistance of Q-7539.

Table 1. Symptoms, genotype, and probable yield loss of sorghums affected by SCMV-JG.

Reaction	Foliage symptoms	Genotype	Maximum yield loss[a] (%)
Mosaic	chlorotic streaks	*kk nn Rlf-*	0–30
Red leaf	necrosis (after cold weather)	*kk nn rlf rlf*	10–90
Red stripe	necrosis	*kk N- —*	60
Resistant (Krish gene)	nil	*K*	0

a. Assuming 100% infection in the seedling stage (Henzell et al. 1979)

Table 2. Evaluation of environments and inoculation methods used for screening sorghums for resistance to SCMV-JG.

	Advantages	Disadvantages
ENVIRONMENT		
Field	natural; normal plant growth	affected by weather, pests, other diseases; field equipment required
Glasshouse	some control of weather, pests, other diseases	expensive; labor intensive
Growth cabinet	good control of weather, pests, other diseases; suitable for red leaf evaluation	very expensive; labor intensive
INOCULATION METHOD		
Aphids	natural; permits detection of low levels of resistance	inefficient; slow; virus-source plants required
Air-blast	efficient; fast	unnatural; equipment expensive; may not detect low levels of resistance
Leaf-rubbing	efficient; inexpensive	unnatural; slow with large numbers of plants; may not detect low levels of resistance

Susceptibility to systemic infection is determined on the basis of mosaic or necrotic symptoms developing in seedlings. This can be confirmed by inoculation to sweet corn or sorghum indicator seedlings, or by negative-stain electron microscopy. Plants are more susceptible when young.

Screening for Tolerance

Plants that produce only mosaic symptoms are tolerant in comparison with plants that produce either red-stripe or red-leaf symptoms. Screening for the red-stripe disease can be done at any temperature, but for the red-leaf disease a low temperature (e.g., 15°C) must be provided. In countries with warm climates, this low temperature must be provided by means of controlled-environment plant-growth facilities.

Lines exhibiting only the mosaic reaction differ in degree of tolerance. At present, yield is the only reliable basis on which screening is done, although symptom severity is a useful guide.

Sources of Tolerance or Resistance to Infection

Until recently almost all sorghums grown in Australia were susceptible to infection by SCMV-JG (Teakle et al. 1970), although some commonly grown varieties were tolerant and experienced little yield loss (Persley et al. 1976; Henzell et al. 1979). The use of tolerance as the sole means of avoiding losses caused by SCMV-JG, however, has not been acceptable, because nontolerant sorghums often outyielded tolerant sorghums when virus incidence was low. Thus a search was made for resistance.

Krish Resistance

The first source of resistance was Krish sorghum (Pritchard 1964), a tall, grassy photoperiod-sensitive cultivar derived from the cross *Sorghum* sp × *S. bicolor* group Roxburghii (*S. halepense* 2n = 20 × *S. roxburghii*) (Krishnaswamy et al. 1956). Krish sorghum was

shown to segregate for virus susceptibility and resistance (Teakle and Pritchard, 1971), and it is probable that only one of the original parents, the Indian *Sorghum* sp (=*S. halepense* 2n = 20 = Q12117), was resistant (Conde et al. 1976). Krish resistance depends on a single gene, K, with resistance dominant over susceptibility. It is effective against all four Australian strains of SCMV. It has now been transferred to 16 breeding lines which have been field tested sufficiently to show that they are not agronomically inferior to the recurrent parents (Henzell et al. 1978, Table 3). In addition, virus-resistant A and B lines of three other sorghums — Tx-378, KS-4, and Tx-618 — should be released in 1979 (Henzell et al. 1978).

Q-7539 Resistance

A second source of resistance to all four strains of SCMV in Australia is Q-7539, a tall, photoperiod-sensitive grain sorghum of Nigerian origin (Persley et al. 1972). In contrast to Krish resistance, which is controlled by a single gene, resistance in Q-7539 is probably multigenic and quantitative. Leaf-rub or air-brush inoculation produces a high proportion of infected plants and is considered too severe a test for screening. However, screening by growing sorghums near air-brush inoculated sweet corn in fields infested by aphids has revealed lines with a low incidence of infection compared with the recurrent parents, KS-4 and TAM-422. A Texas 610 with Q-7539 resistance in both parents will be available for release in 1979 (Henzell personal communication 1978).

Other Sources of Resistance

Of approximately 1000 other lines tested in the world sorghum collection, none were resistant to infection by SCMV-JG (Persley, personal communication 1978). Apparently resistance to this strain is uncommon. However, since there

Table 3. Pedigree of released SCMV-resistant lines

Line	Year of release	Pedigree[a]
QL-1	1972	(Tx-414 × Krish 13)R-7078) TAM-422
QL-2	1972	TAM-422 (Tx-414 × Krish 13) R-7078)
QL-3[b]	1972	KS-4_3 × Krish 21
QL-4	1972	TAM-618 (KS-42 × Krish 21)
QL-6	1977	KS-19_3 × Krish 13 ^{70}/137-3-1-2-11bk-10
QL-7	1977	KS-19_3 × Krish 13 ^{70}/137-3-1-2-11bk-12
QL-8	1977	KS-19_4 × Krish 13 ^{70}/137-102-2-3-1-10
QL-9	1977	KS-19_4 × Krish 13 ^{70}/137-102-2-3-1-26
QL-10	1977	KS-19_4 × Krish 13 ^{70}/137-102-2-3-1-39
QL-11	1977	KS-19_4 × Krish 13 ^{70}/137-102-2-3-1-41
QL-12	1977	KS-19_5 × Krish 13 ^{70}/137-102-2-3-8
QL-13	1977	(Tx-414 × Krish 13)R7078 ^{70}/136-6-7-13bk
QL-14	1977	(Tx-414 × Krish 13)R7078) TAM422 ^{70}/136-6-7A-17-3bk
QL-15	1977	TAM-422_2 (Tx-414 × Krish 13)R7078) ^{70}/136-102bk-3-2-bk
QL-16	1977	TAM-22_2 (Tx-414 × Krish 13)R7078 ^{70}/136-102bk-27-1
QL-17	1977	TAM22_2 (Tx-414 × Krish 13)R7078) ^{70}/136-102bk-27-1-3

a. Tx-414 = R7078$_2$ × Tx-09
R7-78 is the male parent of Tx-610
TAM-422 = R7078$_4$ × Tx-09 and is the male of Tx-610SR
KS-4 = B-3197$_{10}$ × B-398 and is the female parent of Q5161. B-3197 is the female parent of Tx-610SR and Tx-626.
TAM-618 is a head-smut resistant Combine Kafir
KS-19 is the male parent of Q-5161.
QL-6 - QL-17 are homozygous for Krish resistance, whereas QL-1 - QL-4 contain some susceptible plants.
b. QL-3 has been immune to sorghum downy mildew in ICRISAT trials (see report on the International Sorghum Downy Mildew Nursery)

are approximately 13 000 lines in the world sorghum collection so far untested, other resistant sorghums may occur.

Lines with resistance in other countries have often been susceptible in Australia. For instance, of nine lines reported to have resistance in Texas (Toler and Huebner 1978), only one, SC-0097, had a low incidence of infection (3%) in Queensland, and six lines were approximately as susceptible as KS-19 (67%) (Persley, personal communication 1978).

Stability of the Resistance and Variability of the Pathogen

Krish and Q-7539 resistances have been highly stable in Queensland. A possible exception is the discovery of a necrotic red striations disease in a bc_2F_2 population of KS4$_3$×Krish and B51$_3$×Krish in 1972. Characteristics of this were the higher than normal number of plants in the susceptible class and the occurrence of both necrotic and mosaic classes when only a mosaic class was expected. This necrotic red striations disease has not been seen in recent years.

In addition to SCMV-JG, three other strains have been reported, namely the sugarcane, sabi grass, and Queensland blue couch grass strains (Teakle and Grylls 1973). There is no evidence that these infect sorghums in the field, although they can infect sorghum in the glasshouse. Krish and Q-7539 resistances are effective against all four strains of SCMV when tested in the glasshouse.

Suggested International Screening for SCMV Tolerance or Resistance

International screening differs from national screening in that more virus strains and host genotypes are tested and a wider range of biological and physical environments is encountered. The number of locations required for adequate international screening is unknown, because our knowledge of SCMV-caused diseases of sorghum is incomplete. However, preliminary evidence based on the mosaic and necrotic reactions of "differential" sorghums indicates that eleven "strains" of SCMV in seven different countries can be placed in either of two groups (unpublished). Thus, at least two sites, reflecting the Australian and American (or European) johnsongrass strains of SCMV, should be used for screening.

So far, lines identified as resistant to SCMV infection in Australia (Krish, Q-7539, SC-0097) have also been resistant in Texas, but the reverse has not been found (Persley, personal communication 1978). Possibly the Australian johnsongrass strain (SCMV-JG) is more infectious than the American strain (MDMV-A=SCMV-J) to many sorghum lines.

Other Control Measures

There are several feasible approaches. Aphicides could be applied to destroy the vectors. However, this measure is only slightly effective with stylet-borne viruses, and is undesirable because of its cost and polluting effect. Insect repellents, such as aluminum foil, would probably be more effective but too costly. Cultural methods would include (a) altering the time of planting to avoid the worst infection period, (b) eradication of alternative hosts such as johnsongrass, and (c) planting at a considerable distance from known infected weeds or crops. All the above methods are unlikely to be as effective, reliable, convenient, or cheap as the incorporation of genetic resistance by plant-breeding techniques.

Areas Where Research Priority Is Needed

Krish Resistance

Several questions need to be answered:

1. Is Krish resistance effective against all present strains of SCMV in all countries? Preliminary tests in Australia, France, Hawaii, India, Italy, the Philippines, and USA (Texas) have shown that either Krish or a derivative is resistant to one or more strains of SCMV. Additional tests in these and other countries are needed.
2. Will new strains of SCMV arise and overcome Krish resistance? The development of new strains of SCMV which infect previously resistant varieties of sugarcane and maize has been observed in the USA. It is possible that Krish resistance of sorghum

will suffer the same fate, and a close watch should be kept for this.

3. Can Krish resistance be incorporated into other sorghum breeding lines? The K gene for resistance is now present in a number of diploid breeding lines, and its incorporation into others should present no great difficulty. Obtaining homozygous resistant tetraploid sorghums, such as *S. almum*, would be more difficult. The K gene is apparently present in Silk, a tetraploid sorghum.
4. Does incorporation of Krish resistance affect other properties? In Australia the Krish-resistant parental lines QL-6 to QL-17 are generally superior to their recurrent parents in open headedness (conferring resistance to head caterpillar and *Heliothis*), and are resistant to rust (*Puccinia purpurea*), leaf blight (*Drechslera turcica*), and grey leaf spot (*Cercospora sorghi*). Further, hybrids produced using these lines are higher yielding by 10 to 20% (Henzell et al. 1978). In the USA, QL-11 was resistant to sorghum downy mildew Frederiksen, personal communication to Henzell). However, further testing in Australia and other countries under a wide range of conditions is required to indicate strong and weak traits.

Other Sources of Resistance

The same questions concerning Krish resistance need to be answered for Q-7539 resistance and for sources of resistance recently identified in other countries.

Summary

Viral diseases of sorghums in Australia are caused by the Australian maize stripe virus and the johnsongrass strain of sugarcane mosaic virus (SCMV-JG). The latter virus causes three diseases — mosaic, red leaf and red stripe — which cause 0 to 90% loss in yield of plants inoculated in the seedling stage. Disease reaction is determined genetically by the host, and, in the case of red leaf disease, by low temperatures.

Two sources of resistance to SCMV infection have been studied in Australia, i.e., Krish resistance, which is controlled by a single gene, and Q-7539 resistance, which is multigenic. Krish resistance is particularly promising in Australia and other countries, and has generally proven to be stable in Australia.

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Maize Dwarf Mosaic Virus A and Maize Dwarf Mosaic Virus B as Causal Agents of a Varied Symptomatology in Different Cultivars of *Sorghum bicolor* L. (Moench)

E. E. Teyssandier, Delia Docampo, Graciela Laguna, and Laura Giorda*

The maize dwarf mosaic virus (MDMV) induces a variety of symptoms on infected sorghums in Argentina, and each sorghum cultivar may react to the disease in a different way. This variety of symptoms was observed in areas of Pergamino (Buenos Aires province) with remarkably different climatic conditions.

The range of symptoms observed in the two areas were similar, although the sorghum materials examined were different. The situation attracted the attention of technicians of the Cargill Experiment Field (Pergamino), Manfredi Experiment Station (INTA), and the Phytopathology Department of the Agricultural Sciences Institute of the Cordoba National University, who began a joint research project to investigate whether the observed symptoms were caused by a virus or a combination of viruses.

The work included artificial inoculation of numerous breeding lines and precommercial cultivars at Pergamino Experiment Field, Manfredi Experiment Station and the greenhouse of the Phytopathology Department in Cordoba, using controlled sources of inoculum. It was possible to reproduce the range of symptoms, as well as to demonstrate the presence of MDMV in the diseased plants (based on physical properties, the size of the particles, the reaction on a set of differential hosts).

Through serological tests (Van Slogteren technique) it was possible to identify the presence of MDMV strain A and MDMV strain B as causal agents of this range of symptoms.

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Leaf Symptoms

1. Slight mosaic with discontinuous longitudinal stripes. Pale and dark green blotches alternating on young and old leaves.
2. Definite mosaic followed by the formation of chlorotic rings, superimposed, which become coalescent to the point of turning large areas on young and old leaves chlorotic.
3. Small chlorotic rings, which later become necrotic and tend to become reddish arabesque lines on mature leaves. On young leaves (tillers), mosaic is present.
4. Elongated, oblong tan spots ranging in length from 3 to 5 cm and 0.5 to 2 cm in width, with reddish edges. These tend to become coalescent on mature leaves. On young leaves (tillers) mosaic is present.
5. Concentric necrotic tan rings, which remain isolated or generally end in large necrotic areas. As necrotic areas become generalized, all leaf areas are covered.

General Symptoms

1. Affected plants tend to be dwarfed because of shortening of the internodes.
2. Reduction in size of the panicle.
3. Sterility caused by total absence of panicles.
4. Reddish panicles with shrivelled kernels.
5. Death of plants when infected early or in very susceptible materials.

Sorghum Viral Disease Discussion Session

Frederiksen:
Why are viral diseases of sorghum more important in the Americas than in Asia or Africa?

Teakle:
The sugarcane mosaic group of viruses is more important in cooler regions, where they induce red-leaf symptoms. Monoculture of sorghum on large areas in the Americas and Australia is certainly favorable to epidemics. Some of the genotypes used for making hybrids in the Americas develop necrotic reactions. The local varieties grown in small patches in Asia and Africa may have been selected over time for tolerance to the viruses.

Toler:
The type and population of insect vector is important for virus transmission and it varies among continents. For example, in India there are several viruses on millets that can infect sorghum that are leafhopper vectored.

Teyssandier:
In Argentina, lines show very severe symptoms when planted early. This may be so because low temperatures induce severe symptoms (red leaf and necrosis), but also because conditions are favorable for sorghum to be invaded by greenbugs that come from the wheat fields.

The population of *Sorghum halepense* (johnsongrass) around sorghum fields might also be important reservoir for the virus.

Toler:
Sugarcane mosaic virus types, which have a wide distribution and are most important economically, tend to have a wider host range and can be transmitted by aphids and also mechanically. There is no lack of viral reservoirs in the cooler areas.

N. G. P. Rao:
Insect pests are generally more important in the tropics. Diseases are more important in temperate regions.

Frederiksen:
What will happen in the future concerning viral diseases in the tropics?

Toler:
Temperature has a great effect on the type of vectors and the population of vectors. It has no effect on the presence of viruses, and does not hinder viral infection, but it greatly influences symptoms. Viral diseases will probably increase in incidence. Sorghum was first listed as an experimental host for brome mosaic virus; 24 years later this virus was found in Texas on sorghum under natural conditions. As genotypes become more uniform, rare viruses may become important.

K. N. Rao:
In India we seldom observe viral infection on sorghum, and then only on occasional plants. We never see it on a wide scale, or causing significant yield losses. I am curious about sorghum yield losses due to viruses in the Americas.

Teyssandier:
In Argentina, MDMV appeared suddenly and spread rapidly. We have not yet estimated the yield losses. Some seedlings are killed, but many infected plants produce panicles.

Riccelli:
The estimated losses are considerable in Venezuela. In 1977 SCMV caused losses in sorghum production all over the country.

Teakle:
When there is an occasional cold snap of a few days in Australia, we have heavy losses in red-leaf-susceptible sorghums.

Balasubramanian:

How are necrotic lesions produced? Are they produced on systemically infected plants? Do they contain virus?

Toler:

The virus is systemic. Chlorosis and mottling appear first, then necrosis develops.

Teyssandier:

Necrotic areas develop in mature leaves, while mosaic occurs in young tissue.

Malaguti:

Some viruses are seed-transmitted. Are any strains of SCMV transmitted by seeds?

Toler:

SCMV was first reported to be seed-transmitted in maize by Shepard in 1967. In 1977 and 1978 Boothroyd demonstrated MDMV seed transmission in sweet corn. I tested 22 000 seeds from MDMV-infected plants, but I obtained no evidence for MDMV transmission in sorghum.

Toler:

I propose that we develop a set of differentials that would be available for international use, to give an idea of variants of MDMV and other viruses all over the world. We could start with the differential set used by Drs. Teakle, Riccelli, and myself, with which we obtain differential responses to MDMV-A, MDMV-B, Brome mosaic, SCMV-A, B, D, H, and I, and with the virulent virus strains in Venezuela and Argentina.

Utilization of Resistance

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Strategies for Utilization of Disease Resistance in Sorghum

H. Doggett*

The objective of ICRISAT's sorghum program is the production of sorghum cultivars possessing both improved stability and level of yield, with a grain quality acceptable to consumers. ICRISAT is only one member of a number of groups of scientists working together towards the same ends. However, ICRISAT does have a special responsibility to support other scientists in the technical areas of their work.

There are two aspects of yield improvement: (a) the accumulation of positive yield factors, so that genotypes with a more stable, better production potential are developed, and (b) the accumulation of factors that help to frustrate the yield reducers (pests, diseases, *Striga*, and physiological stresses) that prevent the full expression of inherited potential performance. Improved genotypes should ideally be matched to the best economic farming practices.

In the semi-arid tropics as a whole, chemical disease control is uneconomic. The only exception is the use of seed dressings, and even these will take a long time for adoption by many sorghum cultivators who save their own seed. For practical purposes, when we talk of disease control in the sorghums of the developing world, we are talking of breeding for disease resistance. However, the plant breeder cannot confine his attention to disease resistance. He has also to improve yield, adaptation, maturity length, drought resistance, pest resistance, *Striga* resistance, and grain quality, to mention only some of his problems. If we consider the time scale required to take each of these aspects in turn, and to combine them step by step until the perfect sorghum genotype has been created, it is inconceivably long. Further, disease resistance is most certainly not a single factor, but the accumulation of numerous genetic factors that thwart numerous disease organisms. It is therefore absolutely essential to see resistance breeding as one facet, albeit a most essential facet, of the total improvement program. Perhaps the step-by-step approach has seemed superficially attractive because of the amazing success of U.S. scientists in creating the modern North American sorghum cultivars. However, let us not forget that sorghum was a new crop in that country, lifted out of its home environment. Problems tended to arise step by step, as diseases or pests appeared, and were therefore solved step by step. The main problems of the Old World — Quelea birds, *Striga*, shoot fly, and *Chilo* stem borer — have not yet reached the main sorghum areas of the USA. Material developed there has not been subjected to selection pressure for any of these major problems. I often wonder whether the history of pearl millet hybrids in India would not have been very different if the male-sterile pearl millets had been developed in the Old World, rather than in the New World where they were totally isolated from the downy mildew pathogen which prevails in the Old World.

Diseases are caused by parasitic organisms, although by no means all of them are obligate parasites. We can view the history of disease development as the interaction of two genetically variable populations — that of the host plant and that of the pathogen. In the course of this interaction, pathogens developed strains that could parasitize, and these exerted a selection pressure on the host population. If the host population responded successfully, then a resistance mechanism developed which, in turn, exerted a selection pressure on the population of the pathogen, favoring mutations, or genetic recombinations, which could break down this resistance mechanism. The ding-dong struggle has been continuing since life began. And the host plants we have today are those that had been fashioned on the anvil of adversity so that they can flourish and reproduce in the presence of numerous parasitic organisms. Similarly, the parasites, or at least the obligate parasites, have developed such versatility that they have managed to survive successfully on their resource-

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ful hosts. Obligate parasites that have survived as successfully as have the wheat rusts and downy mildews have a flexibility which we ignore at our peril.

The third component of this system is furnished by the environment. Conditions in one area may favor the host relative to the parasite, and in another area the converse may be true. Differing climatic conditions during the course of the year may favor one of the antagonists in this struggle. Historically, the sorghum areas of the Old World consisted of regions, or even pockets, where the host genotypes were in balance with the pathogen genotypes within the system. Today, however, there is much movement of host plants between regions and into pockets, and there has certainly been movement of the pathogens as well. Thus, when dwarf shallu from the USA was grown in 1952 on light land at Ukiriguru, Tanzania, it fell flat on its face from charcoal rot, and provided the first record of the existence of the disease in that country.

How, then, can we tackle the problem of disease resistance? I suggest that our basic approach must be to look at the situation just outlined, and to join it. We should seldom think of adding resistance genes one by one, although there could be particular situations where this approach is correct. We have the situation in which there are differences between the genotypes of the various sorghum cultivars that have developed in different regions, or pockets. We can assume that, down the years, the cultivators have exploited the genetic variation available to them to a large extent, and for the plant breeder to continue to work within that circumscribed system is largely a waste of time. He is not going to do very much better than cultivators have done before him. The plant breeder's basic operation shall therefore consist of crossing together different cultivars developed in different regions which will therefore have differently adapted genotypes. From these crosses, he extracts new lines superior to the parents originally used. However, the parents brought in from other locations will have developed a different spectrum of disease resistances, perhaps against different diseases, but certainly against different levels of those diseases as a result of environmental differences, resulting in different selection pressures. What we need to do is to expose the segregating material from such crosses to as comprehensive an assault as we can organize by the important pathogens from both areas, so that the complementary superior resistances are selected out.

I think that ICRISAT can assist a great deal in making this possible. Multilocational testing of large segregating populations may theoretically be the best way to do this, if the locations are chosen so that there is exposure to every important sorghum disease. This would be an enormous undertaking. However, if we could develop a comprehensive range of disease nurseries, much of the initial screening for resistance to disease could be carried out in the early segregating generations, leaving only manageable quantities of material to be sent out for multilocational tests. Therefore, I see the first of ICRISAT's most valuable supportive functions as the identification of *susceptible* sorghum cultivars, and the development of the technology to create epidemics in the disease nurseries, so that plant breeders in the national programs have the tools for disease-resistance selection available to them.

The multilocational testing starts within countries and within regions, but the second main supportive function of ICRISAT lies in its organization of multilocational testing of crops for the semi-arid tropics. In this way, the best material in the national and regional nurseries can be exposed to the whole range of sorghum-growing conditions. These nurseries result in the identification of both susceptibles for local-disease-nursery purposes, and resistant material for use as parents.

I would not want to make the breeding for disease resistance sound too difficult. As mentioned in my earlier paper, and made clear in the leaf disease session, much can be done in national programs by planting material at a few locations, with the plantings timed so that high levels of disease incidence occur. One then throws out the susceptibles and favors the cleaner plants. However, in dealing with crosses involving exotics, especially crosses of exotic × exotic material, it is essential to test disease resistance adequately, and disease-nursery technology outlined in the leaf diseases session ought to be used.

There is a fundamental difference between plants, which strongly influences the kind of resistance mechanisms that have been de-

veloped under the selection pressure imposed by pathogens. The self-pollinating crops derive from generally self-pollinating ancestors, whose populations approximated to mixtures of pure lines. Selection therefore tended to be for the accumulation of resistance factors in the lines, and of linkages that often resulted in their behaving as major genes. A range of resistance factors was accumulated, which, working together as a group (a mixture of pure lines, each with somewhat different resistance factors), were able to contain the variability of the pathogen so that epidemics did not occur.

Single genotypes occupying large areas are always vulnerable to organisms possessing the flexibility of some of the obligate parasites. When this happens with a crop plant such as wheat, the selection imposed on the population of the pathogen results in new races and causes "a breakdown of resistance." Thus, with the wheat rusts, it has proved necessary — at least with regard to rust resistance — to go back to the ancestral situation, and to grow a mixture of genotypes. These multilines present the pathogen with a similar situation to that which prevailed in the wild ancestors of the wheat crop, and selection pressures on the pathogens to produce new races are greatly reduced. We are simply using the mechanism that the wheat plant had developed in its wild state. However, the development and maintenance of multilines requires much technical input. A series of distinct resistance genes has to be identified, and has to be backcrossed into a common genotype. The rust races and the resistance genes need constant monitoring during the life of the multiline.

The cross-pollinating crops present a different picture, for resistance factors were accumulated within the population and not within individual inbred lines. Therefore, pressures to accumulate and develop major genetic factors were less, and polygenic situations are more common. In situations of considerable pressure, the entire population developed resistance and major genes were accumulated, but often against a broad background of modifier genes.

The hazards of covering large areas with single genotypes are just as great for the cross-pollinating crops as for the self-pollinating crops, and for exactly the same reason. The single genotype imposes a selection pressure on the pathogen, to which it responds by the production of new races. Downy mildew in pearl millet is an excellent example. The plant breeder's response should be to go back to the resistance mechanisms developed by the ancestors of pearl millet during the evolution of the crop. Genetic variability for resistance must be retained. This can be done by taking a series of inbred lines, each showing resistance to different races of the pathogen, and then recombining them into random-mating populations as a synthetic. The parallel with the multiline approach for the inbreeding plant is evident. However, there is a much easier way of doing this, which again is the way in which the plant did it in the wild. A variable population is exposed to the full range of the pathogen, which itself imposes the selection pressure on the population without any need to extract inbred lines, or to identify resistance factors (provided that appropriate recurrent-selection technology is used, and that the full range of the distribution of the pathogen is used to provide the selection pressures).

I see a parallel between the situations where the pathologist and the breeder are working to obtain resistance to many races of one pathogen, and the situation in which they are working for resistance to single races of many pathogens. We can use the technology that the plants themselves have developed during their years of evolution to do a large part of our work for us. By using male-steriles to treat sorghum as a cross-pollinating crop, we can subject populations to intense selection pressure for a whole range of disease-causing organisms, and just accumulate our resistances without lots of genetic studies.

I would therefore regard the second most important task of ICRISAT as being the development of random-mating populations containing a broad spectrum of disease resistances, as well as many other desirable characteristics. In terms of sorghum improvement in the developing world as a whole, I would give this aspect of study the highest priority, because it presents a workable answer to the problem of how we can accumulate these numerous favorable characteristics needed in our improved crop plant. I recognize that ICRISAT has had to become involved in so much national program work that this cannot be given

the priority which I believe it needs. Perhaps this could be the most important place where the USAID-supported Title XII program could be of lasting and very great assistance to the developing world, i.e., the development of populations, and the recurrent-selection technology to go with them, which can take crop improvement in national programs for great distances merely by selection carefully carried out by well-trained, experienced scientists with a thorough knowledge of the crop situation and its needs in their own country. They ought to have started life as farmers, and to have retained the closest possible links with farmers.

I would outline again a few of the ways in which recurrent selection in random-mating populations can be used to handle disease resistances.

1. Mass-selection Systems where a population is developed by random mating, for three generations, parent lines carrying individually some of the resistances required, so that the final population will contain all the desirable resistances. Mass selection for clean plants is then performed, choosing male steriles and male fertiles in alternate generations. Selection pressures of 20 to 25% might be used for the first three cycles, with more intense selection pressure subsequently. The population should be grown for selection under conditions favorable to disease development, and artificial aids should be used to enhance disease levels. Failing this, the population should be grown at two or three sites where disease levels are naturally high. Grain from the selected heads from all sites should be random sampled and mixed in equal proportions at the beginning of each cycle, i.e., the harvest from the male-fertile plants chosen in the second generation of the cycle. Large numbers of plants are rather easily handled by such bulk methods. Thus, 2000 plants can be grown in a block and divided up into a 5 × 4 grid, each containing 100 plants. For 10% selection pressure — which is quite high — the best ten plants are taken from each grid. Carrying forward 200 plants per generation in this way gives plenty of opportunity to select for good agronomic type at the same time.
2. S_2 Testing Systems. In this system, some selection pressure for resistance can be applied both in the S_1 (F_2) and S_2(F_3) generations, and this might prove particularly useful for grain mold resistance. One half of each S_1 row should be inoculated with grain mold, and the selection of the lines to go into S_2 testing should be influenced by the mold reaction. Alternatively, a separate replication of the S_2 evaluation trial should be treated as a grain mold resistance nursery, and the heads actually used for the recombination generation would include some that showed superior mold reaction.
3. It is always permissible to include one or two good grain mold resistance entries in the recombination generation, even if they do not come from the population itself. Evidently the inclusion of several such lines could lead to difficulties, and the figure suggested is of the order of 5%.
4. The sidecar approach is of special value. In this method, a separate population is under strong selection pressure for grain mold resistance in parallel with the main population, which is being improved for agronomic, quality, and yield characters. Crosses are made between the populations in a manner analogous to the conventional backcross method used between individual parents. In this way, a steadily improving standard of grain mold resistance is combined with the agronomic improvements being achieved in the main improvement population.

I have attempted a broad coverage of the subject, and have left the classical resistance breeding until last, because I expect many other people to speak on this. I would add as ICRISAT's fourth task, the identification and creation of sorghum lines which combine high levels of particular disease resistances with good agronomic characters, some level of pest resistance, and an acceptable grain quality. These would be valuable parents for workers in national programs. However, I am firmly convinced that it is the population-breeding approach, combined with good disease nurseries and multilocational testing, which is going to prove to be the most effective answer, not only to breeding for disease resistance but also for the improvement of the sorghum crop as a whole.

Note: After this paper was written, I came across a paper by Frey, Browning, and Simmons on this topic, which is very helpful. Copies are available for those interested. The reference is: Frey, K. J., Browning, J. A., and Simmons, M. D., 1977. Management systems for host genes to control disease loss. Annals New York Academy of Sciences, 255–274.

Needs and Strategies for Incorporation of Disease Resistance in Sorghum Hybrids Compared with Varieties

L. R. House*

Improvement of sorghum may be characterized in three broad aspects: improvement in yield and its stability; improvement in resistance to diseases, insects, *Striga*, and drought; and improvement in quality.

It may not be possible to immediately solve all problems, and frequently varieties and hybrids superior for yield but deficient for other traits are released for farmer use along with a package of management practices. Shifts in disease and insect problems generally occur when there are changes in variety and management. These shifts help mount pressure to expand the research base and resources to take up these problems.

For the small farmer, stability of yield is more important than high yield per se. Stability can be defined as uniform performance over seasons and locations. Some varieties are more stable than others and in sorghum hybrids are almost universally more stable than varieties. Resistance to diseases and other yield-limiting factors contributes to stability.

It is necessary to identify those factors that limit stability and yield and also to identify where such factors are important and where research on these factors can be focused. Viral diseases are important in parts of the Americas but not in India; obviously, research on them should be focused in the Americas. Such examples are many.

The job then is to breed for yield and yield stability and to incorporate resistance and quality traits as necessary. As a solution to these problems is frequently dependent on environments, the establishment of regionally specific networks of stations is required in the case of ICRISAT, such a system of stations is being developed and it is expected that this, in collaboration with country programs, will provide a mutually supporting network. It is through such a network that multilocational evaluation of materials for most traits of economic importance can finally be undertaken.

Varietal Improvement in Relation to the Improvement of Hybrids

Incorporating Disease Resistance into Varieties and Hybrids

Generally, good varieties make good parents for hybrids; the best varieties may not be the best parents, but surely the best parents are good varieties. The improvement in disease resistance in a hybrid is essentially a matter of improving resistance in the parents i.e., varietal improvement. The development of hybrids can be viewed as an extension of a varietal-improvement program.

As elite varieties are developed, they can be crossed to a seed parent for evaluation in hybrid combination. The production of sorghum hybrids depends on the availability of a cytoplasmic male-sterility mechanism. The male-sterile seed parent is referred to as an A-line, the maintainer of the A-line is called a B-line, i.e., A-line × B-line produces the A-line. The restorers of male fertility (R-lines) are used as pollinator parents of hybrids.

The development and evaluation of hybrids requires more time than is required to develop varieties. Usually varieties are developed first and then evaluated in hybrid combination. Unless resistance to a disease is dominant in gene action, it generally will have to be bred into both parents, i.e., the B-line and R-line. The source of resistance, when crossed to the B-line, may interfere with the male-sterility mechanism, i.e., the sources of resistance available may all be R-line in reaction; additional test evaluation

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would be required to maintain the factors for nonrestoration as disease resistance is being incorporated. This amounts to making a testcross onto an A-line and checking for nonrestoration.

Backcrossing

Development of male-sterile seed parents, or A-lines, involves a breeding process requiring eight to ten or more generations. Recently, for example, nonrestoring (B-line) entries have been identified from ICRISAT composites. The best of these will be converted directly into A-B line pairs by backcrossing, but also, a screening and breeding process will begin for resistance to diseases and insects, and for quality. Depending on the mode of inheritance of the trait(s) of interest, and whether or not they are available in already existing B-lines, this may be a relatively fast and easy, or a long and difficult process. While backcrossing, the B-line is the recurrent parent, i.e., it is the constant parent used in each cycle of backcrossing. Time can be saved if it is being improved for resistance during the backcrossing process to develop the A-line. In fact, the maintenance of an A-line is a backcrossing process, so as the B-line improves the A-line can be simultaneously improved. Improvement is a relative thing; good field resistance is generally satisfactory and may be approached over time, i.e., if a new variety or parent has a significantly better level of resistance than that currently available, it is contributing.

Backcrossing may also be useful for the improvement of existing proven parents; for example, the improvement of grain mold resistance in CK 60-A and CK 60-B may be desired. Good progress towards this objective appears possible using a backcross breeding procedure.

Recurrent Selection in B and R Composites

Population breeding techniques are useful for the simultaneous incorporation of many traits. If a breeder is interested in hybrid development he can keep varieties with R-line and B-line reaction separate when composites are formed, i.e., B-lines can form one set of composites, and R-line another set. In as much as possible, it would be worthwhile using source material for resistance and quality traits with B-line reaction in B-type composites. Unfortunately, the availability of resistant types in a B-line background is scarce. This means that R-type source material must frequently be used. Testcrossing onto A-line is required to determine nonrestoration. It is also possible to cross the B-line composite to the source material of interest. Evaluation might be made in F_2 or backcross progeny from such a cross. Evaluation should include a test for nonrestoration and only the nonrestoring segregates with the trait(s) of interest selected for recombination back into the population. We anticipate following such a process as this at ICRISAT.

It is simpler in the case of the R-line composite. As most source material is R-line in reaction, the improvement of the population (incorporating the resistance and quality traits of interest) can proceed in the same way as for normal varietal improvement, i.e., no testcrossing is required to maintain nonrestoring factors.

Population breeding proceeds by cycles of improvement, each cycle involving an evaluation and recombination phase. This cyclic process of breeding is referred to as recurrent selection. Reciprocal recurrent selection is a breeding procedure useful if hybrids are an objective. In this breeding system, each population is used as a tester for the other, i.e., entries in the B-composite would be crossed onto genetic male-steriles (frequently utilizing the ms_3 or ms_7 male-sterility source) in the R-composite and vice versa. The hybrids so formed are evaluated in yield trials and the parents to be recombined in each population identified from the best hybrids. In this way, it should be possible to identify material from both populations that, when combined, will result in good hybrids. Obviously, resistance and quality traits can be strengthened in both populations by screening during the evaluation phase of each breeding cycle.

We can appreciate that improvement of hybrids is essentially a matter of varietal improvement. It is apparent that the identification of good hybrids can be expected to take more time than the development of good varieties because the varieties must first be developed, then evaluated in hybrid combination. Also, it is generally necessary to breed resistance into

both parents of a hybrid and this may involve testcross evaluation of B-line types to be sure that genes for nonrestoration are not lost. The discussion to follow will deal primarily with varietal improvement, as this is the basic entity.

The ICRISAT Sorghum Improvement Program

The program at ICRISAT will be used to illustrate needs and strategies.

If one looks at a list of disease and insect problems on sorghum, it is apparent that dealing with all of them in a breeding program would be a frustrating exercise. Priorities must be established. We have decided initially at ICRISAT Center to concentarate on breeding for resistance to grain molds, charcoal rot, and downy mildew. However, we anticipate being sure that our material is not unusually susceptible to other diseases. We recognize, for example, that some of the bacterial diseases are important in parts of Africa and that anthracnose and viral diseases can be major limiting factors in parts of the Americas. We are still in the process of establishing regional stations, and as these stations develop we will provide and encourage the accumulation of a wide diversity of material that can be evaluated for reaction to major diseases, as well as other traits of economic interest, in the region represented by that station.

Resistance to grain molds is of universal concern and is associated with the desire for earlier maturing varieties that can be expected to perform better than traditional cultivars if rains stop early. It is not so widely recognized that the use of earlier types can result in the buildup of midge that can be devastating on late-maturing traditional types. One way to be ready for this is to breed for midge resistance in early material. We are beginning to make progress on this front.

Charcoal rot has increased dramatically in recent years and, as Hugh Doggett pointed out, probably followed a change to susceptible varietal material. The incidence of downy mildew also seems to be on the increase. These two diseases are of universal concern.

Our approach has been to develop satisfactory screening procedures and evaluate collections and existing breeding stocks. This source material has then been incorporated into our breeding material and, increasingly, breeding material is appearing in our screening programs.

We feel that close interdisciplinary cooperation is a key element of our activities — a team in the best sense of the word. We plan to apply selection pressure on early generation material where variability for traits of interest should be fairly high. Three basic thrusts are identified — the development of agronomically elite types, the development of source material for resistance and quality traits, and the development of types where there is an effort to incorporate multiple traits simultaneously. It is valuable to identify agronomically good material for use per se and as parents for crossing to source material. The development of source material is more or less the building-block approach wherein an attempt is made to improve, individually, the level of resistance or quality in different materials, although an effort is made to obtain multiple traits as far as possible. Severe levels of stress are used and the material is given reasonable protection against pests and diseases not involved. Our effort to simultaneously incorporate many traits is still in a formative stage. The approach in early generation material is to screen in epidemic and epiphytotic conditions similar to a situation as the farmer faces it. With generations or cycles of improvement, depending on the trait, the level of screening severity will be increased. We hope to maximize our chances to accumulate desired genes in this way. Too severe a screen with early generation material may result in the loss of useful genes.

The breeding techniques used in all three aspects depend largely on the mode of inheritance. Trichomes (microscopic hairs on sorghum leaves) for example, have been found in the case of shoot fly to contribute to oviposition nonpreference. The presence of trichomes is controlled by a single recessive gene. The manipulation of this trait is different from one that is polygenically controlled. We are interested in learning more about mechanisms of resistance because this knowledge may enable us to more completely screen the genetic material available. It may also enable us to use different breeding techniques and to strengthen the expression of the resistance trait.

The degree of resistance for different traits

varies. We have decided that direct evaluation of breeding material for resistance to stem borer, charcoal rot, and downy mildew is worthwhile but not for shoot fly. Our approach is changing so that breeders with rosponsibility for different projects will lower selection pressure on the trait involved. The numbers of selections in each project will then be reduced first by screening for resistance to stem borer, charcoal rot, downy mildew, and grain mold. For traits like resistance to shoot fly, where the available levels of resistance are poor and the trait is polygenically controlled and recessive, we feel that the material screened should be crossed to the best source material that we have and the segregates screened, beginning with low shoot fly pressure. We have yet to try these ideas, but it does present a phased approach to the rapid incorporation of many traits into the same material. The ones that appear to be easier are undertaken first.

There is a substantial effort in the breeding program at ICRISAT to develop new elite lines from composites. Composites are also being developed for resistance to mold, *Striga*, and three insect pests — shoot fly, stem borer, and midge. Population breeding provides a valuable mechanism for simultaneous incorporation of many traits. Composites developed earlier were primarily directed toward increasing yield in plants with tan color and pearly white seeds. The objective is now being broadened to incorporate resistance traits. To avoid incorporating poor material into a relatively elite population, bulk pollen from the population will be crossed onto source material, or selections from the composites will be evaluated directly for resistance. If necessary, backcrossing will be undertaken. Segregates will be screened for multiple traits; seed from the source families is being evaluated for yield, disease, and pest resistance. Good selections from each evaluation will be intermated in the recombination phase and the cycle repeated.

We anticipate that sources of resistance identified at one location will be evaluated at other locations, so that we learn about racial variation or differences in reaction. The best material found in these different situations can be recombined. Regional cooperation is necessary for this to succeed. International disease and pest nurseries, and some relevant trials from breeders, have now been distributed for several years. These have already been mentioned at this meeting.

A program for the development of hybrids is gaining strength at ICRISAT. As a service, we expect to concentrate on the development of A-lines adapted to different environmental situations, and to incorporate resistance to diseases and insect pests with good grain quality. We have begun to search our material for B-line reaction with some success. We also have available B-lines developed by the All India Coordinated Sorghum Imporvement Project, and from the USA, and elsewhere. We first screen these as possible source material, particularly for resistance to grain mold, charcoal rot, and downy mildew. For grain quality, it is important to our breeding effort if source material in a B-line background can be identified.

We are also aware that all problems do not appear everywhere. We need not be concerned about resistance to shoot fly for material to be used in the Americas. The problems of sorghum adapted in cool high-elevation areas are quite different from those occurring in the lowland and more tropical situations.

We feel that our program is finding definition in identifying priorities and selecting the best locations from which to undertake research. We feel that our breeding strategy is broad based, using a range of breeding procedures to develop high-yielding stable lines, to identify and strengthen source material, and to attempt simultaneous incorporation of many traits. Our screening techniques are improving and useful material is increasingly being identified.

Breeding Sorghums for Disease Resistance in India

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Breeding for resistance to prevalent diseases or control through fungicide usage have not received adequate attention in attempts to improve traditional subsistence-level culture of sorghums in the semi-arid tropics. Seed treatment through Sulfur or other fungicides to control smuts has been, by and large, the major recommendation. In transforming traditional sorghum culture to productive agriculture and attaining stability at higher levels of production, incorporation of disease resistance in future cultivars assumes particular importance — not only in order to provide resistance to known disease hitherto not considered serious enough to cause economic losses, but also to avoid possible occurrence of diseases in epiphytotic proportions. As explained in its annual reports, combining resistance to more than one disease in future cultivars has been a major objective of the All India Coordinated Sorghum Improvement Project. Some of these efforts will be briefly outlined.

Head Molds and Grain Deterioration

One of the major breeding efforts to avoid climatic vulnerability of rainfed sorghums has been to develop cultivars whose duration would match with the duration of the rainy season, as against the disproportionately long duration of 5 to 6 months for traditional cultivars. While the development of hybrids and varieties that mature in 90 to 110 days confer stability of production, even in years of low rainfall, occasional late rains in October cause grain deterioration and consequent low market price. The problem has been to reconcile the need for earliness for yield stability and necessity of avoiding grain deterioration when rains arrive before harvest (Rao 1977). This situation occurs in several African countries as well.

Grain deterioration is the result of heads getting soaked during the grain-drying stage, and/or development of molds resulting in grain discoloration. The problem of head molds in India has been investigated by various workers including Narasimhan and Rangaswamy (1969a, 1969b); Koteswara Rao and Purnachandrudu (1971), and Ravindranath and Indira (1979). *Curvularia, Fusarium, Helminthosporium, Alternaria*, and *Phoma* have been reported to be the common genera of fungi associated with head molds. *Phoma* has been observed to occur under humid heat, particularly if harvests are delayed. In gooseneck types, only the exposed portion is infected by *Phoma*; the other side is relatively free. Experience also indicates that bagging prevents occurrence of *Phoma*.

From the point of view of breeding, it appears necessary that the total process of grain deterioration (involving fungal *and* physical aspects) should receive attention. Screening for resistance to head molds and grain deterioration has identified the released variety CSV-4 (CS-3541) as the best available. The released hybrids CSH-5 and CSH-6, which have CS-3541 in their parentage, are also desirable. In fact, the availability of CS-3541, CSH-5, and CSH-6 — which corrected for the drawback of grain discoloration of CSH-1 — has now enabled larger coverages in states like Andhra Pradesh and Tamil Nadu where molds are serious. It has also made practical the practice of double-cropping under rainfed conditions in parts of Maharastra, Madhya Pradesh, and Karnataka.

Further screening efforts have also identified CSV-5 (148/168), SPV-35, SPV-81, SPV-102, SPV-126, SPV-141, and SPV-249 as moderately

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resistant to head molds. Besides, IS-3927, IS-9327, IS-9333, and IS-9530 have been reported as resistant by ICRISAT workers, and may be used as genetic stocks.

Selection criteria to breed for resistance to grain deterioration have been developed by Rana et al. (1977). The three characters — tan plant type, low water absorption capacity when soaked for a period of 2 hr, and the breaking strength of grain — are the criteria used for selection of types resistant to grain deterioration.

The inheritance of hardness of seed and rate of water absorption are predominantly additive, and selection for these characters together with the tan plant type is effective. The tan × tan and hard × hard crosses exhibited less deterioration. As the degree of softness increased, the degree of deterioration also increased, and hard × hard crosses within tan types are the best (Rana et al. 1978).

From the crosses involving CSV-4, CSV-5, SPV-81, SPV-102, etc., several advanced generation progenies of economic worth have been isolated and are undergoing yield testing.

Downy Mildew

Downy mildew caused by *Peronosclerospora sorghi* has been more frequently observed in humid areas around Dharwar in Karnataka and adjoining areas of Maharashtra and parts of Tamil Nadu. Occurrence of downy mildew in the yellow locals of Andhra Pradesh was observed during the current rainy season. Even though most locals show a susceptible reaction, SDM rarely assumed epiphytotic proportions outside experimental fields. Even so, it is desirable to avoid release of highly susceptible cultivars in vulnerable areas.

Fortunately, very high levels of resistance have been built up in released hybrids and varieties. The following parents/hybrids/varieties are highly resistant to SDM:

Parents:	..	2219A and B, 2077A and B, 323A and B, CSV-4 (CS-3541), CSV-5 (148/168)
Hybrids: (released and experimental)	..	CSH-5, CSH-6, SPH-24, SPH-107
Varieties: (released and experimental)	..	CSV-4, CSV-5, SPV-35, SPV-81 (Uchv-2), SPV-104, SPV-105, SPV-126, and SPV-238

Among genetic stocks, QL-3, IS-173, and several others have been reported to be highly resistant.

Resistance to SDM appears quantitative in inheritance. F_3 progenies of resistant × susceptible crosses presented normal distribution (Rana et al. 1978a). However, there is a report by Puttarudrappa et al. (1972) indicating two complementary factors governing resistance.

F_1 hybrids between resistant and susceptible parents indicate partial dominance for resistance, their infection percentages falling between mid-parental values and the resistant parent. A detailed genetic analysis of inheritance to downy mildew is presently in progress. Available evidence indicates the highly heritable nature of resistance. Hence there should be no problems in transference.

Charcoal Rot

Charcoal rot caused by *Macrophomina phaseolina* becomes prominent under conditions of moisture stress, usually during the postrainy season, and when low soil moisture and high temperature occur during seed development, as sometimes happens in rainy season situations.

Screening of presently available cultivars identified the following moderately to highly resistant types:

Highly resistant:	..	CSV-5 (148/168) and SPV-104
Moderately resistant:	..	323B, 296B, SPV-34, SPV-35, SPV-105, SPV-106, SPV-126

Some efforts have been made to study the genetic basis of resistance to charcoal rot (Rana et al. 1978a), and further studies are in progress. Hybrids between resistant and susceptible parents show intermediate reaction or tend towards resistant parent. The F_2 range was within parental limits. In F_3, there was transgressive segregation and the distribution was continuous. The relationships between F_1 and F_2 and between F_2 and F_3 were significant at the 10% level only. This low degree of determina-

tion indicates that heritability of resistance may be low. Since F_1 hybrids tend to approach resistant parents, use of moderately resistant parents (or at least one resistant parent) in a hybrid program may confer advantage to the hybrid with respect to reaction to charcoal rot.

Leaf Diseases

Leaf diseases in India rarely cause economic losses. Rust usually appears before harvest and reduces forage quality. Breeding for resistance to several of the leaf spots is generally attempted. Common leaf spot diseases occurring in India include zonate leaf spot (*Gloeocercospora sorghi*), grey leaf spot (*Cercospora sorghi*), rough leaf spot (*Ascochyta sorghi*), blight (*Helminthosporium turcicum*), sooty stripe (*Ramulispora sorghi*), anthracnose (*Colletotrichum graminicola*) and rust (*Puccinia purpurea*).

Among released and advanced generation materials, the following exhibit high degree of resistance to more than one disease.

Parents: .. 2219A and B, 2077A and B, 296A and B, 323A and B, CS 3541 and 168/148
Hybrids: .. CSH-5, CSH-6, and SPH-61
Varieties: .. CSV-4, CSV-5, SPV-96, SPV-104, SPV-105, SPV-106, SPV-192, SPV-220, SPV-224, SPV-232, SPV-257 and several others.

The rust reactions of certain cultivars differ at Dharwar and other centers. Patil-Kulkarni et al. (1972) reported more than two pairs of genes, susceptibility being dominant. At Dharwar, where stocks rated as resistant to rust at other locations succumb to rust, the following were identified as resistant to rust: SPV-9 (SB 411), SPV-13 (SB 803), SPV-34, SPV-35, SPV-73 (SB 1079), SPV-81 (Uchv-2), SPV-126, SPV-193 (SB 905), SPV-248, SPV-257.

The following exhibit moderate degree of resistance to rust:

Parents: .. 2219B, 2077B, 296B, CS 3541
Hybrids: .. CSH-5, CSH-6, SPH-61
Varieties: .. CSV-4, SPV-96, SPV-104.

Genetic analysis of rust resistance by Rana et al. (1976) indicated that susceptibility is dominant and that three major genes govern resistance. Rust resistance is present in the tan and in the purple plant types.

There are indications that resistance to zonate leaf spot is governed by complementary genes. Detailed studies are in progress.

Present indications are that leaf spots may not pose serious problems, and resistance breeding is quite effective.

Multiple Resistance

Genetic stocks and breeding materials exhibiting resistance to more than one disease now

Table 1. Multiple disease-resistant sorghums.[a]

Cultivar		DM	CR	Rust	ZLS	Cer	An	SS	Helm	Asco	SD
SPV-104	(6)	x	x	x	x	x		x			
SPV-126	(6)	x	x	x	x	x					
SPV-178	(6)	x	x	x		x	x				x
CSH-5	(6)	x		x	x	x		x			x
CSV-4	(5)	x	x	x	x	x					
CSV-5	(5)	x	x		x	x					
SPV-193	(5)	x	x	x	x	x					
CSH-6	(5)	x		x	x	x					x
SPH-61	(5)		x	x	x	x					x
SPH-80	(5)	x	x		x	x			x		
SPV-70	(4)	x		x	x					x	

a. Number in parentheses indicates number of diseases to which this cultivar shows resistance; DM - Downy mildew; CR - Charcoal rot; ZLS - Zonate leaf spot; CER - Cercospora; AN - Anthracnose; SS - Sooty stripe; HELM - Helminthosporium; ASCO - Ascochyta (Rough leaf spot); SD - Sugary disease. x indicates resistant.

enable us to breed for resistance to several of the prevalent diseases.

Downy mildew, charcoal rot, and components of grain deterioration are polygenic in inheritance, while several of the leaf spot diseases are governed by relatively few genes. There is evidence that resistance to charcoal rot, SDM and leaf spots is independently inherited. This might hold good for other diseases as well — which should make possible an approach to more than one disease in a resistance-breeding program. It is also evident that, compared to purple-pigmented types, tan-plant types generally exhibit greater levels of resistance to most leaf diseases, and hence the tan plant is an important selection criterion. Biochemical characterization of tan and purple types would be useful.

Keeping some of these criteria in view, it has been possible to develop commercial cultivars, parents, hybrids, and varieties which exhibit high levels of resistance to several diseases. A list of such varieties is presented in Table 1.

Summary

High levels of resistance are available for most sorghum diseases. The nature of inheritance is quantitative for downy mildew and charcoal rot, and qualitative with respect to other diseases. Resistance to various diseases is independently inherited. Heritability of resistance is high, except in the case of charcoal rot. Tan-plant types have generally higher degrees of resistance to foliar diseases. It appears feasible to combine resistance to several diseases together with higher yields in breeding cultivars with higher levels of productivity and stability.

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Current Strategies and Progress in Breeding Disease-Resistant Sorghums in Venezuela

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In the last decade grain sorghum has had a tremendous impact on Venezuelan agriculture (Riccelli 1973c) and it will continue to play a very significant role in the future. Even though it was introduced recently (Campos 1969; Taborda 1972), its acreage has increased faster than that of any other crop, moving from approximately 10 000 ha in 1972 to 300 000 ha in 1978.

Tropically adapted hybrids have been developed (Riccelli 1977a) and their seed is being produced (Riccelli 1978b). However, the supply of local seed is still not sufficient to meet national needs, and large amounts of seed of several temperate hybrids are being imported. It has been repeatedly shown that their performance in the tropics is inferior to that observed in the area for which they were created (Riccelli 1973c, 1974). There is now clear evidence that the generalization of broad adaptation of sorghums is often misleading and too simplifying (Nelson 1967; Riccelli 1973b).

The rapid expansion of grain-sorghum acreage and the introduction of susceptible sorghum hybrids have changed the pattern of disease occurrence, which now appears to be the most important limiting factor when disease resistance is not present in the selected genotype. Furthermore, the warm and humid conditions that occur during the growing season (Table 1) strongly promote disease development. Heavy rains during July and August and large populations of wild sorghums and other natural grasses, which grow and multiply throughout the year and act as a major reservoir for the pathogenic agents (Nass et al. 1977), provide conditions suitable for infection and spread of leaf and stalk diseases and grain deterioration. Because of these conditions, the plant breeder should pay more attention to disease problems in the tropics, than in the sorghum belt of the USA.

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The development of disease-resistant lines and hybrids has been an important aspect of the breeding efforts made in the past few years toward tropical adaptation and yield improvement. The first step was identifying the diseases and determining their distribution in the sorghum-producing areas, and evaluating their virulence. As in other countries in the past, host resistance has been chosen from the great genetic diversity present in the sorghum species. Continuous international cooperation has permitted the assembly of a collection of approximately 3000 items which have been partly evaluated for those diseases of major concern. On the other hand, the international nurseries grown in Venezuela have helped provide additional data on disease resistance of certain selected genotypes, and to identify genetic variability in pathogens.

The screening of large numbers of breeding lines and varieties was followed by the initial determination of the inheritance of resistance to certain diseases, especially those caused by what are supposed to be local variations of the pathogens. The ultimate goal is to combine good disease resistance with good tropical adaptation in the hybrids that will be released.

This paper contains a description and results of the initial work that several authors have done in Venezuela during the last few years.

Sorghum Diseases in Venezuela

The common sorghum diseases that have been reported or observed in Venezuela are presented in Table 2. Perhaps this list is still incomplete, but it certainly contains all the diseases of economic significance. National losses in grain sorghum due to diseases have not been estimated, but they are undoubtedly the most important factor causing yield reductions. At present, in my opinion, the diseases most damaging to production (in order of im-

Table 1. Climatological data of the major sorghum-growing states in Venezuela.

Location	Month											
	Jan	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec
Barinas (Barinitas)[a]												
Max temp (°C)	30.1	30.4	30.4	30.1	29.6	28.9	28.8	29.1	29.3	29.4	29.5	29.6
Min temp (°C)	21.7	21.9	22.1	21.9	21.6	21.2	20.6	21.2	21.4	21.4	21.0	21.4
Rainfall (mm)	34	20	53	197	239	327	311	330	256	352	196	61
Portuguesa (Turen)[a]												
Max temp (°C)	33.5	34.4	35.3	34.6	32.6	30.7	30.5	31.2	31.8	32.3	32.4	32.7
Min temp (°C)	20.7	20.7	21.6	23.1	22.7	22.1	21.6	21.7	21.8	21.9	21.9	21.5
Rainfall (mm)	5	10	9	55	180	304	243	247	180	110	88	27
Guarico (Banos de San Pedro)[a]												
Max temp (°C)	33.4	34.1	34.7	34.5	32.5	30.1	29.7	30.0	30.8	31.5	32.1	32.6
Min temp (°C)	20.9	21.5	21.7	22.9	22.6	22.2	21.8	21.7	21.5	21.5	21.3	20.5
Rainfall (mm)	2	3	4	49	211	282	316	204	192	[illegible]	59	13
Anzoategui (San Tome)[a]												
Max temp (°C)	28.9	30.8	32.1	32.9	32.0	29.9	30.0	30.3	31.5	31.6	31.1	30.0
Min temp (°C)	18.6	18.6	19.3	20.0	20.3	20.0	19.8	20.3	20.2	20.2	19.9	19.3
Rainfall (mm)	16	13	13	16	83	178	238	232	125	103	75	21
Monagas (Maturin)[b]												
max temp (°C)	33.2	34.3	35.4	36.3	36.5	35.6	35.7	34.7	35.4	36.0	34.4	33.3
Min temp (°C)	14.9	16.1	15.9	18.5	16.8	18.8	18.7	17.7	16.9	20.0	18.0	18.5
Rainfall (mm)	58	41	28	39	94	210	202	160	117	99	112	106

Source: Department of Meteorology, CENIAP, Maracay, Venezuela.
a. Average of 10 years (1961–1970)
b. Average of 20 years (1951–1970)

Table 2. Reported or observed sorghum diseases in Venezuela to 1978.

Common Name	Causal organism
Anthracnose	*Colletotrichum graminicola* (Cesati) Wilson
Bacterial stripe	*Pseudomonas andropogoni* (E. F. Smith) Stapp
Charcoal rot	*Macrophomina phaseolina* (Maubl.) Ashby
Covered kernel smut	*Sphacelotheca sorghi* (Link) Clint.
Downy mildew	*Peronosclerospora sorghi* (Weston and Uppal) Shaw
Fusarium	*Fusarium moniliforme* Sheldon
Grain molding	Numerous fungi
Grey leaf spot	*Cercospora sorghi* Ellis and Everhart
Leaf blight	*Exserohilum turcicum* (Pass.) Leonard and Suggs
Rust	*Puccinia purpurea* Cooke
Viral diseases	Aphid-transmitted virus
Zonate leaf spot	*Gloeocercospora sorghi* Bain and Edgerton

portance) are viral diseases, downy mildew, anthracnose, charcoal rot, and grain molding. Downy mildew, the most devastating disease 2 years ago, will probably continue to descend to a lower position when only hybrids resistant to downy mildew are planted. Grain molding can become more important than charcoal rot during years of heavy rains, and vice versa during dry years.

These diseases can eventually be brought under control through the use of genetic resistance. But their eradication is practically impossible because of the prevalence of wild sorghum species (*S. arundinaceum, S. verticilliflorum, S. halepense*, and possibly others) and other grasses, which are frequently infected. They are so abundant in agricultural fields, grassland, roadsides, ditches, etc., that their elimination is impossible.

As expected, the incidence of diseases varies throughout the year. The influence of planting dates on diseases was recently observed by Tovar and Barrientos (1978) who planted eight grain-sorghum hybrids of different maturity during 5 consecutive months (Jun–Oct) in Durigua, Portuguesa. The higher yields were obtained in the June planting, in spite of its being the experiment that showed the most seed deterioration. Chaguaramas 5 and Barinas 3, two local brown-seeded hybrids, had the least weathering and the highest yield. On the other hand cultivar E-57, NK-125, and NK-222 showed a severe deterioration.

The September and October plantings had lower ratings. This could be expected from the fact that rainfall starts to decline gradually from August onward. Incidence of anthracnose was about the same in the plantings of the first 4 months, but diminished significantly in the October planting. In the case of viral diseases the highest incidence was observed in the August planting. This seems to be a consequence of a larger insect population in the 2 months following that planting date. Zonate leaf spot was about the same during the first four periods and did not show up in the last experiment (Oct planting). In the case of grey leaf spot, the highest incidence was observed during the first period and gradually decreased in the plantings of the following months. This seems to indicate that the highest yield can be achieved if the crop is grown during the months of more rainfall, provided resistant hybrids are used.

Screening, International Evaluation, and Breeding

Viral Diseases

Viral diseases were first reported in 1969 and inoculations were made from maize and sugarcane into the variety Midland, which developed clear symptoms of infection (Ordosgoitty and Malaguti, 1969). According to Ordosgoitty (1976, personal communication) there are at least two different types of viral diseases of sorghum in Venezuela: MDMV (maize dwarf mosaic virus) and races A and H of SCMV

(sugarcane mosaic virus) Ordosgoitty and Gonzales 1977).

Starting in 1970, sorghum plants with mottling, chlorosis, red leaf, necrosis, and death of the whorl appeared in the central states (Ordosgoitty and Viera 1973). Ordosgoitty and Viera (Renfro and Frederiksen 1975; J. Viera, personal communication) transmitted the virus mechanically and by means of the aphid *Rhopalosiphum maydis* to several cultivars of corn, grain sorghum, *S. halepense*, *S. arundinaceum*, and *Rottboellia exaltata*, causing the death of the inoculated plants in the sorghum varieties: Yellow Combine Kafir IS 2937 and Combine Kafir 60. *Rottboellia exaltata* L. f., raoulgrass or itchgrass (known in Venezuela as "paja pelua") is a common widely distributed weed which, as well as the wild sorghums, serves as a reservoir for the virus.

The viral particles under the electron microscope are 755 (±10) nm long and 12 to 13 nm wide with a central channel; it is believed to be a new strain of SCMV (Ordosgoitty, personal communication). I am inclined to share this opinion, for the following reasons:

1. The occurrence of necrotic tissue and a high proportion of deaths in breeding lines that show only a mild reaction elsewhere (Riccelli 1978a; Rosenow 1968). Examples are: Tx-2536, Tx-430, Tx-622, Tx-623, Tx-624, Tx-2723, Tx-2724, Tx-2728, Tx-2735, Tx-2737, Tx-2742, Tx-2743, Tx-2744, Tx-2747, Tx-2748, R-5388, SC-48–14, SC-283–14, SC-333 and SC-120×Tx-7000. Sharvelle (1975) pointed out that in 1973 severe necrosis and death, caused by SCMV, was observed in Tx-2536 in Brazil and suggested that the strain involved could be different from the strains of the USA. Susceptibility of Tx-2536 in Brazil and Venezuela and of Tx-430 in Venezuela is significant because these parental lines, or some of their derivatives, are widely used in many currently grown hybrids.
2. The sorghums selected as indicators for MDMV reaction (Toler and Bockholt 1968) show different symptoms from those reported for MDMV-A, MDMV-B, and BMV (brome mosaic virus) when inoculated with the Venezuelan strain (Toler and Bockholt 1968; Toler and Frederiksen 1971, 1977). Reaction observed on those indicators at Macapo, Aragua, when they were 47 days old are presented in Table 3.
3. Immunity of the sorghum cultivar QL-3 with the Krish resistance (from a cross of *S. halepense* × *S. roxburghii*) (Teakle and Pritchard 1971) has not held up in Venezuela under natural or field inoculation, as shown in Table 3. Krish sorghum is resistant to four strains of SCMV in Australia.

Malaguti (1978) described a virus expression in the hybrids Aguassy 1 and Barinas 2, which appeared in the state of Guarico in 1977. It consisted of necrotic spots on the leaves in the form of concentric rings, which would, eventually, cover the whole leaf and pass to the stalk and the panicle. These symptoms were reproduced in the variety Red Swazi (100% of the inoculated plants) and in the hybrid Topaz (22%). In 1978 he used the same strain to inoculate seedlings of several sorghum cultivars and obtained mottling, streaks, and necrotic reactions in the hybrids Topaz, Chaguaramas 5. Barinas 2, and Aguasay 1, but only mottling in the breeding line SL-PR-32650.

In less than a decade since the first observations, the rise of viral diseases has been dramatic and had reached epiphytotic proportions by 1977. They have been reported in every region where sorghum is grown, reaching devastating levels where highly susceptible hybrids are planted. Mena (1978) estimated an incidence of 30% in commercial fields of the hybrid Master 911 during the present year in the state of Guarico. No sorghum was observed by the author to be immune and all kinds of symptoms can be found, from a very mild mosaic to severe stunting, leaf necrosis and deaths. Fortunately, some lines are only slightly affected in terms of plant growth and yield reduction, and they could become the basis for the breeding work.

Mena (personal communication) registered the viral symptoms of the major sorghum hybrids grown in Venezuela in 1978 in a regional test at Villa de Cura, Aragua. Percentages of plants showing different symptoms are presented in Table 4. It is significant that hybrid cultivar Pioneer 8199 was the only hybrid without any viral expression. On the other hand, cultivar Warner Wx-839-DR showed the highest percentage (90%) of plants with necrotic reaction.

Screening nurseries have been planted in Venezuela since the appearance of the disease. Ratings are now registered in each field survey, test, crossing block, or breeding nursery

Table 3. Reaction of sorghums selected as indicators for maize dwarf mosaic in the USA to a viral disease in Macapo, Aragua, Venezuela.

Entry Designation	Variety or kind	Reaction to MDM in Texas	Reaction[a] to viral diseases in Venezuela
B-Tx-378	Redlan	Severe red discoloration	Mild chlorosis No stunting.
SA-7000	Caprock	Severe red discoloration	Necrotic reaction (10%)
Tx-414	7078 der.	Resistant; mosaic or mottle, no stunting	Severe mosaic and mottling. Red streaks.
SA-7078	7078	Resistant; mild mottling	Severe mosaic and chlorotic stunting.
B-Tx-398	Martin	Most resistant; mottling no stunting	Mottling and stunting
Tx-09	Combine White Fet.	Resistant; mottling, no stunting.	Severe mottling and chlorosis.
New Mexico-31	Weskan × Redbine 60	Hypersensitive chlorotic red, necrotic	Necrotic reaction (5.5%)
Tx-412	Tx-09 × Tx-403	Mottling only and stunting.	Mottling, stunting, and necrotic reaction (1.3%)
B-Tx-3197	Combine kafir 60	Mottling and red discoloration.	Necrotic reaction (2.3%)
SA-394	Combine shallu	Mottling and tan discoloration.	Necrotic reaction (6.5%)
PI-35038	Sumac	Severely stunted Chlorosis and orange discoloration	Mild chlorosis
Tx-430	IS-12661 der.	Moderate resistance[b]	Necrotic reaction (15.5%)
QL-3	Combine Kafir der.		Mottling + slight red streaks

Source: Toler and Bockholt, 1968.

a. Rated in PROSECA planting 30 days following inoculation. Last count of dead plants made 47 days following planting. Figures in parenthesis represent percentages of dead plants.

b. As per letter, Dept Soils and Crop Science, Texas A & M Univ., 20 Feb 1976.

planted in an effort to identify good sources of genetic resistance. Publication of the results in tabular form is impractical because of bulkiness, but a listing of the evaluations may be obtained directly from each author. The data presented is from cases, where either artificial inoculations were made, or extensive natural infection developed, providing an excellent opportunity for evaluation of disease reaction.

The International Disease and Insect Nursery (IDIN), a collection of entries with high levels of multiple disease resistance, has been planted and rated on viral-disease reaction in Venezuela since 1975. In 1977 the test was exposed to a severe natural infection in Macapo, Aragua. Table 5 contains the ratings of that test and the results of artificial inoculation of 20 plants of each entry of the same nursery at the same location in 1978. In both cases the selected lines markedly differed in their response. This was the first time that viral symptoms were observed on cultivar QL-3. On the other hand, lines

Table 4. Percentage of plants showing different kinds of viral symptoms in sorghum hybrids grown in Venezuela in 1978.

Hybrid	Reaction				Hybrid	Reaction			
	Red streaks	Necrotic reaction	Mottling	Stunted chlorosis		Red streaks	Necrotic reaction	Mottling	Stunted chlorosis
PROSECA					Golden Acres:				
Chaguaramas 5	60	10	00	00	TE-Hondo	00	00	00	80
Chaguaramas S	00	20	80	00	TE-Plus	40	70	00	00
Aguasay 2	60	60	00	00	TE-Total D	60	00	20	00
Barinas 3	00	00	00	90	TE-Dinero	00	70	00	00
Araure 4	00	00	00	90					
Guanipa 1	00	60	50	00	Werner:				
Perija 3	00	00	00	90	WX-839-DR	00	90	00	00
Pioneer					Wx-832-DR	00	00	00	60
P-815-B	10	00	40	00	Mc Nair:				
P-8311	20	10	00	00	Mc Nair 650 Dr	20	00	00	90
P-8202	30	00	00	00					
8199	00	00	00	00	R. C. Young				
P-816-B	00	00	20	00	Oro Dr.	00	20	00	00
P-W 821-A	30	00	00	00	Oro Dr 11	00	00	80	00
Dekalb					Northrup King:				
D.E-59 +	20	00	30	00	NK-233	00	00	70	00
D.F-64	10	00	40	00	NK-Savanna 3	00	00	20	00
D.D-42a	60	00	20	00	NK-266	00	00	40	00
D.D-59	10	00	40	00	NK-Savanna 5	00	00	90	00
D.D-46	20	00	30	00	NK-1580	00	00	50	00
D.D-55	00	00	70	00	NK-2022	00	00	40	00
Asgrow					Colsemillas:				
Topaz	60	50	00	00	36. Icanataima	00	00	100	00

Source: Hector Mena, 1978. Centro Nacional de Investigaciones Agropecuaria (CENIAP), Maracay (unpublished data).

Table 5. Reaction of sorghum lines contained in the Texas International disease and insect nursery to viral infection in Macapo, Aragua.[a]

Entry	IS No.	1977 ratings[b] from natural infection[c]		1978 ratings[d]	
		Rep. I	Rep. II	Following inoculation	Check
SC 56-14	12568	1	1		
SC 103-12	2403	1	2	3	3
SC 108-14	12608	1	1		
TAM 428	12610	2	1	2	2
SC 110-14	12610	3.5	1	2	2
SC 112-14	12612	1	1	2	2
SC 170-6-17	12661	2	3.5	2	2
SC 170 -6 -8	12661	3	1		
SC 173 -12 -6	12664	2	1	2	2
TAM -2566	12666	1	1		
SC 175 -14	12666	2	2	3	3
SC 237 -14	3071	1	2	3	3
SC 279 -14	7419	2	1	2	2
SC 326 -6	3758	2	2	3	3
SC 717 -12 -E - P1	2508	2	1	3	3
SC 599 -6 -3	17459	1	2		
SC 599 -6 -10	17459	1	1	2	2
SC 748 -5 -3	3552	2	2	(5%)5	3
Tx-430		4	5	(15%)5	(15%)5
B Tx-624		4	1	3	3
NSA-440-12		1	1		
(SC 599-6 × SC 134-6)		•	2		
(B Tx 3197 × SC 170)					
Sel 7505		3.5	1		
SC-103-C	2403	2	1		
QL-3 Selection		3.5	3	3.5	3.5
TAM 2567		1	4		
R 1750	12661 der			2	2
GPR 148				3	3
B 1778				2	2
(SC120 × Tx 7000)	2816 der			(10%)5	3
SC 599-11-E	17459			2	2
SC 170-14	12661			(20%)5	(40%)5
77 CS 1				3	3
CS 3541				2	2
R 5388	12610 der			3	3
B Tx 378	418	1	1	2	2
B Tx 398	412	1	3.5	2	2
Tx 7078	415	1	1	3	3
Tx 2536	10542	1	1	(15%)5	(5%)5
Tx 2748				(20%)5	(5%)5

a. Nurseries planted in the field of PROSECA in Macapo, Aragua on 23 June 1977 and 4 Oct 1978, respectively.

b. 1 = No apparent or very mild symptoms; 2 = leaves with mottling only; 3 = Mottling and significant chlorosis; 3.5 = Mottling with slight leaf necrosis; 4 = Mottling with significant leaf necrosis; 4.5 = As above with stunting; 5 = Theabove accompanied by severe stunting and death.

c. Notes taken 58 days after planting.

d. Notes taken on 20 inoculated plants in each row and 20 check plants. Last rating and count of dead plants was made 43 days after planting. Figures in parenthesis correspond to percentage of dead plants.

otherwise with a certain degree of tropical adaptation (Tx-430, Tx-624, SC-170-6-17) appeared to be seriously affected. IDIN tests planted in previous years partially confirmed these observations — with the exception of Tx-2536, which showed an average reaction of 4 in Guarabao, Yaracuy and 3 in Sabaneta, Barinas in 1975. In the same tests QL-3 appeared without symptoms.

Tovar, Mena, and Barrientos (1978) conducted in 1977 the official Sorghum Hybrids Performance Tests for evaluating commercial genotypes being planted in Venezuela. Ratings on the major diseases, as well as other kinds of data, are recorded in every test. Virus reactions on random groups of varieties and breeding lines have also been taken by Viera (personal communication) and by myself. Table 6 is an extraction of the total data concerning viral diseases only at those locations where significant infection developed. Only a small fraction of the entries of the two tests planted at La Lucia were without symptoms and the majority of the hybrids exhibited between 0 and 20% affected plants. On the other hand, most of the A, B, and R-lines and the varieties rated by Viera (personal communication) showed mottling with an intensity of 3 to 4 in a scale from 0 to 5. Using the scale proposed by Texas workers, I found that most varieties and R-lines rated 2 and 3.

In my experiment, I recorded plant deaths due to viral diseases in a selected group of promising breeding lines under natural infection and field inoculations. (Table 7). It is significant that this data is highly correlated with results obtained in screening a group of experimental hybrids in which these lines serve as either one or both parental lines. In effect, all four hybrids with Tx-430 as a common pollen parent showed a high percentage of dead plants; in the other cases the following proportions were observed: Tx-622 as seed parent: two of four hybrids; Purdue 5976-3 as pollen parent: three of four; Tx-623 as seed parent: nine of 13; SL-PR-10001 as seed parent: six of six. This correlation would seem to indicate a certain degree of dominance of susceptibility to the necrotic reaction.

Table 6. Number of genotypes with different percentages or ratings of viral infection in grain sorghum performance tests and in random groups of varieties and breeding lines.

Test or Nursery	Total Number of Genotypes	Mean Percentage of Natural Infection					
		0	0–5	5–10	10–15	15–20	>20
Grain Sorghum Performance Test[a]	36	8	11	12	4	1	
Grain Sorghum Performance Test[b]	49	10	11	8	7	11	2

		Ratings from Natural Infection[e]									
		Mottling					Red Discoloration				
		1	2	3	4	5	1	2	3	4	5
Random Group of A, B and R-lines[c]	95	1	15	28	25	11	0	2	6	6	1
Random Group of Grain Sorghum Varieties[c]	97	7	5	28	41	11	0	0	1	3	1

		Ratings from Natural Infection[f]						
		1	2	3	3.5	4	4.5	5
Random Group of Grain Sorghum Varieties[d]	164	3	77	50	13	6	3	12
Random Group of R-lines[d]	28	1	11	10	0	1	0	5

a. Test planted on 29 Jun 1977 in La Lucia, Portuguesa, CIARCO.
b. Test planted on 7 Jul 1977 in La Lucia, Portuguesa CIARCO.
c. Lines and varieties planted on 21 Feb 1978 in the field of the School of Agriculture, Universidad Central de Venezuela, Maracay.
d. Varieties and lines planted 20 Aug 1978 in the field of PROSECA, Los Guayos, Carabobo. Ratings made 9 Oct 1978.
e. Ratings according to a scale from 0 = no apparent symptoms to 5 = 100% of plants affected.
f. Ratings according to the following scale: 1 = no apparent symptoms; 2 = leaves with mottling only; 3 = mottling and significant chlorosis; 3.5 = mottling with slight leaf necrosis; 4 = mottling with significant leaf necrosis; 4.5 = as above with stunting; 5 = the above accompanied by severe stunting or death.

Table 7. Evaluation of a selected group of grain-sorghum-breeding lines in relation to plant deaths and other symptoms caused by virus at two locations in Venezuela.

	Guayos		Macapo			
	Ratings from natural infection[a]		Ratings following inoculation[b]		Checks (Natural infection)	
Line	Deaths[c]	Other symptoms[d]	Deaths[e]	Other Symptoms[d]	Deaths[c]	Other Symptoms[d]
IS-12664C		2		2		2
Tx-399		2	5%	5		2
Tx-2728	30%	5	30%	5	20%	5
Combine Sagrain		2		2		2
TAM 428		2		2		2
SL-PR-10001	40%	5	40%	5	5%	5
ICA Natlama		3		3		3
SL-PR-32650		2		2		2
SL-PR-14270		2		3		3
SC 103-12		2		2		2
Tx-623	5%	4.5		3		3
SC-103-C		2		2		2
Purdue 5976-3	10%	5	50%	5	35%	5
Tx 622	10%	4.5	15%	5	5%	5
SC 414-12 E-P1		2	5%	5		3
SL-PR-34651		2		3		3
L-798-A		2		2		2
Tx-430	50%	5	60%	5	45%	5
L-864-R		3		3		3
SC 108-14		2		3		3
SL-PR-35260		2		3		3

a. Nursery planted in the field of PROSECA in Los Guayos, Carabobo, on 18 Aug 1978; rated 3 Oct 1978.
b. Nursery planted in the field of PROSECA in Macapo, Aragua, on 20 Sept 1978. Inoculation performed on 20 plants each on 11 Oct 1978; final rating 14 Nov 1978.
c. Estimated percentage of dead plants in the plot.
d. Ratings according to the following scale: 1 = no apparent or very mild symptoms; 2 = leaves with mottling only; 3 = mottling with significant chlorosis; 3.5 = mottling with slight leaf necrosis; 4 = mottling with significant leaf necrosis; 4.5 = as above with stunting; 5 = the above accompanied by severe stunting or death.
e. Actual percentage of dead plants.

More direct evidence for the above statement comes from a study on stability of performance of temperate and tropical hybrids, their parental lines, and their mixtures when growing in 15 different environments throughout Venezuela. Resistance to major diseases was evaluated and recorded. A good opportunity for viral-disease evaluation occurred in 1977 in La Lucia, Portuguesa, and Macapo, Aragua, and in 1978 at El Jabillo, Portuguesa. Table 8 contains the percentage of infected plants showing necrotic reaction in some susceptible hybrids and their parental lines at three locations. The results are remarkably consistent over locations and seem to indicate that necrotic reaction behaves as a dominant, and sometimes overdominant, character.

Viera and Ordosgoitty (personal communication) made all possible crosses and their reciprocals between Combine Kafir (apparently resistant), KS-24 (red necrotic streaks when infected), Combine Sagrain, and Tam-ga (chlorotic mottling), and inoculated the parental lines and the resulting hybrids. All hybrids, with the exception of those between lines which showed only chlorotic mottling, manifested

Table 8. Percentage of plants with naturally infected necrotic virus reaction in grain-sorghum hybrids and their parental lines at three locations in Venezuela.

Hybrid or parental line	La Lucia, 1977[a]		Macapo 1977[b]		El Jabillo, 1978[c]	
	Plants (No)	Infected plants[d] (%)	Plants (No)	Infected plants[d] (%)	Plants (No)	Infected plants[d] (%)
IS-10240	750	15.07	532	3.19	477	0.17
IS-12608 C	960	0.00	439	0.00	543	0.00
IS-10240 × IS 12608 C	674	41.99	440	6.36	476	11.13
DD-Feterita SA-6649	674	15.73	467	2.14	436	3.96
IS-10240 × DD Feterita SA-6649	784	26.53	425	3.52	575	7.82
IS-2801 C	1013	0.20	412	0.97	509	0.02
IS-10240 × IS-2801-C	682	28.29	364	3.57	602	6.81
IS-12664 C	975	0.00	396	0.03	456	0.00
IS-10240 × IS-12664-C	750	36.40	511	3.52	395	5.82

Source: Data extracted from a study on stability of performance conducted by PROSECA.
a. Planted 29 Jun 1977
b. Planted 2 Jun 1977
c. Planted 10 May 1978
d. Average of three replications in each location.

intense necrosis. These workers then obtained all possible backcrosses to both parents, and these were inoculated, confirming those results reported by Teakle et al. (1970), who demonstrated that the necrotic reaction is controlled by a gene *N* which is dominant over *n* which is responsible for the mosaic reaction. However, they suggested that there might be modifying genes involved. This might also explain my results, presented in Table 8.

Finally, in my work I inoculated one row each of the hybrids "A. Combine Sagrain × Tx-430" and "A, IS 10346 × Tx-430" and the parental lines planted in single-row plots. The results (Table 9) tend to confirm the hypothesis of dominance of susceptibility.

Resistance to the necrotic reaction and the occurrence of mild mosaic only in certain tropically adapted lines is encouraging. Further progress will be made by prohibiting the sale of seed of susceptible hybrids and by developing tropically adopted hybrids in which both parental lines are resistant.

Downy Mildew

Although this disease could have been present in Venezuela before it was noticed for the first time in 1973 (Fernandez et al. 1975; Malaguti et al. 1977; Riccelli 1975) in the state of Yaracuy, by 1974 it was found at damaging levels and had spread to the states of Barinas, Portuguesa, and Aragua (Malaguti et al. 1977). Both forms of infection — systemic, and foliar or local — were observed. The latter has been particularly severe on certain genotypes near boot stage and in sudangrass. Due to the fact that most grain-sorghum hybrids grown at that time were susceptible, levels of infection increased rapidly and the Government reacted strongly. International experts were invited to evaluate the problem in 1975 (Renfro and Frederiksen 1975), and an International Downy Mildew Workshop was held in Maracay in 1977. At the same time, intensive pathological and breeding works were initiated by public and private institutions.

From the beginning, all efforts have been directed toward the development of resistant lines and hybrids. It is fortunate that the strain of *Peronosclerospora sorghi* that prevails in Venezuela, unlike the case of viral diseases, seems to be the same as that present in the USA and other American countries where DM-resistant sorghum genotypes have already been developed. The screening of several sources of resistance from the world collection and from

Table 9. Necrotic reaction in young plants of two sorghum hybrids and their parental lines after inoculation and natural infection in Macapo, Aragua, Venezuela.

Hybrid or parental line	Plants		Plants[a]	
	Inoculated (no.)	Checks (no.)	Inoculated (%)	Checks (%)
A, Combine Sagrain	81	78	0.0	0.0
A, Comb. Sagr. × Tx-430	87	95	3.2	1.9
Tx-430	86	75	4.8	3.6
A, IS-10346	64	51	0.0	0.0
A, IS-10346 × Tx-430	73	67	4.1	2.9
Tx-430	77	72	5.2	3.0

a. Last count of dead plants was made 35 days after planting or 25 days after inoculation. All entries were planted in the PROSECA field in Macapo on 16 Oct 1978.

the Texas-Puerto Rico Conversion Program and the testing of different experimental hybrids of grain and forage sorghums were highly facilitated in 1975 by the construction of a conidial-inoculation chamber. This device, located at PROSECA, Valencia, is a simplified version of the chamber used at College Station, Texas (Craig 1975). Other techniques have been used at CENIAP (Malaguti, personal communication) and in the School of Agriculture in Maracay (Borges, personal communication).

Commercial hybrids have been rated for SDM reaction on a large scale since 1977 (Mena, Barrientos, and Tovar, personal communications, Riccelli, unpublished results). This information is most valuable if used to suggest which downy mildew-resistant hybrids can be imported. By doing so, the disease has been brought under control in the last 2 years. High inoculum potential was observed in a regional test planted at Villa de Cura, Aragua in 1977. Mena (personal communication) rated the 34 hybrids and two varieties contained in that test, results are presented in Table 10. Only eight entries showed 10% or less of SDM infection; this means that the great majority of the hybrids marketed in Venezuela in 1977 were still susceptible. The data contained in this table closely agrees with that published by the Texas Extension Service (Horne 1976), although the percentages of systemically diseased plants here were proportionally higher, probably due to more favorable conditions for infection.

Incidence of SDM in the IDIN was recorded at Sabaneta, Barinas, and Guarabao, Yaracuy (1975), and at Macapo, Aragua (1977). The IDIN entries that exhibited systemic infection are listed in Table 11. Except for NSA 440-12, SC-599-6 × SC-134-6, SC-599-6-10, and B-3197 × SC-170-6 (at Sabaneta only), all entries that showed significant SDM were susceptible checks. The remaining 16 entries were completely resistant at all three locations. Of these, the following were those with some level of tropical adaptation: SC-414-12, SC-112-14, SC-103-12, SC-103-6, TAM-2566, SC-599-6-3, SC-326-6, SC-170-6-17, SC-237-14, SC-423-14, and SC-170-6-8.

Effects of SDM (with natural and with artificial inoculations) on selected sorghum varieties and lines have been studied since 1975. Table 12 lists a group of genotypes noted for their susceptibility or resistance and that seem at the same time to have present or potential breeding value. A large number of resistant genotypes are also listed as such in Texas (Anonymous 1977) and that none of those susceptible in Venezuela are catalogued as resistant in the Texas publication. Fortunately, resistance to downy mildew is not uncommon in tropically adapted sorghums, and SDM-resistant hybrids can be readily obtained.

Data extracted from a study on stability of performance (Table 13) tend to confirm that SDM resistance is somewhat dominant in its inheritance. This finding agrees with previous reports (Frederiksen et al. 1973). Therefore, only one resistant parental line is needed to obtain a

Table 10. Average incidence of systemic downy mildew infection in a group of commercial sorghum hybrids and varieties in Villa de Cura, Aragua, during rainy season 1977.

Hybrid	Systemic SDM (%)
PROSECA	
Chaguaramas 3	30
Chaguaramas 5	60
Barinas 2	50
Barinas 3	30
Aguasay 1	20
Barinas 4	40
Aguasay 2	20
Guarico 1	60
Pioneer	
P-8417	80
P-8311	10
P-8454	70
P-815-B	10
De Kalb	
D-C-42-C	50
D-D-42	10
D-E-57	60
D-E-59 +	30
D-F-64	30
Asgrow	
Bravis	30
Topaz	10
Dorado A	80
Capitan	40
Golden Acres	
T E Honda	10
T E 77-A	20
T E Y-101-R	40
T E Bird-A-Boo II	70
T E Total	50
T E Plus	20
T E 88 A	20
Northrup King	
NK-300	40
NK-222-A	60
NK-265	50
NK-222-G	50
NK-266	10
NK-Savanna-3	10
NK-280	10
Colsemillas	
ICA Nataima	10

Source: Hector Mena, Centro Nacional de Investigaciones Agropecuarias (CENIAP), Maracay.

resistant hybrid. In effect, some of the local hybrid combinations are crosses between one parent that confers tropical adaptation and another that contributes SDM resistance.

The first local SDM-resistant grain-sorghum hybrid (Aguasay I) was made available in 1975. Grain yield of Aguasay I at seven locations, compared with average yields of several imported hybrids used as checks, is presented in Table 14. As expected, the superiority of Aguasay I was most evident at Macapo, the location with the highest level of SDM.

In 1976 a second downy mildew resistant hybrid was released: Aguasay II. Both show high levels of resistance and possess good agronomic characteristics. However, both were dropped because of their susceptibility to viral diseases. Last year a third hybrid in this series (Aguasay III) appeared on the market; it is resistant to both downy mildew and viruses. Another hybrid, Chaguaramas III, has a low incidence of both diseases in the field and will be kept because of its high yield potential.

Foliage Diseases

Foliage diseases have accompanied grain and forage sorghums since their introduction in Venezuela. They are very common and often more than one may be present on the same plant. Some do not seem to interfere with photosynthesis until late in the life of the plant, although under more humid conditions, they may appear earlier and reduce yields.

Anthracnose

Anthracnose affects production significantly only in the warm and humid southwestern states (Portuguesa and Barinas), where occasionally it may become the most damaging of the sorghum diseases there.

Tovar and Barrientos (personal communication) rated 34 commercial hybrids and two varieties for foliage anthracnose in two grain-sorghum-performance tests in the state of Portuguesa in 1977. They found only two tropical varieties — Guarico-1 from Venezuela and ICA Nataima from Colombia — to be free from anthracnose at both locations (Table 15).

The International Sorghum Anthracnose Virulence Nursery (ISAVN) has been planted at Los Guayos, Valencia, since 1975. In 1977 an

Table 11. Percentage of naturally occurring systemic SDM in IDIN entries showing incidence of the disease (from tests conducted by PROSECA at three locations).

Entry	Sabaneta[a] 1975		Guarabao[b] 1975		Macapo[c] 1977		Observations
	Rep I	Rep II	Rep I	Rep II	Rep I	Rep II	
Tx-398	5.6	7.9	0.0	4.0	3.3	4.0	Check
Tx-2566	0.0	0.0	0.0	0.0	1.3	0.0	
NSA-440-12	4.3	4.0	4.3	5.4	8.0	7.5	
Tx-2567	5.7	High	6.0	High	14.9	6.7	Check
Tx-7078	6.0	1.2	5.6	5.0	8.3	6.2	Check
SC-599-6-3	0.0	0.0	0.0	0.0	3.8	4.4	
SC-110-14	0.0	0.0	0.0	0.0	1.2	0.0	
Tx-2536	14.3		Poor	Stand	5.8	0.0	Check
(599-6 × 134-6)	3.2	3.1	6.7	6.0	17.3	6.9	
SC-599-6-10	4.0	3.5	1.2	0.0	3.7	4.6	
SC-326-6	0.0	0.0	0.0	0.0	0.0	1.5	
Tx-378	0.0	0.0	0.0	0.0	0.0	2.2	Check
SC-175-14	1.2	0.9	1.5	0.0	0.0	0.0	
(3197 × 170-6)	28.6	High	0.0	0.0	0.0	0.0	

a. Planted on 13 Jul 1975
b. Planted on 11 Jun 1975
c. Planted on 23 Jun 1977

additional nursery, the Converted Line Anthracnose Nursery was also planted there. Natural infection developed only in 1975 and 1976. In 1977 anthracnose was not observed on any entry (even on susceptible checks), in spite of abundant infection present on nearby wild sorghum (*S. verticilliflorum*). Ratings on the ISAVN of 1975 and 1976 are summarized in Table 16. According to King and Frederiksen (1976) these results give evidence for possible physiologic specialization of *C. graminicola* within the western hemisphere; in effect, the variety Wiley was moderately susceptible in 1975. There is also evidence of physiological specialization in the African continent (Nigeria). Pathogen variability will require the in situ screening of anthracnose resistance and its eventual incorporation into highly adapted high-yielding breeding lines.

Bacterial Stripe, Grey Leaf Spot, Leaf Blight, Rust, and Zonate Leaf Spot

Heavy outbreaks of any of these diseases have not been reported so far, and it may be assumed that they are not limiting factors of sorghum production in Venezuela. However, zonate leaf spot and grey leaf spot, under conditions favoring their development, may occur at very early growth stages of the plant. An estimate of foliage-disease losses is urgently needed.

Zonate leaf spot (ZLS) was reported for the first time in Venezuela in 1949 by Ciccarone (1949) who observed the disease in experimental sorghum plots near Maracay. Not one of the 37 sorghum cultivars evaluated by Malaguti and Tovar proved to be resistant to ZLS; 25 were considered highly and 12 moderately susceptible.

Foliage-disease ratings are routinely registered in every performance test and nursery. Table 17 summarizes only those results obtained through the evaluation of different genotypes grown at locations where infections were promoted by prevailing climatic conditions. Although in no case was any one of the diseases listed capable of destroying any plant, they should not be neglected. Breeding for resistance should be initiated in order to prevent serious outbreaks in the future.

Table 12. Reaction of sorghum lines and varieties selected for breeding value in the tropics to sorghum downy mildew at different locations in Venezuela.

RESISTANT GENOTYPES				SUSCEPTIBLE GENOTYPES			
Line or variety	Introduced from	Line or variety	Introduced from	Line or variety	Introduced from	Line or variety	Introduced from
EC-2147, SB62-2	Colombia	IS-2403	Texas	AK-3003	Arkansas	Tx-2714	Texas
IS-8670	Maracaibo	IS-2508	Texas	KS-9	Kansas	Tx-2717	Texas
PI-267348	Maryland	SC-6	Texas	KS-18	Kansas	Tx-2727	Texas
PI-267504	Maryland	SC-60	Texas	KS-24	Kansas	SC-322	Texas
TSP-72	Bangkok	SC103-12	Texas	Redbine 58	Iowa	Tx-7078	Texas
TSP-342	Bangkok	SC-124	Texas	Wheat land	Kansas	SC599-6-3	Texas
2873	Nigeria	SC-166	Texas	Texioca 63	Kansas	62-142-2	Purdue
IS-8171	India	SC-167	Texas	IS-10348	India	"90"	Purdue
IS-229	Maracaibo	SC-181	Texas	IS-10372	India	68-1-17	Purdue
IS-9623	India	SC-237	Texas	IS-10398	India	68-22-93	Purdue
PI-257595 C	Purdue	SC-326-6	Texas	IS-10475	India	5976-3	Purdue
3927-4	Purdue	SC-328-C	Texas	IS-10588	India	IS-2833	Colorado
4587-3	Purdue	SC-372	Texas	IS-10660	India	IS-1116-C	Texas
954257	Purdue	SC-405	Texas	IS-10254	India	IS-6705-C	Texas
Tx.-430	Texas	SC-414	Texas	IS-10688	India	IS-12684-C	Texas
IS-8236	Purdue	SC-490	Texas	SA-6649-2	Georgia	IS-3922	Purdue
3912-6	Purdue	Tx-2716	Texas	White Sourless	Texas	IS-8168	Purdue
3925-2	Purdue	Tx-2724	Texas	KS-50	Kansas	IS-451	Purdue
IS-2573 C	Texas	Tx-2726	Texas	TAM-618	Texas	IS-949	Purdue
IS-2801 C	Texas	Tx-2728	Texas	Wheat Sel	Purdue	5813	Purdue
IS-2579C	Texas	Tx-2730	Texas	IS-2031	Maracaibo	IS-6383	Purdue
IS-7064 C	Texas	954063, R	Argentina	IS-8622	Maracaibo	IS-8088C	Purdue
IS-12608 C	Texas	68-22-97	Purdue	IS-7382C	Texas	3917-3	Purdue
IS-12610 C	Texas	IS-10352	India	IS-82	Colorado	5799-1	Purdue
IS-12666 C	Texas	IS-10372	India	IS-3691	India	IS-314	Purdue
IS-7193	Purdue	TAM-428	Texas	Ica Nataima	Colombia	4037-1	Purdue
IS-12610 C	Texas	Tx-623	Texas	Comb. Sagrain	Texas	4593-1	Purdue

Source: Evaluations conducted at several PROSECA nurseries with either natural or artificial inoculations.

Bacterial stripe has been only a curiosity and there is no evidence so far of economic damage.

Stalk and Peduncle Rots

At least three pathogens known to be present in Venezuela cause this type of disease: *Macrophomina phaseolina, Fusarium moniliforme*, and *Colletotrichum graminicola*. Unlike the foliage diseases in Venezuela, they cause significant yield reductions. Charcoal rot is quite common in the central and eastern states where rains are more erratic and long periods of moisture stress can occur, while Fusarium stalk rot and red rot are more abundant in the warm and humid southwest.

Lodging is mostly caused by the stalk rots mentioned above. Little has been done in relation with these diseases besides recording lodging and nonsenescence ratings in every performance test. Rosenow (1977) found a sig-

Table 13. Systemic sorghum downy mildew reaction following natural infection in five R-lines; and some hybrid combinations in three trials (extracted from a study on stability of performance conducted by PROSECA).

	Macapo 1976[a]		Macapo 1977[b]		Chaguaramas 1977[c]	
	Plants (No.)	Infected plants (%)	Plants (No.)	Infected plants (%)	Plants (No.)	Infected plants (%)
A-lines						
M_1	681	6.9	532	0.9	657	0.0
M_2	684	76.7	501	20.5	698	5.9
M_3	691	7.1	498	1.6	690	0.3
M_4	683	35.3	448	4.5	707	1.0
M_5	683	25.6	393	6.1	693	0.7
R-lines						
P_1	665	67.0	467	19.5	705	6.5
P_2	669	45.2	401	11.7	676	0.1
P_3	681	4.5	396	0.3	668	0.0
P_4	687	21.9	412	11.9	669	1.0
P_5	677	0.9	439	0.0	721	0.0
Hybrids						
Chaguaramas II ($M_3 \times P_2$)	659	7.3	482	4.1	690	0.3
Chaguaramas III ($M_5 \times P_2$)	675	2.5	452	1.8	738	0.1
Chaguaramas IV ($M_4 \times P_2$)	669	16.3	414	3.4	701	0.8
Perija I ($M_1 \times P_3$)	665	3.5	511	0.8	693	0.7
Araure I ($M_1 \times P_1$)	655	34.7	425	14.6	710	4.1
Barinas II ($M_1 \times P_4$)	661	8.3	364	6.0	680	0.0
Barinas III ($M_2 \times P_4$)	690	22.6	492	5.9	705	3.5
Aguasay I ($M_1 \times P_5$)	667	0.3	440	0.5	737	0.0

a. Planted on 1 Jul 1976
b. Planted on 2 Jun 1977
c. Planted on 8 Jul 1977

Table 14. Grain yield of SDM-resistant hybrid Aguasay-1 and average yield of imported hybrids at seven locations in Venezuela in 1976.

Location	Yield of Aguasay 1 (kg/ha)	Average yield of imported hybrids (checks)[a] (kg/ha)
Barinas (A)	209	058 (7)
Araure	494	605 (7)
Macapo	426	688 (7)
Saman Mocho	653	328 (7)
Chaguaramas (A)	210	583 (7)
Barinas (B)	379	082 (5)
Chaguaramas (B)	901	338 (3)

a. The figure in parenthesis indicates the number of checks present in that particular test.

nificant positive correlation between nonsenescence and lodging resistance. This correlation is of special significance in the Tropics where there is no possibility of plants being killed by low temperatures.

Lodging occurred in nearly every plot in a performance test of a group of experimental hybrids conducted in Chaguaramas, Guarico, in 1977 (Table 18). Lodging and senescence appear to be highly correlated, and the latter seems to be an excellent character to select against when lodging resistance is the goal. Nonsenescence is very common among tropically adapted sorghums and it should be easy to incorporate this character in future breeding programs.

Panicle and Grain Diseases

Head blights incited by *Fusarium, Colleto-*

Table 15. Foliar reaction of commercial sorghum hybrids to natural infection of *Colletotrichum graminicola* at two locations in Venezuela.

Hybrid or Variety	Reaction[a]	
	La Lucia[b]	Durigua[c]
PROSECA		
Chaguaramas I	1	–
Chaguaramas III	1	3
Barinas II	3	6
Barinas III	5	6
Barinas IV	3	4
Aguasay I	1	7
Aguasay II	2	–
Guarico I	1	1
PIONEER		
P-8417	2	3
P-8311	4	4
P-8454	3	4
P-815-B	4	3
DEKALB		
E-57	4	3
E-59 +	4	2
F-64	–	2
C-42-A	4	3
D-42	3	6
ICA-COLOMBIA		
Ica Nataima	1	1
NORTHRUP KING		
NK-300	5	3
NK-222-A	5	4
NK-265	6	–
NK-222-G	5	4
NK-266	5	1
Savanna 3	2	3
NK-280	–	4
ASGROW		
Bravis R	–	3
Topaz	3	1
Dorado A	5	5
Capitan	3	3
TAYLOR EVANS		
TE-Hondo	2	4
TE-77A	4	5
TE-Y-101-R	2	7
TE-Bird-A-Boo	4	3
TE-Total	4	4
TE-Plus	1	2
TE-88-A	3	6

Source: D. Toler and V. Barrientos, CIARCO.

a. On a scale of 1 (no visible spot) to 10 (100% leaf area affected).

b. Planted 29 Jun 1977

c. Planted 1 Jun 1977

Table 16. Reaction of entries in the International Sorghum Anthracnose Virulence Nursery to anthracnose at Los Guayos, Valencia, Venezuela in 1975 and 1976.

Variety or Designation	Foliar symptoms[a]	
	1975[b]	1976
Honey	2.0	1.7
Brandes	1.0	1.0
Wiley	3.3	1.0
MN-960	1.0	1.0
Tx-398	4.0	2.0
N-9040	3.0	–
C-42-Y	3.0	–
NK-233	4.0	–
E-1	–	2.0
E-2	–	2.0
E-3	–	1.7
PB-846	4.0	2.0
Tx-2536	3.0	2.0
Tx-399 × Tx-2536	3.0	1.7
SC-599-6	1.3	1.7
TAM-428	2.0	2.0
Tx-399	–	1.7
Tx-399 × TAM-428	–	1.3
R-1029 A	–	1.0
Combine Sagrain	4.0	–

a. According to a scale from 1 = no infection to 5 = very severe symptoms.

b. Planted in the field of PROSECA on 17 July 1975, rated on 13 Oct 1975.

c. Planted in the field of PROSECA on 28 June 1976; rated on 27 Sep 1976.

trichum, and more recently by viruses, can be observed in sorghum fields and experimental plots throughout the country where they are sometimes severe. There has been no estimate of damage or evaluation of resistance to these diseases in Venezuela. However, since they are only different phases of the same foliar and stalk diseases, resistant genotypes already found in tropical germplasm could be utilized to control head blights.

Covered kernel smut, described many years ago in Venezuela by Ciccarone and Malaguti (1950) has become insignificant since fungicide treatment of sorghum seed has become a common practice.

Table 17. Foliage disease infection ratings of grain sorghum genotypes tested in Venezuela.

Test of Nursery	Genotypes (No.)	Infection rating[a]																			
		Zonate leaf spot					Rust					Grey leaf spot					Leaf blight				
		1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
PROSECA[b] Hybrid Performance Test 75–I[c]	81	2	31	39	9	0	–	–	–	–	–	0	77	4	0	0	5	56	14	6	0
PROSECA Hybrid Performance Test 75–II[d]	21	0	10	11	0	0	–	–	–	–	–	0	20	1	0	0	0	18	3	0	0
PROSECA Hybrid Performance Test 76–VI[e]	64	0	36	25	3	0	0	33	25	6	0	0	62	2	0	0	–	–	–	–	–
PROSECA 1976–77 General Adapt. Test[f]	20	1	8	11	0	0	13	7	0	0	0	0	17	3	0	0	1	18	1	0	0
PROSECA Hybrid Performance Test 77–VI[g]	43	0	0	13	30	0	0	6	21	16	0	0	0	34	9	0	0	20	23	0	0
CIARCO[h] Hybrid Performance Test[i]	34	12	16	5	1	0	5	22	6	0	0	0	2	22	10	0	–	–	–	–	–
CIARCO Hybrid Performance Test[j]	33	3	20	10	0	0	3	12	16	2	0	0	8	25	0	0	–	–	–	–	–
CENIAP (II)[k] Hybrid Performance Test[l]	36	0	23	13	0	0	0	35	1	0	0	0	0	34	2	0	0	23	13	0	0

a. Based on scale of 1 (no infection) to 5 (severe symptoms, death of leaves).
b. Productora de Semillas, C. A., Valencia. *c.* Planted 9 Jul 1975 in Barinas.
d. Planted 11 Jul 1975 in Barinas. *e.* Planted 17 Aug 1976 in Barinas. *f.* Planted 9 Jun 1977 in Macapo.
g. Planted 1 Jul 1977 in Macapo. *h.* Centro de Investigaciones Agropecuarias Region Centro Occidental, Araura.
i. Planted 1 Jun 1977 in Durigua. *j.* Planted 29 Jun 1977 in La Lucia.
k. Centro Nacional de Investigaciones Agropacuarias, Maracay. *l.* Planted in rainy season 1977 in Villa de cura.

Table 18. Relation between nonsenescence and lodging in a grain sorghum performance test[a] of experimental hybrids in Chaguaramas, Guarico.

Nonsenescence rating[b]	Genotypes (no.)	Lodging ratings (average)[c]
3	4	1.50
4	3	2.33
5	12	2.75
6	21	3.19
7	16	4.31
8	13	3.92
9	11	5.18

a. Planted 8 July 1977 in a triple-lattice design. Ratings taken at harvest (96 days after planting).
b. According to a scale from 1 = totally green plant to 9 = totally dried plant.
c. According to a scale from 1 = no lodging observed to 9 = all plants lodged.

Grain mold (weathering) is the most important cause, by far, of kernel damage and losses. Climatic conditions in sorghum production areas of Venezuela are particularly favorable for mold development. The rainy season may continue for even 2 or 3 months after the grain is ready to harvest. If a few dry days occur during this time and the grain cannot be harvested, there can be heavy losses in the form of reduction of quantity and quality. Seed germination in the head, shattering, discoloration, molding, loss of weight, shrivelling, and kernel breakage during harvest are common consequences of a delayed harvest.

Routine evaluation of grain mold is made on all hybrid-performance tests. The results obtained by Tovar and Barrientos (personal communication) on commercial hybrids and by myself on commercial and experimental hybrids indicate that brown-seeded genotypes are, by far, the most resistant materials. Among commercial cultivars, the following are brown-seeded and all were rated as mold resistant: Guarico 1, ICA Nataima, NK 300, all hybrids in the Chaguaramas series, Barinas 3, Aguasay 3, and T. E. Bird-A-Boo. Since brown sorghum is not graded as an inferior type in Venezuela, and because the best sources of tropical adaptation found so far are brown-seeded, this type of sorghum can be a temporary solution to the weathering problem.

Discussion and Conclusion

Disease resistance is essential for successful sorghum production in Venezuela and, probably, in most tropical regions. Right now, no other method of disease control can be visualized. Escape through delayed planting dates does not seem a solution, since it has been shown that the yield potential of a hybrid is better realized if it is grown during the months of higher rainfall. Eradication of such pathogens as *P. sorghi, C. graminicola*, and viruses — by any present method, is not only impractical but is probably impossible because of the presence of infected alternate hosts.

Progress in developing resistance to the major disease has been made. Resistance seems to be more easy to find in tropical exotic sorghums than in the highly improved and sophisticated USA lines. The Texas-Puerto Rico Conversion Program is achieving enormous success in this respect because it makes available a large number of new and effective sources of resistance from the world collection.

In the tropics, disease resistance is useful only if accompanied by tropical adaptation. The agricultural areas of the world located between 15°N and 15°S latitude have a unique environment with specific climatic, disease, and insect problems, and therefore tropical adaptation must be identified in situ. The three recently released females from the Texas Agricultural Experiment Station (Tx-622, Tx-623, and Tx-624) were selected in Texas for tropical adaptation. They perform much better in the tropics than do average female lines from the USA, but are susceptible to a strain of SCMV. Their hybrids are heavily damaged by grain molds under rains, unless the male parent can provide weathering resistance. On the other hand cultivar Chaguaramas 3, a local brown-seeded hybrid whose male parent is a line selected in Venezuela, performed poorly even in a southernmost location of the USA (Weslaco, Texas, approximate 26°10'N lat), while in Venezuela (between 8 and 10°N lat) it has outyielded every temperate hybrid in public and private performance tests. It also had the highest average yield in the PCCMCA tests (Vega-Lara 1978)

conducted at 12 locations in seven Central American countries (between 8 and 15°N lat).

Breeding objectives in the tropics should, by necessity, depart from traditional schemes established by plant breeders working in other parts of the world. In general, the highest yields have been obtained from hybrids that are taller than those grown in the temperate zones. Hybrids 2 m in height can compete much better with weeds and still be combine harvested. They should be resistant, however, to stalk diseases causing lodging. Nonsenescence is also very important, because most of the grain-sorghum producers use sorghum stubble as cattle feed during the dry season. Brown seed seems to be correlated with a number of desirable characteristics, particularly mold and bird resistance. Less attention should be paid to photosensitivity, since most sorghums flower in a reasonable time at these latitudes.

Finally, it is hoped that, by making the appropriate crosses, multiple-disease resistance can be combined with tropical adaptation. A program of straight pedigree selection with field-performance testing will be initiated in PROSECA in 1979. Later, the population-breeding approach will be tried. It seems valuable to achieve longer-term objectives since, through genetic recombination, they might provide a better chance to select genotypes with horizontal resistance. At the same time, artificial-inoculation techniques must be improved to the point where effective screening can be done any time it is needed.

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Current Work on Sorghum Breeding and Diseases in Thailand

Jinda Jan-orn*

In Thailand except for downy mildew, the leaf diseases of maize and sorghum (leaf blight and leaf spot) are common to both crops. Leaf rust (*Puccinia purpurea*) and anthracnose (*Collectotrichum* sp) were found in a relatively smaller percentage (Silayoi et al. 1970). The following diseases were observed during the 1969 crop-season survey conducted by the Department of Agriculture (Silayoi et al. 1970): head molds (*Curvularia lunata, Fusarium* spp, *Penicillium* sp, *Aspergillus* sp); leaf blight (*Exserohilum turcicum*); leaf spots (*Helminthosporium rostratum, Phyllosticta* sp, *Cercospora sorghi, Stagonospora* sp, *Cercospora longipes, Septoria* sp, *Ascochyta* sp); anthracnose (*Collectotrichum* sp); leaf rust (*Puccinia purpurea*); brown spot (*Physoderma* sp), zonate leaf spot (*Gloeocercospora sorghi*); red stripe, charcoal rot (*Macrophomina phaseolina*); kernel rot (*Phoma insidiosa*); and loose smut (*Sphacelotheca cruenta*). In general, most sorghum leaves have been severely affected by leaf blight and zonate leaf spot. Tar spot has been observed at certain locations.

Proliferated plant growth caused by sorghum downy mildew (SDM) rarely appears in sorghum fields, especially in the Hegari types. Probably the first plant with SDM symptoms was found in the late Hegari test plot at Takfa Maize and Sorghum Test Farm, Taklee Field Crops Experimental Station, in 1974, and later at the Maize and Sorghum Center, Pakchong. In the U.S. hybrid materials, infected plants have been found frequently at both Takfa and Pakchong since 1976, with a small percentage in some genotypes. In 1975, Aran Patanothai indicated the prevalence of grey leaf spot (*Cercospora sorghi*) and bacterial stripe in his sorghum nursery at Khon Kaen University.

Leaf blight and zonate leaf spot are the two diseases found frequently in Hegari sorghum. Stalk rot (caused by *Macrophomina phaseolina, Fusarium* sp, *Colletotrichum graminicola*, etc.) has been severe on Hegari plants at the Maize and Sorghum Center, Pakchong. Recently, rust has attacked Hegari sorghum as well as other susceptible lines and varieties, such as the high-lysine cultivar P-721. Boonsue and Harwood (1970) claimed that disease outbreaks were mainly responsible for the failure of Hegari sorghum to ratoon well. As the diseases attacked the Hegari sorghum plant, the leaves turned purple and looked unthrifty while in certain genotypes — such as a yellow-endosperm genotype — the leaves turned yellow and dry, similar to maize leaves.

At present, many yellow-endosperm lines derived from crossing the yellow-endosperm introductions (Indian sorghum), such as KU-257 and other TSS (Thai Sorghum Selection) lines, are quite resistant to leaf diseases. Their leaves are relatively clean and healthy in comparison with those of Hegari. However, the yellow-endosperm lines are susceptible to grain weathering, and they are not grown successfully by Thai farmers because of unfavorable growing conditions and wet periods at grain maturity. Most Thai farmers broadcast sorghum after maize (or even after sorghum in the late rainy season), as it is an easy process and costs less than other methods. After plowing, farmers broadcast the seeds, and then leave the field until harvest time in November or December (sorghum as a second crop is planted in late August or September).

Hegari sorghum, introduced in 1940, has worked well in Thai growing conditions. Other introduced sorghum lines (such as IS-8719, E-173) adapt well to Thai growing conditions, and yield well under rather poor growing conditions. They have been used as parents for developing new lines or varieties. The derived lines are not as good as older lines since they are photoperiod sensitive. Selection is in progress for more adapted lines.

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Recurrent selection in an introduced random-mating sorghum population has been practiced since 1973. Due to an inconvenience confronting random mating in isolation, this population is too tall, probably because of pollen contamination. Besides, the population is poor in resistance to leaf diseases, causing it to look dirty and ugly. Various schemes of recurrent selection, such as mass selection and S_1 family selection, are being practiced for developing a population with more desirable characteristics. A random-mating population of Hegari has been developed, using the NP 3R population as the source of the male-sterile gene (ms_3). Plant height is taller and maturity is later in the constituted population than in the original Hegari material, and the leaves are infected by rust as well as by other diseases, but to a lesser degree than with the original materials.

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Utilization of Disease Resistance Discussion Session

Brhane:

Would Dr. N. G. P. Rao elaborate on his statement that the elite multiple-disease-resistant lines were developed without a directed effort at obtaining multiple-disease resistance? What were the methods used to develop the resistance?

N. G. P. Rao:

The cultivars that have been developed with multiple-disease resistance have not been specifically bred for multiple resistance; they have just turned out that way. We now have a directed program for the identification of multiple-disease resistance, using the good facilities available at our different centers for screening for various diseases.

Williams:

We are concerned about diseases because they reduce yield. Thus selection for high yield across several centers with different disease pressures would contribute to the unconscious development of multiple-disease resistance.

Sundaram:

In the population-improvement program at ICRISAT, the effort has been on incorporation of resistance to grain molds, charcoal rot and SDM. While there is no question that these are important, I suggest that anthracnose, grey leaf spot, and *Striga* resistance are also urgently needed, particularly for material to be grown in West Africa.

House:

Populations developed at ICRISAT Center generally do not perform well in West Africa. West African cultivars will have to be developed there. Problems will have to be worked on at the locations where they are most acute. As *Striga hermonthica* does not occur in India, we cannot work on it here.

Sundaram:

I agree with Dr. Riccelli's observation that for the control of SDM in hybrid sorghum only one parent need be resistant. However, in pearl millet the situation is different, and if one parent is susceptible the hybrid will be susceptible.

Williams:

For pearl millet downy mildew it is desirable for both parents to be resistant, but it is not essential.

Riccelli:

While we have been successful in the control of SDM by crossing a resistant parent with a tropically adapted (susceptible) parent, we have encountered complications in virus control, because this requires both parents to be resistant.

N. G. P. Rao:

I would like to comment on the use of population improvement for incorporation of disease resistance. Population improvement is a long and complicated process, particularly if crosses between the tall late-flowering tropical sorghums and elite temperate sorghums are involved. Progress cannot be made quickly, and such work cannot be done at each station where sorghum-improvement work is conducted. For use of disease resistance in sorghum where resistance factors are not complicated, and where good levels of resistance are available the simple approaches are going to give much faster results. This does not mean that population improvement at a central location is wrong, but it should be kept in perspective.

House:

Population improvement is just one approach used in the ICRISAT's sorghum breeding program. The parents that go into a population have to be carefully selected and should not be a large number — 15 to

20 is about right. I agree that population breeding is a long-term process, and that it should not be carried out by every breeder. We feel that we should do the initial work of assembly and improvement of populations. Only when they have reached an advanced stage should they be given to other breeders.

Brhane:
The elite products from the AICSIP program are almost all tan. Tan plants are not necessarily resistant to diseases. Is there a danger of narrowing the genetic base by breeding almost exclusively for the tan-plant characteristic?

Murthy:
The tan-plant character is simply inherited, and thus its use should not narrow the genetic base. There is a great deal of diversity among plants with tan color.

Gibson:
Is inheritance of tan plant color really that simple? In my crosses between tans and reds, I am not getting a 3:1 F_2 ratio.

N. G. P. Rao:
Intermediates do occur. But the inheritance is quite simple.

Rosenow:
In our experience, the tan color is a single-gene character. The intermediates are due to confounding characters such as glume colors and even pericarp color. If we make two categories, tan and everything that is not tan, we get 3:1 ratios.

Reddy:
What are the appropriate breeding methods for the utilization of disease resistance? Pedigree? Backcross?

House:
We need to use the method that works. It will depend on heritabilities. If, as appears to be the case for shoot fly resistance, we are dealing with a polygenic recessive trait, then a major effort is needed to improve the trait directly. In such a situation the population approach may be the only one that will work.

N. G. P. Rao:
The backcross theory is fine if the resistance is simply inherited. But when it comes to practical breeding, disease resistance is one of many traits being handled. It has to be developed along with a good economic background. Do you spend time on five or six generations of backcrossing, trying to manipulate one specific gene, or do you put your efforts on a more practical level, trying to develop new higher yielding varieties with disease resistance. I favor the latter.

House:
I think that while this is generally so, we need to keep our options open, and backcrossing may be needed in some situations. Where a very good line needs improvement for one simply inherited character, backcrossing can be used. If we can select better material than the original, then this is what should be done. If further backcrossing seems desirable, then we should do it.

Frederiksen:
A good example of the valuable use of the backcross method is the incorporation of greenbug resistance into commercial sorghum cultivars in the USA. This was done essentially through backcrossing, and has revolutionized sorghum production in the areas where greenbug was a problem.

Teakle:
In breeding for virus resistance in Australia, we have identified two very good sources of resistance: (a) the Krish resistance, which is a single dominant gene that can be used easily in a backcrossing program; and (b) the Q-7539 type of resistance, which is recessive and polygenic, and which needs a different strategy of utilization.

Davies:
How do international nurseries contribute to resistance utilization?

House:
I can comment using the example of stem

borer resistance. Different species of stem borers are important in different parts of the world. When we get resistance to one, we should see how it behaves when confronted by another. As we have ICRISAT cooperative programs located in several countries, we have the opportunity to do this. When we recombine the different resistances, these can be tested at the different locations. The international trials, of course, contribute to our knowledge of races of pathogens and stability of resistance. We request national program researchers to contribute entries to the international disease nurseries.

In order not to overload national programs with nurseries and trials from many sources, we believe we should use the ICRISAT regional stations for preliminary evaluation of materials from any source. The materials that are well adapted could then be given to the national programs in the region. This would be for yield as well as for insect and disease reactions.

Recommendations

Recommendations of the Workshop on Sorghum Diseases

Grain Mold

1. Identification and incorporation of resistance to grain mold and other causes of grain deterioration are of prime importance to all sorghum-breeding programs and should receive continued attention.
2. In view of the complex nature of grain deterioration, the techniques for resistance screening need further improvement and refinement. We particularly recommend further examination of laboratory screening procedures, with the objective of developing a simple test or tests, which could be used on large numbers of breeding lines to reduce numbers prior to laboratory and field testing with fungal inoculation.
3. There is a need to study further the importance of the mold-causing fungi at various stages of grain development and maturity. The role of facultative parasites in grain deterioration during the postmaturity stage needs investigation.
4. The effects of interactions of microorganisms on and in sorghum grain are not well understood and need further investigation.
5. There is a need to determine the factors responsible for resistance to grain infection at different stages of development. A greater understanding of such factors could lead to improved screening techniques and a more rapid development of resistance to grain mold and deterioration.
6. While we recognize that several national and international programs have identified sources of resistance to grain mold and grain deterioration, and have successfully used these in breeding programs, we recommend that the search for additional resistance sources be intensified, with particular emphasis in areas where deterioration problems are common.
7. Information on the genetics of grain-mold resistance will be useful in breeding programs, and we recommend studies to obtain this information.
8. There are conflicting reports on the occurrence and importance of mycotoxins in moldy sorghum grain. There is a need for further studies to fully determine the mycotoxin potentials of moldy sorghum, and we particularly recommend studies on the interaction of fungal species as a possible factor in the nonproduction of mycotoxins.
9. The effects of mycologically produced or induced plant growth regulators on kernel structure and composition and on grain sprouting should be examined.

Sorghum Downy Mildew

1. The confusion regarding the identity of the causal agent of the so-called sorghum downy mildew on various hosts in different regions needs clarification. In order to fully characterize the pathogens at the different locations, we recommend studies on:
   a. HOST RANGE. Studies should be made with a standard set of test species from a common seed source. Inoculation procedures should be standardized as far as possible, and observations should be made on type of symptoms and degree of production of conidia and oospores on each host.
   b. MORPHOLOGY AND CYTOLOGY. There is a need for morphological and cytological examination between and within the host-differentiated groups. In order to eliminate differences produced by different techniques, the methodology for those studies will need standardization. We recommend that the sexual and asexual stages be killed and fixed in the same way and sent to a single location for comparison under standardized conditions.
   c. PROTEIN ANALYSIS. The possibility of splitting and grouping based on protein analysis by gel electrophoresis should be examined.

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2. The question of stability of resistance and genetic vulnerability is of major importance. We recommend that known resistance sources be examined, to determine whether there are several resistance types and whether there is a narrow or wide genetic basis of resistance. Methodology should include comparison of reactions to different sources and concentrations of inoculum and different methods of inoculation. Multilocational testing is useful in examining stability of resistance across pathogen populations and environments, and should be continued. Investigation of the biological basis of the apparent differences in cultivar reactions between Texas, USA, and certain other locations is urgently needed.
3. While we recognize that there is some information available on the relationships between reaction of host cultivars to oospores and conidia, and on the relationship between susceptibility to systemic infection and susceptibility to local-lesion infection, we recommend, in order to clarify the relationships, further studies on these relationships at several locations, utilizing a large group of cultivars with varying reactions.
4. In the evaluation of cultivar susceptibility to SDM there is a need to distinguish between systemic infection on the main stalk and systemic infection only in tillers. There is probably little to be gained by recording different severities on the main stalk (i.e., the stage of development at which systemic symptoms first appear) and, therefore, we recommend that at final scoring, which should be made at about the soft-dough stage of grain development, the plants are scored in three categories:
   a. No systemic symptoms;
   b. Systemic symptoms only on tillers, not in the main shoot;
   c. Systemic infection in the main shoot.

   In addition, the presence or absence of local lesions should be indicated on systemically-infected and nonsystemically-infected plants in the row.
5. There appears to be great potential for SDM control with the systemic fungicides in the acylalanine group. We recommend that this potential be fully explored, with evaluation of effectiveness of different application rates of different formulations by different application methods against many SDM pathogen populations in a wide range of environments. Work is also needed on the effects of storage of treated seed under various conditions, on possible phytotoxicity, and on effectiveness of the fungicide. The strategy of the utilization of the fungicide needs to be discussed by appropriate national bodies in countries where SDM control is needed.
6. We recommend continuation of the development of an international communicating and cooperating group of workers on the graminaceous downy mildews, and recommend early initiation of a Downy Mildew Newsletter to help keep downy mildew workers in communication.

Leaf Diseases

1. Leaf diseases can cause considerable damage to the sorghum crop, and can induce a higher susceptibility to other diseases, such as stalk rots. It is important to further evaluate sorghums that have exhibited resistance to one or more diseases in one or more locations and seasons. In order to identify genotypes that have multiple broad-spectrum resistance, we recommend that the multilocational evaluation of resistant cultivars be continued and expanded.
2. The information obtained on apparent pathogenic variability within one pathogen among locations should be followed up with specialized nurseries to test further for the existence of distinct pathotypes. The present International Sorghum Anthracnose Virulence Nursery should, in cooperation with other agencies, be expanded, and similar nurseries established for the study of variability in other pathogens.
3. Test locations should be diverse in distance, elevation, and climate, and should include several locations for reliable reactions to each major leaf disease.
4. In view of the many commitments on resources at most locations, the number of entries in the multilocational leaf disease trial should normally not exceed 30, and efforts should be concentrated on those entries for which there is already evidence of multiple resistance. At all locations a locally

susceptible cultivar should be included as a check on disease pressure.

5. We recommend that in field-screening nurseries a locally susceptible cultivar for each disease be planted strategically throughout the screening area, up to 2 weeks prior to the planting of the test entries, in order to provide a source of inoculum to the particular disease(s). Where necessary, suitable conditions should be promoted for adequate disease pressure; for example, the use of mist irrigation to provide necessary humidity.
6. For some diseases, such as leaf blight, anthracnose, sooty stripe, and grey leaf spot, the natural inoculum provision can be supplemented by direct inoculation. The decision on the necessity for this and for the method of inoculation should be left to the individual cooperator.
7. The timing and method of rating cultivars for susceptibility to leaf diseases is of great importance. The ratings must be done at a meaningful physiological stage of plant development. We recommend that two ratings be made at the appropriate time during the epidemic of the particular disease. We do not see merit in an early scoring, and feel that one scoring at flowering followed by one at the soft-dough stage of grain development will generally provide the necessary data. In mixed-maturity trials, the dates of flowering of all entries are important for the interpretation of cultivar reactions. We recommend the standardization of a 1 to 5 rating scale, based on an estimated percentage of diseased leaf area, for each disease.

Head Blight and Stalk Rots

1. As sorghum stalk rots are greatly affected by environmental stresses, yield potential, and maturity, and since replacement of long-duration lower yielding sorghum cultivars with shorter duration higher yielding cultivars has increased the incidence of stalk rots in several semi-arid tropical countries, we recommend that in the development of improved cultivars and hybrids a critical evaluation be made for stalk rot susceptibility.
2. Breeding for resistance to stalk rots must be closely coordinated with other yield characteristics.
3. The implications of the knowledge on interactions between stalk rot development and postflowering stress, grain-sink size, maturity, etc., should be fully considered in the development of screening techniques and evaluation of varieties for stalk rot susceptibility.
4. In a stalk rot resistance-screening program, as many replications, planting dates, and locations as possible should be used. At any one location, we recommend two planting dates with at least two replications per planting date. If only one planting date is possible, a minimum of three replications is recommended.
5. Flowering-date records are important in stalk rot susceptibility evaluations. Comparisons of stalk rot reactions should be made within test materials of similar maturities.
6. As size of the carbohydrate sink is important in the predisposition of plants to stalk rots, the stalk rot susceptibility evaluation records should include visual yield ratings, perhaps on a 1 to 5 scale, to enable the identification of entries that combine high yield with stalk rot resistance.
7. Direct stalk inoculation with *Macrophomina phaseolina* can be useful, but must be done to at least 20 plants in each replication of each test entry. Border rows and end plants in a row should not be used in these inoculations. Length of internal stem discoloration and number of internodes infected are valid criteria for evaluation, if considered along with stresses, yields, and maturities.
8. As senescence ratings can be used as indicators of stalk rot susceptibility, and as they take less time than splitting the stalks and measuring internal infection, we recommend that senescence ratings be made in all stalk rot susceptibility evaluations.
9. We recommend expansion of the ICRISAT coordinated multilocational cooperative program for the identification of charcoal rot resistance. The experimental design and evaluation should be consistent with the principles outlined in these recommendations. Planting should be timed so that the postflowering period coincides with stress conditions. Local stress interactions must be considered in reaction evaluation.

Universal stalk rot resistance is not expected. Evaluation of adaptability to a location is the strongest value of the international stalk rot resistance screening program. Such adaptability is most accurately predicted following testing in several environments.

10. Future research on stalk rots should emphasize evaluation of the stresses that predispose plants to infection in each geographical region, and the development of efficient techniques to identify sorghums capable of overcoming those stresses.
11. There is a need for basic research on the interactions among the different organisms involved in stalk rots, particularly *M. phaseolina, Fusarium* spp, and *Colletotrichum graminicola.*

Smuts

Head smut can cause major losses when susceptible cultivars are grown intensively. The use of host resistance as the sole strategy for control has led to the erosion of host resistance in some regions. Increased emphasis should be placed on:

1. Developing effective alternative control methods for reducing smut losses, particularly through cultural, biological, and chemical means.
2. Evaluating methods of deploying host resistance in such a way so as to conserve good host genes.
3. Monitoring for variation in pathogenicity through the use of uniform head smut nurseries.

The kernel smuts, both covered (grain) and loose, have been reduced to minor problems following the use of fungicide seed dressings. There are research opportunities, however, to:

1. Encourage the use of these fungicides for control of the sorghum kernel smuts in regions where they are not used.
2. Develop new or alternative means of controlling these diseases.

Long smut may cause modest levels of loss in the more arid sorghum-growing regions of the Sahel and in North Africa. However, there is very limited information on this disease. We strongly recommend:

1. Confirmation studies on the infection process and disease cycle, so as to provide further information on means and methods of testing for resistance.
2. Further evaluation of the importance of the disease in yield reduction.
3. Determination of the susceptibility of new genotypes to this disease.
4. Screening for sources of resistance.

Ergot

1. The factors affecting sclerotial production in ergot infected sorghum inflorescences need to be determined.
2. Effective resistance screening and scoring techniques need to be developed and standardized.
3. The potential hazards of sorghum ergot sclerotia need investigation.

Bacterial Diseases

The importance of bacterial leaf diseases needs further evaluation. Bacterial leaf disease resistance screening should be conducted at those locations with sufficient pressure, following the principles outlined in these recommendations.

Viral Diseases

1. Priority should be given to research on sugarcane mosaic virus in sorghum.
2. Host ranges, serological relations, and other properties of the strains of sugarcane mosaic virus should be studied.
3. A set of sorghum inbred lines selected to differentiate the strains of sugarcane mosaic virus is needed.
4. The sugarcane mosaic differentials should be maintained and distributed by a single institution.
5. Procedures for testing the differential set should be standardized.
6. The importance to sorghum of viruses other than sugarcane mosaic virus should be investigated.
7. The possibility of choosing a set of differential hosts and procedures to identify all viruses naturally occurring in sorghum should be investigated.
8. Breeding of sorghums with multiple sources of virus resistance combined with resistance to other pathogens should be encouraged.

Appendix 1
Participants

465

Front row, seated, left to right: K. M. Safeeulla, Brhane Gebrekidan, G. C. Wall, G. Malaguti, F. J. Schwinn, R. A. Frederiksen, R. C. McGinnis, L. D. Swindale, J. C. Davies, R. J. Williams, L. W. Rooney, D. S. Murthy, B. V. S. Reddy, N. G. P. Rao, H. Doggett.

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